

ASSOCIATIONS BETWEEN MATERNAL BORDERLINE PERSONALITY
DISORDER SYMPTOMS, PARENTING, AND MOTHER-CHILD CORTISOL
LEVELS

by

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A DISSERTATION

Presented to the Department of Psychology
and the Division of Graduate Studies at the University of Oregon
in partial fulfillment of the requirements
for the degree of
Doctor of Philosophy

September 2022

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DISSERTATION ABSTRACT

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Doctor of Philosophy

Department of Psychology

September 2022

Title: Associations between Maternal Borderline Personality Disorder Symptoms, Parenting, and Mother-Child Cortisol Levels

Children of mothers with borderline personality disorder (BPD) are at heightened risk for developing mental health difficulties but the pathway through which this risk is conferred is unclear. Developmental psychopathology theories have highlighted parenting and stress physiology as potential mechanisms for the intergenerational transmission of mental health outcomes that may be particularly relevant to children of mothers with BPD. The aims of this dissertation were to 1) identify associations between maternal BPD symptoms and observed parenting behaviors and 2) examine whether positive parenting behaviors mitigated the risk of disrupted child stress physiology within the context of maternal BPD. To do this, two studies were conducted using both a clinical and community sample of mothers with BPD symptoms to examine both salivary cortisol levels and hair cortisol concentrations as measures of stress physiology.

Across both studies, maternal BPD symptoms were not associated with displays of positive parenting behaviors. This may be a unique strength of mothers with BPD, as maternal psychopathology in other contexts (e.g., maternal depression) has been consistently associated with lower levels of positive parenting behaviors. Maternal BPD symptoms were associated with disrupted child salivary cortisol levels through its

influence on maternal salivary cortisol levels. However, maternal BPD symptoms were not associated with mother-child hair cortisol concentrations which may be due to a lack of sensitivity in hair cortisol concentrations to psychosocial adversity, especially for children. In both studies, positive parenting behaviors were not associated with child stress physiology, which may be due to low power to detect effects as well as limited variability in parenting behaviors. Across both studies, the strongest predictor of child stress physiology was maternal stress physiology.

These findings are contextualized within broader theoretical models of early adversity on child stress physiology, as well as a discussion comparing the use of salivary and hair cortisol collections as measures of stress physiology. As the field continues to develop approaches to reduce suffering and promote healthy child development in families of mothers with BPD, the role of parenting and dyadic stress physiology should continue to be explored as targets for intervention and prevention.

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- Lee, A. H., O'Brien, J. R., Binion, G., Lewis, J. K., & Zalewski, M. (revise & resubmit). Supportive emotion socialization as a moderator of the association between maternal emotion dysregulation and preschooler emotion regulation.
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ACKNOWLEDGEMENTS

I would like to express my deepest gratitude to my advisor, Dr. Maureen Zalewski, for all of her support and mentorship throughout my graduate training. Thank you for taking a chance on me and continuing to see me as a whole person throughout all of the successes and challenges of the past six years. Your endless encouragement, patience, fierce leadership, and timely advice have been a guiding light.

Thank you to the graduate students who have come before me – Nicole Musser, Jenn Lewis, Grace Binion – and those who will come after – Angela Lee, Ana Hernandez, Yoel Everett, and Simone Mendes. Thank you to all my family and friends who have believed in me and championed me along the way, including my partner and our dog, Molly. I hope I can provide the same support to you that you all have given me.

Thank you to the other members of my committee, Drs. Phil Fisher, Jenn Pfeifer, and Geovanna Rodriguez, for your feedback and guidance. Finally, a special thank you to all of the families who participated, as well as our project coordinators and undergraduate research assistants, without whom this work would not be possible.

This dissertation was supported by the Oregon Clinical and Translational Research Institute (TL1TR002371) from the National Center for Advancing Translational Sciences of the National Institutes of Health, as well as the Beverly Fagot Memorial Dissertation Fellowship.

“Your real job is that if you are free, you need to free somebody else. If you have some power, then your job is to empower somebody else.”

— Toni Morrison

This dissertation is dedicated to all of the families who participated in our research. I hope this brings us a small step closer towards providing care and resources to families in need.

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CHAPTER I

GENERAL INTRODUCTION

Children of parents with psychopathology are at risk for a range of socioemotional and behavioral difficulties and are at heightened risk to develop psychopathology at some point in their lives (Barnes & Stein, 2000; Ramchandani & Stein, 2003). This effect is not disorder-specific, such that parental psychopathology is associated with increased risk of children developing any type of mental health disorder (Dean et al., 2010; McLaughlin et al., 2012). The majority of research on child outcomes in the context of parental psychopathology has been most extensively examined in the milieu of maternal depression (Barnes & Stein, 2000; Weissman et al., 1997). The focus on maternal depression has in part been driven by the high prevalence rate of depression (7%) and the impact of depression on interpersonal factors that may impact mother-child relationships, such as negative affect and withdrawal. Despite borderline personality disorder (BPD) having prevalence rates estimated between 2-5% (American Psychiatric Association, 2013) and being characterized by emotion dysregulation, interpersonal chaos, and impulsivity, child outcomes in the context of maternal BPD remain understudied.

Borderline Personality Disorder and Parenting Behaviors

Borderline personality disorder affects over 3 million individuals in the United States each year and for whose treatment places a tremendous burden on our healthcare system (Soeteman et al., 2008). Individuals with BPD are 45 times more likely to commit suicide than the general population (Chesney et al., 2014) and account for up to 20% of psychiatric inpatients (American Psychiatric Association, 2001). Individuals with BPD

are also more likely to develop other chronic medical and mental illnesses (El-Gabalawy et al., 2010). Epidemiological surveys of individuals with BPD have found that up to 83% were diagnosed with a lifetime mood disorder/episode, 85% experienced a lifetime anxiety disorder, and 78% had a co-occurring lifetime substance use disorder (Tomko et al., 2014). Even when controlling for co-morbid diagnoses and sociodemographic factors, a BPD diagnosis significantly predicts impairment of social and emotional function, as well as poorer general health (Tomko et al., 2014). Given the severity of BPD, coupled with a nascent research area, it is important to consider the impacts on children who have a parent with BPD (Stepp et al., 2012).

The leading systematic review on the association between maternal BPD, parenting, and child outcomes found that children of mothers with BPD were more likely to exhibit internalizing (e.g., depression, emotional difficulties) and externalizing behaviors (e.g., aggression, attention deficit hyperactivity), attachment difficulties, emotion dysregulation, and eventually develop their own BPD symptoms (Eyden et al., 2016). These psychosocial and mental health difficulties described were present in infancy and persisted throughout childhood, adolescence, and early adulthood (Eyden et al., 2016). In this review, there were 11 papers that specifically examined outcomes for children ages 3-9 with the majority of these findings focused on child outcomes that are features of BPD such as emotion dysregulation and internalizing/ externalizing behaviors (Eyden et al., 2016). While children of mothers with BPD are at greater risk for developing a range of psychopathologies (Barnow et al., 2013; Barnow et al., 2006), it remains less clear the various pathways through which this risk is conferred.

Research thus far has focused primarily on parenting as a potential mechanism

through which this transmission occurs. This same systematic review showed that mothers with BPD are more likely to engage in negative parenting behaviors such as being insensitive, overprotective, and hostile compared to mothers without BPD (Eyden et al., 2016). These types of negative parenting behaviors may be a potential mediator through which maternal BPD is associated with negative child outcomes (Eyden et al., 2016). Mothers with BPD are more likely to engage in poor quality parenting, which is associated with worse mental and physical health outcomes in offspring (Petfield et al., 2015). However, although this is the pattern of parenting behaviors for mothers with BPD on average, there remains variability in quality of parenting behaviors within this population, such that some mothers with BPD may exhibit parenting behaviors comparable to mothers without BPD.

In support of this, this same systematic review showed that maternal BPD was not associated with differential levels of warmth (Eyden et al., 2016). On average, despite displaying high levels of *negative* behaviors, mothers with BPD may therefore still display high levels of *positive* parenting behaviors such as warmth and responsiveness. Rather than two ends of a spectrum, positive and negative parenting behaviors are independent constructs such that higher levels of positive behaviors (e.g., warmth) are not equated with lower levels of negative behaviors. Effective maternal guidance is an additional dimension of parenting that has important implications for child development, particularly during the preschool period (Goodman & Gotlib, 1999; Hoffman et al., 2006). Maternal guidance shapes child development of self-regulation by providing developmentally appropriate support in order to enhance the child's ability to successfully engage in activities (Blandon & Volling, 2008). Positive parenting behaviors

and guidance are all associated with more favorable child outcomes such as higher levels of socioemotional competence (Barber, 1997; Raby et al., 2015). These behaviors may serve as a protective factor for children of mothers with BPD given that parenting quality is highly responsive to intervention in both normative and clinical samples (Eshel et al., 2006). If mothers with BPD display higher levels of positive parenting behaviors and guidance, this has important clinical implications for parenting intervention targets. This effect may also be a protective factor unique to mothers with BPD, as mothers with other types of psychopathology such as depression have been shown to exhibit lower levels of positive parenting than mothers without psychopathology (Pelaez et al., 2008).

Child Stress Physiology as a Mechanism for Transmission of Mental Health

Outcomes

In addition to parenting, disrupted stress physiology has been another commonly studied mechanism for the relationship between maternal psychopathology and negative child outcomes. For example, blunted maternal and child basal cortisol patterns have been found to be a significant pathway linking maternal depressive symptoms and higher levels of child internalizing behaviors in preschool-aged children (Laurent et al., 2013). While there is limited research examining this pathway in mothers with BPD in particular, emotion dysregulation (a defining feature of BPD) was associated with disrupted stress physiology in both mothers and their children in the sample described in Study 1 of this dissertation (O'Brien et al., 2020). Stress physiology may be a particularly important pathway for the intergenerational transmission of mental health outcomes in the context of BPD, as individuals with BPD experience more stress life events than individuals with major depressive disorder (Pagano et al., 2004) or other types of

personality disorders (Jovev & Jackson, 2006; Pagano et al., 2004). Individuals with BPD demonstrate abnormal hypothalamus-pituitary-adrenal (HPA) axis activation (Zimmerman & Choi-Kain, 2009) and disrupted salivary diurnal cortisol cycles (Lieb et al., 2004). Salivary diurnal cortisol levels indicate day-to-day fluctuations in cortisol levels and are particularly sensitive to *acute* stressors (Dickerson & Kemeny, 2004). Fluctuations in salivary diurnal cortisol levels across days are associated with severity of mental health symptoms (Peeters et al., 2004). Women with BPD have been shown to demonstrate both elevated (Lieb et al., 2004; Rausch et al., 2015) and blunted levels of cortisol (Carrasco et al., 2007), both of which in turn are associated with negative mental and physical health outcomes (Gunnar & Vazquez, 2001; Staufenbiel et al., 2013).

While salivary cortisol is an index of *acute* stress, hair cortisol concentrations may be a more appropriate means of indexing *chronic* stress (Russell et al., 2012) and mental health outcomes (Staufenbiel et al., 2013). Hair cortisol concentrations reflect cumulative levels of cortisol secretion, which makes it an ideal measure of chronic stress and allostatic load (Kirschbaum et al., 2009). Current measures of chronic stress rely mainly on self-report, which is limited by recall and reporting bias issues (Gow et al., 2010). Hair cortisol offers one of the few available measures of objective chronic stress (Gow et al., 2010) and has the advantage of quantifying an accumulation of stress over months as opposed to daily diurnal patterns (Russell et al., 2012). It also measures free, unbound cortisol, which makes it less susceptible to many covariates that influence salivary cortisol such as oral contraceptive use (Dettenborn et al., 2012). However, the research on hair cortisol is predominately cross-sectional and correlational, with little intervention work to establish how and to what extent hair cortisol concentrations can be

altered (Russell et al., 2012). Clinical norms have also not been established, which can make it difficult to compare mean level hair cortisol concentrations across samples (Russell et al., 2012). Because the home environment in which a mother has BPD is characterized by chronic stress and unpredictability (Macfie, 2009), hair cortisol concentrations are ideally suited to quantify this enduring stress. While blunted cortisol levels have been linked to a number of negative outcomes (Gunnar & Vazquez, 2001), the home environment in which a mother has BPD may be more consistent with elevated hair cortisol concentrations (Russell et al., 2012; Staufenbiel et al., 2013). Specifically, the experience of living with a mother with BPD may lead to an accumulation of cortisol secretion in their young children, which in turn may contribute to socioemotional and behavioral problems (Staufenbiel et al., 2013).

There is some evidence that parenting behaviors may interact with BPD symptoms in order to predict child stress physiology. For example, adolescents with BPD who experienced less parental protection exhibited higher baseline cortisol levels than both adolescents with BPD who experienced higher levels of parental protection and non-disordered controls. Perceived parenting quality may therefore influence cortisol stress response in offspring with BPD (Lyons-Ruth et al., 2011). However, it is not yet known how maternal BPD diagnosis interacts with parenting quality to predict child cumulative stress functioning, a more stable indicator of chronic stress. It is therefore important to know if parenting quality in the context of maternal BPD interacts to predict child hair cortisol concentrations, as this would have clear and important implications for intervention and disrupting the transmission of risk in this population. Interventions treating BPD that focus on chronic stress rarely measure stress physiology and

determining whether treatment improves stress physiology could redefine our understanding of how to interrupt the transmission of psychopathology in families.

Knowledge of the stress physiological functioning of children of mothers with BPD could also identify the mechanism for the intergenerational transmission of psychopathology in this population. Child stress physiology such as elevated hair cortisol concentrations has been linked to child behavior problems in other contexts but has yet to be established in this population. While the effect of maternal psychopathology on child hair cortisol concentrations is unclear and warrants further research (Gray et al., 2018; Simmons et al., 2019), child hair cortisol levels have been associated with exposure to traumatic events (Simmons et al., 2016) and low socioeconomic status (Gray et al., 2018; Simmons et al., 2019) that in turn are associated with having a mother with BPD. Given the high degree of impairment and treatment resistance that characterizes BPD, it is crucial to know whether this link is established during early childhood in order to provide as much time as possible to intervene and prevent future psychopathology.

Theoretical Models of Early Life Stress and Child Stress Physiology

There have been several leading theories on how early adverse experiences “get under the skin” in order to impact child stress physiology. The biological sensitivity to context (BSC) model posits that there is a U-shaped relation between early adversity and stress physiological profiles, such that highly reactive profiles are seen when children are exposed to both highly stressful and highly stable/supportive environments (Boyce & Ellis, 2005). Heightened reactivity profiles allow children from high stress environments to react quickly to stressors, while heightened reactivity in children from supportive environments allow them to gain the social and developmental benefits of their

environment (Boyce & Ellis, 2005). The BSC theory came from empirical evidence that children with high reactivity stress profiles showed higher rates of mental and physical health problems than non-reactive peers when they were raised in a high stress environment but had better mental and physical health outcomes than non-reactive peers if they were raised in highly supportive environments (Boyce, 1996; Ellis et al., 2011; Essex et al., 2011; Thomas et al., 1995). This theory was then extended by the adaptive calibration model (ACM) by adding a fourth profile of stress reactivity in addition to the three given by the BSC (1- high reactivity in a supportive environment, 2- high reactivity in a stressful environment, 3- low reactivity in moderate environments). The fourth profile introduced by the ACM described the phenotype of low reactivity following severe adversity (Del Giudice et al., 2011). The ACM also extended research on child stress physiology by including profiles of stress responses across different systems (i.e. the parasympathetic and sympathetic nervous systems and HPA-axis) and considering key developmental stages when exposure to adversity may impact stress physiology (Del Giudice et al., 2011).

However, these models did not distinguish between types of adversity and how different adverse experiences may differentially impact stress response. To fill this gap, recent work on child stress physiology has benefitted from theoretical models emphasizing a dimensional approach to studying childhood adversity that distinguishes between experiences of deprivation and threat (McLaughlin & Sheridan, 2016; McLaughlin et al., 2014; Sheridan & McLaughlin, 2014). Deprivation (i.e., the absence of environmental and social inputs) and threat (i.e., experiences of harm or threat of harm) differentially impact learning processes and stress neurobiology (McLaughlin &

Sheridan, 2016). This model has highlighted the importance of understanding how different types of early life stress (e.g., maternal psychopathology, negative parenting) may differentially impact child stress physiology. In practice however, adverse childhood experiences often co-occur (for review see Herrenkohl & Herrenkohl, 2009) and experiences of deprivation and threat overlap (Smith & Pollak, 2020). For example, living in a high-violence neighborhood represents a threat of harm and is also more likely to be food desert with limited access to nutritious foods. There is also evidence that stress response systems are not sensitive to specific types of stress and that differences in stress responses are due to individual variation rather than stressor type (Korte et al., 2005; McEwen & Gianaros, 2010). One of the newest theoretical model of how early life stress impacts child stress physiology, the Topological Models of adversity, posits that the type of stressful event the child was exposed to has less of an impact on child stress response than factors that influence how the child experiences the event (Smith & Pollak, 2020). The model then describes several important factors that influence how the child experiences the event, including features of the event (e.g., chronicity, intensity); the child's environment (e.g., predictability, stability); social context (e.g., social support, safety), and individual differences (e.g., genetics, temperament; Smith & Pollak, 2020).

While this dissertation does not directly test a specific model of how early life experiences impact child stress physiology, these models provide a useful framework for considering how maternal BPD may influence the development of child stress physiology. Especially when considering how maternal BPD symptoms intersect with child experiences of threat and deprivation, as well as the broader child environment. When examining the impact of maternal BPD on child mental health and stress

physiology, researchers often analytically control for other factors that are known to negatively impact child development. This includes maternal trauma symptoms, income, single parent status, and a range of other risk factors. However in this context, it is important to carefully consider whether these factors should be controlled for as potential third variables contributing to child developmental outcomes or whether these features of risk are a manifestation of maternal BPD that contribute to a disadvantaged environment. By definition, personality disorders are pervasive and cause impairment in multiple domains of social, occupational, and general functioning (American Psychiatric Association, 2001). Borderline personality disorder in particular is characterized by a pervasive pattern of instability in interpersonal relationships, self-image, and affect (American Psychiatric Association, 2001). Mothers with BPD are therefore more likely to be a single parent, unemployed, and low income (McGlashan, 1986; Paris et al., 1987), which are all associated with negative child mental and physical health outcomes. Researchers should therefore be cautious when choosing what factors to include as analytic covariates, and should err on including fewer rather than greater numbers of covariates, as these factors may be mechanisms through which maternal BPD influences child outcomes (Binion, 2020).

Dissertation Aims

The overarching goal of this dissertation is to identify the role of positive parenting in mitigating the effects of maternal BPD on child stress physiology. Specifically, the two aims of this dissertation are to 1) identify associations between maternal BPD symptoms and observed parenting behaviors and 2) examine whether positive parenting behaviors are a protective factor for adaptive patterns of child stress

physiology. To do this, two studies were conducted using both a clinical and community sample of mothers with BPD symptoms to examine both salivary cortisol levels and hair cortisol concentrations as measures of stress physiology.

Study 1 Aims

The first study will use a community sample of mothers with a full range of BPD symptoms to examine how positive parenting may mitigate the risk of disrupted child stress physiology. Bivariate associations between maternal BPD symptoms and observed positive parenting behaviors will first be examined in line with aim 1 of this dissertation. Two specific measures of positive parenting will be used (positivity and guidance) in order to explore two potential aspects of positive parenting that may serve as protective behaviors. This study will then characterize the association between maternal BPD symptoms and diurnal levels of mother and child cortisol to examine acute stress physiology and fluctuations in daily salivary cortisol levels. In line with aim 2 of this dissertation, maternal positivity and guidance will then be examined as moderators to test whether higher levels of positive parenting mitigate the risk of maternal BPD symptoms and maternal cortisol levels on disrupted child stress physiology.

Study 2 Aims

The second study will directly extend the findings from Study 1 by further examining the aims of this dissertation using a clinical sample of mothers with BPD. First, group differences between mothers without BPD symptoms and mothers with elevated BPD symptoms will be examined in relation to four parenting behaviors — positivity, guidance, negativity, and intrusive control. A strength-based framework will then be used by examining how positive parenting behaviors (i.e., positivity and

guidance) may mitigate the risk for disrupted child stress physiology in the context of mothers with BPD symptoms. Hair cortisol concentrations will be used as a measure of stress physiology in place of salivary cortisol levels, as it has the benefit of quantifying chronic stress levels that may more accurately characterize the child environment of having a mother with BPD (Macfie, 2009).

CHAPTER II

STUDY 1: ASSOCIATIONS BETWEEN MATERNAL BPD, PARENTING, AND MOTHER-CHILD SALIVARY CORTISOL LEVELS

Disrupted child stress physiology has implications for development across the lifespan and has been linked to a number of adverse mental and physical health outcomes in children (e.g., Adam et al., 2017). Identifying areas of prevention and intervention to improve child stress physiology has the potential to alter the trajectory of child development (Fisher et al., 2016). Several important factors have been identified in influencing the development of adaptive or disrupted child stress physiology, including maternal stress physiology, maternal psychopathology, and parenting. Maternal stress physiology can provide early scaffolding in helping infants regulate their own stress (Feldman, 2007, 2012). However, maternal psychopathology can cause disruptions to maternal stress physiology (e.g., LeMoult et al., 2015). In this context, similarity between child and maternal stress physiology may be a risk factor for negative child health outcomes (Creaven et al., 2014). Despite being conceptualized as a disorder marked by increased vulnerability to stress (Zimmerman & Choi-Kain, 2009), maternal borderline personality disorder (BPD) has not yet been examined in association with child stress physiology. There is a growing evidence base on the association of maternal BPD symptoms on parenting behaviors, which have the potential to mitigate the effects of maternal stress physiology and psychopathology on child stress physiology. Research on parenting and child stress physiology has often focused on negative parenting behaviors as a risk factor for the disruption of child stress physiology (e.g., Taylor et al., 2013) rather than positive parenting behaviors that may be protective factors. The aim of this

study was to examine how positive parenting behaviors may mitigate the risk of disrupted child stress physiology within the context of maternal stress physiology and symptoms of maternal BPD using a community sample of mothers with a range of BPD symptoms. Specifically, this study focuses on diurnal salivary cortisol levels as a measure of stress physiology that is particularly sensitive to acute stressors.

Development of Child Stress Physiology

Stress physiology has commonly been studied within the context of the hypothalamus-pituitary-adrenal (HPA) axis, by examining hormonal cortisol output. The HPA axis helps to regulate the body's reactions to both acute and chronic stressors, and follows a natural diurnal rhythm where cortisol peaks in the morning and gradually decreases throughout the day (Sapolsky et al., 2000). This diurnal rhythm has been observed consistently throughout early childhood, and in children as young as two years old (Gunnar & Donzella, 2002). In response to childhood stressors, this system can become disrupted such that cortisol levels fail to increase in the morning or remain elevated in the evening (e.g., Cicchetti & Rogosch, 2001). Early childhood adversity has been associated with both elevated (Cicchetti & Rogosch, 2001; Gunnar et al., 2001) and blunted (Gunnar & Vazquez, 2001) patterns of diurnal cortisol. In turn, both elevated and blunted cortisol levels have been associated with negative mental health outcomes such as ineffective emotion regulation (Laurent, 2014), higher levels of behavior problems (Alink et al., 2008), and lower psychosocial functioning (Kushner et al., 2016).

Mothers play an important role in the development of their children's stress physiology. Mother-child co-regulation and similarity in physiological patterns has been considered an important developmental milestone for adaptive functioning (Feldman,

2007, 2012; Jaffe et al., 2001). However, children who display similar physiological profiles as their mothers may be at risk for negative physical and psychological outcomes if their mothers display disrupted stress physiology, as this could lead to a dually dysregulated sample (Creaven et al., 2014). Psychopathology can be a risk factor for maternal disrupted stress physiology, as research has consistently found that mothers with depression exhibit higher levels of morning and total cortisol output compared to mothers without a history of depression (e.g., Vreeburg et al., 2009; LeMoult et al., 2015). While twin studies indicate that genetics play a moderate role in diurnal cortisol patterns (Gustafsson et al., 2011; Kupper et al., 2005), parental psychopathology can influence child cortisol levels even when accounting for genetic heritability (Laurent et al., 2013; Schreiber et al., 2006). For example, children and adolescents of mothers with depression have been shown to exhibit disrupted cortisol patterns, such as lower diurnal cortisol output (Laurent et al., 2013), higher peak morning cortisol levels (LeMoult et al., 2015), and greater variability in morning cortisol levels (Halligan et al., 2004). Disrupted stress physiology may therefore be an important pathway for the intergenerational transmission of risk for psychopathology.

Stress Physiology in the Context of BPD

Disrupted stress physiology has been commonly researched as a mechanism for the intergenerational transmission of risk for maternal depression and anxiety. However, there has been limited research examining stress physiology within the context of maternal borderline personality disorder (BPD) despite the high rates of intergenerational transmission of BPD. Individuals with BPD may be especially at risk for disrupted stress physiology given that core symptoms of the disorder (e.g., emotion dysregulation,

impulsivity, self-injury) can be conceptualized as both increased vulnerability and maladaptive responses to stress (Zimmerman & Choi-Kain, 2009). Individuals with BPD have also been shown to experience more stressful life events compared to individuals with other types of personality disorders (Jovev & Jackson, 2006; Pagano et al., 2004) and when compared to individuals with major depressive disorder (Pagano et al., 2004). While women with BPD have been shown to exhibit both blunted (Carrasco et al., 2007) and elevated (Lieb et al., 2004; Rausch et al., 2015) diurnal cortisol levels, to our knowledge there has been no research examining child stress physiology within the context of maternal BPD. An emerging area of related research has shown that adolescents with BPD display disrupted cortisol response to stressors (Walter et al., 2008) and elevated baseline cortisol levels (Lyons-Ruth et al., 2011) compared to non-disordered controls. Several features of BPD, including emotion dysregulation and executive functioning, develop rapidly during the preschool period (Cole et al., 2009; Carlson & Moses, 2001). This developmental period may be a particularly important time in which to examine stress physiology within the context of maternal BPD.

Parenting as a Protective Factor

Parenting has been examined as an important area of prevention and intervention through which the impact of early adversity on child stress physiology can be mitigated (e.g., Fisher et al., 2007). While poorer quality parenting such as lower levels of warmth have been associated with disrupted child stress physiology (e.g., Zalewski et al., 2012), parenting quality is highly responsive to intervention (Eshel et al., 2006). Parenting interventions have also been shown to improve child stress physiology (Fisher et al., 2007), with parenting interventions during infancy continuing to be positively associated

with child diurnal cortisol functioning in preschool (Bernard et al., 2015). Parenting quality such as higher levels of maternal responsiveness has been shown to weaken the similarity in disrupted mother-child cortisol patterns (Williams et al., 2013), which may serve to mitigate the transmission of risk for maladaptive stress physiology. The healthy development of early child stress physiology relies on external cues for regulation, including both maternal stress physiology (Feldman, 2007) and parenting behaviors (Karreman et al., 2006). In terms of specific parenting behaviors, maternal guidance or scaffolding most closely resembles this external regulatory function by developmentally supporting and enhancing the child's ability to successfully engage in activities (Karreman et al., 2006). Effective maternal guidance has been positively associated with children's ability to self-regulate (Karreman et al., 2006) and is posited to be especially important for the development of independent child regulatory functioning during the preschool period (Goodman & Gotlib, 1999; Hoffman et al., 2006). Child stress physiology may therefore be particularly influenced by maternal guidance.

Within the context of maternal psychopathology, lower quality parenting associated with maternal symptoms of depression (Dougherty et al., 2011) and BPD symptoms (Lyons-Ruth et al., 2011) has been linked to disrupted child cortisol functioning. On average, mothers with elevated BPD symptoms are more likely to engage in lower quality parenting characterized by high levels of insensitive, overprotective, and hostile parenting behaviors than mothers without BPD (Eyden et al., 2016). However, this same systematic review found that mothers with elevated BPD symptoms exhibited similar levels of maternal warmth as mothers without BPD (Eyden et al., 2016). This may be a unique strength of mothers with BPD, as maternal psychopathology in other contexts

(e.g., maternal depression) has been consistently associated with lower levels of warmth (Pelaez et al., 2008). Positive parenting may therefore serve as a protective factor mitigating the negative impact of maternal BPD symptoms on child cortisol functioning. Importantly, this review used a dimensional approach that included maternal BPD symptoms at subclinical levels rather than exclusively mothers with a BPD diagnosis (Eyden et al., 2016), as even one symptom of BPD has been associated with significant functional impairment (Ellison et al., 2016).

Current Study

This study uses a community sample of mothers with a full range of BPD symptoms to examine how positive parenting behaviors can moderate the associations between maternal stress physiology and BPD symptoms to mitigate the risk of disrupted child stress physiology. Specifically, diurnal salivary cortisol levels will be used to measure acute stress physiology in both mothers and their preschool-aged children. Maternal positivity and guidance will be used as two distinct measures of positive parenting that may serve as protective factors for adaptive patterns of child stress physiology. In order to contextualize the relationship between maternal and child stress physiology, the association between maternal BPD symptoms and maternal salivary cortisol levels will first be tested in order to examine whether higher levels of maternal BPD symptoms are associated with disrupted maternal stress physiology. Consistent with prior findings in this sample that examined maternal emotion dysregulation (O'Brien et al., 2020) and prior research finding that evening cortisol levels are more heavily influenced by daily stressors than morning cortisol levels (Gustafsson et al., 2011; Kupper et al., 2005), we hypothesize that maternal BPD symptoms will be positively

associated with higher levels of maternal and child evening cortisol. A direct effect of maternal stress physiology is also hypothesized, such that higher maternal evening cortisol levels will be associated with higher child evening cortisol levels. If evidence of these pathways are found, exploratory analyses will be used to test the indirect effect of maternal BPD symptoms on child evening cortisol levels through its influence on maternal evening cortisol levels. We further hypothesize that maternal positivity and guidance will moderate the associations between maternal cortisol levels and BPD symptoms to mitigate the risk of disrupted child cortisol levels. Specifically, higher levels of maternal positivity and guidance will serve as a protective factor mitigating the positive association of maternal cortisol levels and BPD symptoms on child cortisol levels.

Methods

Participants

Mothers and their preschool-aged children ($N = 68$; $M_{age} = 48$ months, $SD = 7.6$ months, 46% girls) were recruited from a variety of sources, including Craigslist, community mental health centers, and a university developmental database. Mothers were recruited based on the presence or absence of BPD symptoms using the McLean screener (Zanarini et al., 2003). Mothers endorsing a range of BPD symptoms were included, such that on the McLean screener (range 0-9), 27% ($n=18$) endorsed 0-1 items, 24% endorsed 2-4 items ($n=16$), and 49% endorsed 5 or more items ($n=33$). To ensure a range of BPD symptoms were present in the sample, a cutoff score of 5 was used rather than the typical cutoff of 7. Given that mothers with elevated BPD symptoms were more likely to identify as low income, mothers with low income and low BPD symptoms were

oversampled in order to offset this association. Total family annual income of this sample represented in quartiles is: (1) less than \$17,000, (2) \$17,000-\$29,000, (3) \$29,001-\$50,000 and (4) \$50,000 or more.

Eligible mothers were over the age of 18 and had shared or full custody of a 3-4 year old child. Children with known developmental disabilities were excluded. The majority of children in this sample were European American (63.2%), with an additional 5.9% identifying as Latino or Hispanic, 1.5% African American, and 29.4% identifying as having multiple racial and ethnic backgrounds or a racial/ethnic background not listed. Educational distribution included 5.9% mothers with some high school attainment, 4.4% completed high school, 36.8% with some college, 14.7% technical school or professional school, 26.5% college graduates, and 10.3% with post-graduate education. Sixty-two percent of mothers in this sample were married or had long-term partners, 25% were never married, and 13% were separated, divorced or widowed and were single heads-of-household. Overall this sample can be described as a low income, community sample exhibiting a wide range of BPD symptoms.

Procedure

All procedures received Institutional Review Board approval prior to data collection. After mother consent and child assent were obtained, dyads participated in a 2.5-hour assessment in university campus offices. Mothers completed questionnaires with the guidance of an experimenter while children completed a series of tasks with an additional experimenter in an adjacent room. Dyads then reunited to complete a number of parent-child interaction tasks. At the end of the assessment, experimenters demonstrated cortisol collection procedures and trained mothers in home cortisol data

collection. Mothers then collected morning and evening salivary cortisol samples across three consecutive days for themselves and their child. Dyads received \$50 for participating in the assessment, and an additional \$30 for cortisol collection.

Measures

Maternal Borderline Personality Disorder Symptoms

Mothers reported on their BPD symptoms using the Personality Assessment Inventory - Borderline Features Scale (PAI-BOR; Morey, 1991), a 24-item questionnaire where mothers rated how accurate each statement was in describing themselves on a scale of 0 (False) to 3 (Very True). Items included statements such as “My mood can shift quite suddenly”, “Sometimes I feel terribly empty inside”, and “I’m a reckless person”. Possible scores range from 0 to 72, with higher scores indicating higher levels of BPD symptoms. In non-clinical samples, a 6-factor has been shown to best fit item covariances (Jackson & Trull, 2001), where items map onto the following 6 factors: impulsivity/dyscontrol, mood instability, chronic emptiness, separation concerns, negative relations, and reckless spending. The PAI-BOR has demonstrated good internal consistency (Morey, 1991), as well as construct and convergent validity (Gardner & Qualter, 2009). The PAI-BOR has also shown good discriminant validity when comparing individuals with BPD and major depressive disorder, major depressive disorder without a comorbid BPD diagnosis, and a control group (Kurtz & Morey, 2001). In this sample, mean scores on the PAI-BOR indicated non-clinical levels of BPD symptoms on average ($M= 22.61$), with a wide range of BPD symptoms present ($SD = 12.26$). Fourteen percent of the sample ($N = 9$) scored above the suggested clinical cut-off of 38.

Parenting

Parenting behaviors were observed during a 7-minute dyadic interaction task (adapted from Kerig & Lindahl, 2000). During this task, dyads were instructed to complete a Lego figure that is considered too complex for the child to complete independently. Mothers were instructed not to touch any of the Lego pieces and assist the child using only verbal commands. Parenting behaviors were then coding using the Parent-Child Interactions Observational Coding Manual that was adapted from previously established coding systems (Cowan & Cowan, 1992; Lindahl & Malik, 2000; Rubin & Cheah, 2000). Behaviors were coded in 1-minute epochs and then averaged across the task. For each parenting behavior, 20% of cases were randomly assigned for double coding by independent coders to calculate interrater reliability.

Maternal positivity. Maternal displays of positivity were coded as both the intensity and frequency of verbal and non-verbal displays of positivity, comfort, and happiness. Positivity showed excellent reliability in this sample (Cronbach's alpha = .870). Mothers displayed high levels of positivity ($M= 3.62$, $SD= 0.95$) that was normally distributed (skewness = -.805, kurtosis = .615).

Maternal guidance. Maternal guidance was operationalized as efforts to guide and structure the task to facilitate child's successful engagement. This included maternal efforts to explain the task and offer self-initiated assistance without the child needing to request assistance. Maternal guidance showed excellent reliability in this sample (Cronbach's alpha = .813). Mothers displayed high levels of guidance ($M= 3.90$, $SD= 1.10$) that was slightly skewed towards higher levels of maternal guidance (skewness = -1.10, kurtosis = .624).

Mother and Child Salivary Cortisol Levels

Saliva samples were stored at -20° C and then sent to Salimetrics LCC (State College, PA) for assay using High-Sensitivity Cortisol Salivary Enzyme Immunoassay Kits. Duplicate samples for each assay were used, such that 25 µL of saliva from each sample were transferred into two wells. Sample values were then averaged. Cortisol concentrations were then extrapolated from a standard curve that was generated in each test plate and the results were averaged in order to give an adjusted result. All samples were assayed once all cortisol had been collected, and all samples from the same participant were included in the same assay batch in order to minimize inter-assay and within-subject variability. The intra-assay cortisol value (CV) was 6.0% and the inter-assay CV was 7.1%.

Daily questionnaires were completed by mothers for both themselves and their children regarding wake and bed times, sampling times, health, medication use, and eating times on sampling days. All questionnaires were reviewed to ensure compliance, and all families were called on the first evening of collection to review procedures. At this time, mothers were also reminded to avoid sampling on days when they or their child were ill or using steroid-based medications. In order to accurately measure peak morning cortisol levels, morning samples taken less than 15 minutes or more than 45 minutes after waking were excluded from analyses (Stalder et al., 2016). Evening samples taken more than 90 minutes before bed time were also excluded.

Cortisol values were positively skewed, so log transformations were applied to all values to meet assumptions of normality. The correlation between child cortisol levels across the three sampling days ranged from $r = .156$ ($p = .385$) to $.294$ ($p = .092$) in the

morning and from $r = .355$ ($p = .105$) to $.548$ ($p = .005$) in the evening. The correlation between mother cortisol values also varied in significance across the three sampling days, ranging from $r = .069$ ($p = .697$) to $.518$ ($p = .006$) in the morning and from $r = .356$ ($p = .104$) to $.747$ ($p < .001$) in the evening. For each member of the dyad, an average morning and evening cortisol value was calculated in order to create a more stable measure of cortisol levels across the three sampling days. The average morning untransformed cortisol value was $.273$ ($SD = .111$) for mothers and $.243$ ($SD = .100$) for children. The average evening untransformed cortisol value was $.049$ ($SD = .049$) for mothers and $.064$ ($SD = .087$) for children.

Missing Data

Of the 68 families who participated in the study, 16 families (23.53%) did not return any samples; an additional 21 families (30.88%) were missing at least one sample; and 9 families (13.24%) were missing samples due to non-compliance with sampling time. Families who returned at least one morning sample and one evening sample were retained in all analyses. Consistent with prior research (Ashman et al., 2002), cortisol data for one family was excluded due to biologically implausible values ($> 2 \mu\text{g/dl}$). The cortisol data was also visibly inspected for outliers, which led to an exclusion of one dyad's cortisol data ($> 3 SD$'s above the mean). Families with cortisol samples were compared to those missing cortisol samples on measures of maternal BPD symptoms and found that maternal BPD symptoms was not associated with missing samples, $t(64) = .381, p = .705$.

Analytic Plan

All analyses were conducted in SPSS (ver. 26, IBM Chicago, IL, USA). Bivariate correlational analyses were used to identify associations between all variables of interest (see Table 1). Potential covariates were examined prior to inclusion, including child gender, pregnancy, and use of hormonal birth control, which were all non-significant (p 's $> .05$). Household income was significantly related to maternal BPD symptoms ($r(66) = -.319, p = .009$) and was therefore included as a covariate in all analyses. Associations between mother and child wake and bed times; latency to collect samples; and maternal and child cortisol values were examined. Maternal wake ($r = -.533, p < .001$) and collection ($r = -.522, p < .001$) times were significantly correlated with maternal morning cortisol levels and were included as covariates in models examining maternal morning cortisol levels. Child collection ($r = -.295, p = .049$) and bed time ($r = -.335, p = .024$) were significantly related to child evening cortisol levels and were included as covariates in models examining child evening cortisol levels. No other significant associations between mother and child wake and bed times; latency to collect samples; and maternal and child cortisol values were found (p 's $> .05$). Curvilinear associations between maternal BPD symptoms and maternal cortisol levels were also explored by plotting maternal BPD symptoms against maternal mean-centered morning and evening cortisol levels. No evidence of a curvilinear relation was indicated (see Figures 1 and 2).

A series of hierarchical linear regressions were then conducted with all covariates entered at level 1. First, the association between maternal BPD symptoms and maternal salivary cortisol levels was examined. Second, an interaction term was computed as the multiplicative of maternal BPD symptoms (mean centered) and maternal positivity (mean centered) as predictors of child salivary cortisol levels. A parallel multiple linear

regression model was then conducted with maternal guidance. Finally, an interaction term was computed as the multiplicative of maternal salivary cortisol levels (mean centered) and maternal positivity (mean centered) as predictors of child salivary cortisol levels. A parallel multiple linear regression model was then conducted with maternal guidance. Bonferroni corrections were applied to reduce the inflation of Type 1 error rates in order to adjust for the number of regression models tested. Given the very conservation nature of this approach, Bonferroni corrections were applied to each set of dependent variables rather than the total number of regressions. Therefore, the criterion for the four models examining child morning cortisol levels, as well as the four models examining child evening cortisol levels, was adjusted to 0.0125.

Table 1

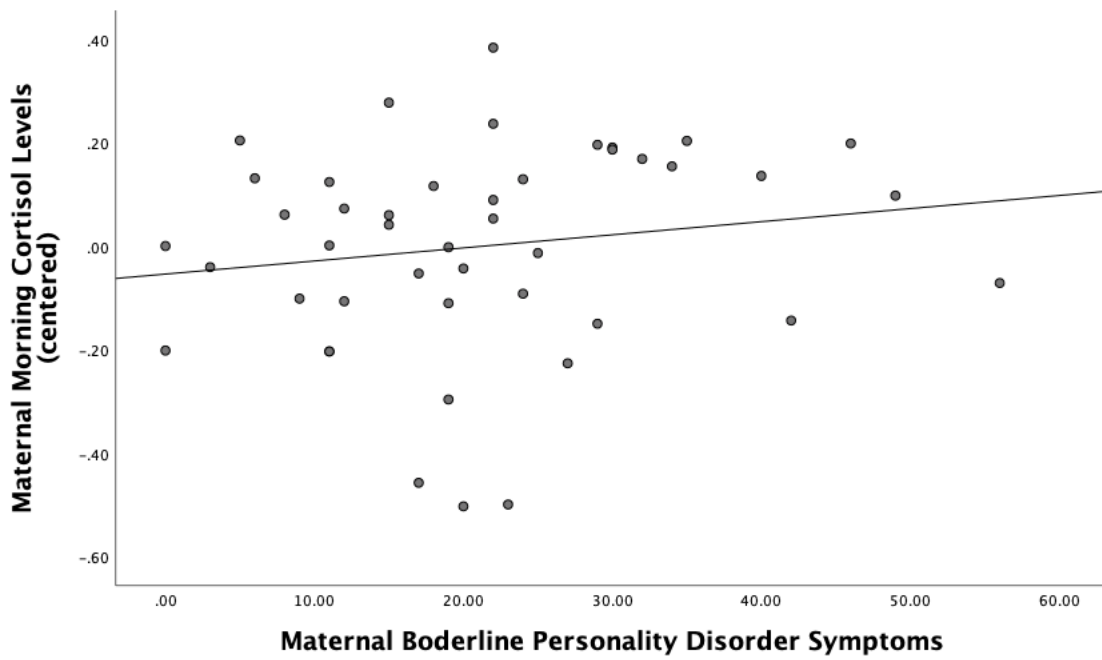
Study 1 - Bivariate Pearson's Correlations Between Variables of Interest

	Income	Maternal BPD	Maternal AM Cort	Child AM Cort	Maternal PM Cort	Child PM Cort	Positivity
Income	1						
Maternal BPD	-.319**	1					
Maternal AM Cort	.027	.160	1				
Child AM Cort	-.109	.229	.175	1			
Maternal PM Cort	-.243	.496**	.172	.281†	1		
Child PM Cort	-.185	.253†	.009	.207	.599**	1	
Positivity	.140	-.243†	.108	-.024	-.194	-.197	1
Guidance	.029	.171	.083	-.093	.106	.088	.193

† $p < .1$, * $p < .05$, ** $p < .01$

Figure 1

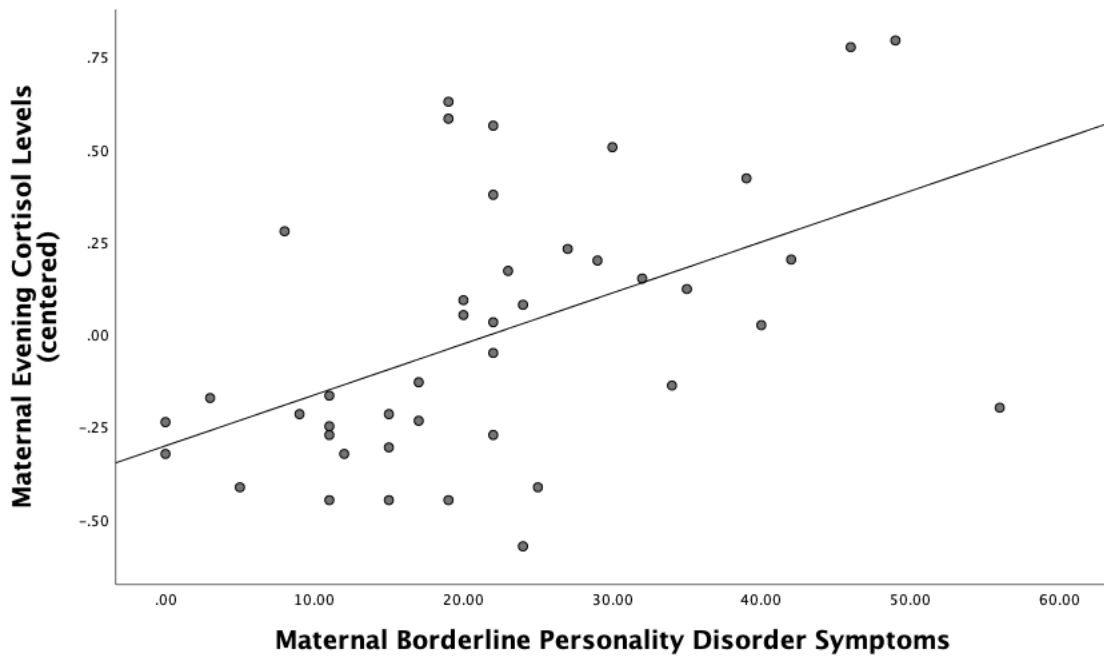
Study 1 – Associations between Maternal BPD Symptoms and Maternal Morning Cortisol Levels



Note. Associations between maternal borderline personality disorder (BPD) symptoms and mean-centered maternal salivary morning cortisol levels (log transformed) in a community sample of mothers with a range of BPD symptoms. No evidence of a curvilinear association is indicated.

Figure 2

Study 1 - Associations between Maternal BPD Symptoms and Maternal Evening Cortisol Levels



Note. Associations between maternal borderline personality disorder (BPD) symptoms and mean-centered maternal salivary evening cortisol levels (log transformed) in a community sample of mothers with a range of BPD symptoms. No evidence of a curvilinear association is indicated.

Results

Maternal BPD Symptoms Predicting Maternal Cortisol Levels

Maternal BPD symptoms were not significantly related to maternal morning cortisol levels when controlling for income, wake time, and latency to collect (see Table 2). However, higher levels of maternal BPD symptoms were associated with higher levels

of maternal evening cortisol levels ($\beta = 0.502$, $t(39) = 3.106$, $p = .004$), significantly accounting for 24.5% of the variability in maternal evening cortisol levels, $R^2 = .246$, $F(2, 39) = 6.354$, $p = .004$.

Table 2

Study 1 - Maternal BPD Symptoms as a Predictor for Maternal Cortisol Levels

Model Predictors	R^2	(df)	F	p	β	t	p
Maternal AM Levels	.340	(4, 39)	5.022	.002**			
Intercept						2.826	.007**
Mom Avg Wake Time					-.569	-.442	.661
Mom Avg Time Collected					-.003	-.003	.998
Income					-.048	-.294	.770
Maternal BPD Symptoms					.212	1.335	.189
Maternal PM Levels	.246	(2, 39)	6.354	.004**			
Intercept						.050	.960
Income					.012	.075	.941
Maternal BPD Symptoms					.502	3.106	.004**

† $p < .1$, * $p < .05$, ** $p < .01$

Maternal BPD Symptoms and Parenting Behaviors Predicting Child Cortisol Levels

In the series of parallel linear regressions examining the effects of maternal BPD symptoms, parenting behaviors, and the interaction between maternal BPD symptoms and parenting behaviors on child cortisol levels, no significant effects were found (see Tables 3 and 4). There were no main effects of maternal BPD symptoms, positivity, or guidance, on child morning or evening cortisol levels, and no significant interactions.

Table 3*Study 1 - Maternal BPD Symptoms and Positivity as Predictors of Child Cortisol Levels*

Model Predictors	R^2	(<i>df</i>) F	p	β	t	p
Child AM Levels	.059	(4, 38) 0.591	.671			
Intercept					.105	.917
Income				-.020	-.104	.918
Maternal BPD Symptoms				.239	1.267	.213
Positivity				.059	.345	.732
BPD x Positivity				-.062	-.388	.700
Child PM Levels	.324	(6, 36) 2.882	.021*			
Intercept					3.458	.001**
Child Avg Time Collected				.673	1.327	.193
Child Avg Bed Time				-1.181	-2.253	.030*
Income				-.155	-.892	.378
Maternal BPD Symptoms				.227	1.374	.178
Positivity				-.247	-1.610	.116
BPD x Positivity				.139	.942	.352

† $p < .1$, * $p < .05$, ** $p < .01$ **Table 4***Study 1 - Maternal BPD Symptoms and Guidance Predicting Child Cortisol Levels*

Model Predictors	R^2	(<i>df</i>) F	p	β	t	p
Child AM Levels	.085	(4, 38) 0.885	.482			
Intercept					-.080	.936
Income				.019	.102	.919
Maternal BPD Symptoms				.234	1.219	.230
Guidance				-.114	-.706	.485
BPD x Guidance				.131	.818	.418
Child PM Levels	.298	(6, 36) 2.547	.037*			
Intercept					3.203	.003**
Child Avg Time Collected				.478	.915	.366
Child Avg Bed Time				-.933	-1.773	.085
Income				-.246	-1.387	.174
Maternal BPD Symptoms				.254	1.430	.161
Guidance				.095	.646	.522
BPD x Guidance				-.149	-1.004	.322

† $p < .1$, * $p < .05$, ** $p < .01$

Maternal Cortisol and Parenting Behaviors Predicting Child Cortisol Levels

In the series of parallel linear regressions, higher levels of maternal evening cortisol were found to significantly predict higher levels of child evening cortisol in models including maternal positivity ($\beta = 0.473$, $t(31) = 3.280$, $p = .003$) as well as maternal guidance ($\beta = 0.490$, $t(31) = 3.322$, $p = .002$). Maternal morning cortisol levels did not predict child morning cortisol levels in any models (see Tables 5 and 6). There were also no main effects of maternal positivity, guidance, or the interaction between maternal cortisol levels and parenting behaviors found (see Tables 5 and 6).

Table 5

Study 1 - Maternal Cortisol and Positivity Predicting Child Cortisol Levels

Model Predictors	R^2	(df) F	p	β	t	p
Child AM Levels	.046	(4, 37)	0.449			.773
Intercept					.424	.674
Income				-.113	-.638	.528
Maternal AM Cortisol				.179	1.103	.277
Positivity				.002	.013	.990
AM x Positivity				.021	.124	.902
Child PM Levels	.513	(6, 31)	5.440			.001**
Intercept					2.029	.051
Child Avg Time Collected				1.037	1.519	.139
Child Avg Bed Time				-1.298	-1.900	.067
Income				-.145	-.998	.326
Maternal PM Cortisol				.478	3.280	.003*
Positivity				-.168	-1.133	.266
PM x Positivity				.228	1.749	.090

[†] $p < .1$, * $p < .05$, ** $p < .01$

Table 6*Study 1 - Maternal Cortisol and Guidance Predicting Child Cortisol Levels*

Model Predictors	R^2	$(df)F$	p	β	t	p
Child AM Levels	.062	(4, 37) 0.606	.661			
Intercept					.491	.626
Income				-.115	-.712	.481
Maternal AM Cortisol				.203	1.258	.216
Guidance				-.116	-.717	.478
AM x Guidance				-.062	-.384	.703
Child PM Levels	.479	(6, 31) 4.743	.002*			
Intercept					1.817	.079
Child Avg Time Collected				.962	1.300	.203
Child Avg Bed Time				-1.192	-1.611	.117
Income				-.228	-1.535	.135
Maternal PM Cortisol				.490	3.322	.002*
Guidance				.120	.866	.393
PM x Guidance				-.071	-.505	.617

† $p < .1$, * $p < .05$, ** $p < .01$

Exploratory Analyses: Indirect Effect of Maternal BPD Symptoms on Child

Evening Cortisol Levels

Exploratory bootstrapping analyses were conducted to examine the indirect effect of maternal BPD symptoms on child evening cortisol levels through its influence on maternal evening cortisol levels, given that maternal BPD symptoms was associated with higher levels of maternal evening cortisol levels ($B= 0.014$, $t(40) = 3.255$, $p= .003$), which in turn was associated with higher levels of child evening cortisol levels ($B= 0.633$, $t(40) = 2.974$, $p= .005$). When controlling for income as well as child bed and collection time, the unstandardized direct effect of maternal BPD symptoms on child

evening cortisol levels remained non-significant ($B = -0.001$, $t(40) = -0.089$, $p = .930$). The unstandardized indirect effect of maternal BPD symptoms on child evening cortisol levels via maternal evening cortisol levels was significant using a 95% confidence interval based on 5,000 bootstrap samples ($B = .009$, 95%CI= [.002, .017]). While maternal BPD symptoms were not directly associated with child evening cortisol levels, this provides preliminary evidence that maternal BPD symptoms was indirectly associated with elevated levels of child cortisol through its influence on maternal evening cortisol levels. Given the cross-sectional design of this study, these exploratory findings should be interpreted with caution and replicated in a longitudinal study where temporal precedence can be established.

Discussion

This study was the first to examine child stress physiology within the context of maternal BPD, using a community sample of mothers with a range of BPD symptoms. Maternal BPD symptoms were found to be positively associated with higher levels of salivary cortisol in the evening for mothers. In turn, higher maternal evening cortisol levels were associated with higher levels of child evening cortisol. Maternal BPD symptoms, positivity, and guidance were not directly associated with child cortisol levels. Maternal positivity and guidance also did not moderate the associations between maternal cortisol levels and BPD symptoms to mitigate the risk of disrupted child cortisol levels. These results suggest that while maternal BPD symptoms directly impact maternal diurnal cortisol patterns, disrupted stress physiology in mothers was the strongest predictor for disrupted cortisol patterns in children.

These findings are consistent with prior research showing that maternal cortisol levels are a strong predictor of child cortisol levels, over and above socioeconomic status (Schreiber et al., 2006), maternal psychopathology (Merwin et al., 2017), and genetics (Schreiber et al., 2006). Mother-child physiological concordance has historically been considered a positive influence on child development (Feldman, 2007, 2012). However, while maternal physiology can positively influence child physiology and aid in helping the child to self-regulate (Feldman, 2007; Jaffe et al., 2001), children of mothers who exhibit disrupted stress physiology may be at risk for physiological dysregulation themselves. As research on the development of child stress physiology continues to develop, disrupted maternal stress physiology may be an important area of prevention and intervention. Future studies should examine how intervening with mothers to improve maternal stress physiology may positively impact the development of child stress physiology. In this sample, maternal BPD symptoms did not have a direct effect on child cortisol levels. However, higher levels of maternal BPD symptoms were associated with higher levels of maternal evening cortisol, which in turn predicted higher levels of child evening cortisol. The indirect effect of maternal BPD symptoms on child evening cortisol levels through its influence on maternal evening cortisol levels was also significant. Targeting maternal BPD symptoms may improve maternal stress physiology and act as a prevention tool for the intergenerational transmission of disrupted stress physiology. This approach may be particularly effective given that BPD can be conceptualized as a disorder characterized by increased vulnerability and maladaptive responses to stress (Zimmerman & Choi-Kain, 2009).

While maternal BPD symptoms did not directly impact child cortisol levels, prior research in this sample found that maternal emotion dysregulation (a key feature of BPD) was directly associated with elevated child evening cortisol levels (O'Brien et al., 2020). In this sample, maternal emotion dysregulation and BPD symptoms were correlated at .72 indicating that these are overlapping, yet distinct constructs. Emotion dysregulation may therefore be a more proximal risk factor through which maternal BPD symptoms impact the child's daily experience and stress physiology. Evening cortisol levels in particular have shown to be more influenced by daily stressors than morning cortisol levels, which have a higher genetic contribution (Gustafsson et al., 2011; Kupper et al., 2005). Maternal and child evening cortisol levels were highly correlated in this sample ($r = .599$), even compared to prior research of basal cortisol levels in dyadic samples of mothers with psychopathology (LeMoult et al., 2015; Merwin et al., 2017; Williams et al., 2013). Models that included maternal evening cortisol levels and parenting also significantly accounted for approximately 50% of the variation in child evening cortisol levels, which is notable considering that even 23% of variability in cortisol levels is considered significant (Williams et al., 2013).

In this sample, maternal displays of positivity and guidance were not associated with child cortisol levels. It may be that this sample was underpowered to detect such effects. This study was able to recruit an overall modest sample of mothers with a range of BPD symptoms and the 76% compliance for cortisol collection is comparable to other at-risk samples. However, post-hoc power analyses showed that this study only had 62-67% power to detect medium effects given the range of cortisol samples returned for each time point ($N = 40-45$). While there is no meta-analysis on the overall effect of positive

parenting behaviors on child salivary cortisol cross-sectionally, a meta-analysis on the longitudinal associations of parental warmth on child salivary cortisol found that small effects of parental warmth on child cortisol levels emerged only within the context of distinct moderators such as socioeconomic status and study design (Hackman et al., 2018). Although these effects were small, they are clinically meaningful as areas of intervention given that parenting quality is highly responsive to intervention (Eshel et al., 2006). However, this study was underpowered to detect small effects within the context of moderators.

This study had several strengths, including being the first study to examine child cortisol levels within the context of maternal BPD symptoms and parenting behaviors. Children of mothers with BPD symptoms are more likely to exhibit internalizing and externalizing behaviors (Eyden et al., 2016), and are at greater risk for developing a range of psychopathologies (Barnow et al., 2006; Barnow et al., 2013). However, children of mothers with BPD symptoms remain largely understudied compared to children of mothers with depression or anxiety. This study also used a strength-based approach to focus on positive parenting behaviors that mothers with a range of BPD symptoms display. Borderline personality disorder is a highly stigmatized disorder, even among mental health providers (Sansone & Sansone, 2013). Focusing on unique areas of strength mothers with BPD symptoms display can be a powerful tool to combat the stigma that mothers with BPD symptoms face. Parenting behaviors were also assessed using observational methods in order to reduce common method variance and reliance on maternal report for both maternal BPD symptoms and parenting behaviors.

These findings should be replicated in a larger sample in order to have appropriate power to identify small effects that may have important implications for child development. A larger sample size would also allow for additional analyses to explore whether specific symptoms or risk factors for child development that are associated with maternal BPD moderate or mediate the association between maternal BPD and child stress physiology. For example, prior research has found that 45-90% of individuals with BPD report a history of trauma (Zanarini et al., 1997; Zanarini et al., 1998), and a review on HPA-axis functioning in individuals with BPD found that posttraumatic stress disorder symptoms can moderate the association between BPD symptoms and cortisol levels (Zimmerman & Choi-Kain, 2009). Mothers with BPD are also more likely to be unemployed, a single parent, and low income (McGlashan, 1986; Paris et al., 1987). Higher powered samples would be able to better identify how controlling for these known risk factors for child development influence the unique contribution of maternal BPD symptoms on child stress physiology.

This study used a community sample of mothers with a range of BPD symptoms. Given this preliminary evidence for the impact of maternal BPD symptoms on maternal and child stress physiology, future research should explore these associations using a more robust measure of BPD such as diagnostic interviews. A more robust measure of BPD would also allow for a closer examination between parenting behaviors that are particularly influenced by BPD symptoms, in order to inform treatments that specifically target aspects of parenting. As a personality disorder, BPD by definition is pervasive and impacts functioning in multiple domains including interpersonal relationships, occupational, and general functioning (American Psychiatric Association, 2001).

Children of mothers with BPD are therefore more likely to live in an environment that is characterized by chronic stress and unpredictability (Macfie, 2009). Future research should therefore explore how maternal BPD impacts chronic child stress physiology. Hair cortisol concentrations measure the accumulation of cortisol secretion and is less influenced by acute daily stressors compared to salivary cortisol levels (Russell et al., 2012). The association between maternal BPD symptoms and elevated levels of evening cortisol in this sample, rather than blunted morning levels, provides some preliminary evidence that maternal BPD may be associated with elevated levels of chronic stress physiology.

In summary, this study was a novel examination into the association between maternal BPD symptoms, parenting behaviors, and mother-child diurnal cortisol levels using a community sample. Maternal BPD symptoms were associated with higher levels of maternal evening cortisol levels, which in turn predicted higher child evening cortisol levels. Positive parenting behaviors were not associated with maternal BPD symptoms, which may indicate a unique area of strength in mothers with elevated BPD symptoms compared to mothers with other types of psychopathology such as depression. Maternal evening cortisol levels were the strongest predictor of child evening cortisol levels, even in models that included parenting behaviors. This offers preliminary evidence that interventions targeting maternal BPD symptoms may improve maternal stress physiology and in turn prevent the intergenerational transmission of disrupted stress physiology.

CHAPTER III

STUDY 2: ASSOCIATIONS BETWEEN MATERNAL BPD STATUS, PARENTING, AND MOTHER-CHILD HAIR CORTISOL CONCENTRATIONS

Chronic stress in childhood is associated with disrupted child stress physiology that has implications for child mental and physical health outcomes across development (Lupien et al., 2009). In childhood, chronic stress has been conceptualized to include not only socioeconomic adversity and trauma, but also adversity that is unique to the experience of being a child such as living with a mother with mental health difficulties and exposure to negative parenting behaviors. Children of mothers with psychopathology exhibit similar disrupted stress physiology as their parents, such that disrupted stress physiology has been conceptualized as an important pathway for the intergenerational transmission of risk for negative mental health outcomes (Creaven et al., 2014). Parenting behaviors have also been highlighted as an important mechanism for the intergenerational transmission of psychopathology (Calkins et al., 2013), and exposure to negative or harsh parenting has also been associated with disrupted child stress physiology (e.g., Hastings et al., 2011). These stressors co-occur such that mothers with psychopathology often display higher levels of negative parenting behaviors. For example, mothers with depression display high levels of insensitive parenting (Trapolini et al., 2008) and mothers with borderline personality disorder (BPD) display high levels of negativity and intrusive control (Eyden et al., 2016). Disrupted stress physiology may be a particularly important pathway within the context of maternal BPD, as it has been conceptualized as a disorder characterized by vulnerability and maladaptive responses to stress (Zimmerman & Choi-Kain, 2009). In recent years, hair cortisol concentrations have emerged as an

indicator of chronic stress that has the advantage of quantifying an accumulation of stress over months by measuring cortisol secretions over time (Gow et al., 2010; Russell et al., 2012). This study aims to examine positive and negative parenting behaviors in a clinical sample of mothers with elevated BPD symptoms, and test whether positive parenting behaviors mitigates the risk of maternal BPD status and stress physiology on disrupted child stress physiology, using hair cortisol concentrations as a measure of chronic stress. A more comprehensive review of associations between maternal BPD symptoms, parenting behaviors, and child stress physiology broadly can be found in chapters 1 and 2 of this dissertation. For this chapter, a briefer overview is provided that highlights research examining hair cortisol concentrations as a measure of chronic stress physiology specifically.

Elevated child hair cortisol concentrations have been associated with low socioeconomic status (Gray et al., 2018; Simmons et al., 2019), exposure to traumatic events (Simmons et al., 2016), and maternal trauma history (Slopen et al., 2018). While the effect of maternal psychopathology on child hair cortisol concentrations is unclear (for reviews see Gray et al., 2018, and Bryson et al., 2021), emerging evidence shows that child hair cortisol concentrations may mediate the association between parental symptoms of depression and anxiety and presence of major depression and attention-deficit hyperactivity in children (Ferro & Gonzalez, 2020). Child hair cortisol levels are consistently associated with maternal hair cortisol concentrations (Dauegaard et al., 2020; Hollenbach et al., 2019; Kao et al., 2018; Kao et al., 2019; Schloß et al., 2019). In turn, women with psychopathology have been shown to exhibit elevated hair cortisol concentrations within the context of depression and lower hair cortisol concentrations

within the context of anxiety (for review see Staufenbiel et al., 2013). Hair cortisol concentrations have not yet been examined within the context of BPD, but individuals with BPD are more likely to experience stressful life events (Jovev & Jackson, 2006; Pagano et al., 2004), be low income (Paris et al., 1987), and be unemployed (McGlashan, 1986), which in turn are associated with higher levels of hair cortisol concentrations in adults (Staufenbiel et al., 2013). Children of mothers with BPD are also more likely to be exposed to traumatic experiences (Feldman et al., 1995) and be in a home environment characterized by chronic stress (Macfie, 2009), which is also consistent with higher levels of hair cortisol concentrations (Gray et al., 2018; Simmons et al., 2019).

Parenting behaviors are also associated with child hair cortisol concentrations and may be an important area of intervention given that parenting quality is highly responsive to intervention even in clinical samples (Eshel et al., 2006). Higher levels of parental sensitivity (Kao et al., 2019), maternal nurturing (Simmons et al., 2019), and maternal emotion coaching (Simmons et al., 2019) have been associated with lower levels of preschool hair cortisol concentrations. Parenting quality has also been shown to moderate the association between mother-child hair cortisol concentrations, but the direction of this effect is unclear. Higher levels of positive parenting behaviors were associated with a stronger relationship between mother-preschooler hair cortisol concentrations (Schloß et al., 2019), while higher levels of negative parenting behaviors were associated with a stronger relationship between hair cortisol concentrations in mothers and their elementary-aged daughters (Ouellette et al., 2015). Given established associations between parenting behaviors and diurnal child cortisol patterns (e.g., Martin et al., 2014), as well as previous research showing that parenting interventions can improve child stress

physiology (e.g., Fisher et al., 2007), understanding the effect of parenting behaviors on child hair cortisol concentrations has important implications for both prevention and intervention of child chronic stress physiology.

Current Study

This study extends findings from Study 1 of this dissertation by examining associations between maternal BPD status, parenting behaviors, and mother-child stress physiology using a clinical sample of mothers with BPD and hair cortisol concentrations as a measure of chronic stress physiology. Maternal BPD status will first be examined in relation to observed positive and negative parenting behaviors. Consistent with Study 1 of this dissertation and a previous review on parenting behaviors in the context of maternal BPD symptoms (Eyden et al., 2016), we hypothesize that mothers with elevated BPD symptoms will display higher levels of negativity and intrusive control than mothers in the non-disordered control group, but that no significant differences in positivity and guidance will be found. The effect of maternal BPD status on mother-child hair cortisol concentrations will then be examined using a subset of dyads that provided hair samples. The home environment of a mother with BPD is characterized by chronic stress and unpredictability (Macfie, 2009), so it is hypothesized that maternal BPD status will be associated with higher levels of mother-child hair cortisol concentrations. A strengths-based framework will then be used to examine whether higher levels of positivity and guidance mitigates the positive association of maternal BPD status and cortisol levels on child cortisol levels.

Methods

Participants

Mother-preschooler dyads ($N = 166$) participated in a larger two-site, longitudinal randomized control trial examining the extent to which dialectical behavior therapy for mothers with BPD improves the development of emotion regulation in their preschool age children ($M = 42$ months, $SD = 4$ months). Detailed description of the larger randomized control trial can be found in the data protocol paper available by request (Zalewski et al., 2021). Data collected during the Time 1 assessment at both the University of Oregon and University of Pittsburgh Medical Center sites were included in the present analyses examining the associations between maternal BPD and parenting behaviors. A subset of the University of Oregon sample ($N = 84$) also provided hair cortisol samples. Briefly, dyads were recruited from various sources including a developmental database maintained by the university psychology department, mailings, social media, community mental health centers, Department of Human Services, and Head Start. Recruitment materials indicated that mothers with both high and low levels of mental health symptoms as well as custody of a 3-year-old child were needed. Families recruited via mailings received an additional \$5 incentive for completing and returning forms. Community providers also received information on study eligibility criteria and detailed instructions on how to refer families to the current study.

All mothers were required to be over the age of 18 years, have at least partial custody of a 3-year-old child, be fluent in English, and have a standard IQ score of 70+ in order to participate. Mothers completed an initial phone screen to confirm custody eligibility requirements and child developmental disability status, as children with known developmental disabilities were not eligible to participate. Maternal BPD symptoms were then assessed using the McLean Screener (Zanarini et al., 2003). Mothers who endorsed

at least 7 items on the McLean Screener were scheduled for the next stage of eligibility screening, where a trained clinician further assessed eligibility criteria as a mother with elevated BPD symptoms. Mothers with BPD were required to meet criteria for 3 or more BPD symptoms on the Structured Interview for DSM-IV Personality (SID-P; Pfohl et al., 1997), one of which must include either affective instability or uncontrollable anger. Mothers with BPD were also required to consent to randomization procedures pertaining to the larger randomized control trial.

Mothers who endorsed 0-2 symptoms on the McLean Screen over the phone were assessed for further eligibility as a non-disordered control. Mothers who denied experiencing emotional or psychiatric difficulties, or participating in alcohol or drug treatment since the child was born, were scheduled for the next stage of eligibility screening, where a trained clinician further assessed for eligibility as a non-disordered control. Mothers who had no symptoms of BPD (of which affective instability and uncontrollable anger could not even be subclinical) and no mental health diagnoses currently or since the conception of the participating child, were eligible to participate as a non-disordered control.

Demographic information by site is presented in Table 7. Dyads recruited from the University of Pittsburgh Medical Center were more likely to be Black or African American, have a relatively higher household income, and higher maternal educational achievement than dyads recruited from the University of Oregon, which are reflective of demographic differences by region. Overall, this sample can be described as low income and predominantly non-Hispanic White dyads.

Table 7*Study 2 - Sociodemographics by Site*

	UPMC		UO		Total	
	N	%	N	%	N	%
Child Gender						
Male	38	46.3	45	53.6	83	50
Female	44	53.7	39	46.4	83	50
Maternal Ethnicity						
Hispanic	1	1.2	5	6.0	6	3.6
Non-Hispanic	81	98.8	78	94	159	96.4
Maternal Race						
African American or Black	23	28	2	2.4	25	15.2
American Indian or Alaskan Native	0	0	1	1.2	1	0.6
European American or White	55	67.1	69	84.1	124	75.6
Multi-Racial	4	4.9	10	12.2	14	8.5
Annual Household Income (\$)						
≤ 22,310	14	17.1	30	35.7	44	26.5
22,311–30,044	11	13.4	14	16.7	25	15.1
30,045–37,777	2	2.4	11	13.1	13	7.8
37,778–45,510	5	6.1	10	11.9	15	9.0
45,511–53,243	4	4.9	6	7.1	10	6.0
53,244–60,976	4	4.9	5	6.0	9	5.4
60,977–68,709	5	6.1	2	2.4	7	4.2
68,710–76,442	3	3.7	1	1.2	4	2.4
≥ 76,443	34	41.5	5	6.0	39	23.5
Maternal Education						
Some high school	1	1.2	3	3.6	4	2.4
High school graduate/GED	9	11.0	8	9.6	17	10.3
Associate degree	6	7.3	0	0	6	3.6
Professional/Technical/Some college	20	24.4	37	44.6	57	34.5
University graduate	20	24.4	27	32.5	47	28.5
Some graduate school	2	2.4	1	1.2	3	1.8
Master's degree	18	22.0	7	8.4	25	15.2
Doctoral degree	6	7.3	0	0	6	3.6
Maternal Relationship Status						
Single	19	23.2	27	32.5	46	27.9
In a relationship	63	76.8	56	67.5	119	72.1

UPMC = University of Pittsburgh, Medical School; UO = University of Oregon

Procedure

Prior to data collection, study procedures were approved by the university's Institutional Review Board, as well as the study's Data Safety and Monitoring Board. Mother consent and child assent were obtained at each stage of the study. Mothers who passed the phone screen were given additional details of the study prior to scheduling an intake with a trained clinician.

Mothers completed a questionnaire to collect information regarding demographics and service utilization during the clinical intake. Clinicians then administered the SID-P to assess for BPD symptoms, as well as the Structured Clinical Interview for DSM-5 (SCID-5; First et al., 2015) to assess for other mental health disorders. The Peabody Picture Vocabulary Test Fourth Edition (PPVT-IV; Dunn & Dunn, 2007) form A was also administered to assess eligibility regarding maternal verbal ability. Consistent with the aims of the larger randomized control trial, eligible mothers with BPD were randomized to receive either Dialectical Behavior Therapy (DBT) Skills treatment or Family Services As Usual. Randomization procedures were not applicable to eligible mothers in the non-disordered control group.

Participating dyads then completed 4 assessments lasting approximately 3-4 hours each over the course of 1 year. Only data from the Time 1 assessment is presented here. Mothers also completed a battery of questionnaires about their mental health, parenting, and their child's mental health. During the assessment while mothers completed these questionnaires, children completed a number of tasks with a trained experimenter to assess child's emotion regulation, executive functioning, and theory of mind in an adjacent room. Dyads were then reunited to complete a series of interaction tasks. All

families were compensated \$40 for participating in the initial intake, as well as \$40 for the Time 1 assessment. Transportation costs for families, including taxis, bus tickets, and gas stipends (\$5), were provided as needed.

Data collection was disrupted in March 2020 due to the coronavirus (COVID-19/SARS-CoV-2) pandemic and remote data collection procedures were initiated. Only data collected prior to March 2020 were included in the present analyses, as it was no longer feasible to collect observed parenting behaviors or hair cortisol measures after this date. Specifically, remote procedures were not reset until Fall 2020 when hair cortisol samples were scheduled to be sent for assay. Additional funds to collect hair samples remotely (e.g., postage fees, staff time) were also not available as cortisol collection was not proposed in the original parent grant.

Measures

Maternal Borderline Personality Disorder Status

Trained clinicians delivered the Structured Interview for DSM-IV Personality (SID-P; Pfohl et al., 1997) in order to assess maternal BPD status. The temporal period was modified to reflect the 4-5 years “since the conception of your child” for the aims of this study. Symptoms were each rated on the following scale: 0 = absent, 1 = subthreshold, 2 = present, and 3 = strongly present. Each symptom was then categorized as either present (score of 2 or 3) or not present (score of 0 or 1). Independent coders assessed interrater reliability for approximately 20% of the sample, showing strong interrater reliability across both the University of Pittsburgh Medical Center (Krippendorff’s $\alpha = .91$) and the University of Oregon (Krippendorff’s $\alpha = .90$) sites. The distribution of maternal BPD symptoms were examined in order to determine whether it would be

appropriate for maternal BPD symptoms to be treated as a continuous variable. On average, the whole sample reported elevated levels of BPD symptoms with high levels of variability ($M = 3.60$, $SD = 3.60$). However, this was a bimodal distribution given that mothers in the non-disordered control group could not endorse any symptoms of BPD. The subset of mothers who endorsed BPD symptoms showed elevated levels of clinically assessed BPD symptoms on average ($M = 6.84$, $SD = 1.61$). Given this bimodal distribution and clinically meaningful differences in BPD symptoms, maternal BPD status was treated as a dichotomous variable for all analyses. Mothers who met criteria for presence of at least three BPD symptoms, including affective instability or uncontrollable anger, were categorized as BPD while mothers with no symptoms of BPD, including subclinical levels of affective instability and uncontrollable anger, were categorized as non-disordered control.

Parenting

Parenting behaviors were observed during a 5-minute mother-child interaction task (adapted from Kerig & Lindahl, 2000). During the task, mothers were asked to help the child build a Lego figure that was considered too complex for the child to complete independently. While assisting the child, mothers were asked to use only verbal commands and to not touch any of the pieces. Parenting behaviors were then coded using the Parent-Child Interactions Observational Coding Manual which was adapted from previously established coding systems (Cowan & Cowan, 1992; Lindahl & Malik, 2000; Rubin & Cheah, 2000). Behaviors were coded in 1-minute epochs and then averaged across the five epochs. To assess interrater reliability, twenty percent of the files were double coded. The following four behaviors were coded during each epoch on a scale of

0 (no evidence of the behavior) to 5 (extremely prominent evidence of the behavior). Cronbach's alpha ranged from .71 to .89 across all domains, indicating acceptable to excellent interrater agreement.

Maternal positivity. Positivity was operationalized as the intensity and frequency of verbal and non-verbal displays of positivity, comfort, and happiness. This included displays of warmth and affection, as well praise and compliments. Low levels of positivity include flat or neutral affect, as well as disinterested or withdrawn demeanor. In this sample, mothers displayed high levels of positivity ($M= 3.27, SD= 0.95$).

Maternal guidance. Maternal displays of guidance/structure were coded as the frequency and quality of maternal instructions during the task. This included developmentally appropriate assistance that enhance the child's ability to engage or successfully complete the task. Displays of guidance should help facilitate child functioning and serve to enhance the activity. Mothers in this sample showed extremely high levels of guidance ($M= 4.63, SD= 0.59$).

Maternal negativity. Maternal negativity was coded as the frequency and intensity of negative vocalizations, para-vocal (tone) cues, and non-verbal expressions of frustration or annoyance, with an emphasis on negativity directed towards the child. This included behaviors that are dismissive, rejecting, or invalidating of child's feelings. In this sample, mothers displayed low levels of negativity ($M= 0.29, SD= 0.62$).

Maternal intrusive control. Intrusive control was coded as the frequency and intensity of maternal control of child behavior which inhibited child-directed behavior or task completion. This included verbal and physical intrusiveness where mother dominated the conversation or activity, as well as maternal directives that limit child

autonomy. Mothers in this sample displayed low levels of intrusive control ($M= 0.34$, $SD= 0.79$).

In this sample, maternal positivity (skew= -0.910) and guidance (-2.235) were negatively skewed, while maternal negativity (2.859) and intrusive control (2.878) were positively skewed. Log and square root transformations were both tested as potential strategies to reduce skew. In this case, square root transformations reduced skew to a greater extent than log transformations, but guidance (skew= -1.750), negativity (1.571), and intrusive control (1.739) remained non-normally distributed. Consistent magnitude and pattern of results were found in all analyses when square root transformed parenting behaviors were used in place of untransformed parenting data. Results are therefore presented using untransformed parenting data for ease of interpretability.

Mother and Child Hair Cortisol Concentrations

During the assessment, mother and child hair samples (15-30 mg) were collected from the posterior vertex using the 3 cm closest to the scalp, measuring cortisol output from the preceding 3 months. Additionally, mothers completed a questionnaire for inclusion of potential covariates, such as hair maintenance behaviors, oral steroid use, and medications for both themselves and their child. Hair samples were then placed in aluminum foil pouches and stored at -20 degrees C until extraction.

Cortisol samples were assayed at Dr. Elizabeth Shirtcliff's lab at Iowa State University, one of the leading laboratories on hair cortisol measurement. Samples were weighed, washed twice in isopropanol to remove contaminants, dried, and grinded into a fine powder. Cortisol was extracted into methanol, which was evaporated, and the sample was then reconstituted in assay buffer. Samples were assayed along with standards and

quality controls using a commercial enzyme immunoassay kit provided by Salimetrics LLC. Final values were converted to pg cortisol per mg hair weight. Samples from the same participant were assayed on the same plate to minimize inter-assay within-subject variability, and all samples were assayed in duplicate. The overall intra-assay coefficient of variation (CV) was 5.1% and the inter-assay CV was 3.2%. All duplicates that varied with a CV greater than 7% were re-assayed to ensure reliability. Two samples (1.8%) showed elevated levels of cortisol ($> 3.149 \mu\text{g}/\text{dl}$) that were above sensitivity even after re-assay, so a cortisol value could not be calculated.

Cortisol data was then visually inspected for outliers. Four mother cortisol samples (94.65, 95.40, 102.75, 185.85 pg/mg) and three child cortisol samples (116.40, 505.20, 1615.20 pg/mg) were considerably higher than average and considered outliers. These outliers were clustered such that these seven samples, as well as the two samples that had elevated levels of cortisol that were above sensitivity for assay, came from only five families. Of these five families, three were categorized as non-disordered control group and two were categorized as BPD group. Given that these outliers were evenly distributed across BPD status, appeared to be clustered within families, and were re-assayed if above sensitivity, outliers were retained for analyses as they appeared to reflect true differences in the rank ordering of cortisol levels. In order to reduce the influence of these outliers and reduce the Type 1 error rate, these values were winsorized to three standard deviations above the mean consistent with previous methods (Brummelte et al., 2011; Dixson & Yuen 1974; Seltzer et al., 2010; Wainer, 1976). Retaining these winsorized values has been recommended as a more robust method of addressing outliers where there is confidence in the rank ordering of these values (Wilcox et al., 2014).

Consistent with previous research, children showed higher levels of hair cortisol concentrations ($M = 24.68$ pg/mg, $SD = 25.40$) than mothers ($M = 12.79$ pg/mg, $SD = 16.52$). As is common with cortisol data, values remained positively skewed even following winsorization. All cortisol values were therefore log transformed and mean centered for analyses.

Missing Data

Across the sample, 5% ($N= 9$) of dyads were missing parenting data because they missed their time 1 assessment. An additional 11% of dyads ($N= 18$) were missing parenting data due to technical malfunctions, and one dyad (1%) was missing parenting data due to child task refusal. For dyads at the University of Oregon, 75% of mothers ($N= 63$) and 58% of children ($N= 49$) had usable cortisol data. Children were more likely to be missing cortisol data than mothers due to insufficient hair length for collection that was exacerbated by gender differences. Boys were more likely to be missing hair cortisol samples than girls ($\chi^2(1, N= 89) = 6.292, p= .012$), given that boys more frequently had hair that was too short for collection. Families with cortisol samples were compared to those missing cortisol samples on maternal BPD status to determine whether BPD status was associated with missing cortisol samples. Maternal BPD status was not significantly related to the missingness of mother ($\chi^2(1, N= 89) = 1.939, p= .164$) or child ($\chi^2(1, N= 89) = 1.077, p= .299$) cortisol samples. Even though more samples were missing for boys, given that there were no gender differences in hair cortisol concentrations ($t(47)= 1.570, p= .123$), child gender was not included as a covariate. Due to low rates of ethnic and racial diversity at the University of Oregon site where hair cortisol samples were collected, missingness of cortisol samples by specific racial and ethnic group could not be

conducted. As an imperfect measure of racial and ethnic differences in hair cortisol collection, missingness of samples was tested between white and non-white families. There was no significant differences in missingness of cortisol samples between white and non-white mothers ($\chi^2(1, N= 89) = 3.360, p= .067$) and children ($\chi^2(1, N= 89) = 0.091, p= .763$).

Analytic Plan

All analyses were conducted in SPSS (ver. 26, IBM Chicago, IL, USA). Covariate testing was first conducted using bivariate correlational analyses or independent samples t-tests between the BPD and non-disordered control groups, as appropriate. Given that BPD symptoms have been associated with both elevated and blunted levels of cortisol, curvilinear versus linear associations between maternal BPD symptoms and hair cortisol concentrations were examined prior to hypothesis testing. Further details on covariate testing and curvilinear associations are described below.

Bivariate correlational analyses and descriptive statistics for all variables of interest are presented in Table 8. One-way analysis of variance tests (ANOVA) were first conducted to assess group differences in parenting behaviors between mothers in the BPD and control groups across both sites. Hierarchical linear regressions were then conducted with all covariates entered at level 1 using the sample of dyads from the University of Oregon that provided hair cortisol samples. First, the association between maternal BPD symptoms and maternal hair cortisol concentrations was examined. Second, an interaction term was computed as the multiplicative of maternal BPD status and maternal positivity (mean centered) as predictors of child hair cortisol concentrations. A parallel multiple linear regression model was then conducted with maternal guidance.

Finally, an interaction term was computed as the multiplicative of maternal hair cortisol concentrations (mean centered) and maternal positivity (mean centered) as predictors of child hair cortisol concentrations. A parallel multiple linear regression model was conducted with maternal guidance.

Table 8

Study 2 - Bivariate Pearson's Correlations Between Variables of Interest

		BPD			Negat-	Intrusive	Child
	Income	Symptoms	Positivity	Guidance	ivity	Control	Cort
Income	1						
BPD Symptoms	-.398**	1					
Positivity	.103	-.205*	1				
Guidance	.203*	-.171*	.331**	1			
Negativity	-.220**	.257**	-.327**	-.112	1		
Intrusive Control	-.161†	.267**	-.271**	-.035	.472**	1	
Child Cort	-.024	.090	-.004	-.246†	-.127	-.095	1
Maternal Cort	-.228†	.149	.028	-.277*	.085	.192	.540**

† $p < .1$, * $p < .05$, ** $p < .01$

Covariate Testing

Maternal BPD status was examined in association with household income level and maternal educational attainment using Pearson chi-square analyses. Maternal BPD status was significantly associated with lower household income ($\chi^2(8, N= 176) = 27.237$, $p= .001$) and lower maternal education ($\chi^2(7, N= 175) = 37.248$, $p < .000$). Specifically, the household income of mothers in the BPD group was more likely to be less than \$22,310 a year while mothers in the non-disordered control group were more likely to

have a household income of more than \$76,442 a year. In terms of educational attainment, mothers in the BPD group were more likely to have some high school or a high school diploma/GED while mothers in the non-disordered control group were more likely to have a master's or doctoral degree. Given that maternal educational attainment is a more distal measure of socioeconomic status compared to income, income was included as a covariate in all analyses. Income as a continuous variable was also significantly associated with lower levels of negativity and higher levels of maternal guidance (see Table 8).

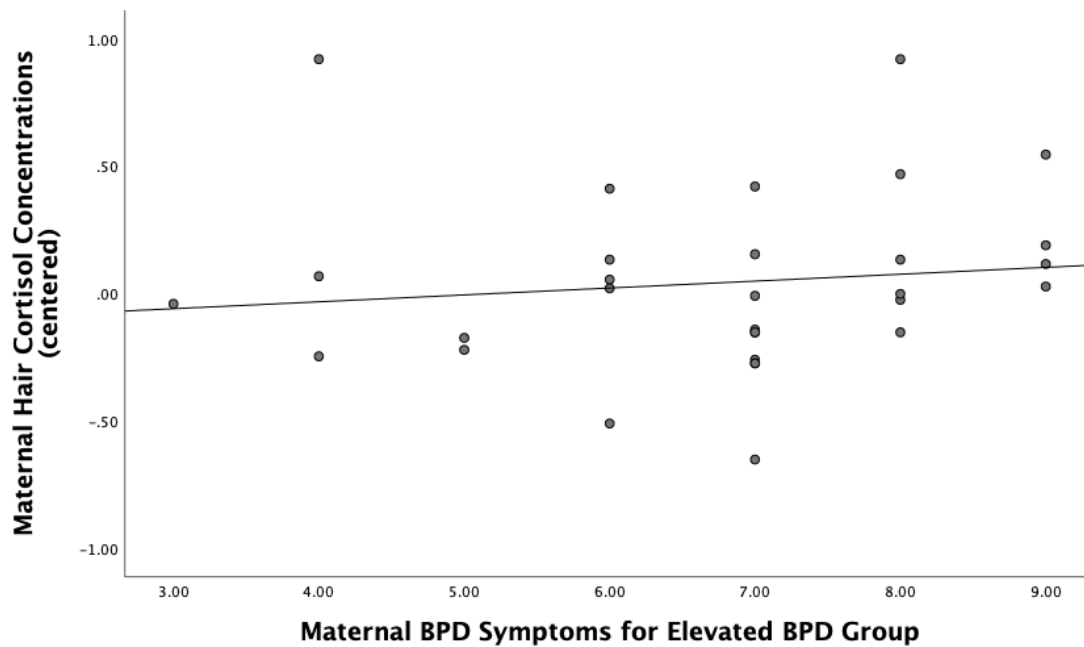
The association between hair cortisol concentrations and potential covariates were also examined. Household income was not associated with maternal or child hair cortisol concentrations (see Table 8). Child age ($r(49) = -.152, p = .296$) and gender ($t(47) = 1.570, p = .123$) were also not associated with child hair cortisol concentrations. For mothers and children, use of topical creams, steroids, medications, or hair treatments (e.g., hair coloring) were not associated with hair cortisol concentrations (p 's $> .05$). The weekly frequency in which hair was wet was associated with maternal ($r(50) = -.316, p = .025$) but not child ($p > .05$) hair cortisol concentrations. For mothers, hormonal contraceptive and tobacco use was not significantly associated with hair cortisol concentrations (p 's $> .05$). However, mothers who were breastfeeding had significantly lower levels of untransformed cortisol values ($M = 6.75$ pg/mg, $SD = 3.29$) than mothers who were not breastfeeding ($M = 16.51$ pg/mg, $SD = 20.92$), $t(48) = 2.713, p = .010$. Maternal frequency of hair getting wet and breastfeeding status were therefore included as covariates in all analyses involving maternal hair cortisol concentrations.

Curvilinear Associations Between Maternal BPD Symptoms and Maternal Cortisol

Curvilinear associations between maternal BPD symptoms and maternal hair cortisol concentrations were explored in two ways. First, the number of BPD symptoms endorsed by mothers in the BPD group were plotted against maternal mean-centered cortisol values (see Figure 3). A curvilinear association was not indicated, as there was an even distribution of mothers with higher and lower than average cortisol values across BPD symptoms. Maternal cortisol levels were then recoded into quartiles and examined in conjunction with maternal BPD status. Pearson chi-square analyses showed no significant difference between mothers in the BPD and control groups being in the highest or lowest quartiles of hair cortisol concentrations ($\chi^2(2, N= 63) = 4.627, p= .099$).

Figure 3

Study 2 - Maternal BPD Symptoms by Maternal Hair Cortisol Concentrations



Note. Associations between maternal borderline personality disorder (BPD) symptoms and mean-centered maternal hair cortisol concentrations (log transformed) in the group of mothers with elevated BPD symptoms. No evidence of a curvilinear association is indicated.

Results

Maternal BPD Status Predicting Parenting Behaviors

Group differences in parenting behaviors across the BPD and non-disordered control groups were tested using the full sample of participants from both the University of Pittsburgh Medical Center (UPMC) and University of Oregon (UO) sites. When controlling for household income and site, mothers in the BPD group showed significantly higher levels of negativity compared to mothers in the control group (see Table 9). There was also some evidence that mothers in the BPD group displayed higher levels of intrusive control and lower levels of guidance, but this difference was not statistically significant (see Table 9). These findings were consistent with bivariate correlational analyses that showed that number of BPD symptoms endorsed was negatively associated with maternal positivity and guidance, and positively associated with maternal negativity and intrusive control (see Table 8).

Table 9

Study 2 - Group Differences in Parenting Behaviors by Site

Measure	Site	Control Group		BPD Group		$F(4, 137)$	p	η_p^2
		M	SD	M	SD			
Positivity	UPMC	3.156	.177	2.948	.150	2.157	.144	.015
	UO	3.668	.158	3.368	.171			
Guidance	UPMC	4.751	.111	4.636	.094	2.880	.092 [†]	.021
	UO	4.704	.100	4.448	.108			
Negativity	UPMC	.310	.108	.650	.091	3.969 [*]	.048 [*]	.028
	UO	.030	.096	.111	.104			
Intrusive	UPMC	.365	.145	.667	.122	2.993	.086 [†]	.021
Control	UO	.055	.129	.243	.140			

Note. F statistics presented for main effect of BPD status; [†] $p < .1$, ^{*} $p < .05$

There were also significant site differences such that mothers at UPMC were rated lower in positivity ($F(4, 137) = 15.200, p < .001, \eta_p^2 = .100$), and higher in negativity ($F(4, 137) = 15.200, p < .001, \eta_p^2 = .100$) and intrusive control ($F(4, 137) = 15.200, p < .001, \eta_p^2 = .100$) than mothers at UO (see Table 9). These site differences may be driven by racial and ethnic differences between the two sites, as the majority of mothers who identified as Black or African American (92%) participated at the UPMC site (see Table 7). At the UPMC site, Black or African American mothers who participated were more likely to be in the BPD group ($N = 17, 74%$) compared to the non-disordered control group ($N = 6, 26%$), which is consistent with epidemiological research showing higher prevalence rates of BPD in Black communities (Tomko et al., 2014) but further confounds the association between race and BPD status at the UPMC site. These site differences need to be further examined in order to elucidate whether the variation in parenting behaviors is due to distinct features of the site, racial bias by coders and/or the coding scheme, differences in BPD presentation or comorbidities, or other factors.

Maternal BPD Status Predicting Mother and Child Hair Cortisol Concentrations

The effect of maternal BPD status on mother and child hair cortisol concentrations was tested using a subset of families from the University of Oregon who provided hair cortisol samples. When controlling for household income, maternal frequency of hair getting wet, and breastfeeding status, maternal BPD status was not significantly associated with maternal hair cortisol concentrations, $\beta = -.111, t(45) = -.698, p = .489$.

In the series of parallel linear regressions examining the effects of maternal BPD status, parenting behaviors, and the interaction between maternal BPD status and

parenting behaviors on child hair cortisol concentrations, no significant effects were found (see Table 10). There were no main effects of maternal BPD status, positivity, or guidance on child hair cortisol concentrations, and no significant interactions.

Table 10

Study 2 - Maternal BPD Status and Parenting Predicting Child Cortisol Levels

Model Predictors	R^2	(df)	F	p	β	t	p
Positivity	.014	(4, 44)	0.156	.959			
Intercept						-.510	.613
Income					.042	.233	.817
Maternal BPD Status					.140	.771	.445
Positivity					.056	.291	.828
BPD x Positivity					-.052	-.209	.836
Guidance	.113	(4, 44)	1.399	.250			
Intercept						-.628	.534
Income					.048	.284	.778
Maternal BPD Status					.141	.813	.421
Guidance					.116	.417	.679
BPD x Guidance					-.406	-1.509	.138

† $p < .1$, * $p < .05$, ** $p < .01$

Maternal Cortisol Levels and Parenting Behaviors Predicting Child Cortisol Levels

In the series of parallel linear regressions, higher levels of maternal hair cortisol concentrations were found to significantly predict higher levels of child hair concentrations in models including maternal positivity ($\beta = 0.647$, $t(41) = 4.905$, $p < .001$) as well as maternal guidance ($\beta = 0.527$, $t(41) = 3.860$, $p < .001$). There were no main

effects of maternal positivity or guidance on child hair cortisol concentrations, and no significant interaction between maternal hair cortisol concentrations and guidance to predict child cortisol concentrations (see Table 11). There was a significant interaction between maternal hair cortisol concentrations and positivity to predict child hair cortisol concentrations, such that as predicted, the positive association between maternal and child hair cortisol concentrations is weakened for mothers who display higher levels of positivity (see Figure 4).

Table 11

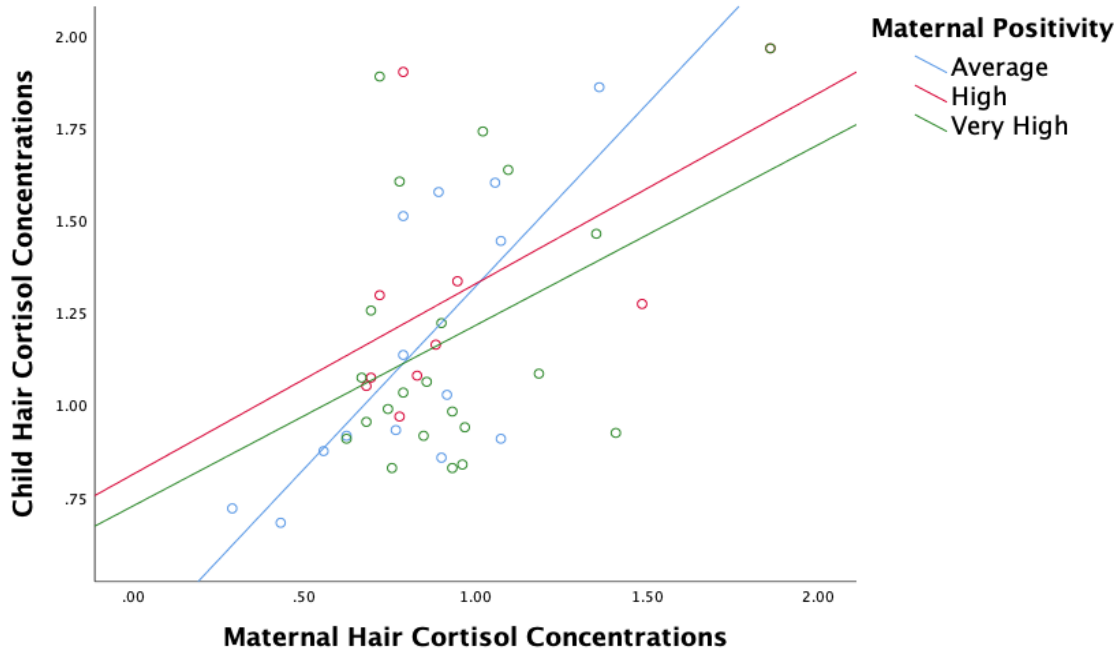
Study 2 - Maternal Cortisol Levels and Parenting Predicting Child Cortisol Levels

Model Predictors	R^2	(df)	F	p	β	t	p
Positivity	.374	(4, 41)	6.121	.001**			
Intercept						-.353	.726
Income					.109	.855	.397
Maternal Cortisol					.647	4.905	.001**
Positivity					-.181	-1.349	.185
Maternal Cortisol x Positivity					-.274	-2.018	.050*
Guidance	.324	(4, 41)	4.902	.003**			
Intercept						-.646	.522
Income					.126	.949	.348
Maternal Cortisol					.527	3.860	.001**
Guidance					-.166	-1.023	.312
Maternal Cortisol x Guidance					.036	.225	.823

† $p < .1$, * $p < .05$, ** $p < .01$

Figure 4

Study 2 – Interaction Between Maternal Hair Cortisol and Positivity on Child Hair Cortisol



Note. Effect of maternal hair cortisol concentrations on child hair cortisol concentrations, as a function of level of maternal positivity displayed during a mother-preschooler interaction task. As predicted, the association between mother-child hair cortisol concentrations is weaker for dyads whose mothers displayed higher levels of positivity.

Discussion

This study examined parenting behaviors and mother-child chronic stress physiology within the context of maternal BPD using a clinical sample. Maternal BPD symptoms were positively correlated with displays of negativity and intrusive control, and negatively correlated with displays of positivity and guidance. Group differences between mothers with elevated BPD symptoms and the non-disordered control group

were only found for displays of negativity, such that mothers with elevated BPD symptoms displayed higher levels of negativity than mothers in the control group. Maternal BPD status was not associated with maternal or child hair cortisol concentrations, and maternal positivity and guidance was not associated with child hair cortisol concentrations. The strongest predictor of child hair cortisol concentrations across all models were maternal hair cortisol concentrations. There was also a significant interaction between maternal cortisol and positivity, such that the association between mother-child hair cortisol concentrations was weaker for dyads whose mothers who displayed higher levels of positivity. Overall, these results suggest no main effect of maternal BPD symptoms on maternal-child hair cortisol concentrations but are consistent with prior research findings that maternal hair cortisol concentrations are a strong predictor of child hair cortisol concentrations.

When controlling for annual household income, there were no differences in displays of positive parenting behaviors between mothers with elevated BPD symptoms and the non-disordered control group. However, mothers in the BPD group displayed higher levels of negativity than mothers in the control group. It is worth noting that mothers in this sample were required to have custody of the participating child in order to be eligible to participate, and that mothers with elevated BPD symptoms are overrepresented in involvement with Child Protective Services (CPS; Perepletchikova et al., 2012). While this does not mean that all mothers with elevated BPD symptoms engage in child maltreatment practices, it is worth noting that custody eligibility requirements may have led to the exclusion of mothers with BPD symptoms who particularly struggle with effective parenting practices. While only a small percentage of

mothers with BPD engage in parenting behaviors that lead to the removal of the child from the home, it is important to recognize that these findings regarding parenting behaviors within the context of maternal BPD may not generalize to all mothers with elevated BPD symptoms.

Overall, however, these results on parenting behaviors within the context of maternal BPD are consistent with findings from a systematic review showing that maternal BPD symptoms were associated with higher levels of negative parenting behaviors but were not associated with lower levels of positive parenting behaviors (Eyden et al., 2016). This may indicate a unique strength of mothers with BPD compared to mothers with other symptoms of psychopathology, as mothers with depression (Pelaez et al., 2008) and anxiety (Murray et al., 2012) have been found to display lower levels of positive parenting behaviors than mothers without psychopathology. While mothers with BPD may display similar levels of positivity as mothers without psychopathology, mothers with BPD are also displaying higher levels of negativity. This pattern of parenting behaviors (high levels of positivity and negativity) may result in an unpredictable home environment for children where mothers are equally likely to respond with positivity or negativity in a situation. In this context, higher levels of positive parenting behaviors may not buffer against the risk of an unpredictable home environment, as the unpredictability of the home environment is theorized to influence child stress response across adverse experiences (Smith & Pollak, 2020). Future work in this area would benefit from a profile approach to examine patterns of parenting behaviors in mothers with elevated BPD symptoms to determine whether the same mothers who display high levels of positivity also display high levels of negativity.

The strongest predictor of child cortisol levels across all models was maternal hair cortisol concentrations. This is consistent with prior research showing strong associations between mother-child hair cortisol concentrations (Dauegaard et al., 2020; Hollenbach et al., 2019; Kao et al., 2018; Kao et al., 2019; Schloß et al., 2019). While there is evidence of a strong genetic component between maternal and child hair cortisol levels (Tucker-Drob et al., 2017), the association between mother-child hair cortisol concentrations can be impacted by environmental influences such as parenting (Ouellette et al., 2015; Schloß et al., 2019). Similarity in chronic stress profiles of mothers and their children have important implications for the intergenerational transmission of risk for negative mental health outcomes when maternal psychopathology is associated with specific chronic stress responses. In this study, no significant differences in hair cortisol concentrations were found between mothers with elevated BPD symptoms and mothers in the non-disordered control group. While mothers in the control group did not have significant mental health concerns, they still faced significant socioeconomic adversity and were likely to have a history of trauma given that women from Oregon experience higher rates of sexual trauma, intimate partner violence, and childhood victimization than the national average (Smith et al., 2017). It may be that in this context, the effect of maternal mental health symptoms on maternal hair cortisol concentrations was overshadowed by the high levels of adversity experienced across both groups, which had a stronger influence on maternal hair cortisol levels. While this study did not examine multiple indicators of socioeconomic adversity or trauma history, this theory is consistent with bivariate correlations showing that higher levels of maternal hair cortisol concentrations were associated with lower household income across both groups (see Table 1).

Further, while child hair cortisol concentrations have been a growing area of study, little research has been conducted on how child hair cortisol concentrations are associated with child mental health outcomes. There is some evidence that elevated hair cortisol concentrations in children of mothers with a history of early life maltreatment are associated with higher levels of behavior problems (Fuchs et al., 2018), but other studies have found no association between child hair cortisol concentrations and internalizing or externalizing behaviors (Kao et al., 2018; Ursache et al., 2017). Low hair cortisol concentrations during preschool have been associated with symptoms of attention-deficit/hyperactivity disorder (ADHD) in boys cross-sectionally (Pauli-Pott et al., 2017), as well as predictive of an ADHD diagnosis at age 8 for both boys and girls in the same sample (Pauli-Pott et al., 2019). This same study found no association between preschooler hair cortisol concentrations and symptoms of anxiety, depression, conduct disorder, or oppositional defiant disorder cross-sectionally or at age 8 (Pauli-Pott et al., 2019). Given that the association between child hair cortisol concentrations and child mental health outcomes is unclear, similarity in mother-child hair cortisol concentrations may not have the same implications for negative child mental health outcomes as diurnal or reactivity measures of cortisol. This month, a systematic review was published on the associations between social adversity on hair cortisol concentrations in young children up to 8 years old (Bryson et al., 2021). Across all domains of socioeconomic, psychosocial, and early life adversity, 70% of the findings showed no association with child hair cortisol concentrations (Bryson et al., 2021). Of the 13 studies that specifically examined maternal mental health and hair cortisol levels in young children, eight of these (62%) found no association, while three (23%) found negative associations and two (15%)

found positive associations between maternal mental health symptoms and child hair cortisol concentrations (Bryson et al., 2021). Overall, the impact of maternal mental health on child hair cortisol concentrations, as well as the implications of child hair cortisol levels on child mental health outcomes remains unclear.

There are several possible methodological and theoretical reasons that no associations between parenting behaviors and child hair cortisol concentrations were found in this sample. First, disruptions in data collection due to the COVID-19 pandemic, as well as insufficient hair length for collection, led to a smaller sample size than expected. Post-hoc power analyses showed that to detect medium effects, this study had 68% power in dyadic cortisol analyses ($n= 47$), 70% power in child hair cortisol analyses ($n= 49$), and 78-79% power in maternal hair cortisol analyses ($n= 61-63$). There was also limited variability in parenting behaviors displayed, as most mothers in the sample displayed high levels of positive parenting behaviors and low levels of negative parenting behaviors. This may have obscured associations between parenting behaviors and child hair cortisol concentrations due to ceiling and floor effects. Theoretically, prior research has found that child characteristics can moderate the associations between parenting behaviors and child hair cortisol concentrations. For example, preschooler emotional reactivity has been found to moderate the association between parental sensitivity and child hair cortisol concentrations, such that parental sensitivity more strongly predicted child hair cortisol concentrations in children who exhibited higher levels of emotional reactivity (Kao et al., 2019). Similarly, preschooler emotion regulation skills (Kao et al., 2019) and ADHD symptoms (Schloß et al., 2019) have been found to moderate the association between mother-child hair cortisol concentrations. Parenting is an inherently

transactional process such that as parenting behaviors influence child development, parenting behaviors in turn are influenced by child characteristics such as child temperament and emotion regulation skills. Future work in this area should continue to use high-powered samples to detect small effects of parenting behaviors on child stress physiology and examine how child characteristics can moderate associations between maternal mental health symptoms, parenting behaviors, and child stress physiology.

This study had several strengths, including being the first to examine chronic stress physiology within the context of maternal BPD using a clinical sample. This was also one of the largest sample sizes of mothers with elevated BPD symptoms to examine parenting behaviors within the context of maternal BPD. This study used a multimethod approach that allowed for rigorous measurement of clinician-assessed maternal mental health symptoms and observed parenting behaviors. Mothers in the control group were not eligible to participate if they met criteria for any symptom of BPD or any mental health disorder since the conception of the participating child. This led to a rigorous examination on the impact of maternal BPD symptoms on parenting behaviors and mother-child stress physiology. Borderline personality disorder is a highly stigmatized disorder, even among treatment and healthcare providers (Aviram et al., 2006; Sansone & Sansone, 2013). A systematic review found that providers have more negative views towards individuals with BPD, including feeling more apathetic, less caring, and expressing less empathy towards these patients (Sansone & Sansone, 2013). This stigmatization can lead to providers minimizing symptoms and undervaluing strengths of individuals with BPD (Aviram et al., 2006). Strength-based approaches such as the one used in this study can provide a powerful tool in order to combat stigma and ensure that

research frameworks are not reinforcing negative stereotypes for mothers with BPD. As the field continues to develop tools of prevention and intervention to reduce suffering and promote healthy child development in families of mothers with BPD, it is important to continue leveraging existing areas of strengths that these mothers display.

Overall, this study found that mothers with elevated BPD symptoms display similar levels of positive parenting behaviors and higher levels of negativity than mothers with no mental health concerns. Maternal BPD was not associated with disrupted chronic stress physiology in mothers or their preschoolers, which may in part be due to the significant socioeconomic adversity these families faced that overshadowed the influence of maternal mental health symptoms. Emerging evidence in child chronic stress physiology highlight that hair cortisol concentrations may not be a sensitive measure of psychosocial and socioeconomic adversity in young children (Bryson et al., 2021). Future work should continue to identify areas of prevention and intervention in families of mothers with elevated BPD symptoms by leveraging maternal strengths that may help disrupt the intergenerational transmission of negative mental health outcomes.

CHAPTER IV

GENERAL DISCUSSION

The aims of this dissertation were to 1) identify associations between maternal BPD symptoms and observed parenting behaviors and 2) examine whether positive parenting behaviors mitigated the risk of disrupted child stress physiology within the context of maternal BPD. Using both a community and clinical sample of mothers with BPD symptoms, we found that across studies maternal BPD symptoms were not associated with displays of positive parenting behaviors. Associations between maternal BPD symptoms and negative parenting behaviors were examined using the clinical sample, where mothers with elevated BPD symptoms were found to display higher levels of negativity than mothers in the non-disordered control group. Maternal BPD symptoms were found to be positively associated with maternal salivary evening cortisol levels but were not associated with maternal hair cortisol levels. While there was no direct effect of maternal BPD symptoms on child stress physiology across both samples, there was a significant indirect effect of maternal BPD symptoms on higher child evening salivary cortisol levels through its influence on higher maternal evening salivary cortisol levels. There was a similar magnitude of association for mother-child salivary evening cortisol levels ($r = .599$) and hair cortisol concentrations ($r = .540$), while no significant association between mother-child salivary morning cortisol levels was found ($r = .175$). In both studies, there were no direct effects of maternal displays of positivity or guidance on child stress physiology. However, a significant interaction was found between maternal displays of positivity and maternal hair cortisol predicting child hair cortisol concentrations, such that as predicted, the association between mother-child hair cortisol

concentrations was weakened for dyads whose mothers displayed higher levels of positivity. Across both studies, the strongest predictor of child stress physiology was maternal stress physiology.

Salivary diurnal cortisol measures circulating cortisol levels that are heavily influenced by daily fluctuations in acute stress (Dickerson & Kemeny, 2004). In contrast, hair cortisol concentrations measure the cumulative level of free, unbound cortisol that is able to quantify chronic stress over the previous months (Dettenborn et al., 2012; Kirschbaum et al., 2009; Russell et al., 2012). Because hair cortisol concentrations measure an accumulation of cortisol secretion, it is most closely associated with total measures of salivary cortisol rather than acute time points. For example, in adults, cortisol concentrations in 1cm segments (indicative of approximately one month of cortisol secretion) was found to be strongly associated with 30-day averages of area-under-the-curve (AUC) diurnal salivary cortisol but not associated with salivary cortisol awakening response or diurnal slope (Short et al., 2016). In preschoolers, hair cortisol concentrations have been strongly associated with AUC measures of salivary cortisol following a stressor task (Kao et al., 2018; Ouellette et al., 2015). Hair cortisol concentrations in elementary school children have also been associated with salivary diurnal AUC measures but were not associated with diurnal slope or cortisol levels at awakening (Vanaelst et al., 2012b). Consistent with the findings from this dissertation, strong associations have been found for both mother-child salivary cortisol levels (e.g., LeMoult et al., 2015; Stenius et al., 2008) and hair cortisol concentrations (e.g., Kao et al., 2019; Schloß et al., 2019). Twin studies show a strong genetic component for morning salivary cortisol levels (60%, Gustafsson et al., 2011) and hair cortisol

concentrations (72%, Rietschel et al., 2017), and a weaker genetic component for evening salivary cortisol levels (8%, Gustafsson et al., 2011). However, both salivary (Gustafsson et al., 2011) and hair cortisol concentrations (e.g., Tucker-Drob et al., 2017) can be influenced by environmental factors even when accounting for genetic contribution.

In addition to the aims of the research study, there are several advantages and disadvantages to using diurnal salivary and hair cortisol collections that may influence a researcher's decision to use one over the other (see Table 12). The following is a brief review that includes our experiences collecting data for this dissertation. For a more comprehensive comparison of cortisol collection methods see Vanaelst and colleagues (2012a). Salivary and hair cortisol collections are both non-invasive and have the advantage of being collected by either research staff in the lab or by parents at home. However, there are several downsides to using diurnal salivary cortisol measures. First, while it is a sensitive measure to daily stressors, this means that it is also easily influenced by timing of the collection and daily events such as exercise, food consumption, medication use, and other daily activities. Meticulous and accurate tracking of these influences, as well as rigorous compliance protocols, are needed so that covariates can be carefully assessed and controlled for as needed. It also requires significant coordination by lab staff to ensure families are adhering to compliance protocols and follow-through with mailing samples back to the research lab. In order to get a stable measure of diurnal patterns, at least two samples per day (30 minutes after waking and evening) need to be collected for at least three days. More detailed estimates of cortisol awakening response and diurnal decline throughout the day require 3-5 samples per day, and stable measures of diurnal slope require up to 10 days of cortisol

collection (Segerstrom et al., 2014). While each sample is therefore inexpensive to assay (~\$12 per sample), costs can quickly add up as at least six samples are required per individual.

Table 12

Pros and Cons of Salivary and Hair Cortisol Collections

	Pros	Cons
Saliva	<ul style="list-style-type: none"> ○ Non-invasive collection ○ Can be collected at home or in lab ○ Inexpensive assay costs per sample 	<ul style="list-style-type: none"> ○ Easily influenced by daily events ○ Rigorous compliance protocols needed ○ High levels of administrative oversight required ○ Minimum of 2 samples per day for a minimum of 3 days needed per person ○ Overall costs become high
Hair	<ul style="list-style-type: none"> ○ Non-invasive collection ○ Can be collected at home or in lab ○ Low administrative oversight required ○ Only one sample needed ○ Overall cost low 	<ul style="list-style-type: none"> ○ Similar compliance rates due to insufficient hair length ○ May not be sensitive to influences of socioeconomic and psychosocial adversity

In contrast, hair cortisol collections require only a single sample per individual so overall cost is drastically reduced even though hair samples typically have higher assay fees (~\$35 per sample). Because hair cortisol is not as susceptible to daily covariates that

influence salivary cortisol levels (Dettenborn et al., 2012), compliance protocols are less extensive. Hair cortisol can also be easily collected in the lab at a single time point which requires fewer administration resources to ensure samples are correctly collected by participants at home and returned. However, despite more simplified and less time-intensive protocols, in this dissertation hair cortisol collections showed lower compliance rates for children (58%) than salivary cortisol (76%) due to insufficient hair length. Hair that was too short for collection was particularly prevalent among boys in this sample, who often had buzzcuts. While there are some strategies for collecting hair samples that are shorter in length, this requires the assay lab to statistically account for differences in length and approximate cortisol concentrations for 3cm of hair using only a 1cm piece (Staufenbiel et al., 2013). This strategy may not be ideal as it could result in less accurate cortisol measurement, but it may be preferable to high levels of missing hair cortisol data, especially for young boys. Finally, while hair cortisol concentrations are a promising measure of stable cortisol concentrations over time, it may not be sensitive enough to influences of socioeconomic and psychosocial adversity, particularly in young children whose stress physiological systems are continuing to develop (Bryson et al., 2021). The most recent review of hair cortisol concentrations in young children recommends using a multidimensional assessment that measures both chronic stress using hair cortisol concentrations and acute stress physiology using salivary cortisol (e.g., Tarullo et al., 2020) in order to more clearly assess the influence of socioeconomic and psychosocial adversity on child cortisol functioning (Bryson et al., 2021).

The generalizability of the findings from this dissertation are limited by the narrow range of racial and ethnic diversity present within the samples, particularly for the

samples who participated in cortisol collection. While children in the samples reflected a wider range of racial and ethnic diversity than their mothers, this is particularly a limitation for findings around observed parenting measures given that there is evidence of racial and ethnic differences in parenting behaviors (see Kotchick & Forehand, 2002 for overview) and that these parenting behaviors are differentially associated with optimal child outcomes within distinct cultural contexts. For example, authoritarian parenting styles high in parental control have been associated with higher academic competence and lower levels of school misconduct and substance use in African American adolescents, but lower levels of self-reliance and self-esteem in European American adolescents (Lamborn et al., 1996). These differences in parenting behaviors can also be adaptive to the sociocultural context of living in the United States of America. For example, African American parents are more likely to discourage child displays of negative emotions which may be an adaptive strategy for avoiding racial bias (Dunbar et al., 2017) and associated with positive child outcomes (Smith & Walden, 2001) due to systemic racism in America. While there is evidence that specific parenting behaviors are associated with positive child outcomes across sociocultural contexts (e.g., Amato & Fowler, 2002; Bradley & Corwyn, 2000; McLoyd & Smith, 2002; Rowe et al., 1994), it would be harmful to neglect the unique strengths of parents within specific racial, ethnic, and cultural groups (e.g., Brody & Flor, 1998).

When measuring parenting behaviors in samples of mothers with elevated BPD symptoms, there is considerable variation in the parenting constructs that are examined as well as the operationalization of these terms (for review see Eyden et al., 2016). In part, this reflects an obstacle in the field of parenting research more broadly where labels such

as “positivity”, “warmth”, and “responsiveness” each measure overlapping constructs with little distinction between terms. Mothers with elevated BPD symptoms often struggle with affective instability, impulsivity, and anger which is likely to impact their parenting behaviors (Stepp et al., 2012). Behaviorally, this may be related to an oscillation in parenting behaviors but the exact pattern and timing of this oscillation in parenting behaviors is unclear and difficult to capture (Zalewski & Lengua, 2012). In this dissertation there were also significant ceiling and floor effects, as most mothers in the sample displayed high levels of positive parenting behaviors and low levels of negative behaviors, irrespective of BPD symptoms. Levels of maternal guidance were particularly skewed, which may have been accentuated by task demand, as mothers were instructed to help their child build the Lego figure. Future research in this area should continue to consider how maternal BPD symptoms correspond to specific patterns of parenting and operationalize parenting behaviors accordingly. For example, oscillation in parenting may be better captured by examining changes in parenting behaviors across time or using a profile approach to test patterns of parenting behaviors. A profile approach would require a large sample size in order to examine patterns of parenting behaviors but would offer valuable insight into whether the same mothers with elevated BPD symptoms that display high levels of positive parenting behaviors also display high levels of negative parenting behaviors.

While this dissertation did not directly test models of early adversity on child stress physiology, the results of this dissertation can be contextualized within these broader theories. Across both studies, there was no evidence of curvilinear associations between maternal BPD symptoms and mother-child cortisol levels that would be

consistent with the biological sensitivity to context (BSC) or adaptive calibration model (ACM). However, there is a substantial heterogeneity within BPD symptom presentation as well as significant levels of comorbidity with other mental health disorders.

Curvilinear associations may therefore be present within a subset of mothers with elevated BPD symptoms (e.g., mothers who present with both BPD and PTSD) that are not visible within the larger sample. Similarly, curvilinear associations between maternal BPD symptoms and child cortisol levels may appear for children with certain characteristics, such as low levels of emotion regulation, that are obscured within the larger sample. Children of mothers with BPD are at high risk for experiencing maltreatment and neglect (Perepletchikova et al., 2012), as well as exposure to domestic violence (Whisman & Schonbrun, 2009). Families of mothers with BPD are also more likely to be low income and under-resourced (Ullrich et al., 2007). The high rates of childhood adversity in children of mothers with BPD make it difficult to distinguish between experiences of deprivation and threat that may influence child stress physiology. Prior research has shown high prevalence rates of elevated BPD symptoms in mothers involved with Child Protective Services (CPS; Perepletchikova et al., 2012). To more closely test the deprivation and threat model within the context of maternal BPD, future research could examine stress physiology between children of mothers with elevated BPD symptoms that become involved with CPS and those who have no history of CPS-involvement.

Given the pervasive nature of personality disorders, the effect of maternal BPD on child stress physiology may best be understood within the context of the Topological Models of adversity. Maternal BPD likely impacts all factors theorized to influence how

the child experiences a stressful event, including features of the event, the child's environment, social context, and individual child differences. Specifically, maternal BPD is associated with high levels of chronic stress and frequent experiences of stressful life events (features of the event; Pagano et al., 2004). The environment of children of mothers with BPD is also characterized by unpredictability and instability (child's environment; Macfie, 2009). Children of mothers with BPD are likely to come from single parent households or be exposed to domestic violence (social context; Whisman & Schonbrun, 2009). Children of mothers with BPD also often exhibit emotional difficulties and externalizing behaviors such as aggression (individual differences; Eyden et al., 2016). While this framework highlights the many pathways in which maternal BPD may negatively impact child stress physiology, it also identifies specific areas of intervention as each of these pathways can be targeted for prevention and intervention.

There are three specific future directions that will extend the findings from this dissertation.

- 1. Screening for parenting behaviors at enrollment.** Given the limited variability in parenting behaviors displayed by mothers in this sample, future research in this area should consider screening for self-reported parenting difficulties during enrollment to ensure a range of parenting behaviors are represented. Intervention trials testing the effectiveness of incorporating parent training into existing treatments for BPD could also use this screening when randomizing mothers into treatment groups. For example, there is growing interest in incorporating parent training into dialectical behavior therapy (DBT; the gold standard treatment for BPD) in order to better support

mothers with elevated BPD symptoms (Zalewski et al., 2020). Child development outcomes could then be compared for mothers with elevated BPD symptoms who receive DBT alone and for those who receive DBT with parent training. In order to test these effects, it will be important to ensure an adequate range of parenting behaviors are present in the sample or small effects may be obscured.

- 2. Mother-child stress physiology in response to treatment for BPD.** This dissertation examined baseline associations between maternal BPD symptoms and mother-child stress physiology. Future research should examine how mother-child stress physiology changes in response to treatment for maternal BPD symptoms in order to test psychotherapy intervention as a potential mechanism for improving stress physiology in these families. Specifically, in addition to baseline collections of hair cortisol, hair cortisol samples were also collected one year later for the dyads in Study 2 of this dissertation. At baseline, half of the mothers with elevated BPD symptoms were randomized to receive one year of DBT skills treatment while the other half received family services as usual. Although sample sizes will be small due to COVID-19 related disruptions to data collection, this will provide an exploratory examination into changes in mother-child hair cortisol concentrations in response to treatment targeting BPD symptoms. Importantly, even though there were no group differences in hair cortisol concentrations between dyads in the elevated BPD symptom group and those in the control group at baseline, differences in response to treatment may be found.

3. Parenting and stress physiology within the context of involvement with Child Protective Services. Mothers with elevated BPD symptoms are overrepresented in involvement with Child Protective Services (CPS), yet current services for parents involved with CPS do not target emotion dysregulation and interpersonal difficulties that are associated with core features of BPD (Chaffin et al., 2001; Perepletchikova et al., 2012). Mothers involved with CPS may particularly struggle with effective parenting practices and may display different patterns of parenting behaviors than those found in this dissertation. Stress physiology in children of mothers with elevated BPD symptoms is also likely to differ based on history of CPS-involvement, due to the higher levels of adversity that is associated with being involved with CPS. Identifying how elevated BPD symptoms and CPS-involvement interact to predict parenting behaviors and mother-child stress physiology is the first step in developing targeted interventions for these families.

Overall, this dissertation found evidence that maternal BPD symptoms are associated with disrupted child salivary cortisol levels through its influence on maternal salivary cortisol levels. However, maternal BPD symptoms were not associated with mother-child hair cortisol concentrations which may be due to a lack of sensitivity in hair cortisol concentrations to psychosocial adversity, especially for children. Positive parenting behaviors were not associated with child stress physiology, which may be due to low power to detect effects as well as limited variability in parenting behaviors. Mothers with elevated BPD symptoms displayed similar levels of positive parenting behaviors as mothers without BPD symptoms but displayed higher levels of negativity.

Profile approaches, using a larger sample size, examining patterns of parenting behaviors may better be able to capture how displays of positive and negative parenting behaviors co-occur. The strongest predictor of child stress physiology was maternal stress physiology. Improving maternal stress physiology by targeting maternal BPD symptoms may serve as a preventative tool for reducing the intergenerational transmission of disrupted stress physiology, especially as BPD is a disorder characterized by increased vulnerability and maladaptive responses to stress (Zimmerman & Choi-Kain, 2009). Within the context of maternal BPD, future research should explore the benefits of screening for parenting behaviors at enrollment, changes in mother-child stress physiology in response to intervention, and parenting and stress physiology within the context of involvement with Child Protective Services. Ultimately, children of mothers with BPD are at greater risk for developing mental health difficulties, and the role of parenting and dyadic stress physiology should continue to be explored as targets for intervention and prevention to disrupt the intergenerational transmission of negative mental health outcomes in these families.

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