MATERNAL BORDERLINE PERSONALITY DISORDER AND CHILD DEVELOPMENT: AN EXAMINATION OF RISK TRANSMISSION AND STATISTICAL APPROACHES TO INFERENCE

by

GRACE ELIZABETH BINION

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Student: Grace Elizabeth Binion

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This dissertation has been accepted and approved in partial fulfillment of the requirements for the Doctor of Philosophy degree in the Psychology Department by:

Maureen Zalewski	Chairperson
Sanjay Srivastava	Core Member
Lou Moses	Core Member
Elizabeth Skowron	Institutional Representative

and

Kate Mondloch	Interim Vice Provost and Dean of the Graduate School

Original approval signatures are on file with the University of Oregon Graduate School.

Degree awarded June 2021

DISSERTATION ABSTRACT

Grace Elizabeth Binion

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Borderline Personality Disorder (BPD) is a serious mental illness and children whose mothers have BPD are at elevated risk for poor psychosocial outcomes across their lifespans. A growing body of research endeavors to elucidate mechanisms by which this risk is conferred. Because BPD is associated with many other risk contexts, research in this area must contend with multiple confounding variables. The research contained in this dissertation advances knowledge in this field through two empirical contributions. First, a systematic review of covariate adjustment in statistical analyses examining maternal BPD was conducted. Results suggest substantial heterogeneity in covariate practices, including which variables are treated as covariates and how many covariates are included in statistical models. Recommendations for best practices, including increased reliance on substantive theory for covariate selection and use of graphical causal models, are discussed. Second, the cross-sectional correspondence between maternal BPD, children's Executive Function (EF) and Theory of Mind (ToM), and children's psychosocial outcomes were examined. Results revealed significant associations between maternal BPD, children's EF, and children's social competence and symptoms of psychopathology. These findings indicate children's disrupted development

of EF may be a mechanism by which risk for poor psychosocial outcomes is conferred. Together, these works contribute to the field of maternal BPD by examining potential risk-conferring mechanisms and elucidating methodological and analytic approaches which might improve inferences in this area. To further advance this field, future research should employ longitudinal designs to examine the co-development of transdiagnostic risk processes, consider experimental designs (such as treatment trials) to rigorously test mechanistic models, and ground methodological and analytic choices in well-articulated causal models whenever possible.

CURRICULUM VITAE

NAME OF AUTHOR: Grace Elizabeth Binion

GRADUATE AND UNDERGRADUATE SCHOOLS ATTENDED:

University of Oregon, Eugene
Georgia Gwinnett College, Lawrenceville, GA

DEGREES AWARDED:

Doctor of Philosophy, Clinical Psychology, 2021, University of Oregon
Master of Science, Psychology, 2015, University of Oregon
Bachelor of Science, Psychology, 2014, Georgia Gwinnett College

AREAS OF SPECIAL INTEREST:

Developmental Psychopathology
Methodology
Replicability and Open Science

PROFESSIONAL EXPERIENCE:

Predoctoral Intern, Marcus Autism Center, Emory University, 2020-2021

Doctoral Trainee, Child Development and Rehabilitation Center Eugene, Oregon Health Sciences University, 2018-2019

Data Manager, R01-MH111758-01 (PI: Maureen Zalewski), University of Oregon, Eugene, 2017-2018

Doctoral Student Therapist, Early Intervention in Foster Care Program, Oregon Community Programs, 2017

Doctoral Student Therapist, Depressed Parents of Adolescents Intervention, ADAPT Lab, University of Oregon, Eugene, 2016-2017

Doctoral Student Therapist, Filming Interactions to Nurture Development (FIND) Program, SNAP Lab, University of Oregon, Eugene, 2016-2017

Doctoral Student Therapist, Psychology Clinic, University of Oregon, Eugene, 2015-2018

Graduate Teaching Fellow, Department of Psychology, University of Oregon, Eugene, 2014-2020

GRANTS, AWARDS, AND HONORS:

Future Fellows Research Conference, St. Jude Children's Research Hospital, 2020

Carolyn M. Stokes Memorial Scholarship, College of Arts and Sciences, University of Oregon, 2019

Beverly Fagot Memorial Dissertation Fellowship, Psychology Department, University of Oregon, 2019

Distinguished Teaching Award, Psychology Department, University of Oregon, 2018

Developmental, Child, & Family Psychology Scholarship, Inter-university Consortium for Social and Political Research Summer Program in Quantitative Methods in Social Science Research, 2018

Committee for an Inclusive Community Travel Award, Psychology Department, University of Oregon, 2018

Student Travel Award, Society for Research in Child Development, 2017

Psi Chi Annual Prize, Georgia Gwinnett College, 2014

Psi Chi Regional Travel Grant, Psi Chi International Honor Society, 2014

Outstanding Registered Student Organization Officer, President of Georgia Gwinnett College Chapter of Psi Chi, Georgia Gwinnett College, 2014

Outstanding Student Scholarship, School of Liberal Arts, Georgia Gwinnett College, 2014

Reproducibility Project Grant, Center for Open Science, 2013

PUBLICATIONS:

Musser, N., Zalewski, M., Lewis, J., Rogers, M., Binion, G., & O'Brien, J. (under review). *Bidirectional Associations of Child Psychopathology Symptoms with Maternal Emotion Dysregulation and Implications for Parenting*

Lewis, J., Binion, G., Rogers, M., & Zalewski, M. (2019). Disengaging from dysregulation: Maternal emotion dysregulation as a predictor of early childhood dissociation. *Journal of Trauma and Dissociation*

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CHAPTER I

GENERAL INTRODUCTION

Children whose parents have mental health disorders are at elevated risk for a range of poor psychosocial outcomes, including behavior problems (Breaux et al., 2014), poor academic performance (Shen et al., 2016), and social difficulties (Eyden et al., 2009). These difficulties may emerge early in life and difficulty in one domain of development may exacerbate risk in another, leading to a cascade of risk that increases the likelihood that these children will develop psychopathology (Forsman et al., 2016; Masten & Cicchetti, 2010). In fact, children whose parents have psychopathology are at significant risk for developing psychopathology at some point in their lives (Burstein et al., 2012; McAdams et al., 2015).

Transmission of risk cuts across diagnoses, meaning a child with a parent who has psychopathology is at risk for developing *any* type of mental disorder (McLaughlin et al., 2012). In part because of this, there is an increasing emphasis on examining the intergenerational transmission of features common across multiple forms of psychopathology as opposed to focusing on specific disorders (Mansell et al., 2008). Further, understanding the transmission of these *transdiagnostic features* of psychopathology may assist in creating prevention and early intervention efforts by focusing on impairments in cognitive, emotional, behavioral, and social processes.

Borderline Personality Disorder (BPD) is a serious mental illness characterized by affect instability, turbulent relationships, identity disturbances, impulsivity, and chronic suicidality (American Psychological Association, 2013). Parents who have BPD are more likely than non-disordered parents to experience low socioeconomic status (Walsh et al.,

2013), have lower educational attainment (Bagge et al., 2004; Trull et al., 1997), and to experience marital distress and dissolution (Whisman & Schonbrun, 2009). Unsurprisingly, children whose parents have BPD are at elevated risk for developing a wide range of mental disorders across their lifespans (Stepp et al., 2012).

Executive Function (EF) and Theory of Mind (ToM) are two cognitive processes that develop rapidly during the preschool years (Harris, 2006; Zelazo & Muller, 2002). EF and ToM are impaired in several forms of psychopathology (Brüne & Brüne-Cohrs, 2005; Calkins & Keane, 2009) and thus can be considered transdiagnostic features. Further, many of the deficits observed in BPD closely parallel key developmental processes which emerge during the preschool period (Macfie, 2009), such as EF and ToM. Thus, children whose parents have BPD may be disadvantaged in their acquisition of these skills, as parental EF and ToM deficits may impair a parent's ability to scaffold these skills in their offspring. Given this, it is possible that risk for psychopathology may be conveyed to offspring from mothers who have a diagnosis of BPD via disrupted development of preschoolers' EF and ToM.

The literature examining children's outcomes in the context of parental BPD has predominantly focused on maternal, as opposed to paternal, BPD. This is the product of two important factors. First, Widiger and Weissman (1991) found that BPD is most commonly diagnosed in women. Although it is unclear whether uneven rates of diagnosis between men and women results from a true difference in phenomenology or from uneven rates of detection, mental health providers and patients alike are more likely to recognize and identify BPD symptoms in women. Second, many individuals with BPD are likely to be single parents due in part to the nature of core BPD features (for example,

a chronic pattern of stormy relationships). Despite increasing rates of single fatherhood, single parents are currently disproportionately women (U.S. Census Bureau, 2016). For these reasons, scholars exploring child development in the context of parental BPD are far more likely to identify mothers who have a diagnosis of BPD than fathers.

Scholars working in the area of maternal BPD face a significant number of challenges identifying effects of interest. First, the lion share of the empirical work is interested in causal questions but must rely on observational data (Eyden et al., 2016). Second, causal questions related to BPD must contend with many confounding variables in the form of high comorbidity of mental health disorders (Grant et al., 2008) and many co-occurring risk contexts (Bagge et al., 2004; Trull et al., 1997; Moore et al., 2017; Walsh et al., 2013; Whisman & Shonbrun, 2009). Third, developmental psychopathology research must balance control for risk contexts which are not of interest with concerns related to multicollinearity (Foster, 2010). Together, these challenges make the selection of appropriate covariates in analyses testing the association of maternal BPD and child outcomes exceptionally difficult.

This dissertation is comprised of two manuscripts, each addressing the intergenerational transmission of risk features from mothers who have BPD to their children. The first manuscript is a methods-focused systematic review which examines covariate adjustment practices in research on child development in the context of maternal BPD. The second manuscript is substantive in focus and is derived from an on-going randomized controlled trial of mother-child dyads examining the transmission of emotion dysregulation from mothers who have a diagnosis of BPD to their preschool-aged children. Prior to introducing these manuscripts, I summarize:

- Definitions of EF and ToM with emphasis on their operationalization in my dissertation;
- The associations of EF and ToM deficits with psychopathology and poorer social competence;
- Inferential challenges in research addressing maternal BPD;
- The randomized controlled trial which provides data for the present substantive work.

Defining Executive Function

Executive Function (EF), sometimes referred to as executive control, is a collection of cognitive processes which, together, enable the regulation of "lower level" processes, such as attention and motor impulses (Snyder et al., 2015). EF abilities are essential for navigating situations which require the regulation of behavior or attention, as when one is asked to remember and follow rules, to comply with behavioral norms, or to refrain from socially inappropriate actions. Three core processes are thought to constitute EF: inhibition, cognitive flexibility, and working memory (Diamond, 2013).

Inhibition, sometimes referred to as inhibitory control, is the ability to exert control over attentional, emotional, or behavioral impulses, particularly in the context of strong environmental "lures" or internal predispositions (Diamond, 2013). Tasks which assess inhibition present participants with an appealing stimulus which must be resisted, or a stimulus designed to elicit a prepotent response which must then be overridden. For example, children's inhibition is often assessed using the Gift Delay task (Kochanska et al., 1996) in which children must resist peeking while a prize is loudly wrapped. Other tasks assessing children's inhibition include Bear/Dragon (Kochanska et al., 1996) in

which children are required to inhibit a prepotent motor response (by ignoring behavioral commands given by one puppet while heeding those given by another) and Day/Night (Gerstadt et al., 1994) in which children must put forth a non-dominant verbal response (by saying "day" when shown a picture of the moon and stars and "night" when shown a picture of the sun). In the present work, children's inhibition is assessed with the Bear/Dragon and Day/Night tasks.

Cognitive flexibility, sometimes referred to as set shifting or mental flexibility, is the ability to flexibly adapt to new rules or demands (Davidson et al., 2006). Tasks which assess cognitive flexibility ask participants to perform actions based on rules or demands which intermittently change and thus require participants to flexibly switch between rule sets. For example, the present work assesses children's cognitive flexibility using the Dimensional Change Card Sort Task (Zelazo, 2006), in which children are asked to sort cards into stacks based on various properties of the cards (e.g. color, shape). The properties to which children are asked to attend, however, change and become more complex and conditional as the task proceeds, thus increasing the flexibility required for successful task execution.

Finally, working memory is the ability to mentally represent and manipulate (e.g. relate to one another) units of information which are no longer perceptually present (Baddeley & Hitch, 1994). Working memory supports both of the other EF processes, as the ability to inhibit a response in service of a goal or to update behavior following a rule change is predicated on the ability to represent a goal, retain conditions necessary for goal acquisition, and/or recall that a rule has been changed. In the present body of work,

working memory is not directly assessed, although it is understood to be necessary for task performance on other EF assessments.

Defining Theory of Mind

Theory of Mind (ToM) is a broad term referring to the ability to recognize and represent the minds or internal states of others. ToM abilities entail an understanding that others may have knowledge, beliefs, intentions, and emotions separate from one's own (Imuta et al., 2016) and an awareness that mental states influence actions (Moses & Sabbagh, 2007). These abilities enable the understanding and anticipation of social behavior. This ability is central for successfully navigating interpersonal situations and relationships (Slaughter et al., 2015).

An array of terms is used to refer to various components of ToM, such as false belief understanding, affective perspective-taking, and mentalization. Although not interchangeable, these terms refer to processes which fall under the umbrella of ToM, as they are all related to the ability to represent an internal state (belief, emotion, intention) of an external actor. In addition to the range of states which may be represented, components of ToM vary in degree of sophistication. For instance, the ability to represent an actor's mistaken belief (false belief understanding) is more rudimentary than the ability to represent the emotional reactions of participants in a social faux pas (Wellman & Liu, 2004).

Given this range, ToM assessments vary in both component assessed and degree of difficulty. For example, in a classic assessment of early ToM abilities (contents false belief; Gopnik & Slaughter, 1991), young children are presented with a clearly labeled box (e.g. bandaids) which they are shown does not actually contain the item it appears to

contain (e.g. actually contains a stuffed animal). Children are then asked what someone who has never seen inside the box believes it contains, requiring them to represent the mistaken belief of a naïve other. Somewhat more challenging assessments (belief-desire reasoning and belief-location reasoning) require children to make inferences about an actor's behavior based on that actor's inaccurate beliefs. Assessments of more sophisticated forms of ToM also incorporate social and affective norms, such as the concept of hiding emotions to maintain social acceptance or avoid embarrassment (Wellman & Liu, 2004). To assess children's ToM, the present work utilizes a 7-item battery consisting of common ToM queries for children which was assembled to represent a range of components and complexity (Wellman & Liu, 2004).

Association of EF and ToM with Psychopathology and Social Competence

Together, EF and ToM abilities support regulated social and emotional behavior. EF abilities are essential for rule following, retaining and generalizing social norms, and inhibiting inappropriate responses (Riggs et al., 2006). ToM abilities are central to supporting sensitive, appropriate social responding and prosocial behavior (Couture et al., 2011; Imuta et al., 2016). The intersection of these abilities is particularly consequential for social functioning, as routine but cognitively complex social tasks (such as understanding and appropriately responding to the diverse desires of others) hinge on successfully using many elements of both EF and ToM. Deficits in these abilities, therefore, are implicated in impaired social competence (Holmes et al., 2016; Slaughter et al, 2015).

The inability to control behavior and attention and to understand the mental states of others also confers risk for a wide range of mental disorders. For example, core

symptoms of externalizing disorders such as ADHD and ODD reflect persistent deficits in the ability to control attention and to inhibit inappropriate behavioral impulses (Biederman et al., 2004; Hobson et al., 2011). Further, deficits in the ability to shift attention away from distressing stimuli, to think flexibly about environmental demands, and to accurately represent the mental states (e.g. intentions) of others are implicated in the mood disruption evinced in internalizing disorders such as Major Depressive Disorder (Inoue et al., 2006; Taylor-Tavares et al. 2007). Therefore, early disruptions in these processes, are understood as signals of emerging risk for psychopathology.

Inferential Challenges in Maternal BPD

Given these known associations, poorer EF and ToM may be mechanisms conferring risk for the development of psychopathology in children who have mothers who have a diagnosis of BPD. As mentioned previously, children whose mothers have a diagnosis of BPD are at significant risk for a range of poor life outcomes, including developing psychopathology themselves (Stepp et al., 2012). A growing literature has examined the outcomes and parenting experienced by children whose mothers meet diagnostic criteria for BPD, broadly seeking to answer the questions: 1) *what is the causal effect of maternal BPD on child outcomes?* and 2) *What mechanisms explain this effect?* Although research addressing these questions is often necessarily observational, causal questions remain of central interest due to their immense relevance to public health.

Causal inferences related to BPD are complicated by the nature of the disorder itself. Although comorbidity, or the co-occurrence of mental disorders, is a challenge for all research on specific mental disorders, BPD has an exceptionally high rate of

comorbidity. It is known to co-occur at high rates with internalizing disorders (mood and anxiety disorders), externalizing disorders (substance use disorders, conduct disorder), eating disorders, trauma-related disorders, and personality disorders (Eaton et al., 2011; Grant et al., 2008; Sharp et al., 2015; Zimmerman et al., 2005). In addition, mothers who have a diagnosis of BPD are more likely than non-disordered mothers to experience additional risk contexts known to compromise parenting and child development. Specifically, mothers who have a diagnosis of BPD are more likely to be of low socioeconomic status (Walsh et al., 2013), be involved with the criminal justice system (Moore et al., 2017), experience marital distress and dissolution (Whisman & Schonbrun, 2009), and have lower educational attainment (Bagge et al., 2004; Trull et al., 1997). Importantly, these comorbidities and co-occurring risk contexts may be related to the core symptomology of BPD. Core features of the BPD diagnosis, such as affect instability, impulsivity, and tumultuous relationships, likely increase the chances that mothers who have a diagnosis of BPD will meet criteria for multiple mental disorders and experience contextual risk. Thus, disentangling the causal effect of BPD on child development is complicated by causal pathways which may include mediating contexts of risk known to be deleterious to child development.

Overview of Data Source and Subsequent Chapters

The research presented across the following two chapters is designed to advance research on the intergenerational transmission of risk from mothers who have a diagnosis of BPD to their preschool children. Each chapter is meant to be a stand-alone manuscript and the terms “chapter” and “manuscript” are used interchangeably. Although distinct in

approach, the goal of each of these manuscripts is to elucidate risk processes implicated in the emergence of psychopathology in children whose mothers have BPD.

Chapter II addresses this goal through a systematic review of methodological and analytic practices related to causal inference in the literature examining child development and parenting in the context of maternal BPD. Chapter III addresses this goal through the collection of original data. These data are drawn from a larger longitudinal, two-site clinical trial (R01-MH111758-01) enrolling mothers and preschool children (aged 36-48 months at enrollment). This trial began enrolling participants in October of 2017 and data collection is ongoing. Two-thirds of the total sample is being recruited on the basis of mothers' elevated symptoms of BPD. The remaining third of the sample is being recruited as a non-disordered control group and is comprised of mother-preschooler dyads matched on income with high-risk dyads. Half of mothers who have elevated BPD symptoms will be assigned to receive a year-long course of Dialectical Behavior Therapy (DBT), a highly effective treatment for BPD (Linehan et al., 2006), while the other half will be assigned to receive Family Services as Usual. All mothers complete a clinical intake to assess their mental health symptoms and to determine eligibility for the study. Mothers and preschool children participate in four assessments (comprised of extensive behavioral and self-report batteries) over a one-year period (at 0-, 4-, 8-, and 12-months). Data for Chapter III are drawn from the clinical intake and baseline assessment. Relevant measures and methods are detailed in that chapter.

Subsequent chapters are presented as follows: Chapter II addresses the use of covariates to improve inferences in research examining child outcomes in the context of maternal BPD. Next, Chapter III leverages insights from Chapter II to address the

concurrent (single-timepoint) correspondence between maternal BPD, child EF and ToM abilities, and children's symptoms of psychopathology. Finally, Chapter IV provides a general discussion of these results, with a focus on limitations of the present studies and directions for future research.

CHAPTER II

INFERENCE ABOUT INTERGENERATIONAL TRANSMISSION: A SYSTEMATIC REVIEW OF COVARIATE ADJUSTMENT IN MATERNAL BORDERLINE PERSONALITY DISORDER

The literature addressing maternal Borderline Personality Disorder (BPD) has grown substantially in the last two decades (Eyden et al., 2016). Broadly, the goal of this research is to better understand child outcomes and mechanisms explaining the intergenerational transmission of risk for mental disorders from parents to children. BPD is a serious mental illness characterized by affect instability, turbulent relationships, identity disturbances, impulsivity, and chronic suicidality (American Psychological Association, 2013). Children whose parents have BPD have been found to be at elevated risk for developing a wide range of mental disorders across their lifespans (Stepp et al., 2012). A systematic review by Eyden and colleagues (2016) synthesized the research examining the parenting and child outcomes associated with maternal BPD. This review described that children whose mothers have a diagnosis or elevated symptoms of BPD consistently score lower on a wide range of social, emotional, and cognitive assessments and score higher on measures of psychopathology. Further, Eyden and colleagues found that mothers who have a diagnosis or elevated symptoms of BPD engaged in parenting practices characterized by greater hostility, insensitivity, and overprotection as compared to mothers who do not have a diagnosis of BPD or BPD symptoms. Thus, authors concluded that risk for poor child outcomes in the context of maternal BPD may be due in part to maladaptive parenting practices.

In addition to substantive conclusions, Eyden and colleagues provided a descriptive synthesis of the methods used in this research, including study design and measurement choices. This inventory revealed substantial heterogeneity across many methodological domains including treatment of BPD as categorical (present/absent) or continuous, use of comparison groups (no comparison, non-disordered comparison, differently disordered comparison), number of measurement occasions, and operationalization of parenting behaviors and child outcomes. The variability was considered so extensive that it precluded a meta-analysis of substantive findings, thereby limiting the scope of conclusions about the literature.

Given that the primary focus of Eyden and colleagues' review was on synthesizing parenting and child outcomes, an exhaustive synthesis of all methodological and statistical choices was beyond the scope of their work. One such choice which was not addressed and spans both methodological and statistical domains is covariate adjustment, or the practice of including variables which are not of primary interest in statistical models. The practice of covariate adjustment has significant implications for the substantive conclusions reviewed by Eyden and colleagues. In fact, covariates are often included in statistical analyses for the explicit purpose of strengthening inferences and better estimating effects of interest (Steiner et al., 2010). Thus, whether to include covariates in statistical analyses, which variables to treat as covariates, and how many covariates to include are all choices which are inextricable from substantive findings. Although covariate adjustment decisions are important across all areas of research, such choices are especially complex in research examining maternal BPD and child outcomes. This complexity is a result of 1) reliance on predominantly observational data and 2) the

co-occurrence of risk contexts which increase the likelihood of poor outcomes for both mothers and children. In such dual-generation designs, potential confounders related to mothers alone, children alone, and the dyad together must be considered, resulting in many potential covariates. Given the high-risk nature of child development in the context of maternal BPD, and the marked complexity of methodological decisions in this area, particularly as related to covariate adjustment, a critical evaluation of current covariate practices in the field is necessary. To this end, the current systematic review builds upon Eyden and colleagues' review through an exclusive and detailed focus on the practice of covariate adjustment within the field of maternal BPD. The goal of this work is to provide practical recommendations to improve the methodological and statistical practices which undergird this important research area.

Covariate Adjustment

Covariate adjustment merits particular consideration among methodological and statistical choices as the practice is 1) common and 2) consequential for the interpretation of substantive findings. Although covariates are broadly employed in many fields, they are particularly favored in observational (as opposed to experimental) social science research (Steiner et al., 2010) where they are included in statistical models in efforts to better isolate causal effects (Foster, 2010). It is impossible, and unethical, to randomly assign research participants to life circumstances which might result in the emergence of a mental disorder in efforts to examine etiological pathways, for example by randomly assigning some children to be raised by a mother with a diagnosis of BPD in order to determine the child's likelihood of developing psychopathology. Further, it is often not possible to manipulate the degree to which various risk contexts and causal variables

which may not be of interest to a given research question covary with maternal BPD. Thus, the majority of research addressing these topics is constrained to be observational in nature. Nevertheless, causal questions remain of central interest in this research area, given their immense relevance to public health and the wellbeing of these vulnerable dyads. The inclusion of potential confounding variables as covariates in statistical models works to isolate the causal effect in which we are interested.

Control for possible confounders can be addressed in both statistical and methodological approaches. As noted above, *analytic control* is the inclusion of participant-, family-, child-, or environmental-level variables other than primary variables of interest (covariates) in statistical models. By contrast, *methodological control* consists of recruitment methods, inclusion and exclusion criteria, and group assignment (in the case of clinical trials) that attempt to balance or offset the occurrence or degree of certain confounding variables, ensuring groups do or do not contain specific features. For example, individuals with BPD often experience low socioeconomic status (Walsh et al., 2013) and low income is associated with many poor outcomes for children (Singh-Manoux et al., 2004). Methodological control for this confound might be exercised through the recruitment of a comparison group which is also predominantly low income. Such an approach would strengthen inferences that observed parenting or child outcomes associated with BPD are truly related to this diagnosis and not to socioeconomic status. It should also be noted that methodological and statistical approaches to control are non-exclusive, in that control for a single variable in a study may be exercised both methodologically and statistically. For example, a single study might control for income through the recruitment of predominantly low-income families as well as the inclusion of

family income in statistical models. Despite this independence of approaches, rates of statistical or methodological control may be related to the degree of control exercised via the other approach, such that studies which exercise more statistical control rely less on methodological approaches and vice versa. Although the primary focus of this review is analytic control, it is important to acknowledge that methodological treatment of confounders offers a valid alternative to inference.

A comprehensive review of the statistical and inferential properties of covariates has been extensively addressed elsewhere (Foster, 2010; Judd & McClelland, 1989; Steiner et al., 2010; Yzerbyt et al., 2004) and is beyond the scope of this paper. Instead, a primer on two key elements of covariate use is provided here, accompanied by illustrations from the maternal BPD field. First, covariates are typically included for three primary reasons – 1) improvement of statistical power through the reduction of residual variance; 2) increased precision of effect size estimation for the primary effect of interest; and 3) overcoming limitations associated with causal inference in observational research (Rohrer, 2018; Yzerbyt et al., 2004). Briefly, statistical power is improved when a covariate is strongly related to the outcome variable, but not to the independent variable. Using an example from the maternal BPD literature, a study examining child theory of mind in the context of maternal BPD might experience improvements in power by controlling child verbal ability, provided that child verbal ability is not strongly related to maternal BPD. By contrast, inclusion of covariates which are more strongly related to the independent variable will function to decrease estimates of the primary effect of interest, resulting in a more precise estimate. For example, a study examining child theory of mind in the context of maternal BPD might obtain a more accurate estimate of effect size of the

relationship between these variables, with no appreciable gains in statistical power, by controlling for maternal depression, a variable that is strongly associated with maternal BPD. These statistical properties work in conjunction to achieve the third aim, by providing an estimate which is not inflated by confounding variables nor undetectable due to low power. For example, a study examining child theory of mind in the context of maternal BPD might strengthen confidence in a causal association by controlling for family socioeconomic (SES) status given the assumption that low SES does not cause the emergence of BPD.

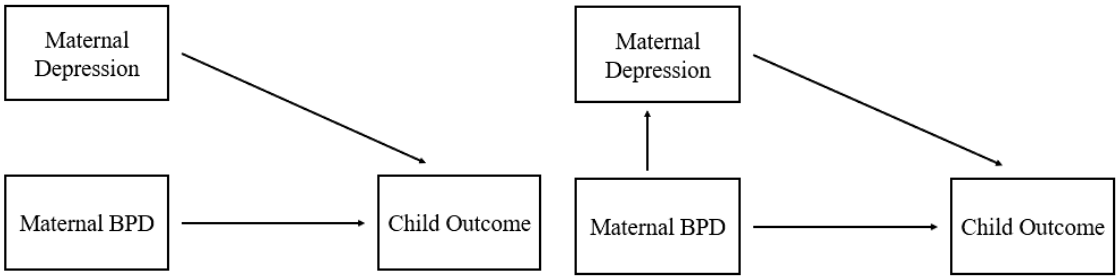
Second, the utility of covariates for causal inference in observational research is predicated on specific causal models. When a researcher includes (or omits) covariates in a statistical model, they are, knowingly or unknowingly, subscribing to a causal model which specifies relationships between particular variables. That is, the underlying directional, causal relationships among a set of variables suggests a particular covariance matrix and provides indications about which variables ought to be treated as confounders and thus controlled (Foster, 2010; Meehl, 1971; Pearl, 1995). Given different causal assumptions, very different covariates might be indicated. Thus, selection of covariates is not atheoretical, but rather is inherently predicated on causal theories. As noted in the examples above, the statistical benefits of covariate inclusion are only observed *given specific assumptions* about the relationships among variables (e.g. that SES does not cause BPD). Given different assumptions, the inclusion of those same covariates can work counter to these aims, introducing spurious relationships, suppressing true relationships of interest, and generating unstable effect size estimates (Foster, 2018; Rohrer, 2018; Pearl, 1995; Spector & Brannick, 2011; Meehl, 1971). A graphical

notation system developed by computer scientists, called Directed Acyclic Graphs (DAGs; Pearl, 1995), can be useful in depicting these relationships and assumptions. DAG models resemble path diagrams or structural equation models (SEMs) in that they are comprised of variables (depicted in boxes) and directional arrows linking variables in "causal chains." DAGs are distinct from mathematical models in that they do not assume or imply linear or even parametric relationships between variables. For this reason, DAGs can be understood as non-parametric SEMs (Elwert, 2013). DAGs are distinguished from SEMs and path models in that they are directional, depicting causal relationships which end at a terminal outcome, and acyclic, with no reciprocal or circular relationships between variables permitted. Variables depicted in DAGs represent a single point in time (measurement instance), thus transactional and reciprocal relationships are permitted only through the modeling of such interactions over time (e.g. Child Psychopathology at Age 5 → Parental Hostility at Age 5 → Child Psychopathology at Age 6). Together, these properties produce independence assumptions about pairs of variables, resulting in clearly traceable causal "chains." These chains can then be leveraged to understand the influence of including particular variables in the chain in statistical models.

To illustrate these properties, let us consider maternal depression, maternal BPD, and some unspecified child outcome. Figure 1a depicts a possible causal relationship between these variables. This model specifies that maternal BPD causally affects the child outcome, that maternal depression causally affects the child outcome, and that there is no causal relationship between maternal BPD and depression (that is, depression does not cause BPD and BPD does not cause depression). Under these assumptions, including

maternal depression as a covariate in statistical models "blocks" its confounding effect on the child outcome.

Figures 1a and 1b. Comparison of DAG models with a) no causal relationship between maternal BPD and depression and b) causal relationship between maternal BPD and depression



By contrast, we might believe that mothers' levels of depression are causally impacted by (is a causal descendent of) maternal BPD, as depicted in Figure 1b. Under this assumption, controlling for maternal depression removes an amount of causal effect otherwise attributable to BPD by "blocking" the causal pathway between maternal BPD and depression, thus reducing our effect size estimate. That is, controlling for maternal depression under this model removes part of our effect of interest (maternal BPD → child outcome), by blocking one avenue for the transmission of this effect. In this way, controlling for mediating variables risks covarying away the exact effect in which we are interested. Both models may be theoretically defensible, but each implies a different statistical approach. As is readily apparent, this example is oversimplified for illustrative parsimony. This figure does not depict causal antecedents of maternal BPD and maternal

depression[1], which would likely implicate additional variables as covariates and may further dissuade us from including depression as a covariate.

Although not exhaustive, these examples illustrate the utility of DAGs for depicting underlying causal assumptions and the impact of such assumptions on covariate selection. These examples additionally highlight the way covariate selection can change the interpretation of outcomes. Despite this, there remains little formal guidance regarding the selection of statistical covariates in psychology broadly, in subdisciplines specifically, or even within research areas (Foster, 2010). Decisions regarding which covariates to include in statistical models are particularly difficult in research examining child development and parenting in the context of maternal BPD. As stated earlier, this complexity stems from 1) the high rate at which BPD co-occurs with other disorders and risk contexts and 2) the nature of dual-generation developmental psychopathology research in which multiple contextual risk factors co-occur (Cicchetti, 1993; Lanza et al., 2010). In combination, these factors make the selection of covariates and the clear articulation of the rationale for such selection especially challenging in this subfield.

Confounding Issues in BPD

Theoretical and empirical work modeling the meta-structure of mental disorders often aims to generate a finite number of diagnostic categories in an attempt to 'carve nature at its joints.' Efforts to this end have consistently produced diagnostic systems in which the categorization of disorders into discrete phenotypes is challenged by comorbidity, or the co-occurrence of multiple discrete diagnoses. Such difficulties persist

[1] In full DAG notation, it is important to depict any variable which is causally related to two or more other variables in the model, as resulting causal chains can introduce backdoor paths and highlight collider variables. The present example excludes such variables for the sake of simplified illustration.

in the DSM-5, under which rates of comorbidity remain high (Goldstein et al., 2016; Ruscio et al., 2017). Thus, research on specific mental disorders is tasked with parsing the influence of a specific mental disorder from general poor mental health or other disorders. To this end, comorbidity presents a challenge for the isolation of an effect of interest. This challenge is often addressed by treating co-occurring mental health symptoms or diagnoses which are not of interest as covariates in statistical models (e.g. Kiel et al., 2011; Macfie et al., 2014; Zalewski et al., 2014). Comorbidity of mental health symptoms presents a challenge regardless of specific disorder being examined but is especially pronounced in research on BPD, as rates of comorbidity in BPD are exceptionally high (Grant et al., 2008). In both epidemiological and outpatient samples, BPD has been found to covary at high rates with internalizing disorders (mood disorders, anxiety disorders) and externalizing disorders (substance use disorders, conduct disorder) (Eaton et al., 2011; Grant et al., 2008; Zimmerman et al., 2005). BPD has also been found to co-occur at high rates with other personality disorders (Sharp et al., 2015). Thus, research examining the unique effect of BPD must contend with the co-occurring influence of a broad array of mental disorders.

Beyond comorbid mental disorders, research on BPD must further disentangle the influence of BPD on outcomes of interest from that of other co-occurring contexts known to elevate risk for poor interpersonal, health, and mental health outcomes. Individuals with BPD are more likely than non-disordered individuals to experience risk factors for a host of adverse life outcomes including future mental disorders and medical complications. Specifically, individuals with BPD are more likely to be of lower socioeconomic status (Walsh et al., 2013), have lower educational attainment (Bagge et

al., 2004; Trull et al., 1997), and to have experienced greater early life adversity (Pietrek et al., 2013). Thus, isolating the unique influence of BPD requires the consideration of many potential confounders related to individual characteristics and life history in addition to the consideration of co-occurring mental disorders. In this way, isolating the effect of BPD is complicated due to the substantial number of potential variables associated with BPD which might be controlled.

Confounding Issues in Developmental Psychopathology

In addition to these challenges, scholars working in the area of maternal BPD must also grapple with complexities related to research in developmental psychopathology. The fields of child development and developmental psychopathology have identified a multitude of risk factors which increase the likelihood that children will experience poorer developmental outcomes and these risk factors can be categorized as occurring at various levels of influence (i.e. home, school, neighborhood, etc.) (Bronfenbrenner, 1979; Evans et al., 2013; Lanza et al., 2010). Children whose parents have BPD are more likely to experience these risk contexts than their peers with non-disordered parents. Specifically, parents who have BPD are more likely to experience marital distress and dissolution (Whisman & Shonbrun, 2009), have lower educational attainment (Bagge et al., 2004; Trull et al., 1997), experience involvement with the criminal justice system (Moore et al., 2017), and be of lower socioeconomic status (Walsh et al., 2013) than non-disordered parents. Thus, children whose parents have BPD face additional challenges beyond parental psychopathology which increase the likelihood of poor outcomes. For this reason, research in this area must consider

confounders related to multiple levels of contextual risk which might be associated with child outcomes.

Given that parental BPD is associated with many contexts known to negatively impact child development, isolation of the unique influence of BPD may be approached through the inclusion of these variables as covariates in statistical models. Problematically, however, the inclusion of highly correlated independent variable, known as multicollinearity, reduces statistical power and results in unstable estimates (Cohen et al., 2003; Myers & Wells, 2003). Concerns related to multicollinearity may be particularly relevant in the field of maternal BPD, given that many risk contexts are highly correlated with the diagnosis (Bagge et al., 2004; Moore et al., 2017; Walsh et al., 2013; Whisman & Shonbrun, 2009). Inclusion of these covariates without careful examination of collinearity with parental BPD threatens to undermine the very goals for including covariates – improved causal inference, more accurate estimates of effects, and gains in statistical power.

Current Study

Research addressing child development in the context of maternal BPD generally seeks to answer the questions: *what is the causal effect of maternal BPD on child outcomes and what mechanisms explain this effect?* To this end, scholars working in this area must contend with the reality that child development in the context of maternal BPD is fraught with multiple confounding risk factors. Given this, it is beneficial for the field to further consider covariate adjustment, as this practice is an invaluable tool for obtaining more precise estimates of effects and, ultimately, for advancing theory. Unsurprisingly, there is no documentation of covariate use in this literature. The present

study aims to address this gap by reviewing covariate use in this research to date with a goal of providing best practices guidelines for covariate use in this field. To achieve these goals, we conducted a systematic review of studies examining parenting and child development in the context of maternal BPD which were published from 1980-2019 with the goal of generating a descriptive inventory of covariate use and practices. This review aims to explore the scope and variability of covariate use with regard to the following three general questions: 1) What variables are being treated as covariates?; 2) How many covariates are being included in analyses?; 3) What explanation is being provided for inclusion of covariates?

Method

The Preferred Reporting Items for Systematic Reviews and Meta Analyses (PRISMA; Moher et al., 2009) were used to guide this review. The methods employed in a systematic review of the child outcomes and parenting of mothers who have elevated symptoms or a diagnosis of BPD published in 2016 by Eyden and colleagues were used as the basis for search string, database selection, and inclusion/exclusion criteria. This review was selected for its recency, methodological rigor, and identical search and inclusion goals. The lead author of the present study (G.B.) consulted with the lead author of the 2016 review (J.E.) in order to ensure methodological comparability. The previous review was deemed a sufficiently thorough search of the literature dated 1980 to July 2015. Given this, the present review extracts data from the 33 articles included in the Eyden and colleagues (2016) review as well as articles which met search criteria and were published between July 2015 and May 21st, 2019. A preregistration of these search

and data extraction methods (posted prior to search date) can be found at https://osf.io/7zu85.

Data Sources

PsycINFO, PubMed, Scopus, Web of Science, EMBASE, and ASSIA were searched to identify empirical studies of mothers who have a diagnosis of BPD or BPD symptoms and their children published between 2015 and May 21st, 2019. The year 2015 was selected as the earliest year to search as Eyden and colleagues (2016) are noted to have searched 1980 to July 6th, 2015.

Search Terms

The following search strings were used to search Titles and Abstracts in each of the above databases: (borderline* OR "emotionally unstable personality" OR BPD) AND (mother* OR parent* OR maternal*) AND (child* OR infant* OR infancy OR offspring OR bab* OR adolescen* OR famil* OR girl* OR teenager* OR youth* OR young* OR toddler* OR daughter* OR son*). Both Titles and Abstracts were searched in order to ensure articles which reported on relevant populations were not overlooked when such populations were not the primary subject of a study.

Eligibility Criteria

This review included retrospective, cross-sectional, and longitudinal quantitative studies which met the following criteria:

1) Includes mothers who have a diagnosis of BPD or BPD symptoms and/or offspring (of any age) of mothers who have a diagnosis of BPD or BPD symptoms
2) BPD or BPD symptoms were assessed via standardized measure

3) Reports on maternal parenting and/or offspring outcomes
4) Parent sample consists of at least 70% mothers

Studies were excluded if they met the following criteria:

1) Were reviews, expert opinion commentaries, or individual case studies
2) Examined personality disorders broadly rather than BPD or BPD symptoms specifically
3) Reported on extreme outcomes resulting in external intervention (e.g. filicide)
4) Were not written in English
5) Were not published (e.g. conference abstracts)

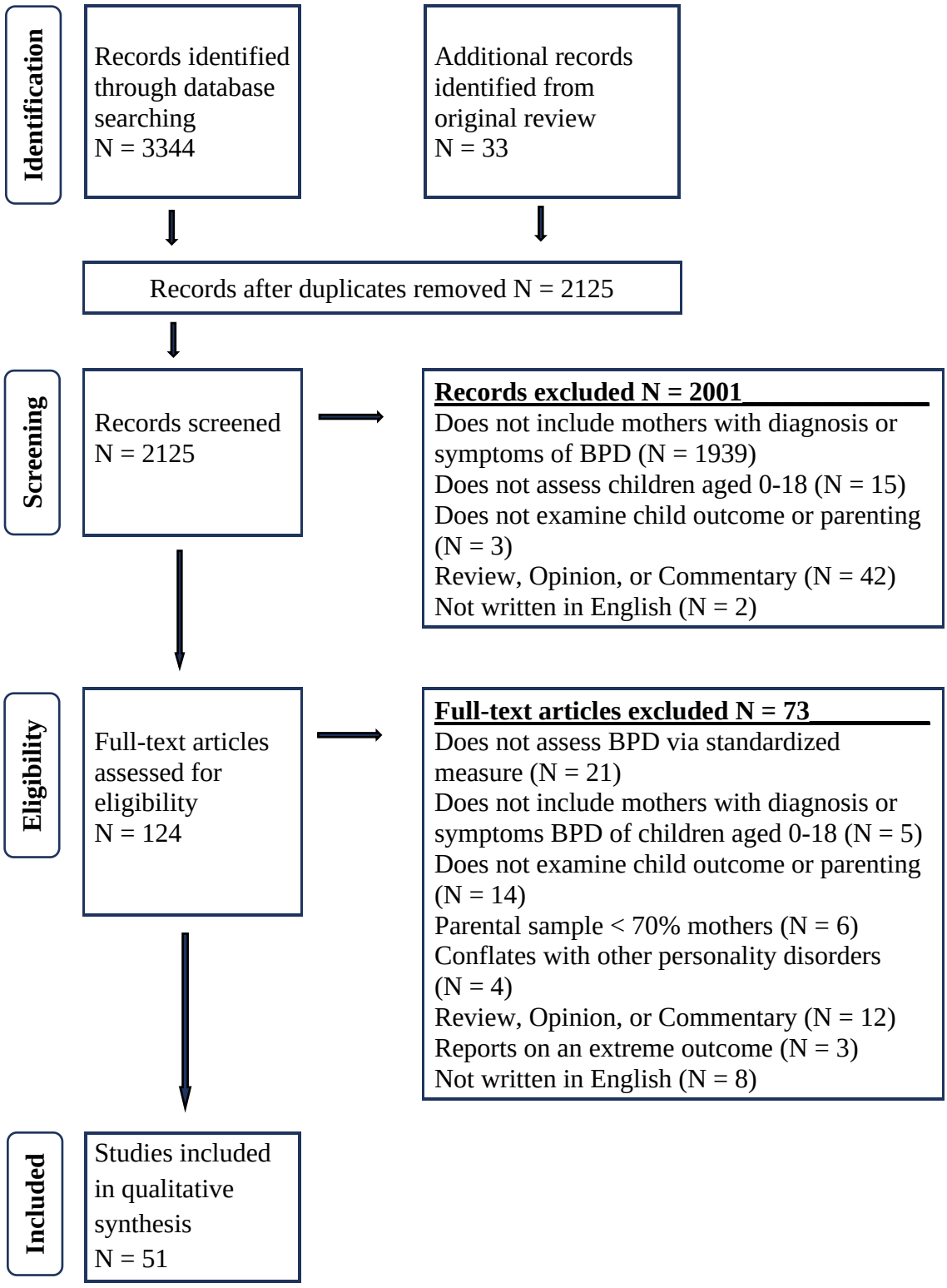
Screening Procedures

DisillerSR (Evidence Partners, Ottawa, Canada), an online platform for storage, screening, and data extraction in systematic reviews, was used in this review. A diagram of the article search and screening process is depicted in Figure 2. Screening was carried out at two levels. First, the titles and abstracts of all articles retrieved were screened by one of four trained coders to determine initial eligibility. Second, full-text copies of articles which met inclusion criteria or for which eligibility could not be determined via title and abstract were retrieved and screened by this same team of four authors. At the full-text screening stage, all articles were screened by G.B. and at least one other author. Disagreements regarding eligibility were resolved with discussion between co-authors.

Data Extraction

A data extraction form (see Appendix) was created to extract information related to study population, aims, methods, and focal analyses including covariate use in these analyses. Study descriptive information extracted included year of publication, country in

Figure 2. A diagram of the search and screening process (PRISMA; Moher et al., 2009)



which the study was conducted, age range of children, and what proportion of the sample was reported to be Caucasian. Methodological information extracted included study design, inclusion and exclusion criteria, and methods used to assess mothers and children. Exclusion criteria which were exact inverses of inclusion criteria (e.g. inclusion "Must speak English" and exclusion "Does not speak English") were not coded. Thus, all exclusion criteria represented unique criteria. In order to extract data related to statistical covariates, coders identified a single focal analysis in each study. The focal analysis of a study was identified as the statistical model which tested the hypothesis around which the study was framed. When no single or primary study frame was apparent, the analysis which tested the study's first listed hypothesis was treated as the focal analysis. Analysis-level information extracted included what variables were treated as covariates in the focal analysis, whether or not covariates were reported in the final statistical model, total number of covariates included in the focal analysis, and authors' stated rationales for covariate inclusion (if any). To assess rationales for covariate inclusion, coders recorded whether any of the following explanations were used to explain why covariates were included in the focal statistical model – correlations between variables in the current data, theory-based decision, theory-based decision which the authors stated was made after looking at the data, other reasons (which coders explicated), or no rationale. Coders selected a rationale (or indicated there was none) for each covariate included in the focal analysis. Finally, because the purpose of this review was to describe statistical and methodological approaches to statistical control, an overall assessment of study quality was deemed outside the scope of the current review and was not assessed. Twenty

percent of articles were double coded for reliability. For non-exclusive categorical codes, percent agreement was calculated, as chance agreement was highly unlikely. For number of covariates included in focal analysis, intraclass correlation coefficient was calculated. Estimates of reliability (percent agreement 94-100%; ICC = 0.99) suggest excellent agreement on all codes.

Data Synthesis

In line with the goals of this review, all studies were qualitatively synthesized. A meta-analysis was not conducted as such methods do not address the qualitative goals of this review. Results are presented in four primary sections – 1) description of characteristics of included articles; 2) descriptive inventory of number of covariates and which variables are treated as covariates; 3) rationale for covariate inclusion; and 4) methodological factors related to covariate use.

Results

Description of Included Articles and Study Characteristics

Prior to addressing review aims, characteristics of included studies were examined in efforts to better understand publishing patterns in this literature. Fifty-one total papers were retrieved. The rate at which studies were published increased across the three decades examined (Table 1). Specifically, five studies were published between 1980 and 1999, 10 studies were published between 2000-2010, and 36 studies were published between 2011-2019. The year with the greatest number of publications is 2018 (n = 7, 14%). Nearly half of all papers (n = 24, 47%) included samples and author teams based in the United States. Eight papers (16%) included Australian samples and author teams, five (10%) included German samples, five included UK samples, four (8%)

Table 1. Summary of covariate use in studies examining maternal BPD

Year	Authors	Number of Covariates	Covariate Rationale(s)	Number of Inclusion/ Exclusion Criteria	Study Design	Focal Outcome Category	Offspring Age Groups
1991	Jellinek et al.	0	None	7	Cross-Sectional	Child Outcome	School-Aged
1991	Marantz & Coates	0	None	2	Case-Control	Child Outcome	Preschool; School-Aged
1995	Howard et al.	0	None	3	Cross-Sectional	Child Outcome	Infancy
1995	Feldman et al.	0	None	3	Case-Control	Child Outcome	Preschool; School-aged; Adolescence
1996	Weiss et al.	0	None	5	Case-Control	Child Outcome	School-Aged; Adolescence
2003	Crandell et al.	0	None	2	Case-Control	Child Outcome	Infancy
2005	Hobson et al.	0	None	2	Case-Control	Child Outcome	Infancy
2005	Abela et al.	1	Theory	2	Case-Control	Child Outcome	Adolescence
2006	Barnow et al.	2	Theory	2	Case-Control	Child Outcome	Adolescence
2007	Newman et al.	0	None	5	Case-Control	Child Outcome	Infancy
2008	Herr et al.	0	Correlations, Theory	1	Cross-Sectional	Child Outcome	Adolescents
2008	Delavenne et al.	4	None	1	Case-Control	Child Outcome	Infancy
2009	Macfie & Swan	1	None	2	Case-Control	Child Outcome	Preschool; School-Aged

Table 1. (continued)

Year	Authors	Number of Covariates	Rationale(s) for Covariate Inclusion	Number of Inclusion/ Exclusion Criteria	Study Design	Focal Outcome Category	Offspring Age Groups
2009	Hobson et al.	0	None	2	Case-Control	Parenting	Infancy
2010	Crittenden & Newman	0	None	2	Case-Control	Parenting	Infancy
2011	White et al.	0	Correlations (lack thereof)	7	Case-Control	Parenting	Infancy
2011	Kiel et al.	0	None	3	Case-Control	Parenting	Infancy
2011	Harvey et al.	1	Theory	3	Cross-Sectional	Parenting	Infancy
2011	Cheng et al.	1	Theory	9	Case-Control	Child Outcome	Adolescence
2012	Wilson & Durbin	1	Correlations	1	Cross-Sectional	Child Outcome	Preschool
2012	Bertino et al.	2	Correlations	5	Cross-Sectional	Child Outcome	Preschool; School-Age; Adolescence
2013	Stepp et al.	3	Correlations	2	Longitudinal	Child Outcome	Adolescence
2013	Reinelt et al.	1	None	0	Cohort	Child Outcome	Adolescence
2013	Barnow et al.	4	Correlations, Theory	2	Cohort	Child Outcome	Adolescence
2013	Schacht et al.	1	Correlations, Theory (post-hoc)	1	Case-Control	Child Outcome	Infancy
2014	Zalewski et al.	4	None	3	Longitudinal	Parenting	Adolescence

Table 1. (continued)

Year	Authors	Number of Covariates	Rationale(s) for Covariate Inclusion	Number of Inclusion/ Exclusion Criteria	Study Design	Focal Outcome Category	Offspring Age Groups
2014	Macfie et al.	6	Correlations	3	Case-Control	Parenting	Preschool; Adolescence
2014	Gratz et al.	0	None, Theory	9	Case-Control	Child Outcome	Infancy
2014	Elliot et al.	4	None	0	Case-Control	Parenting	Infancy
2015	Whalen et al.	2	Correlations	1	Cross-Sectional	Child Outcome	Infancy
2015	Frankel-Waldheter et al.	0	None, Correlations	1	Case-Control	Child Outcome	Adolescence
2015	Conway et al.	1	None	1	Cohort	Child Outcome	Adolescence
2015	Blankley et al.	6	None	3	Case-Control	Child Outcome	Infancy
2015	Haabrekke et al.	1	Theory	3	Longitudinal	Child Outcome	Infancy
2016	Pare-Miron et al.	9	None	3	Case-Control	Child Outcome	Infancy
2016	Kaufman et al.	4	None	2	Cohort	Child Outcome	School-Age
2017	Mena et al.	4	Correlations, Theory	4	Case-Control	Child Outcome	Preschool; School-Age
2017	Macfie et al.	3	Theory	6	Case-Control	Parenting	Preschool; School-Age
2017	Kiel et al.	4	Correlations	3	Case-Control	Parenting	Infancy

Table 1. (continued).

Year	Authors	Number of Covariates	Rationale(s) for Covariate Inclusion	Number of Inclusion/ Exclusion Criteria	Study Design	Focal Outcome Category	Offspring Age Groups
2017	Huntley et al.	0	None, Theory	3	Longitudinal	Child Outcome	Infancy
2017	Marcoux et al.	1	None	3	Case-Control	Parenting	Infancy
2017	Apter et al.	9	None	0	Case-Control	Child Outcome	Infancy
2018	Kurdziel et al.	1	None	2	Case-Control	Child Outcome	Adolescence
2018	Kluczniok et al.	5	Correlations, Theory	10	Case-Control	Parenting	Preschool; School-Age
2018	Trupe et al.	0	None	4	Case-Control	Parenting	Preschool; School-Age
2018	Mahan et al.	2	Correlations, Theory	2	Case-Control	Parenting	Adolescence
2018	Dittrich et al.	4	None, Theory	8	Cross-Sectional	Parenting	Preschool; School-Age
2018	Hoivik et al.	7	Theory	6	Cohort	Parenting	Infancy
2018	Zalewski et al.	4	Theory	5	Cross-Sectional	Child Outcome	Preschool
2019	Lyons-Ruth et al.	0	Correlations	2	Case-Control	Child Outcome	Infancy
2019	Hatzis et al.	3	Theory	6	Case-Control	Parenting	Infancy

included Canadian samples, two (4%) included samples from France, two included samples from Norway, and one included a Chinese sample and author team.

Study characteristics which might be related to covariate use, namely age range of children who participated and focal outcome, were also extracted. Samples of children ranged in age from newborn to late adolescence (21 years). Age range examined was coded as belonging to the following categories – infants (0-2 years), preschoolers (3-5 years), school-aged (6-12 years), and adolescents (13-21 years). The majority of studies (n = 40, 78%) included participants from a single age category. Nine studies (18%) examined two age categories and two studies (4%) examined three. Twenty-four samples included infants, 12 included preschoolers, 13 included school-aged children, and 15 included adolescents.

Although not traditionally considered a study characteristic, focal analysis was extracted to inform whether number and type of covariates might differ by outcome studied. Thirty-four papers (67%) included focal analyses related to children's outcomes in the context of maternal BPD and 17 (33%) included focal analyses related to parenting in the context of maternal BPD, suggesting this literature to date has focused more on child outcomes. Within parenting or child outcomes, there was little consistency with regard to what was measured or assessed for each focal analysis. The most frequently examined child outcome was symptoms or a diagnosis of a mental disorder, which was examined in 15 (44%) of the studies which focused on child outcomes. The next most frequent category of child outcomes could be broadly categorized as social outcomes, encompassing social competence, language development and vocalizations, responsiveness to caregiver, family satisfaction, and narrative representations. Such

outcomes were examined in seven (21%) studies. The remaining child outcome categories and the rate they were examined were: temperament and emotionality examined in four (12%) studies, physical development examined in three (9%) studies, attachment in two (6%) studies, and emotion regulation, theory of mind, and executive function in one study each. Similarly, parenting behaviors examined ranged widely, with the most frequently examined behavior being positive parent behaviors such as sensitivity, warmth, responsiveness, and positive affect which were examined in six (41%) of the 17 studies examining parenting behavior. The next most common outcome was negative parenting behavior (such as harshness, hostility, negative affect, and control) which was assessed in four (24%) studies. Maternal attachment-related behaviors (mind-mindedness, working model of the child, recognition of child's emotions) and emotion-related parenting (emotion labeling, emotion socialization, affective communication) were assessed in three (17%) studies each. Finally, maternal potential for child abuse was assessed in one study.

Inventory of Statistical Covariates

The first and second aims of this review were to provide an inventory of how many covariates are included in analyses related to maternal BPD and of which variables are being treated as covariates in these analyses. Of the 51 papers included in this review, 34 (67%) included at least one covariate in the paper's focal analysis. Of the 34 studies that used covariates, ten studies (29%) included one covariate, nine studies (18%) used four covariates, five studies (10%) included two covariates, three studies (6%) included three covariates, two studies (4%) each included five, six, and nine covariates, and one study included seven covariates. This indicates that most studies employ at least one

statistical covariate and, of studies which included covariates, it is most common to include one or four covariates. Number of covariates included by year of study publication was examined to better understand how covariate use might be changing over time. Although the number of studies which included no covariates has increased slightly over the last four decades, these studies constitute a shrinking proportion of total work. All five studies published prior to the year 2000 included no statistical covariates in focal models. Of the 10 studies published between the years 2000 and 2010, six included no covariates (range 0-4). Of the 36 studies published in the last decade, only seven included no covariates (range 0-9, $M = 2.72$).

During initial coding, covariates were classified as belonging to one of three categories – mother-related, child-related, or family-related. Of the 34 studies which included covariates, 28 included covariates related to mothers. The most common mother-related covariate was maternal depression (included in 21 studies) with maternal anxiety controlled in three studies, maternal substance use controlled in seven studies, maternal trauma controlled in two studies, and maternal mental health symptoms not described in those categories (e.g. dysthymia, antisocial traits, comorbid personality disorders) controlled in 9 studies. Maternal education was controlled in 10 studies and maternal age was controlled in 4 studies. Fourteen additional mother-related covariates were included in only one study. These covariates included variables related to maternal physical characteristics (race, obesity), maternal resources (type of hospital at which mothers gave birth, type of insurance, employment status), parenting behaviors (parenting style, expressed negative affect in parenting interactions, intrusiveness), life

history (age at first pregnancy), transdiagnostic characteristics (emotion regulation), and cognitive abilities (intelligence).

Child-related covariates were included in analyses less frequently. Of the 34 studies which included covariates, 17 included covariates related to children. The most common child-related covariate was child age (included in 9 studies) with child sex controlled in 6 studies. All other covariates were controlled in only 1 study. These covariates included variables related to infant temperament (fear, soothability, negative emotionality), symptoms of psychopathology (depression, BPD, number of personality disorders), intelligence (cognitive ability, verbal ability), and child ethnicity.

Family-related covariates were the least common, controlled in 14 studies. The most common family-level covariate was family income (included in 10 studies) with single parent status controlled in 4 studies, a global environmental risk score controlled in 2 studies, and number of siblings controlled in 1 study.

Rationale Provided for Covariate Use

The third aim of this review was to assess whether studies provided rationales for covariate use and what type of rationales were provided. Of the 34 papers which included covariates in the focal analysis, 10 provided no rationale for the inclusion of at least one covariate in their analytic models. In 7 of these papers, no explanation or justification was provided for any covariates included in the focal analysis.

Theory was the most commonly provided rationale for inclusion of covariates. Eighteen of the 34 papers which included covariates reported an a priori theoretical decision to include at least one covariate in their analytic models. Examples of a priori theoretical decisions included disentangling the effect of BPD from confounding mental

disorders, isolating BPD relative to environmental risk, and disentangling the effects of third variables related to child outcomes (such as verbal ability). In 10 of these papers, theoretical rationales were the sole explanation provided for including covariates in statistical models. In one additional paper, authors reported a post-hoc theoretical decision – a theoretical decision made after looking at relationships between variables – for the inclusion of a covariate.

Correlations among variables was another rationale provided for covariate inclusion. In 15 papers, the presence of statistically significant correlations between predictor or outcome variables and other variables in the dataset was used to explain the inclusion of covariates. In nearly half of papers which included this rationale (n = 7), this was the sole explanation provided. In one additional paper, which did not include any covariates in the focal statistical model, an absence of statistically significant correlations between predictor variables and other variables was provided as the rationale for not including covariates in the focal analysis.

Two thirds of studies (n = 24) which included covariates provided a single rationale for covariate inclusion. Ten studies provided multiple rationales. That is, the inclusion of some covariates was explained by either theory or correlations in the data and the inclusion of another covariate was either not explained or was explained by theory or correlations.

Methodological Control

As noted previously, control for potential confounders can be approached both statistically and methodologically. In the present review, methodological control was estimated through the number of reported inclusion and exclusion criteria. Number of

reported inclusion criteria employed by studies ranged from 0-5, with three studies not detailing any inclusion criteria. Twenty-six (51%) studies specified that mothers must have a child of a certain age, 11 (22%) studies specified language requirements, nine (18%) studies specified mothers had to have symptoms or a diagnosis of another disorder (e.g. Major Depressive Disorder, Substance Use Disorder), and 8 (16%) studies specified that mothers must have full or primary custody of their child. All other inclusion criteria (income requirements, child and parent are biologically related, maternal age, child symptoms or characteristics) were included in 5 or fewer studies. Number of reported exclusion criteria employed by studies ranged from 0-7, with 24 studies not detailing any exclusion criteria distinct from inclusion requirements. As a reminder, in this review, exclusion criteria which were exact inverses of inclusion criteria (e.g. inclusion "Must speak English" and exclusion "Does not speak English") were not counted, thus all exclusion criteria represented unique requirements. Twelve (24%) studies excluded families in which the focal child had a developmental disability, 8 (16%) studies excluded mothers who exhibited psychotic symptoms, and 7 (14%) studies excluded mothers who had symptoms or a diagnosis of another mental disorder from comparison groups. All other exclusion criteria (substance use/abuse, physical or neurological disability, intelligence criteria, severe/active suicidal ideation, hospitalization, inability to consent) were included in 5 or fewer studies.

Study design decisions, such as use of comparison groups in case-control designs, can also work to control for confounding variables. Of the 51 included studies, the vast majority employed a case-control design. In these studies, efforts were made to recruit at least two distinct groups of participants with one group characterized by elevated

maternal BPD symptoms or a maternal diagnosis of BPD. The comparison groups varied and included mothers who have no mental health symptoms, mothers who have elevated symptoms of depression or a diagnosis of Major Depressive Disorder, mothers who have symptoms of substance abuse, and mothers who have multiple comorbid mental health conditions. Nine studies employed a cross-sectional design, five employed cohort designs, and four employed longitudinal designs. For the purposes of coding, cohort studies which included at least 3 assessments were deemed longitudinal.

Finally, it is possible that methodological and statistical approaches to control for confounders are not independent. Study designs which employ more methodological control might rely less on statistical control. To probe this possibility, the average number of covariates employed in different study designs was examined. Cross-sectional studies and case-control studies tended to have slightly more inclusion and exclusion criteria (Cross-sectional mean = 3.89; Case-control mean = 3.48), on average, than did studies employing other designs (Cohort mean = 2.6; Longitudinal mean = 1). By comparison, case-control studies included, on average, fewer covariates in analyses (mean = 1.58) compared to other study designs (cross-sectional mean = 2.2; cohort mean = 2.8; longitudinal mean = 4.2). This suggests that, on average, studies employing a case-control design tend to employ greater methodological and less statistical control than studies employing other designs.

Although average amount of methodological and statistical control appears to differ by study type, averages can obscure meaningful variability within groups. To address this limitation and to further probe the association between statistical and methodological control, we examined the bivariate association between number of

inclusion/exclusion criteria and number of covariates included in analyses. Pearson correlation revealed functionally no association (r = .03) between number of inclusion/exclusion criteria and number of covariates. Thus, although case-control studies tended, on average, to employ more methodological and less statistical control, studies did not systematically balance control exerted between methodological and statistical approaches.

Discussion

Despite the immense utility of covariate adjustment for observational research, and the pervasiveness of this practice in psychological research, there is little research documenting the nature of covariate adjustment practices in specific subdisciplines. The present work extended a systematic review of research examining the parenting and child outcomes associated with maternal BPD by providing a qualitative synthesis of covariate use in this literature. Broadly, results from this review suggest that the number of studies addressing child development and parenting in the context of maternal BPD is increasing rapidly, with over half of this literature published in the last 10 years. The majority of these studies (approximately two-thirds) included covariates in statistical analyses, with the average number of covariates increasing over time. Although average number of covariates used varied by study design (with case control studies using the fewest covariates, on average), number of covariates was not related to amount of methodological control employed by studies. Among variables measured in these studies, those related to maternal mental health symptoms or disorders are the most frequently included as covariates. Theory is the most common rationale for covariate inclusion.

Importantly, a significant number of studies cited the presence of correlations with other variables as the rationale for covariate inclusion or provided no rationale at all.

Results suggest that there is a general trend towards increasing covariate use in this literature. This trend signals a shift towards more complex statistical models in which many possible confounding variables are included in analyses in efforts to better isolate the causal effect of maternal BPD. Importantly, this trend is not associated with reduced methodological control. This indicates that researchers are not merely shifting their attempts to control confounders from methodological to statistical approaches but are generally attempting to exert greater control or to control for more confounders. Such a shift might reflect increased sophistication and specification of a causal model, with authors sharing a causal framework and carefully controlling for an increasing number of variables known to confound results. Another possibility is that this trend is part of a growing convention in psychological research to include many covariates in statistical models (Rohrer, 2018). For many decades, methodologists have cautioned against the tendency to include potential confounding variables in analyses without serious consideration (Meehl, 1971). Rather than better isolating an effect of interest and strengthening inferences, such approaches can suppress effects of interest, introduce spurious relationships, and lead to imprecise estimates of effects (Rohrer, 2018; Greenland et al., 1999). Even so, the myth that greater control for confounding variables is attained through the inclusion of many covariates in statistical analyses persists (Achen, 2005). Although it is possible the nascent field of maternal BPD has converged around a shared causal framework, it is more plausible that it is following the broader trend of including a greater number of covariates.

The understanding that this literature is participating in a trend towards greater covariate use is consistent with the substantial heterogeneity of covariates included in this literature. Field-wide convergence around a shared causal model would suggest little heterogeneity of covariates included in analyses. Results indicate, however, that, with the notable exception of maternal mental health symptoms, there were no consistent covariates or covariate clusters in this literature. This may result from the substantial variability in outcomes examined. There was little consistency in either parenting behaviors or child outcomes examined and, even when studies examined the same construct (e.g. childhood symptoms of mental disorders) operationalizations within these constructs varied (e.g. externalizing symptoms, internalizing symptoms, symptoms of specific mental disorders, overall number of symptoms). It is therefore possible that highly variable use of covariates is related to highly variable outcomes examined. It is also possible that covariate variability is attributable to idiosyncratic causal models or norms. Given that there is little formal guidance around covariate selection, many scholars working in this area must rely on their training experiences to guide covariate selection (Rohrer, 2018; Achen, 2005). This can produce norms or practices which are consistent within labs (e.g. “In our lab, we control for child age in all analyses”), but which vary between labs. Such varied approaches are not, in and of themselves deleterious to a research area. In fact, a diversity of models which are empirically pitted against one another can work to advance knowledge, resulting in novel lines of research which might not otherwise have been explored (Platt, 1964). Without clear declaration of the underlying models and where models differ, however, these possible novel lines remain unexplored. Further, causal models which differ a great deal might be conflated

should readers (and, at times, authors) not appreciate the varied models that clusters of covariates convey.

The present review also found that maternal mental health symptoms are statistically controlled in over half of studies which include analytic covariates. The most common mental health symptom for which studies control is maternal depression. BPD is associated with a wide array of mental health difficulties including substance use, anxiety disorders, and eating disorders (Grant et al., 2008). It is worth exploring, then, why depression, among other disorders, is so commonly controlled. This may result in part from the history of research exploring the links between maternal mental health, parenting, and child outcomes. This research area first gained significant traction examining maternal depression (e.g. Weissman & Paykel, 1974; Weissman et al., 1972) and quickly revealed that children born to depressed mothers are both at elevated risk of developing mental disorders themselves (Goodman et al., 2011) and are more likely to encounter socioemotional adversity throughout development (Murray et al., 2006; Murray et al., 1999; Priel et al., 2019). Given these known associations, scholars working to parse the effect of maternal BPD from maternal psychopathology broadly may attempt to control (methodologically or analytically) for the effect of maternal depression. Given that maternal BPD has been found to be associated with many other mental health diagnoses and symptoms, each of which is understood to be negatively related to parenting quality and optimal child outcomes, it is unsurprising that authors are treating these symptoms as covariates in statistical models. Although laudable, such motives must be weighed critically against an author's stance on the general structure of BPD as a disorder. A core feature of BPD is a persistent pattern of affective instability which

includes wide mood swings. Given that emotional lability is core to this disorder, it is unsurprising that individuals with BPD often report elevated symptoms of depression (Beatson & Rao, 2013) and anxiety (Zanarini et al., 2004) relative to non-disordered individuals. It is possible, then, that depression in the context of BPD may not function so much as a distinct mental health phenomenon as it does a component of the disorder itself. Certainly, the experience of elevated mental health symptoms is not sufficient for a BPD diagnosis, but it does serve as an indicator in a larger constellation of symptoms. In this view, the treatment of maternal depression as a covariate does not serve to reduce confounding, but to partition variance away from the construct in which authors are interested[2]. That is, in this view, authors controlling for mental health symptoms may be covarying out the effect in which they are interested, portioning them into small pieces ascribed to "nuisance" variables. Despite the common treatment of maternal depression as a covariate, it is unclear the extent to which authors are aware of the causal assumptions implied by such treatment, as underlying causal models/assumptions are not well articulated in this literature.

A final key finding is that many studies are reporting theoretical justification for inclusion of covariates in statistical models. The reason most commonly articulated is a desire to remove extraneous effects theorized to be associated with constructs of interest. Such decisions are presented nearly unilaterally as being made prior to looking at study results. This suggests that many authors are in the practice of presenting theoretical reasoning and may recognize the importance of this information for consumers of their research. Importantly, however, a significant number of studies relied on correlations

[2] Authors interested in comparing maternal depression and maternal BPD could do so through the recruitment of distinct groups with no need to statistically covary for depression.

amongst variables to explain covariate use, without describing the theoretical implications of the variables' inclusion. Further, nearly a third of papers which included analytic covariates did not provide an explanation for the inclusion of at least one variable in the model. In a fourth of these studies, no explanation was provided for *any* covariates in the statistical model. Given that the selection of covariates is inherently theoretical (Foster, 2010; Hall & Mast, 2009; Meehl, 1971), the lack of any theoretical rationale for their use in many studies obfuscates the degree to which authors appreciate the implications of their use. It is possible, and perhaps likely, that decisions to include covariates are being made on the basis of norms within a research group or research area (Rohrer, 2018). It is possible, further, that such norms are themselves predicated on theory. Without clear articulation of underlying theory or causal models, however, it is impossible to determine the extent to which this may be the case.

Recommendations

Given these findings, how should scholars working in this area best proceed? First, authors are encouraged to resist the temptation and trend to control for many variables without careful consideration of the influence this might have on the interpretability of results. Although the use of increasingly sophisticated statistical models enables authors to "correct" for several variables, and the desire to disentangle the true causal effect from spurious noise is admirable, doing so very likely has unintended and unanticipated consequences (Achen, 2005; Greenland et al., 1999; Hall & Mast, 2009; Rohrer, 2018). These consequences include invocation of implausible causal models (Rohrer, 2018; Foster, 2010), covarying away the construct of interest (Greenland et al., 1999; Rohrer, 2018), and the introduction of spurious relationships (Achen, 2005;

Greenland et al., 1999; Rohrer, 2018). Further, authors may not achieve the control they desire through simple linear corrections, as the effect of confounding variables may follow other functional forms or may depend on (interact with) other variables (Rohrer, 2018). For example, the effect of children's age may be quadratic or may depend on child verbal ability. In such an instance, a single linear term only removes a portion of the effect associated with age. Thus, the assumption that more control is gained through inclusion of many covariates in linear models, although well-motivated, must be counterbalanced by clear, careful consideration of the consequences of and theoretical rationale for doing so.

A second and related recommendation is that the results of statistical models be reported with and without covariates. Such a practice helps make clear to the reader the sensitivity of presented effects to other variables. Reporting effects estimated when controlling for covariates provides useful information about the magnitude and behavior of those effects *given the unique set of covariates also employed* (Hall & Mast, 2009). By also reporting models without covariates included, authors communicate greater information about the sensitivity of these effects to particular covariate structures. Further, there are many plausible causal models of maternal BPD's influence on parenting and child development. The current state of widely varied application of covariates attests to scholars' lack of consensus in this area. Reporting results of statistical models with and without covariates included provides additional information regarding the pattern of correlations between variables which might plausibly play a role in the broader causal structure. In this way, this practice further supports the resolution of theoretical disputes with minimal additional effort and journal space.

Finally, authors are encouraged to use causal models to guide their selection of covariates and, when possible, to make these models and the associated assumptions explicit. A significant portion of studies published in this area do articulate theoretical (if not always causal) justification for covariate inclusion. Authors are encouraged to continue articulating theory and to broaden this articulation to include causal assumptions, particularly of the links between constructs of interest and variables which are being considered for inclusion as covariates. One relatively approachable and straightforward method for doing so is the use of DAGs (addressed in more depth above). It is unlikely most psychologists will develop comprehensive fluency with graphical causal models and the tenants on which they are based (and perhaps unreasonable to expect they would). Thankfully, such mastery is not necessary for authors to glean the utility of these models for informing covariate selection. Psychologists interested in leveraging these models in their own work may find the following two resources beneficial:

1) Rohrer (2018) provides a concise, approachable primer on the use of DAGs in psychological research. This work extends the ideas presented in the current work's brief introduction of DAGs to include consideration of more complex causal structures. Specifically, this work addresses important topics such as collider bias, the effect of statistically controlling for mediators, and "backdoor" causal paths introduced by statistical overcontrol, all of which are highly relevant to research on maternal BPD.
2) The statistical software R hosts multiple packages, such as the "daggity" package (Textor et al., 2016), which enable researchers to generate a causal structure based

on their own beliefs about the directional associations between variables. These packages, then, can produce recommendations about which variables ought to be controlled (included as covariates) and, perhaps more crucially, which should not, in order to obtain an accurate estimate of a plausible causal effect. The use of such packages has immense potential for clarifying and simplifying the vast decision space in which authors working to understand the effect of maternal BPD on parenting and child development are working.

Strengths and Limitations

The primary strength of the present review is methodological similarity to previous work. The current review was undertaken in close consultation with the lead author of a previous systematic review addressing parenting and child development in the context of maternal BPD (Eyden et al., 2016). This consultation was aimed at ensuring identical search strategy and criteria for study inclusion/exclusion, ensuring the present review examined the same literature as the original review. The present review seamlessly builds upon previous work elucidating substantive results in this field through the examination of important methodological and analytic practices. Thus, the present review aids in the interpretability of these findings by providing increased methodological and analytic context.

In addition, we are not aware of any similar work documenting patterns of covariate use in a highly focused substantive research area. Although many studies have addressed covariate use in various social science disciplines (for examples, see Achen, 2005; Foster, 2010; Rohrer, 2018; Yzerbyt et al., 2004), the present study is the first of its kind to systematically review covariate adjustment as manifest in research addressing a

distinct question. To structure this review, the present work integrated guiding principles from the domains of substantive psychological research, methodology, and philosophy of science. The resulting best practices recommendations are therefore predicated on this interdisciplinary perspective and a clear inventory of current practices in a focused research area. In this way, the present work advances research in this domain by providing the first tailored guidelines for covariate adjustment to scholars working to conduct rigorous research in the causally complex area of maternal BPD.

The present review is also marked by three primary limitations. First, this review did not assess unpublished work. The decision to not solicit unpublished manuscripts and conference proceedings represents a divergence from the methodology of Eyden and colleagues (2016) review and limits the generalizability of these results beyond published works. Given that the goal of this review was to assess covariate use in the published literature addressing parenting and child outcomes in the context of maternal BPD, unpublished manuscripts were deemed outside of the scope of this review. Published manuscripts are known to differ from unpublished work in many dimensions (Trespidi et al., 2011). It is possible that one such dimension is covariate use. Unpublished studies may have differed from published work with regard to number of covariates used, which covariates were included, and rationales provided for such inclusion or omission. Given the substantial range and heterogeneity of covariate use in the present review, it is unlikely that patterns not discussed in the present work would have been observed. Even so, it is possible that unpublished work is characterized by *more* systematic and theoretical use of covariates. The current results cannot be interpreted as representing such work.

Second, the present review did not assess quality of theoretical rationale for covariate inclusion. Although many studies reported that covariates were included in statistical models for theoretical reasons, it is unclear the extent to which such rationales were connected to underlying causal theories. The data extraction form did not include a free-response space for indicating quality of rationale, nor was a systematic assessment for rationale quality developed. Though it would be informative to examine differences in covariate use between studies which provided cursory rationales and those which provided highly articulated, theoretically grounded rationales, the absence of a rationale quality assessment precludes such examination.

Finally, the present review did not code the statistical manner in which covariates were controlled. As noted previously, inclusion of a covariate as a linear term in a multivariate model assumes a linear relationship between the covariate and the outcome. Should this relationship not, in fact, be linear, but follow some other functional form, inclusion of a linear term would insufficiently control for this covariate's effect (Rohrer, 2018; Meehl, 1971). Similarly, the effect of a covariate on the outcome may depend on a predictor or other covariate. In such instances, the inclusion of the covariate alone removes only a portion of its effect on the outcome. Thus, statistical treatment of covariates, like number and type of covariates employed, is another manner in which covariate use can vary between studies. The present review is unable to address the extent to which the maternal BPD literature is characterized by these more sophisticated treatment of covariates.

Conclusion

In sum, the present study provides a succinct inventory of the use of statistical covariates in studies examining maternal BPD, parenting, and child development. Use of statistical covariates appears to be increasing over time and the limited consistency of covariate patterns suggest disparate causal models, many of which are not articulated. Maternal characteristics, particularly maternal mental health symptoms, are the most common variables treated as covariates, raising concerns about the degree to which studies may be unintentionally suppressing effects of interest. Given this, authors are encouraged to consider using fewer covariates, to present models with and without covariate adjustment, and to explicate their causal models through directed acyclic graphs. Such steps will increase the utility of published results and support generative theoretical debate through increased clarity.

CHAPTER III

BORDERLINE PERSONALITY DISORDER, CHILDREN'S EXECUTIVE FUNCTION AND THEORY OF MIND, AND PSYCHOSOCIAL RISK

Epidemiological research indicates that children whose parents have a mental disorder are at elevated risk of developing a mental disorder themselves (McLaughlin et al., 2012). Further, transmission of disorders from parents to children are not necessarily yoked together (i.e. that a child is more likely to have bipolar if his/her parent had bipolar), highlighting the importance of attending to transdiagnostic features of mental disorders rather than discrete diagnostic categories (e.g. Major Depressive Disorder, Generalized Anxiety Disorder). Transdiagnostic features are atypical or risk-conferring processes which are common across multiple disorders (Mansell et al., 2008). When considering the transmission of psychopathology from parents to children, there is a growing emphasis on studying transdiagnostic features rather than diagnoses.

To date, research examining the transmission of transdiagnostic features has focused predominantly on emotion regulation (Binion & Zalewski, 2017; Rogers et al., 2016; Suveg et al., 2011). Receiving relatively less empirical study in the transmission of transdiagnostic risk are Executive Functioning (EF) and Theory of Mind (ToM). EF is broadly defined as a set of cognitive control processes (e.g. response inhibition, working memory, attentional shifting) which enable the regulation of "lower level" processes (e.g. motor activity) and thereby enable self-regulation (Snyder et al., 2015). EF develops rapidly during the preschool period (Garon et al., 2008; Zelazo et al., 2003) with gains resulting in increased, observable self-regulatory behavior (Hughes et al., 1998). ToM is broadly defined as the ability to understand the diverse mental states of others (Goldman,

2012) and is implicated in social functioning (Hughes & Leekam, 2004; Slaughter et al., 2015). As with EF, the preschool period is marked by rapid growth in ToM abilities such that by the early school years many children understand sophisticated concepts such as possible discrepancies between emotional experience and emotional expression (Hughes & Leekam, 2004).

Research has elucidated factors which increase risk for atypical development in EF and ToM (Pears & Fisher, 2005; Sarsour et al., 2011). Among these factors is parental mental health symptoms (Hughes et al., 2013; Rhoades et al., 2011; Zalewski et al., 2019). Given this, it is possible that EF and ToM deficits are a mechanism by which psychopathology in parents increases risk for psychopathology in children. The present study aims to examine the association between mothers' and preschoolers' EF and ToM in a sample of mothers who have psychopathology. First, evidence regarding EF and ToM as related to psychopathology in childhood is reviewed and then this evidence as related to psychopathology in adulthood is presented.

Childhood EF, ToM, Psychopathology, and Social Competence

There is strong evidence for an association between deficits in EF and a broad range of mental disorders, behavior problems, and poor social competence in childhood (Calkins & Keane, 2009). Concurrently, lower EF abilities in childhood are strongly associated with antisocial behaviors (Morgan & Lilienfeld, 2000) and symptoms of Attention Deficit Hyperactivity Disorder (Willcutt et al., 2005). In a large-scale study of over 2,000 Brazillian children (ages 6-12), EF abilities were concurrently associated with children's symptoms of psychopathology broadly (Martel et al., 2017). A meta-analysis of 22 studies of preschool children found a medium association (ESz_r = 0.22) between

preschoolers' EF and externalizing behavior problems (e.g. hyperactivity, aggression) and found that this effect was stronger for older compared to younger children (Schoemaker et al., 2013). Beyond concurrent effects, longitudinal evidence suggests that preschoolers' EF abilities (effortful control and delay ability) are positively related to social competence (cooperation, sensitive responding to others' emotions, forming and fostering peer relationships) and negatively related to externalizing behavior problems during the early school years (Lengua et al., 2015). EF deficits are also negatively related to children's emerging social competence both concurrently (Riggs et al., 2006) and longitudinally (Nigg et al., 1999).

A substantive body of research spanning several decades has demonstrated ToM deficits in children (and adults) with Autism Spectrum Disorders (see Baron-Cohen, 2000 for a comprehensive review). By contrast, evidence linking ToM deficits in neurotypical children with mental health disorders is less well developed. Regarding concurrent associations, a recent study provided evidence that preschoolers with Oppositional Defiant Disorder (ODD) exhibit atypical response patterns to emotion-related ToM items (de la Osa et al., 2016). In older children and adolescents, ToM deficits have also been associated with bipolar disorder (Schenkel et al., 2008). ToM abilities have been associated with children's social competence (Liddle & Nettle, 2006) and a recent meta-analysis found that young children's (preschool and early school years) ToM was positively correlated with popularity with peers (Slaughter et al., 2015). Prospectively, evidence from a recent longitudinal study suggests that children's ToM abilities at age 3 are negatively correlated with children's externalizing behaviors at ages 5-6 (Olson et al., 2017).

Crucially, in both literatures, longitudinal evidence links deficits in these processes to *later* mental health symptoms, behavioral difficulties, and social competence. These findings provide support for the role of EF and ToM deficits as conferring risk for later psychopathology. Further, the diverse nature of adverse outcomes associated with EF and ToM deficits provides support for the transdiagnostic nature of such risk.

Adult EF, ToM, Psychopathology, and Social Competence

There is a robust literature on EF deficits and psychopathology in adults (Snyder et al., 2015). Regarding mood disorders, meta-analyses have found evidence of moderate associations between EF deficits and both Major Depressive Disorder (Rock et al., 2013; Snyder, 2013) and Bipolar Disorder (Mann-Wrobel et al., 2011), even when correcting for publication bias. For Obsessive Compulsive Disorder, a meta-analysis revealed the diagnosis was associated with three separate domains of EF: planning (d = -0.44), response inhibition (d = -0.49), and cognitive flexibility (d = -0.52) (Abramovitch et al., 2013). A large meta-analysis examining the performance of individuals with Attention Deficit Hyperactivity Disorder on several measures of EF found significant meta-analytic effect size estimates ranging from Cohen's d of -0.29 to -0.72. Together, these studies provide strong evidence of impaired EF abilities in adults with both internalizing and externalizing disorders.

A significant portion of literature exploring ToM deficits and psychopathology in adulthood has examined schizophrenia (Brüne & Brüne-Cohrs, 2004). Although theories regarding the origins of deficits differ, there is strong evidence of ToM deficits in individuals with schizophrenia (Frith, 2004; Langdon et al., 2001; Pickup & Frith, 2001)

which may persist following symptom remission (Inoue et al., 2006). Beyond schizophrenia, evidence suggests that ToM deficits are pronounced in individuals with Bipolar Disorder (Bora et al., 2016) and that these deficits may also persist after depressive and manic episodes have remitted (Inoue et al., 2004).

EF and ToM in Borderline Personality Disorder

One particularly impairing form of psychopathology marked by alterations and/or deficits in EF and ToM is Borderline Personality Disorder (BPD). BPD is characterized by substantial difficulty with emotion regulation, instability of self-image, tumultuous interpersonal relationships, and marked impulsivity (Lieb et al., 2004). Alterations in EF and ToM are theorized to underlie many of these interpersonal and self-regulatory challenges and in recent years focus has shifted to elucidating the extent and specificity of these EF and ToM impairments. Evidence has accumulated for BPD-related deficits in several aspects of EF, including attentional shifting and mental flexibility (Lenzenweger et al., 2004), working memory (Stevens et al., 2004; Krause-Utz et al., 2012), and inhibitory control (Nigg et al., 2005).

The relationship between BPD and alterations in ToM has been somewhat less consistent. A small number of studies have found no evidence of deficits (Arntz et al., 2009) and at least one has found evidence of superior ToM abilities (Fertuck et al., 2009) in individuals with BPD. More commonly, however, studies have differed in indicating the *specificity* of impairments, with some indicating general impairments (Bouchard et al., 2010) and others suggesting these impairments are limited to affective components of ToM (Harari et al., 2010). Recently, evidence has begun to emerge suggesting that observed deficits are better explained by *excessive* attention to and interpretation of cues

used in mental state inference (Sharp et al., 2011; Sharp et al., 2013). This heightened attention is posited to produce a specific type of ToM deficit – excessive inference about the internal states of others – sometimes referred to as *hypermentalizing* (Sharp, 2014).

Present Study Aims

Given the evidence of atypical and/or impaired EF and ToM in individuals with BPD, it is likely that preschoolers reared in this context face significant challenges in their own EF and ToM development owing both to genetic (Friedman et al., 2008; Hughes & Cutting, 1999) and environmental factors (Bernier et al., 2012; Hughes et al., 2005). A preponderance of literature has established the high risk for psychopathology experienced by offspring of mothers who have a diagnosis of BPD (Eyden et al., 2016) and it is possible that disruptions in EF and ToM development may be mechanistic in conferring this risk, given that deficits in these processes are understood to confer risk for psychopathology. A recent study examining maternal BPD and preschooler psychosocial development found evidence for deficits in preschoolers' EF and emotion-related facets of ToM (Zalewski et al., 2019), providing partial support for such a mechanistic model. The present study builds upon this work by Zalewski and colleagues by seeking to replicate findings in a larger, more diverse sample of mother-preschooler dyads recruited for over-representation of maternal symptoms of BPD. Specifically, we hypothesize that: 1a) maternal symptoms of BPD will be associated with lower child EF, 1b) maternal symptoms of BPD will be associated with lower child ToM, 2a) lower child EF will be associated with increased symptoms of psychopathology, 2b) lower child ToM will be associated with increased symptoms of psychopathology, 3a) lower child EF will be

associated with poorer social competence, and 3b) lower child ToM will be associated with poorer social competence.

Method

Participants

Participants in the present study are 144 mothers and their preschool children (aged 36-48 months at time of enrollment) participating in a larger, two-site study (R01-MH111758-01) examining the relation between maternal emotion dysregulation and child development for which data collection is ongoing. The goal of the larger study is to recruit two-thirds of mothers on the basis of mothers' elevated symptoms of BPD and the remaining mothers on the basis of having no mental health disorders since conception of their child (non-disordered control group). Participants were recruited in Lane County, Oregon and Pittsburgh, Pennsylvania in efforts to maximize sample size and diversity. Participants for the present study are those who completed an initial (baseline) assessment by January 1st, 2020. Roughly equal numbers of participants in the present sample were enrolled at each site. Overall, mothers ranged in age from 22-47 years (M = 32.05, SD = 4.96) and children ranged in age from 37-51 months[3] (M = 42.13, SD = 4.41) at the time of assessment. Roughly equal numbers of boys and girls participated (51%, n = 73 girls). The racial/ethnic composition of mothers included 72% European American or White, 15% African American, 2% Latino or Hispanic, and 11% multiple racial/ethnic backgrounds. A small minority of the sample reported they had not completed high school (2%), 10% reported they had completed high school, 36% reported they had completed some college (or had earned a terminal associates degree), 30% reported they

[3] Families were engaged in recruitment when children were 36-48 months. Due to rescheduling efforts, some baseline assessments were delayed and 9 children were aged 49-51 months at the time of assessment.

had earned a 4-year college degree, and 22% reported having earned or studied for a post-graduate degree. Totally family income was assessed categorically. Twenty-five percent of the sample reported a total family income of $22,000 or less, 26% of the sample reported a total family income between $22,000 and $53,000, 22% of the sample reported a total family income between $53,000 and $76,000, and 26% reported a total family income above $76,000. Income data was missing for one dyad.

Over three-quarters of mothers with elevated BPD symptoms reported a total family income of less than $53,000 per year, with nearly half reporting an annual income of less than $22,000 (Table 2). By contrast, over three-quarters of mothers in the Non-Disordered group reported a total family income greater than $53,000, with over one third reporting an annual income greater than $76,000.

Table 2. Number of participants in each income category in the BPD and Non-Disordered groups

	BPD (n = 71)	**Non-Disordered (n = 72)**
Less than $22,000	31	5
$22,000 - $53,000	26	11
$53,000 - $76,000	4	28
Greater than $76,000	10	28

Note: Income data missing for one mother in the BPD group

Procedure

Recruitment, Screening, and Eligibility

Given the need to recruit both disordered and non-disordered mothers, multiple recruitment methods were used. Across sites, participants were recruited from developmental databases, local community mental health clinics, social media postings, and letters mailed to families receiving social services (e.g. Early Head Start, Department

of Human Services). Interested mothers first completed a detailed phone screen to verify eligibility to participate in a more thorough clinical intake where final study eligibility was determined. To be eligible for clinical intake, all mothers must be at least 18 years of age, have at least partial custody of a 36-48-month-old child, and report that the child has no known developmental disabilities. Further, mothers had to meet either BPD or non-disordered control group criteria on the McLean Screening Instrument (Zanarini et al., 2003). To be eligible for intake in the BPD group, mothers had to endorse 7 or more symptoms while mothers eligible for the non-disordered group had to endorse 2 or fewer less symptoms.

Following initial screening, eligible mothers then completed a clinical intake to assess their mental health symptoms more thoroughly. Mothers were assessed by trained clinicians using the Structured Clinical Interview for the DSM-5 (SCID-5; First et al., 2015) and the Structured Interview for the DSM-IV Personality (SIDP-IV; Pfolh et al., 1995). Additionally, mothers' verbal ability was assessed using the Peabody Picture Vocabulary Test, Fourth Edition (PPVT-IV; Dunn & Dunn, 2007). Mothers who were assessed as having no mental health diagnoses since conception of their child were eligible to participate as part of the non-disordered group. Mothers who were assessed as having 3 or more symptoms of BPD on the SID-P, at least one of which must be affective instability or uncontrollable anger, were eligible to participate as part of the BPD group. For both groups, mothers were ineligible if they had a PPVT score of less than 70, endorsed an active suicide plan, and/or were actively psychotic or manic. Eligible mothers and their child were then scheduled to complete an initial assessment within 4 weeks of their clinical intake.

Assessment

Dyads were assessed in offices on university campuses at the University of Oregon and the University of Pittsburgh. With approval by each institution's Institutional Review Board, both mother's consent and child assent were secured prior to participating in a 2.5-hour session. During this session, children completed behavioral assessments while mothers completed questionnaires in an adjacent room. Next, mothers and children were reunited for parent-child interactions. Families receive $40 for this laboratory visit. Enrolled dyads completed 4 such assessments over the course of one year. Data for the present study were drawn from the clinical interview and baseline assessments only.

Measures

Maternal BPD Symptoms

The Structured Interview for DSM-IV Personality (SIDP-IV; Pfolh et al., 1995) was used to assess mothers' symptoms of BPD. The SIDP is a structured clinical interview which assesses the presence or absence of symptoms of ten personality disorders within the last 5 years. In the present study, seven disorders were assessed – Paranoid, Schizotypal, Narcissistic, Antisocial, Borderline, Avoidant, and Obsessive Compulsive. The SIDP was further modified for this study to assess the presence or absence of symptoms since the conception of the target child to be enrolled in the study as opposed to lifetime incidence. Symptoms of each disorder are rated by trained clinicians on a scale from 0-3 in which 0 indicates the symptom is "Absent", 1 indicates a symptom is present at a subthreshold level, 2 indicates a symptom is "Present," and 3 indicates a symptom is "Strongly Present." Mothers were classified as having elevated symptoms of BPD if they were rated as having at least 3 symptoms of BPD present or

strongly present, at least one of which must be affective instability or uncontrollable anger. Mothers were classified as being non-disordered if no symptoms of BPD were rated as present, they received a score of 0 (not present) for both the affective instability and uncontrollable anger items, and they were rated as having no other mental health diagnoses on the SCID-5 at clinical intake. In the present sample, 72 mothers were classified as having elevated symptoms of BPD and 72 were classified as non-disordered controls. Interrater reliability of group classification was assessed on the basis of 8% of files, with results indicating perfect agreement.

Preschooler EF

Preschoolers' EF abilities were assessed with three tasks, each tapping a different domain. Tasks were selected for their capacity to capture children's growth of EF abilities over the year-long study period. Thus, children's scores on these measures at baseline assessment were expected to be somewhat low. Further, in order to better depict this growth, raw scores on each task were converted to proportion scores. Proportion scores additionally facilitate the generation of a composite EF score. Thus, the use of proportion and composite scores in the present work enables straightforward comparison to future works modeling EF growth over time.

Day/Night Task. Children's ability to inhibit a prepotent verbal response (cognitive inhibitory control) was assessed with the Day/Night Task (Gerstadt et al. 1994). In this task, children are shown one of two cards – a black card depicting a moon and stars or a white card depicting a sun. Children are instructed to say "day" when shown the black card and to say "night" when shown the white card. Successful task performance requires children to inhibit the prepotent response to the image on the cards.

Children then complete 16 trials and their responses are recorded. Incorrect responses of "day" or "night" are not corrected; however, children are prompted ("In this game we say day or night") if they produce an off-task response (e.g. "Sunny!"). To facilitate the generation of an EF aggregate score, proportion scores were generated and are the primary unit of analyses. Total correct responses, therefore, are the proportion of correct responses out of 16 trials. Proportion correct ranged from 0-1 (M = 0.45, SD = 0.31). Twenty percent of files were double scored for reliability. Resulting intraclass correlation (ICC = 0.99) suggests excellent agreement.

Bear/Dragon Task. Children's ability to inhibit a prepotent motor response (behavioral inhibitory control) was assessed with the Bear/Dragon Task (Kochanska et al., 1996). In this task, which is similar to "Simon Says," children are instructed to heed the instructions of a bear puppet and to ignore the instructions of a dragon puppet. The puppets alternate in commanding the child to engage in motoric responses (e.g. "touch your nose") and children's behavioral responses are scored on a scale from 0 – 3 indicating the degree to which the rule (obey/don't obey) was followed. A total of 10 commands are given, and children are reminded of the rules ("Remember, in this game we listen to the nice bear, but we don't listen to the naughty dragon") between administration of the first five and final five items. For each dragon command (which require children to inhibit a behavioral response), children receive 3 points for not moving, 2 points for an incorrect movement, 1 point for a partial command movement, and 0 points for a full response. For each bear command (which require children to execute a behavioral response), children receive 3 points for a full response, 2 points for a partial response, 1 point for a wrong movement, and 0 points for no movement. Response

scores were summed across bear and dragon trials. Total scores are the proportion of the summed score out of the total possible score (30). Proportion correct ranged from 0-1 (*M* = 0.72, *SD* = 0.24). Twenty percent of files were double scored for reliability. The resulting estimate (ICC = 0.98) suggests excellent agreement.

Dimensional Change Card Sorting Task. Children's cognitive flexibility was assessed using the Dimensional Change Card Sorting Task (Zelazo, 2006). In this task, children are asked to sort cards by competing properties and to switch which property is used to sort the cards. Children are first shown cards with either a red or blue background which display either a truck or a star (all shapes are solid black) and are then asked to sort the cards based on shape displayed. Children are asked to do this for six trials and the sorting rule (e.g. "if it's a truck put it here but if it's a star put it here) is reiterated between each trial. Then, children are asked to disregard the previous sorting rule (shape) and to sort cards instead by card color (red or blue). This is again repeated for six trials, with the new sorting rule repeated between each trial. Next, children are shown a new set of cards with white backgrounds displaying solidly colored (either red or blue) shapes (either trucks or stars). Children are then asked to sort by shape for six trials as before and then by color for six trials, with the appropriate sorting rule repeated between each trial. Finally, children are shown a new set of white cards with colored shapes (as before) in which some cards have borders and some cards do not have borders. Children are instructed to sort by shape when a card does *not* have a border and to sort by color when the card *does* have a border. Children are then presented with 12 trials in which they must flexibly sort cards based on these properties, with the sorting rule again repeated between trials. The task is discontinued after a child incorrectly sorts four or more cards in a set,

thus it is uncommon for children to complete all 36 task trials. Children are scored on number of items correctly completed (range 0-36) with higher scores indicating better EF abilities. A proportion score was computed as the number of correct items out of total possible correct items. Proportion scores ranged 0-1 (M = 0.47, SD = 0.25). Twenty percent of files were double scored for reliability. The resulting estimate (ICC = 0.93) suggests excellent agreement.

In keeping with common approaches to children's EF, preliminary steps to create a composite measure of EF abilities were undertaken (Lengua et al., 2015; Zalewski et al., 2019). Namely, we examined correlations between measures of EF (see Table 4 for correlations among child tasks, child outcome variables, and child age). Performance on Bear/Dragon and DCCS were moderately correlated. However, performance on Day/Night was not meaningfully correlated with performance on either Bear/Dragon or DCCS. Given this, Bear/Dragon and DCCS proportion scores were averaged to create a composite score. Composite scores ranged from 0.13 – 0.95 (M = 0.59, SD = 0.20). For the purpose of analyses predicting child EF performance, the EF composite and Day/Night were examined separately. For the purpose of analyses in which EF was treated as an independent variable, the EF composite and Day/Night were both included as predictors.

Preschooler ToM

Preschooler's ToM abilities were assessed with a scale of common ToM queries for children constructed by Wellman & Liu (2004) to reflect a range of complexity and difficulty. This seven-item scale assesses children's understanding of diverse desires, diverse beliefs, others' access to knowledge, contents false belief, location false belief,

belief-emotion reasoning, and real-apparent emotion reasoning. In each task, children are read a short vignette which is acted out with toy dolls and are asked to make inferences about how a character will behave or feel given that character's (false) beliefs, desires, or emotions. Tasks presented earlier in the battery (e.g. diverse desires, knowledge access) represent elements of ToM understood to be acquired earlier in development, while tasks at the end of the battery (e.g. real-apparent emotion) assess more advanced ToM abilities. Each task is scored 0 (incorrect) or 1 (correct) and more complex items include a control/comprehension question to ensure children are not responding at random. Scores on each task are then summed to yield a single continuous score (ranging 0-7), with higher scores representing more developed ToM abilities. Theory of mind scores ranged 0-7 (M = 2.30, SD = 1.38). Twenty percent of files were double scored for reliability. The resulting estimate (ICC = 0.99) suggests excellent agreement.

Social Competence

Preschoolers' social competence was assessed using the Preschool and Kindergarten Behavior Scales 2nd Edition (PKBS-2; Merrell, 2002). The PKBS-2 is a ratings scale for preschool- and kindergarten-aged (3-6 years) children's behavior comprised of two subscales – social skills (SS) and problem behaviors (PB). In the present study, mothers completed the SS subscale only (34 items), rating statements about the frequency of behaviors related to children's social cooperation, social interaction, and social independence on a 4-point scale ranging from "Never" (0) to "Often" (3). Higher scores on this measure indicate greater social skills. Social competence scores ranged 16-99 (M = 83.5, SD = 12.84).

Symptoms of Psychopathology

Children's problem behaviors and symptoms of psychopathology were assessed using the Child Behavior Checklist for Ages 1.5 -5 (CBCL/1.5-5; Achenbach & Edelbrock, 1983). The CBCL is a 99-item ratings scale for children's problem behaviors which provide subscales for both internalizing (e.g. anxious, sad) and externalizing (e.g. hyperactive, aggressive) behaviors. The scale consists of statements about children's behavior (e.g. "afraid to try new things") and mothers are asked to indicate whether this statement is Never True (0), Sometimes True (1), or Often True (2) of the child being assessed. Higher scores on this measure indicate greater problem behaviors and symptoms of psychopathology. In the present sample, total scores ranged 0- 144 (M = 32.52, SD = 25.55).

Measures Collected but Not Analyzed

The present study also assessed children's delay ability (Gift Delay; Kochanska et al., 1996) and verbal ability (PPVT-IV; Dunn & Dunn, 2007). Delay ability is often considered an element of children' EF (Carlson, 2005) and was collected with the goal of including scores in children's EF composite. Children's verbal ability is a characteristic known to influence children's performance on EF and ToM (Happé, 1995; Hughes & Ensor, 2005; Milligan et al., 2007). Given these associations, verbal ability was collected with the intent of including scores as a covariate predicting children's performance on EF and ToM measures. Unfortunately, data for these measures were unable to be accessed and processed in a timely fashion due to the SARS-CoV-2 global pandemic. Data on these important metrics is not included in the present study.

Data Analysis

Missing Data

Missing data occurred at non-ignorable levels in all EF tasks (7% - 16% missing). Missing data also occurred at somewhat lower levels for ToM (3% missing) and PKBS (4% missing). Missingness on child tasks occurred due to child refusal (0.5-10%), technical errors preventing video coding (5-6%), and experimenter error (0.5%). Missingness on PKBS occurred due to responses of "Prefer not to answer" on at least one item. Missing data on all measures was not related to child age, family income, or maternal BPD status. Missing data was treated with multiple imputation through chained equations using the *mice* package (van Buuren & Groothuis-Oudshoorn, 2011) in R version 3.6.3. Missing values were replaced via predictive mean matching using values of maternal education, family income, BPD status, child age, CBCL, PKBS, and all child tasks as predictors. Missing values for all measures were imputed at the level of total (as opposed to item) scores. The EF composite score was created based on imputed values.

Analytic Plan

To test hypothesis 1a, we conducted two parallel regressions in which maternal BPD status was regressed on the EF composite and Day/Night proportion scores, respectively. To test hypothesis 1b, maternal BPD status was regressed on ToM performance. To test hypothesis 2a, child EF composite and Day/Night proportion scores were regressed on CBCL total score. To test hypothesis 2b, child ToM scores were regressed on CBCL total score. To test hypothesis 3a, child EF composite and Day/Night proportion scores were regressed on PKBS total score. To test hypothesis 3b, child ToM scores were regressed on PKBS total score.

In analyses predicting child task performance, total family income and child age were included as covariates. Neither variable is theorized to be causal antecedents of BPD and child age is not theorized to be a causal descendent of BPD. Family income is theorized to be a causal descendent of BPD (and other life history factors), thus its inclusion in these models may lower estimates of BPD's association with child performance. In analyses predicting child psychosocial outcomes, total family income was included as a covariate. Total family income is theorized to be a causal antecedent of child symptoms of psychopathology as well as child EF and ToM abilities (Hackman et al., 2015; Hughes & Ensor, 2005). Given this, its inclusion in these models is expected to lower the estimates of the association between child tasks and ratings of children's behavior. For all models, results are presented as bivariate, unadjusted associations as well as with covariate adjustment to aid in the interpretability of covariance patterns.

Results

Descriptives

Prior to testing study hypotheses, group differences in key variables (ToM, EF, child outcomes, family income) were examined. Group means for all child characteristics are displayed in Table 3. Child age was roughly equal across groups. Children whose mothers were in the BPD group were rated as having greater symptoms of psychopathology and fewer social skills than children whose mothers were in the Non-Disordered group. Although children whose mothers were in the BPD group consistently performed worse on EF and ToM tasks, group differences were largest for cognitive flexibility.

Table 3. Means (standard deviations) of child variables in the BPD and Non-Disordered groups

	BPD (n = 72)	**Non-Disordered (n = 72)**
Child Age	42.55 (4.37)	41.71 (4.44)
CBCL	47.19 (27.9)	18.06 (10.51)
PKBS	77.94 (14.44)	88.94 (8.03)
Day/Night	0.44 (0.33)	0.47 (0.3)
Bear/Dragon	0.66 (0.22)	0.78 (0.24)
DCCS	0.37 (0.24)	0.56 (0.22)
ToM	2.18 (1.28)	2.41 (1.47)

Note: Day/Night, Bear/Dragon, and DCCS are proportion scores

Bivariate associations between continuous variables (children's performance on EF and ToM tasks, symptoms of psychopathology, social competence, and child age) are displayed in Table 4. Children's symptoms of psychopathology were strongly inversely related to social competence. Modest positive associations were observed between all child tasks, with the exception of children's cognitive inhibitory control, where correlations were small to negligible. Children's symptoms of psychopathology were moderately related to children's task performance on all measures except cognitive inhibitory control, such that better performance was associated with fewer symptoms of psychopathology. Children's social competence was also moderately associated with children's task performance on all measures except cognitive inhibitory control, however, this association was positive, such that greater social competence was associated with better task performance. Finally, child age was moderately related to child task performance on behavioral inhibitory control, cognitive flexibility, and theory of mind.

Table 4. Bivariate association among child variables

	1	2	3	4	5	6	7
1. Child Age	-	-.031	.003	.065**	.272**	.148**	.337**
2. CBCL		-	-.675**	-.024	-.304**	-.212**	-.191**
3. PKBS			-	.078**	.26**	.283**	.213**
4. Day/Night				-	-.034†	.101**	.106**
5. Bear/Dragon					-	.36**	.367**
6. DCCS						-	.381**
7. ToM							-

† p <.05, * p < .01, ** p < .001

Child EF & ToM

Children's composite EF abilities were significantly associated with maternal BPD status and child age in months, suggesting that children whose mothers have BPD perform worse on EF tasks and that older children perform slightly better on these tasks (Table 5). Children's cognitive inhibitory control was not significantly associated with maternal BPD status or child age, but was significantly associated with maternal income, such that family income between $53,000 and $76,000 was associated with better performance scores. Maternal BPD status was not significantly associated with children's ToM abilities (p = .06). ToM abilities were, however, significantly associated with children's age in months, suggesting older children performed better on ToM.

Child Outcomes

Higher composite EF was associated with fewer symptoms of psychopathology (Table 6). Children's symptoms of psychopathology were significantly associated with composite EF but not cognitive inhibitory control (p = .86), suggesting cognitive inhibitory control is a poor predictor of children's internalizing and externalizing symptoms. Children's ToM abilities were not significantly associated with symptoms of

psychopathology (p = .068), however, higher family income was significantly associated with lower CBCL scores (Table 7).

Table 5. Regressions predicting child task performance from maternal group status, total family income, and child age

Predictors	**Executive Function Composite**				
	R^2	**B**	**SE**	**95% CI**	
				Lower	**Upper**
	.267			.146	.399
Maternal BPD		-.139**	.034	-.207	-.072
Total Annual Income $22-53k		-.01	.041	-.091	0.071
Total Annual Income $53-76k		.047	.058	-.068	.162
Total Annual Income > $76k		.08	.046	-.01	.175
Child Age		.014**	.004	.007	.022
	Cognitive Inhibitory Control				
	.057			.004	.159
Maternal BPD		.017	.063	-.108	.141
Total Annual Income $22-53k		.036	.079	-.122	.194
Total Annual Income $53-76k		.25†	.111	.029	.471
Total Annual Income > $76k		.073	.09	-.105	.252
Child Age		.005	.007	-.009	.018
	Theory of Mind				
	.155			.054	-.396
Maternal BPD		-.156	.253	.285	1.301
Total Annual Income $22-53k		.34	.301	-.658	-.05
Total Annual Income $53-76k		.452	.429	.345	1.325
Total Annual Income > $76k		.638	.347	-.257	.059
Child Age		.114**	.028	.937	.17

† p <.05, * p < .01, ** p < .001

As with symptoms of psychopathology, children's social competence scores were significantly associated with EF composite but not cognitive inhibitory control (p = .56), suggesting higher EF composite scores are associated with greater social skills. Children's ToM abilities were also significantly associated with social competence, suggesting that children with greater ToM abilities are rated as having greater social skills.

Table 6. Regressions predicting child psychosocial outcomes from EF task performance and family income

Predictors	Symptoms of Psychopathology				
	R^2	B	SE	95% CI	
				Lower	Upper
	.142			.051	.262
EF Composite		-32.022*	10.807	-53.405	-10.638
Cognitive Inhibitory Control		1.214	7.121	-12.904	15.333
Total Annual Income $22-53k		-6.988	5.272	-17.417	3.442
Total Annual Income $53-76k		-13.569	7.721	-28.840	1.703
Total Annual Income > $76k		-14.693†	5.866	-26.296	-3.09
	Social Competence				
	.122			.037	.238
EF Composite		19.205**	5.454	8.412	29.997
Cognitive Inhibitory Control		2.157	3.686	-5.162	9.475
Total Annual Income $22-53k		0.920	2.671	-4.364	6.205
Total Annual Income $53-76k		2.365	3.917	-5.384	10.114
Total Annual Income > $76k		2.914	2.956	-2.934	8.763

† p <.05, * p < .01, ** p < .001

Table 7. Regressions predicting child psychosocial outcomes from ToM task performance and family income

Predictors	Symptoms of Psychopathology				
	R^2	B	SE	95% CI	
				Lower	Upper
	.107			.029	.218
Theory of Mind		-2.805	1.523	-5.818	0.209
Total Annual Income $22-53k		-7.173	5.342	-17.738	3.393
Total Annual Income $53-76k		-15.846†	7.592	-30.861	-0.831
Total Annual Income > $76k		-17.846*	5.787	-29.095	-6.206
	Social Competence				
	.07			.011	.17
Theory of Mind		1.765†	0.772	0.237	3.292
Total Annual Income $22-53k		1.112	2.736	-0.989	10.65
Total Annual Income $53-76k		4.397	3.86	-4.3	6.525
Total Annual Income > $76k		4.831	2.942	-3.236	12.03

† p <.05, * p < .01, ** p < .001

To contextualize the magnitude of these effects, exploratory regressions were conducted in which children's social competence and symptoms of psychopathology were separately regressed on maternal BPD status and family income (Table 8). Results suggest that maternal BPD status is significantly associated with both symptoms of psychopathology and social competence while family income was not associated with these outcomes.

Table 8. Regressions predicting child psychosocial outcomes from maternal group status and family income

Predictors	**Symptoms of Psychopathology**				
	R^2	**B**	**SE**	**95% CI**	
				Lower	**Upper**
	.333			.209	.458
Maternal BPD		28.734**	3.989	20.846	36.622
Total Annual Income $22-53k		2.63	4.818	-6.899	12.159
Total Annual Income $53-76k		-0.159	6.943	-13.889	13.571
Total Annual Income > $76k		-1.987	5.489	-12.842	8.868
	Social Competence				
	.07			.011	.17
Maternal BPD		-11.224**	2.217	-15.61	-6.839
Total Annual Income $22-53k		-2.468	2.682	-7.775	2.84
Total Annual Income $53-76k		-1.386	3.824	-8.95	6.177
Total Annual Income > $76k		-0.917	3.03	-6.911	5.076

† p <.05, * p < .01, ** p < .001

Discussion

The current study sought to examine the associations between maternal BPD and children's EF, ToM, symptoms of psychopathology, and social competence. Results suggest that maternal BPD is associated with children displaying poorer social skills and greater symptoms of psychopathology. Maternal BPD was also associated with poorer child EF abilities for some domains and was not associated with children's ToM abilities. Better ToM abilities are associated with greater social competence, but not with fewer

symptoms of psychopathology. By contrast, stronger EF abilities (composite) are associated with better outcomes in both domains. Surprisingly, cognitive inhibitory control was not meaningfully associated with maternal BPD, child symptoms of psychopathology, or child social skills. Together, these results suggest that children whose mothers have BPD have greater symptoms of psychopathology and poorer social competence and that EF abilities may be a mechanism by which this risk is conferred.

Consistent with previous literature, the present study observed a large association between maternal BPD and children's symptoms of psychopathology. These findings also replicate previous work suggesting that maternal BPD is associated with poorer EF abilities in preschool children (Mena et al., 2017; Zalewski et al., 2019). The effects observed in the present study were slightly larger than those observed in previous work (Zalewski et al., 2019), suggesting that children whose mothers have BPD fare worse on metrics of EF and ToM than children from low-income backgrounds (see Pears & Moses, 2003; Lengua et al., 2015). The present study observed these effects in a larger sample collected in two different geographic locations, which afforded more racial and ethnic diversity compared to the pilot work supporting the current study. Further, the present study observed these effects in a sample recruited based on a theoretically focused age range (3-4 years), lending support to the understanding that disruptions in transdiagnostic socioemotional and socio-cognitive processes emerge during the preschool period. The consistency of these effects in a more diverse, representative sample increases confidence in the robustness of these findings. In a departure from previous work (Schacht et al., 2013; Zalewski et al., 2019), the present study did not observe an association between maternal BPD and children's ToM abilities. This may result from measurement

differences, as previous work has reported associations between maternal BPD and children's affect-related ToM abilities (Zalewski et al., 2019). Although the present study utilized a ToM battery with affect-laden items, it did not include a separate measure of affect perspective taking or affect-related ToM. In addition, the ToM battery used in the present study may not have been sensitive enough to detect emerging rank order differences at this early developmental stage. Although ToM scores ranged widely, with some children obtaining perfect scores, over three-quarters of the present sample got fewer than half of the items correct, with 25% receiving a score of 0 or 1. Thus, it is possible that meaningful rank order differences in ToM abilities are not yet present or that the measure employed in the present study was not sensitive enough to detect these emerging differences. Given this, it is possible that the current study would have observed these effects if different measures were employed.

Beyond measurement differences, there are also theoretical reasons which might explain the lack of association between maternal BPD and children's ToM. First, it is possible that alterations in ToM may not be observable until later in development. Individuals with BPD have demonstrated a unique form of ToM alteration called *hypermentalizing* or excessive inference about the mental states of others (Preißler et al., 2010; Sharp & Vanwoerden, 2015). ToM abilities develop hierarchically, with more rudimentary abilities appearing earlier in development. Thus, it is possible that more foundational ToM skills are preserved in children whose mothers have BPD and that deficits (including hypermentalizing) are observable only in more advanced ToM domains. Second, the literature demonstrating alterations in ToM in individuals with BPD is inconsistent, suggesting true effects may be absent, sensitive to unknown

contextual factors, or variable within the population. The present study did not include an assessment of maternal ToM and thus it is unclear the extent to which maternal BPD was associated with alterations or deficits in ToM in the present sample. It is possible that no association is present, in which case an association between maternal BPD and children's ToM would not be expected. Alternatively, it is also possible that ToM abilities are variable within the BPD population. As with many other symptoms of BPD, there is substantial heterogeneity in estimates of social skills impairments in individuals with BPD (Wright et al., 2013). Thus, it is possible that variable ToM abilities may be related to variable symptom presentations. Given these possibilities, future research in this area should include direct assessments of ToM abilities in mothers who have a diagnosis of BPD.

The current study also extends previous research by examining the association between maternal BPD, children's EF, ToM, and symptoms of psychopathology with children's emerging social competence. Findings from the present study extend previous research in several novel ways. First, findings suggest that maternal BPD is associated not only with increased symptoms of psychopathology but with reduced social competence. To our knowledge, this is the first study demonstrating an association between maternal BPD and children's emerging social skills. Given known associations between deficits in early social competence and later life educational, occupational, and social functioning (Jones et al., 2015), it is possible that social competence represents another mechanism by which risk for poor outcomes is transmitted from mothers who have a diagnosis or symptoms of BPD to their children. Second, as noted previously, research has suggested that children's ToM and EF skills are associated with emerging

social competence (Liddle & Nettle, 2006; Riggs et al., 2006; Nigg et al., 1999). Findings of the present study bolster this literature by demonstrating these associations are present concurrently and in young preschool children. Such findings further highlight the non-independence of EF and ToM in shaping behavioral outcomes, even very early in development. Finally, the present study found a strong, negative association between social competence and symptoms of psychopathology. Such associations have been observed prospectively and concurrently in older children (Dennis et al., 2007), with some models suggesting a pathway from poor social competence to elevated internalizing and externalizing symptoms (Burt et al., 2008). The current study adds to this literature by observing concurrent associations between social competence and psychopathology in young preschool children, suggesting these effects may begin earlier in development than previously recognized.

Observed associations between maternal BPD, children's EF, and children's symptoms of psychopathology may be explained by heritability of trait-level impulsivity. Difficulty with impulse control is a hallmark feature of the diagnosis of BPD (Lieb et al., 2004) and individuals with BPD have been shown to perform poorly on behavioral measures of inhibitory control (Nigg et al., 2005). As previously mentioned, preschoolers' deficits in EF broadly are prospectively associated with emerging psychopathology (Lengua et al., 2015). Although EF and impulsivity are not the same construct and are considered substrates from distinct neurobiological processes, research suggests these domains are related, with impulsivity serving as an antipode for elements of EF (Bickel et al., 2012). Thus, it is possible that EF deficits, particularly in domains associated with behavioral inhibition, may signal elevated trait-level impulsivity. Further,

impulsivity has been shown to be heritable (Coccaro et al., 1993; Niv et al., 2012). Therefore, EF deficits in children of mothers who have a diagnosis or symptoms of BPD may in part be accounted for by genetic variations in impulsivity and that this impulsivity, in turn, accounts for the emergence of psychopathology. This genetic pathway may be bolstered by co-occurring environmental risk contexts. EF development is known to be compromised by low family income, low maternal educational attainment, and low-quality parenting interactions (low positivity, high intrusiveness) (Hughes & Ensor, 2005; Hughes & Ensor, 2009; Noble et al., 2007; Rhoades et al., 2011). Mothers who have a diagnosis or symptoms of BPD are at elevated risk for experiencing each of these risk contexts (Bagge et al., 2004; Eyden et al., 2016; Trull et al., 1997; Walsh et al., 2013). Thus, children whose mothers have BPD may be exceptionally at risk for poor EF outcomes owing to both genetic and environmental factors.

It is important to note that a core measure of children's EF abilities – Day/Night – was not associated as expected with other measures. Previous studies have demonstrated modest correlations between Day/Night and other EF measures (Lengua et al., 2015; Zalewski et al., 2019). By contrast, Day/Night did not meaningfully correlate with any other EF measure in the present sample. Further, scores on the Day/Night task were not meaningfully associated with children's age. Because a robust literature demonstrates that children's performance on EF measures improves with age (Zelazo et al., 2003) and because other measures of EF in the present sample were associated with age as expected, this finding is surprising. It is also noteworthy that rates of missing data were highest on Day/Night as compared to other measures. Although it is possible that low correlations may be due to uncertainty in imputations, associations between measures did

not meaningfully shift when only complete cases were analyzed, thus such an explanation is unlikely. It is possible that the nature of the Day/Night task interacted with task order effects and assessment fatigue to produce highly variable task performance. In contrast to other child tasks, which use colorful dolls or puppets, Day/Night relies on relatively simple pictures. Further, Day/Night was embedded near the midpoint of a much larger battery of tasks. Together, these factors may have amplified assessment fatigue and children's difficulty engaging in the task.

Strengths of this study include use of measures capable of capturing development of children's EF and ToM abilities. Although the present study did not employ a longitudinal design, it established cross-sectional associations between these constructs, laying the groundwork for future longitudinal examinations. Specifically, the present study employed well-validated behavioral assessments of children's EF and ToM which captured a broad range of performance on these tasks. Thus, this baseline assessment serves to establish expected initial associations using measures which might later be used to model growth. Further, the present study closely replicated previous work in this domain with a larger, more diverse sample, bolstering confidence in such findings. Studies of clinical phenomena often face challenges recruiting large samples. The present study sought to overcome these challenges by recruiting from two sites, enabling the recruitment of a sample both larger and more diverse than has often been examined in previous work. Finally, the present study recruited individuals who present with a high level of clinical severity, thus better representing the population of individuals presenting with BPD. Challenges recruiting sizable samples are often most pronounced in studies examining clinical BPD presentations as opposed to BPD features in community samples

(Eyden et al., 2016). Thus, the present work provides a valuable contribution through the examination of a relatively large and clinically severe sample.

Although design and sampling choices are among study strengths, some elements of these choices are also limitations. Specifically, data from the present study are not longitudinal and thus cannot rigorously test a mechanistic model by which maternal BPD results in poorer child outcomes via deficits in children's EF. Further, a rigorous test of such mechanisms necessitates the experimental manipulation of maternal BPD and/or children's EF, which this study was not able to do. Similarly, although previous work has demonstrated an association between BPD and poorer performance on EF tasks and variable performance on ToM tasks, this study did not directly assess mothers' EF or ToM. In addition, the present study did not assess children's working memory. Working memory is understood to be a component of EF and correlations with other EF tasks are evident in both childhood (Senn et al., 2004) and adulthood (McCabe et al., 2010). Even so, efforts to comprehensively assess EF abilities were necessarily balanced with concerns about the length of the task battery and possible associated decrements in task performance. Thus, the EF battery employed in the current study does not constitute a comprehensive assessment of these abilities. Finally, maternal income and BPD were both assessed categorically in the present study. Although categorical approaches aid in the comparison of qualitatively distinct groups, they are known to reduce statistical power (Altman & Royston, 2006). Further, categorical approaches may not well represent the underlying structure of maternal BPD. To advance research in this area, future work should employ longitudinal designs which include direct assessments of maternal EF and ToM abilities, comprehensive assessments of children's EF, and both categorical and

dimensional assessments of maternal BPD. In addition, future studies should consider the use of clinical trials to experimentally manipulate maternal BPD symptoms and/or children's EF to test the roles of EF and BPD in risk transmission more rigorously.

The present study is also limited by lack of inclusion of child verbal ability in statistical models. Child verbal ability has been shown to relate to performance on both EF and ToM tasks (Happé, 1995; Hughes & Ensor, 2005; Milligan et al., 2007). Inclusion of child verbal ability in these models would thus improve overall variance explained. As previously mentioned, inclusion of verbal ability in the present sample was thwarted due to data access issues related to the SARS-CoV-2 global pandemic. Future work should consider controlling for child verbal ability given its known associations with core constructs of interest.

In sum, the present study provides further support for the association between maternal BPD and children's atypical development in transdiagnostic socio-cognitive domains. Children of mothers who have a diagnosis or symptoms of BPD showed deficits in EF performance, elevated symptoms of psychopathology, and lower social skills relative to children of non-disordered mothers, even when controlling for family income and child age. Although maternal BPD was not significantly associated with children's ToM abilities, ToM abilities were associated with children's emerging social competence and EF abilities. These results provide initial support for a mechanistic model of risk transmission by which maternal BPD disrupts children's development of EF which in turn results in the emergence of psychopathology. These findings provide the necessary foundation for future research to more rigorously test such a model through the use of longitudinal designs and clinical trials.

CHAPTER IV

GENERAL DISCUSSION

This dissertation leveraged a systematic review and original data collection to advance research about children's development in the context of maternal BPD. Chapter II reviewed covariate adjustment in this literature, revealing a trend over time towards use of more covariates in statistical models. Apart from maternal depression, there was little consistency in which variables were treated as covariates and many studies did not provide theoretical rationales for the inclusion of covariates. Chapter III examined the cross-sectional associations between maternal BPD, children's EF and ToM, and children's social competence and symptoms of psychopathology. Results revealed that children whose mothers had BPD had elevated symptoms of psychopathology and poorer social competence than did children whose mothers had no mental health disorders. Further, children's EF abilities were negatively associated with both maternal BPD and psychosocial outcomes, suggesting disrupted EF abilities may be a mechanism conferring risk from mothers to children. Together, these results suggest that children whose mothers have a diagnosis or symptoms of BPD are at elevated risk for disruptions in key developmental processes related psychosocial outcomes. Understanding this risk, however, necessitates contention with many confounding risk contexts, challenges which might be partially addressed through careful use of covariate adjustment in the context of clear causal models.

As discussed in Chapter II, research addressing maternal BPD is growing, with nearly three-quarters of this literature published within the last 10 years. Research about this population is vital, as being raised by a mother with a diagnosis or symptoms of BPD

constitutes a high-risk context. As Chapter II illustrates, such research is challenged by the need to contend with multiple co-occurring risk factors. Chapter III contributes to this important literature by examining children's development of key transdiagnostic processes known to be compromised in individuals with BPD. The goal of understanding these processes is to test mechanistic models by which disruptions in these processes result in poor psychosocial outcomes. Although the contribution of Chapter III to elucidating mechanisms by which risk is conveyed is modest, it aims to draw from findings of Chapter II to lay a firm theoretical and methodological framework on which future research might build.

Future Directions

Given these findings, promising future directions for research in this area bear mentioning. These future directions are presented in two broad sections. First, future directions for the field of maternal BPD are discussed. Second, future directions as they pertain to my personal research program are presented.

Research Addressing Maternal BPD

First, future research should employ longitudinal and prospective designs to examine children's development of socio-emotional and socio-cognitive processes in the context of maternal BPD. The research in this dissertation adds to literature demonstrating that these processes are compromised in this population and that such deficits are concurrently associated with poor outcomes. Although deficits in these domains have been longitudinally associated with poor psychosocial outcomes in other at-risk and normative populations (Lengua et al., 2015; Nigg et al., 1999; Olson et al., 2017), no work has yet established such a prospective link in children whose mothers

have BPD. Further, we are not aware of any research with this population examining the co-development of socio-emotional and socio-cognitive processes over time. Although previous research has demonstrated the interdependence of these processes (Carlson & Moses, 2001; Devine & Hughes, 2014; Zelazo & Cunningham, 2007), their interplay in this high-risk context is not yet understood. Longitudinal and prospective designs can thus substantially advance our understanding of children's development in this context by further elucidating potential risk-conferring mechanisms as well as the developmental interplay between such mechanisms.

Second, future research in this area should consider employing experimental paradigms to more rigorously test mechanisms by which risk is conferred from mothers to children. Although naturalistic longitudinal examinations of potential risk mechanisms are immensely informative, such designs cannot test the extent to which theorized processes are truly mechanistic. To do so, experimental manipulation of such processes is needed. Clinical trials represent powerful tools for such work, as a variety of targeted interventions can be used to experimentally manipulate theorized mechanisms. Such designs thus not only provide a rigorous test of theory but also inform the extent to which such mechanisms are modifiable. In this way, experimental designs further advance this research area through the identification of malleable processes which might be targeted for the prevention or reduction or risk to these vulnerable dyads.

Finally, future research should continue to critically integrate theory with approaches to inference. The research in this dissertation elucidates the challenges associated with covariate adjustment in the complex risk context of maternal BPD. Covariate adjustment can be potent for overcoming challenges associated with

observational research and the practice remains an effective tool in longitudinal and experimental designs as well. It should be noted, however, that increased use of these more complex designs necessitates increased complexity and specificity of underlying theoretical causal models. Thus, future research should continue to carefully employ covariates for causal inference and to support such use with clearly articulated causal frameworks. To this end, future research should also continue to conduct work which examines the degree to which current methodological practices reflect underlying theory to ensure tests of such causal models are rigorous and their results accurately interpreted.

Personal Research Program

It is my aim that a portion of the above described research be my own. Given that the dissertation is an academic milestone signifying the preparedness of a scholar to conduct independent research, discussion of future directions for my own research program is warranted. Broadly, I aim to examine substantive and methodological questions related to the role of transdiagnostic socio-cognitive processes in the emergence of psychopathology following exposure to early life adversity.

In the immediate future, I plan to examine the longitudinal co-development of EF and ToM abilities in children whose mothers have BPD. This work will elucidate the contribution of growth in one domain to growth in the other domain. Further, this work will examine the degree to which these growth patterns are related to later social competence and symptoms of psychopathology. Further, I plan to leverage the on-going clinical trial of mothers who have elevated symptoms of BPD from which my dissertation's study data was drawn to rigorously test the role of EF in conferring risk for psychopathology. This work will examine the extent to which reductions in maternal

BPD symptoms are associated with restored normative growth patterns in children's EF abilities as well as the extent to which such growth is associated with reduced risk for psychopathology. Finally, I plan to examine the extent to which maternal characteristics affect the convergence of measurement methods. This work will assess the correspondence of self-report and behavioral indices of parenting practices and assess the degree to which maternal emotion dysregulation is associated with the magnitude of this correspondence.

Beyond these works, my research program will continue to develop along two lines. The first line of research will interrogate the role of social competence and the successful recruitment of social support as potential mitigators of the association of early life adversity with the emergence of later psychopathology. This research will continue to explore EF and ToM, as these skills underpin social competence, and will broaden risk contexts to include other forms of parental psychopathology. The second line of research will explore design, measurement, and analytic methods which have the potential to advance the substantive research described above. Specifically, this research will address the measurement of constructs which qualitatively change across development (e.g. ToM, emotion regulation, social competence) with particular attention to methods which might advance the longitudinal study of these processes, such as planned missingness designs and vertical equations. Further, this line of research will examine the behavior of cornerstone measures across populations and settings to inform underlying theories about both these measures and the populations examined.

APPENDIX

DATA EXTRACTION FORM

1. List all author last names as they appear in the paper

   E.g. Gratz, Kiel, & Roemer

2. Year in which the study was published

3. Offspring age group (e.g. 8-11 years, 12-24 months)

4. Country in which the study was conducted

○ US ○ Canada ○ UK ○ France ○ Germany ○ Australia ○ China ○ Other

5. The study presents the racial/ethnic breakdown of which members of the sample:

☐ Children ☐ Mothers ☐ Overall ☐ None of the above

9. Select all recruitment/inclusion criteria described

☐ None reported ☐ Parent meets diagnostic criteria for BPD ☐ Parent has elevated symptoms of BPD ☐ Parent meets diagnostic criteria for other mental disorder ☐ Parent has elevated symptoms of other mental disorder ☐ Parent of a child of a certain age ☐ Income criteria ☐ Primary/sole custody ☐ Intelligence criteria ☐ English-speaking ☐ Child has no known developmental disabilities ☐ Parent & Child are biological relatives ☐ Other

10. Select all exclusion criteria described

    If exclusion criteria are the exact inverse as inclusion criteria, do not report here. Only report additional criteria.

☐ None reported ☐ Parent does not have primary/sole custody ☐ Parent meets criteria for other mental disorder ☐ Child has known developmental disabilities ☐ Intelligence criteria ☐ Severe/active suicidality ☐ Psychotic symptoms ☐ Parent & child are not biologically related ☐ Other

11. Study design

12. Is this sample referred to by a proper name/title (e.g. The Pittsburgh Girls Study or The Griefswald Family Study) and/or does the paper state that the sample has been analyzed elsewhere?

○ Yes ○ No **Clear Response**

14. Please select all methods used to measure maternal symptoms:

☐ Self-report (mother) ☐ Self-report (child) ☐ Maternal report of child behavior ☐ Other caregiver/teacher report of child behavior ☐ Standardized interview ☐ Non-standardized interview ☐ Physiological measure ☐ Dyadic discussion task ☐ Dyadic interaction task ☐ Standardized lab task ☐ Unstandardized lab observation ☐ Other ____

15. Please select all methods used to measure parental behavior/parenting (if applicable):

☐ Self-report (mother) ☐ Self-report (child) ☐ Maternal report of child behavior ☐ Other caregiver/teacher report of child behavior ☐ Standardized Interview ☐ Non-standardized interview ☐ Physiological measure ☐ Dyadic discussion task ☐ Dyadic interaction task ☐ Standardized lab task ☐ Unstandardized lab observation ☐ Other ____

16. Please select all methods used to measure child outcomes/child behavior (if applicable):

☐ Self-report (mother) ☐ Self-report (child) ☐ Maternal report of child behavior ☐ Other caregiver/teacher report of child behavior ☐ Standardized interview ☐ Non-standardized interview ☐ Physiological measure ☐ Dyadic discussion task ☐ Dyadic interaction task ☐ Standardized lab task ☐ Unstandardized lab observation ☐ Other ____

17. Child outcome (if applicable)

This is the child outcome examined in the *FOCAL ANALYSIS*. The abstract & paragraph introducing hypotheses are a good place to look to determine what analysis/question is focal to the study. One analysis may test multiple hypotheses (e.g. a mediation analysis may test both a direct and indirect effect).

☐ None reported ☐ Executive function ☐ Theory of mind ☐ Emotion regulation ☐ Temperament ☐ Impulsivity ☐ Attachment ☐ Social competence ☐ Substance use ☐ Internalizing symptoms ☐ Externalizing symptoms ☐ Symptoms of psychopathology (undifferentiated OR both internalizing &

externalizing) ☐ Specific mental disorder (note which) ☐ Other

18. Parenting (if applicable)

    This is the aspect of parenting or parenting variable examined in the *FOCAL ANALYSIS*. The abstract & paragraph introducing hypotheses are a good place to look to determine what analysis/question is focal to the study. One analysis may test multiple hypotheses (e.g. a mediation analysis may test both a direct and indirect effect).

    Please select the variable as reported/framed in the paper. For example, control/laxness may be on a spectrum. Please select the one(s) that the paper states it is examining.

☐ None Reported ☐ Psychological Control ☐ Behavioral Control ☐ Warmth/Sensitivity ☐ Harshness/Hostility ☐ Autonomy Support ☐ Insensitivity ☐ Disrupted Affective Communication ☐ Laxness ☐ Over-reactivity ☐ Expressed Positive Affect During Parenting Interaction ☐ Expressed Negative Affect During Parenting Interaction ☐ Emotion Recognition (of offspring emotion) ☐ Family Environment ☐ Other (Describe)

19. Mother-related covariates included in focal analysis

    One analysis/test may test multiple hypotheses (e.g. a mediation analysis may test both a direct and indirect effect). Covariate selection may be uniform across analyses OR it may vary. Please carefully ensure you are selecting only those covariates included in the ***focal analysis***.

☐ None ☐ Depression Symptoms ☐ Anxiety Symptoms ☐ Trauma Symptoms ☐ Trauma History ☐ Maternal Age ☐ Maternal Education ☐ Maternal Employment Status ☐ Maternal Substance Use/Substance Use Severity ☐ Other (describe)

20. Child-related covariates included in focal analysis

    One analysis/test may test multiple hypotheses (e.g. a mediation analysis may test both a direct and indirect effect). Covariate selection may be uniform across analyses OR it may vary. Please carefully ensure you are selecting only those covariates included in the ***focal analysis***.

☐ None ☐ Child Age ☐ Child Gender ☐ Other (describe)

21. Family/environment/demographic/other covariates included in focal analysis

One analysis/test may test multiple hypotheses (e.g. a mediation analysis may test both a direct and indirect effect). Covariate selection may be uniform across analyses OR it may vary. Please carefully ensure you are selecting only those covariates included in the ***focal analysis***.

☐ None ☐ Number of Siblings ☐ Family or Focal Parent Income/SES ☐ Both Parents In Home ☐ Other (describe) _____

22. Justification for covariate inclusion

Please select all that were used to justify covariate inclusions in the *focal analysis*. For each justification you select, please list the covariates associated with this justification in the box provided.

☐ None Provided _____

☐ Correlations In Current Data _____

☐ Theoretical (post-hoc; e.g. added after looking at data) _____

☐ Theoretical (a priori) _____

☐ Field-Norm (e.g. cites previous article(s) indicating this is normative/typical) _____

23. Total number of covariates included in focal analysis

_____

24. Is/are the covariate(s) reported in the final model?

○ Yes ○ No ○ Not reported **Clear Response**

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