Parent Depression & Child Mental Health Problems in Families with Autistic Children: Examining Potential Mediators

By

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Dedication

For Chad, Mariah, and Avery. Chad, your unwavering support and constant encouragement helped guide me to the finish line. The countless dishes you washed and dinners you cooked have not gone unnoticed or unappreciated. Mariah and Avery, I hope you always know the depth of my love and support and know that you can accomplish anything you set your mind to. "Mom" will always be my favorite title.

Acknowledgments

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This research would also not be possible without the wonderful families who participated in the study. I am grateful for their time and dedication to our research efforts. Finally, thank you to the National Institute of Mental Health [NIMH R01MH099190] and the National Institute of Child Health and Human Development [NICHD P50HD105353] for supporting this research.

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Chapter 1: Introduction

The overarching goal of my dissertation is to understand the pathways connecting parent depression and child mental health problems in families with autistic children. Autism spectrum disorder (ASD) is a lifelong condition, estimated to occur in 1 in 36 children in the United States (Centers for Disease Control and Prevention, 2023), and characterized by impairments in social-emotional reciprocity, challenges with social communication and interactions, and restricted and repetitive behaviors (American Psychological Association, 2020). In addition to these traits, autistic children are at an increased risk for mental health problems compared to their typically developing (TD) peers (Bauminger et al., 2010; Hudson et al., 2019; Vasa & Mazurek, 2015). Experiencing these co-occurring mental health conditions predict poorer objective and subjective well-being in adulthood for autistic individuals (McCauley, Elias, & Lord, 2020; Scheeren et al., 2021). There is a critical need to identify factors, including *modifiable* family processes, that contribute to mental health problems in autistic children to inform the development of prevention and intervention strategies. Parents of autistic children also face a high risk for depression compared to parents of TD children (e.g., Al-Farsi et al., 2016; Schnabel et al., 2020). There is also an important need to understand *modifiable* factors, including family processes, that drive the high risk for depression in parents of autistic children.

My dissertation determined the role that the parent-child and the parent-couple subsystems play in shaping both parent depression and child mental health problems within families of autistic children. My dissertation is made up of three manuscripts. The first manuscript has already been published in the *Journal of Autism and Developmental Disorders* and is included in Appendix 1. This paper demonstrated strong and bidirectional ties between parental depression and child mental health problems in families of autistic children. Across the

other two studies that make up my dissertation, I sought to examine whether the *parent-child relationship quality* - measured through the constructs of warmth and criticism -and *parent-couple relationship satisfaction* act as mediators that account for within-family connections between parent depression and child mental health problems in these same families of autistic children. Research on both TD and autistic children has shown links between parent depression and child mental health problems (e.g., Benson, 2018; Goodman et al., 2010; Letourneau et al., 2010), with children of depressed mothers reporting higher rates of internalizing mental health problems (e.g., anxious and depressed affect) than children of nondepressed mothers (Goodman et al., 2010). Conversely, child mental health problems have also been associated with an increase in parent depression symptoms in both TD and ASD populations (Benson, 2018; Stone et al., 2016).

Manuscript 1 of my dissertation also found important bidirectional associations between parent depression and the internalizing mental health problems of autistic children (Piro-Gambetti et al., 2022; see Figures 1 and 2). More specifically, mother-report of parent depression predicted child internalizing mental health problems 12 months later, and in the opposite direction, mother-report of child internalizing mental health problems also predicted parent depression 12 months later. Father-report revealed that parent depression predicted child internalizing mental health problems 12 months later, but bidirectional effects were not found. Building on these findings, my dissertation explored the underlying mechanisms that explain these bidirectional associations. Figures 3 and 4 illustrate basic conceptual pathways for each manuscript. This work is informed by both the developmental psychopathology (Cummings et al., 2000a; Davies & Cicchetti, 2004) and family systems (Cox & Paley, 1997) theoretical frameworks, while also recognizing the importance of transactional exchanges (e.g., Sameroff,

2009; Sameroff & Mackenzie, 2003; Werner, 1992). Figure 5 provides a depiction of the merging of these frameworks, a recommended approach within the field of family research (Kerig, 2016; Restifo & Bögels, 2009; Sameroff & Mackenzie, 2003).

My dissertation used data from the first four time points (T1-T4) of the Family Outcomes in Autism Study (R01MH199091; Hartley), which has IRB approval through the University of Wisconsin-Madison. At T1, 188 families (N = 376 parents) with at least one autistic child participated in the study. For both manuscript 2 and 3, parent depression was measured using the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977), and child mental health problems were assessed via the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2000, 2001), using both the preschool form (ages 1.5-5 years) and the school age form (ages 6-18 years).

Manuscript 2: Does parent-child relationship quality mediate the connection between child and parent mental health in families with autistic children?

The aims of manuscript two of my dissertation were to: 1) determine whether parent-child relationship quality (via the constructs of warmth and criticism) is associated with parent depression and child mental health problems; and 2) evaluate whether warmth and/or criticism within the parent-child relationship serves as a mediator for the association between parent depression and child mental health problems. Warmth (i.e., the number of positive remarks that express interest or empathy) and criticism (i.e., the number of negative comments that may express disappointment) were measured using Five Minute Speech Samples (FMSS; Magaña et al., 1986), in which mothers and fathers separately speak about their autistic child and their relationship with him/her for 5 minutes.

Within both the developmental psychopathology (Cummings et al., 2000a; Davies & Cicchetti, 2004) and family systems (Cox & Paley, 1997) theoretical frameworks, parent and child mental health problems are hypothesized to be interconnected. These theoretical connections have been supported by research. There is evidence that parent depression often influences child mental health through parent-child interactions (e.g., Beardslee et al., 2011; Ho & Swain, 2017). A depressed parent is often withdrawn from the parent-child relationship and lacks the motivation necessary for fostering a positive and warm relationship with their child (Kamis, 2021; Wilson & Durbin, 2010). Further, depressed parents tend to struggle to engage in sensitive parenting (i.e., being responsive in timely and appropriate ways) and/or have difficulty considering their child's perspective (Taraban & Shaw, 2018), instead resorting to harsher, more disengaged parenting (Wilson & Durbin, 2010). These maladaptive parent-child relationship behaviors may lead to increased child mental health problems across time. In the other direction, there is evidence that child mental health problems contribute to parent depression via altered parent-child relationship quality (e.g., Benson, 2018). Specifically, child mental health problems can create stress, fatigue, and negativity within the parent-child relationship, which in the absence of effective coping strategies, contributes to increased parent depression symptoms (e,g., Mackler et al., 2015).

My dissertation was the first study, to our knowledge, to longitudinally examine whether parent-child relationship quality mediates – or accounts for - the association between parent depression and child mental health problems in families with autistic children, examining both parent and child driven effects. Knowledge gained from this work will help inform the development of family-centered interventions focused on addressing parent and child mental health problems for families with autistic children. Specifically, this work will inform whether

interventions that target parent-child relationship quality may be a way to improve the mental health of both parents and children in these families.

Paper one analyzed two multi-group (e.g., grouped by parent gender) complete longitudinal mediation models using structural equation modeling (SEM), drawing on data from T2-T4 of the Family Outcome in ASD study. These time points were selected because T2 was the first time point that the FMSS was introduced into the study protocol. For publication purposes, we refer to the timepoints in this paper as T1-T3. The first model examined whether criticism (assessed via the FMSS) mediates the association between parent depression and child mental health problems across the time points. The second model investigated whether warmth (assessed through the FMSS) mediates the association between parent depression and child mental health problems.

Drawing on principles of the developmental psychopathology and family systems frameworks, and based on previous research, the following hypotheses were formed: 1) Higher parent depression will predict a decreased level of warmth and increased level of criticism in the parent-child relationship 12 months later; 2) Higher child mental health problems will predict a decreased level of warmth and increased level of criticism in the parent-child relationship 12 months later; 3) Warmth/criticism in the parent-child relationship will mediate the association between parent depression and child mental health problems. Examples of hypothesized mediation pathways include: a) higher parent depression at T1 → lower warmth and higher criticism in parent-child relationship at T2 → higher child mental health problems at T3; and b) higher child mental health problems at T1 → lower warmth and higher criticism at T2 → higher parent depression at T3.

Manuscript 3: Parent-couple relationship satisfaction and child and parent mental health in families with autistic children

The aims of manuscript three of my dissertation were to: 1) examine associations between parent depression, parent-couple relationship satisfaction, and child mental health problems; and 2) determine whether parent-couple relationship satisfaction mediates the association between parent depression and child mental health problems for families with autistic children. Within the family system, the parent-couple (marital or romantic partner relationship) subsystem is highly influential in shaping the well-being of both parents and children (e.g., Cummings et al., 2000b). In support of these theorized connections, research suggests that increased parent depression may contribute to lower parent-couple satisfaction (e.g.,Whisman, Uebelacker, & Weinstock, 2004), such that parents who are experiencing depression symptoms may withdraw from their significant other or become more irritable, leading to more destructive behaviors, greater tension within the parent-couple dyad, and decreased parent-couple relationship satisfaction (e.g., Cummings et al., 2014; Cummings & Davies, 1994; Cummings et al., 2000c; Johnson et al., 2018; Whisman et al, 2004). In the opposite direction, research has found that being in a dissatisfying parent-couple relationship can lead to greater depression symptoms (e.g., Shi & Whisman, 2023; Yang et al., 2023).

Conversely, child mental health problems may create stress and negative affect that spills over into the parent-couple subsystem, leading to increased parent-couple conflict and lower parent-couple relationship quality (Jenkins et al., 2005; Vannier et al., 2018), and in turn parent depression (Choi & Marks, 2008). However, research demonstrating these connections has largely been cross sectional and on TD populations. Little is known about whether parent-couple relationship satisfaction is an important mediator of the links between parent depression and

child mental health in families with autistic children. Yet these pathways may be of particular relevance given that parents of autistic children have a higher risk of divorce (Berg et al., 2016; Hartley et al., 2010), report lower parent-couple satisfaction (Gau et al., 2012) and report more frequent and severe parent-couple conflict and unresolved problems when compared to parents of TD children (Hartley et al., 2017).

Parent-couple relationship satisfaction was measured using The Couple Satisfaction Index (CSI; Funk & Rogge, 2007). Mothers and fathers separately report on their level of parent-couple relationship satisfaction on the CSI. An example item is, “In general, how often do you think that things between you and your partner are going well?”. Child mental health problems were examined by using the internalizing and externalizing scales of the CBCL. An example internalizing item is, “feels worthless or inferior,” and an example externalizing item is, “destroys things belonging to his/her family or others.”

To address the study aims, two multi-group (grouped by parent gender) complete longitudinal mediation models were conducted in SEM, utilizing data from T1-T4 of the Family Outcomes in ASD study. The models examined parent-couple relationship satisfaction as a mediator for the association between parent depression and child mental health problems, broken into models of child internalizing and externalizing mental health problems. The following hypotheses were formed based on research on TD populations (e.g., Johnson et al., 2018; Shi & Whisman, 2023): 1) higher parent depression will predict decreased parent-couple relationship satisfaction 12 months later; 2) higher child mental health problems will also predict decreased parent-couple relationship satisfaction 12 months later; 3) parent-couple relationship satisfaction will mediate the association between parent depression and child mental health problems across time. Example pathways of primary hypotheses include: a) higher parent depression at T1 →

lower parent-couple relationship satisfaction at T2 → higher child mental health problems at T3 (similar path T2→T3→T4); and b) higher child mental health problems at T1 → lower parent-couple relationship satisfaction at T2 → higher parent depression at T3 (similar path from T2→T3→T4).

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Figures.

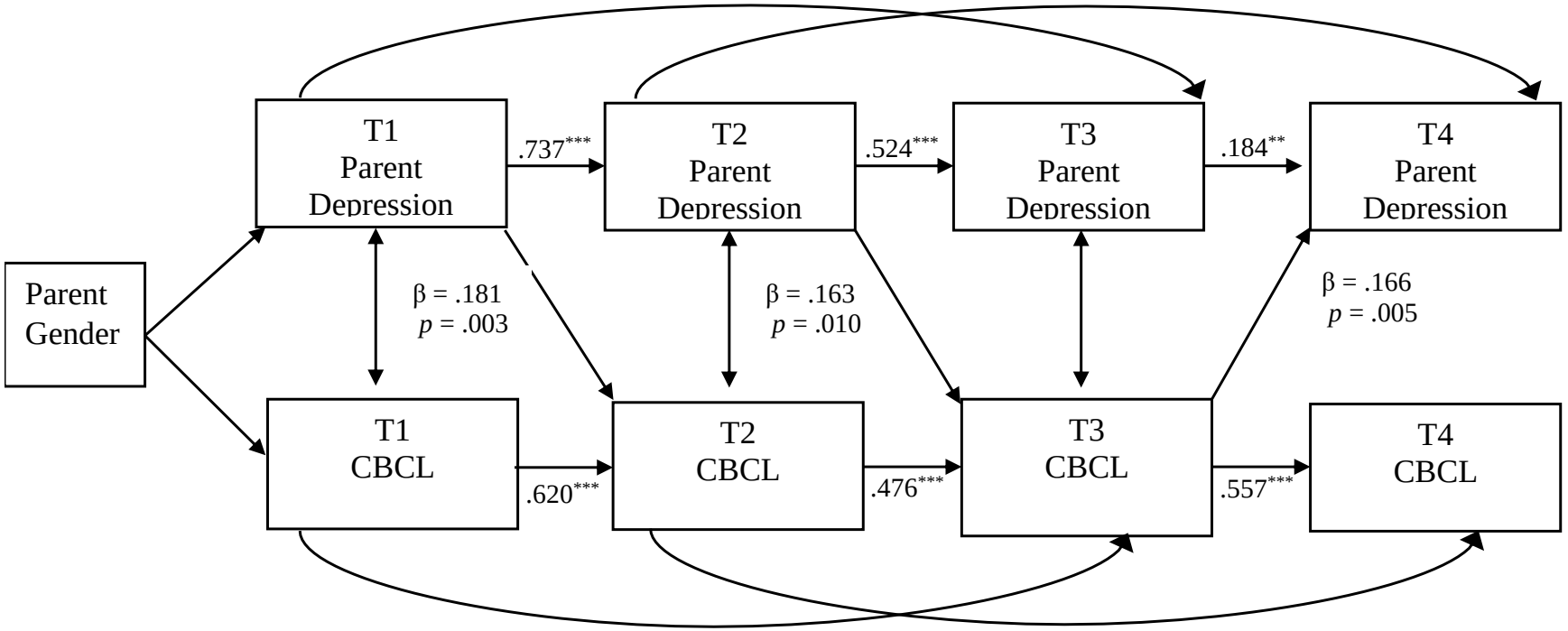


Figure 1. From Piro-Gambetti et al., (2022). Results of the cross-lagged panel model for *mother-reported* parent depression and youth internalizing mental health problems, controlling for child age and household income. Values are standardized path estimates.

$^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$

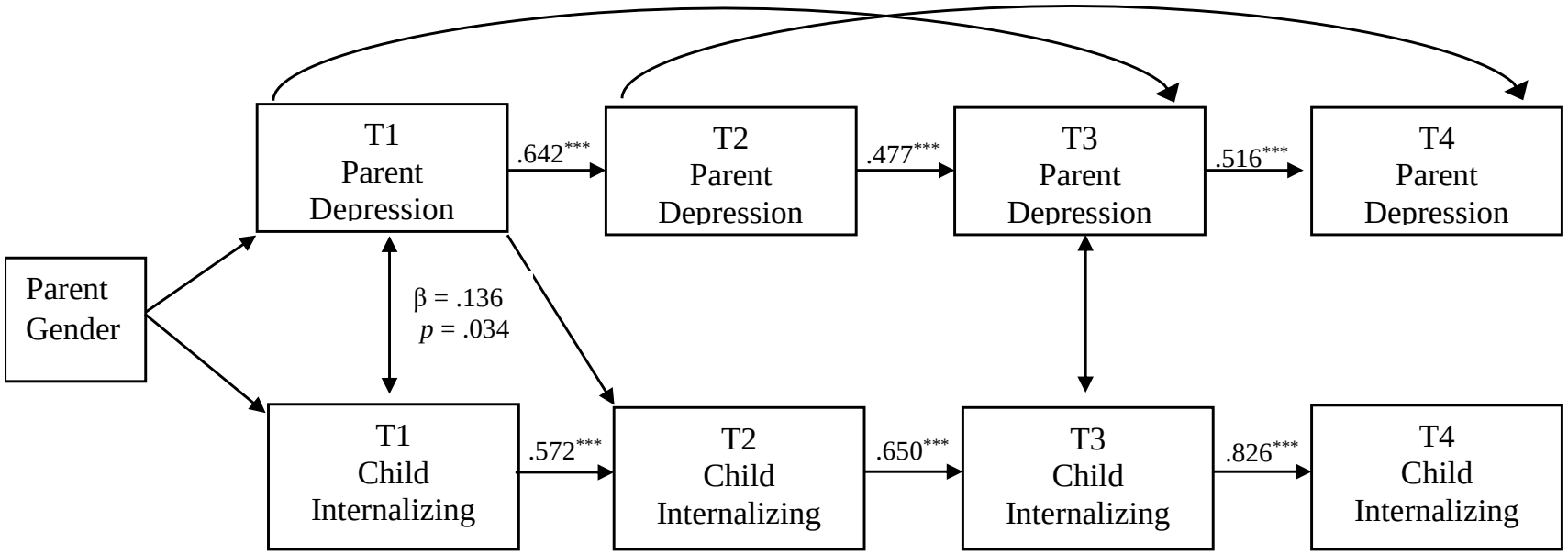


Figure 2. From Piro-Gambetti et al., (2022). Results of the cross-lagged panel model for *father-reported* parent depression and youth internalizing mental health problems, controlling for child age and household income. Values are standardized path estimates.

$^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$

Figure 3. *Possible Developmental Pathways Within the Parent-Child Subsystem [Manuscript 2]*

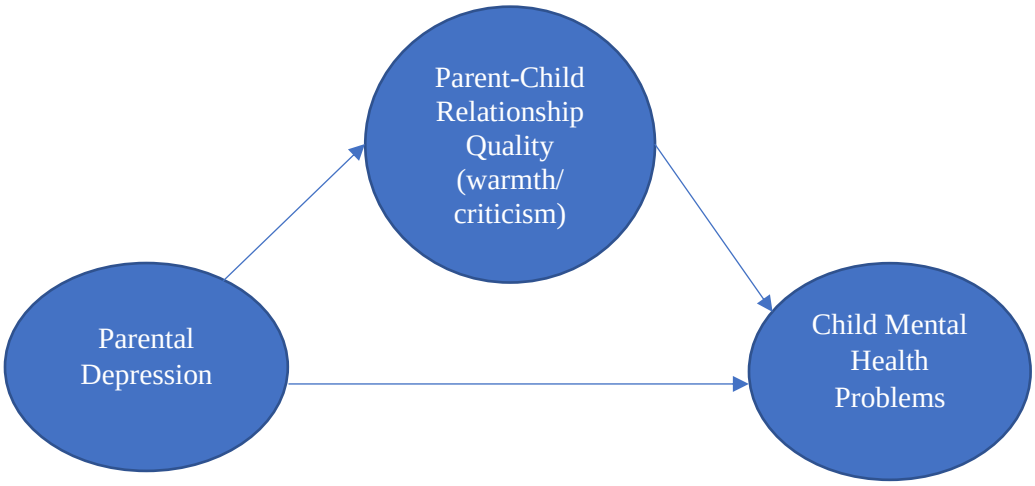


Figure 4. *Possible Developmental Pathways Within the Parent-Couple Subsystem [Manuscript 3]*

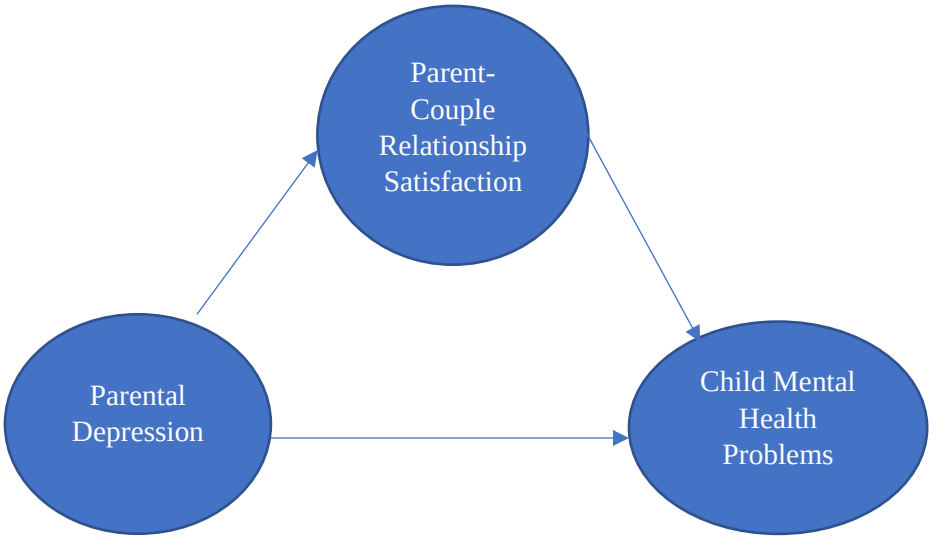
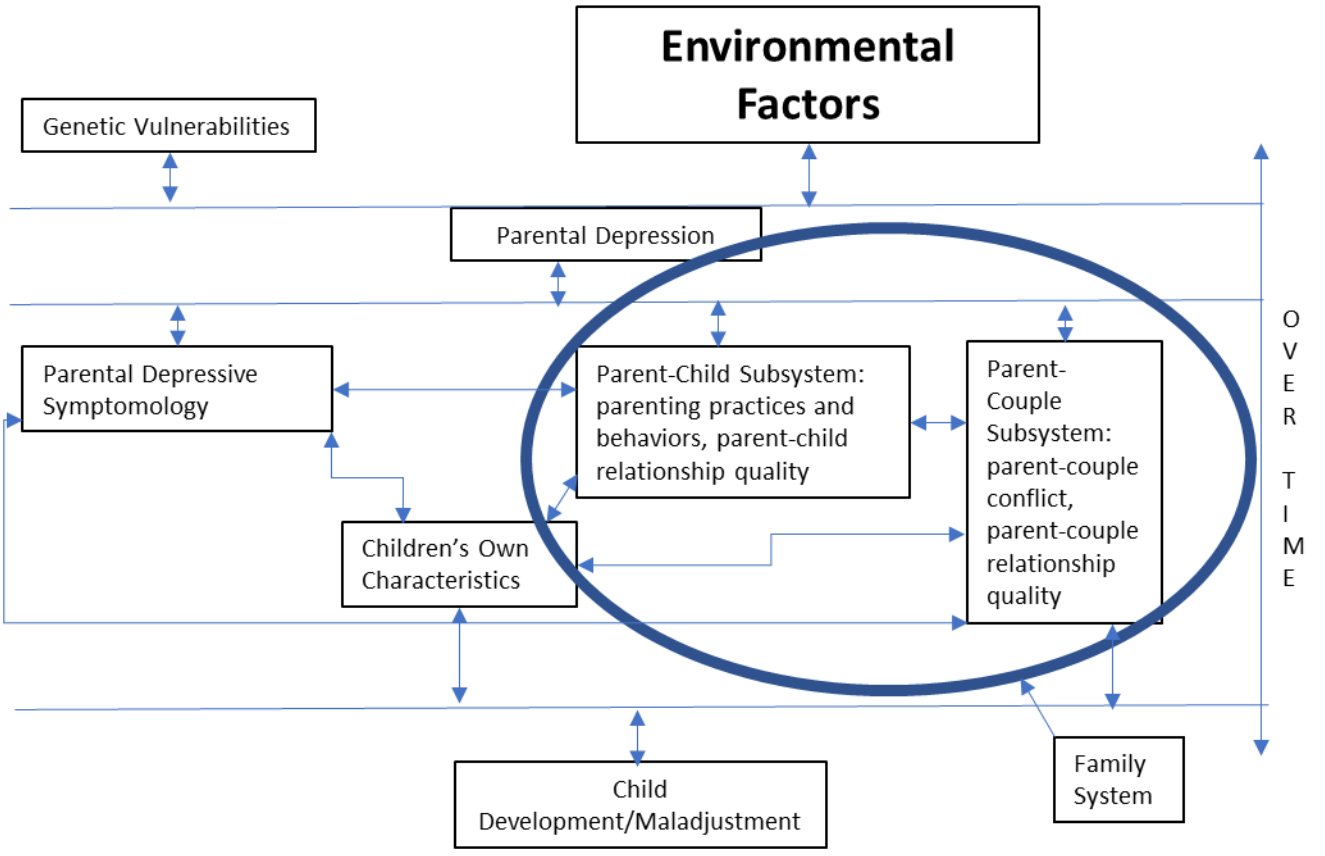


Figure 5. *Merging of Developmental Psychopathology, Family Systems, and Transactional Exchanges*



Note. This figure was inspired by Cummings et al. (2000c) and was modified to illustrate how the family system may influence one's developmental trajectory. Note that the arrows represent transactional exchanges.

Abstract: Manuscript 2

Both autistic children and their parents are at an increased risk for mental health problems. Yet little is known about the within-family processes through which the mental health of autistic children is connected to that of parents. The present study examined the potential role of the quality of the parent-child relationship in mediating a pathway between child mental health problems and parent depression within 162 families of autistic children (aged 6-13). Analyses leveraged three time points of data collection, spanning 2 years. Warmth and criticism in the parent-child relationship were assessed via the Five Minute Speech Sample. Two multi-group (grouped by parent gender) complete longitudinal mediation models were conducted using structural equation modeling and controlled for parent age, household income, and child intellectual disability status. In support of our hypothesis, father's use of positive remarks mediated the connection between father depression and child mental health problems. Additionally, father depression mediated the link between child mental health problems and father's critical comments, and at trend level, child mental health problems mediated the association between father depression and father's critical comments. Results also indicated robust direct associations between the main study variables for both mothers and fathers. Strategies that improve parent-child relationship quality may help reduce the negative effect of parent depression on the mental health of autistic children.

Keywords: autism, parent-child relationship, depression, child mental health problems

Chapter 2: Manuscript 2

Does parent-child relationship quality mediate the connection between child and parent mental health in families with autistic children?

Autism spectrum disorder is a neurodevelopmental condition that occurs in approximately 1 in 36 children in the United States, with boys four times more likely to receive this diagnosis than girls (Centers for Disease Control and Prevention, 2023, 2022). Autism involves restricted and repetitive behaviors and impairments in social communication and social interactions (American Psychological Association, 2020). Autistic children face a high risk for mental health problems compared to typically developing (TD) children (Bauminger et al., 2010; Bos et al., 2018; Hudson et al., 2019; Vasa & Mazurek, 2015). Indeed, it is estimated that 70-95% of autistic children experience a co-occurring mental health condition such as depression, anxiety, or attention deficit hyperactivity disorder (Joshi et al., 2010; Lever & Geurts, 2016; Mosner et al., 2020). Parents of autistic children also have a high risk for mental health problems, with approximately 31-49% of parents of autistic children reporting clinically significant depression (Al-Farsi et al., 2016; Schnabel et al., 2020). Parent depression and child mental health problems are linked in bidirectional ways within families in research on both TD and autistic child samples (e.g., Benson, 2018; Goodman et al., 2010; Letourneau et al., 2010; Piro-Gambetti et al., 2022). For example, Goodman and colleagues (2010) found that children of depressed mothers have higher rates of internalizing mental health problems than children of nondepressed mothers by middle school. Child mental health problems (in both TD and autistic samples) have also been associated with an increase in parent depression symptoms across time (Benson, 2018; Piro-Gambetti et al., 2022; Stone et al., 2016). For example, tracking a sample of

110 mothers of autistic children over a 12-year period, Benson (2018) reported that increased child-related stressors (e.g., higher severity of child behavior problems) led to increased maternal depression. The underlying mechanisms that explain the association between parent depression and child mental health, however, are less clear.

Developmental psychopathology posits that development (both typical and atypical) occurs as a result of a complex interplay between one's genetic vulnerabilities and psychosocial environment (Cummings et al., 2000a; Davies & Cicchetti, 2004). Developmental psychopathology focuses on the underlying processes or mechanisms that gradually occur over time to shape development (Cummings et al., 2000a; Kerig, 2016). A transactional worldview is embedded in developmental psychopathology's idea of a complex genetic-environment interplay that influences one's developmental trajectory (Sameroff & Mackenzie, 2003). Transactions, such as the interactions between parents and children, are described as the mechanism through which one individual (e.g., parent) influences or changes the behavior of another individual (e.g., child) (Sameroff & McKenzie, 2003). Family systems theory complements developmental psychopathology through the concept of transactional exchanges by proposing that a family's main goal is to manage transactions with the environment while maintaining the integrity of the family system (Cox & Paley, 1997). Families are seen as interactive and reactive (Cridland et al., 2014), meaning that what affects one individual or one subsystem (e.g., parent-child subsystem or parent-couple subsystem) within a family will also affect other individuals or subsystems within the system (Cox & Paley, 1997; Kerig, 2016). Maladjustment is thought to occur when disruptions arise within an individual and at least one subsystem (Kerig, 2016). In line with recommendations to merge these theoretical perspectives (e.g., Kerig, 2016; Restifo & Bögels, 2009; Sameroff & Mackenzie, 2003), the present study is rooted in the integration of the

developmental psychopathology and family systems frameworks, with a focus on the transactional exchanges.

Within these frameworks, the connection between the mental health of parents and their autistic child is posited to be driven by both genetic and environmental pathways. From a genetic standpoint, the heritability of depression is estimated to be approximately 40% (Breen et al., 2011; Fernandez-Pujals et al., 2015), with children of depressed mothers being 1 to 5 times more likely to develop depression by adolescence than those of non-depressed mothers (Tirumalaraju et al., 2020), and children of depressed fathers having a 33-70% increased risk for an internalizing or externalizing mental health problem compared to children of nondepressed fathers (Weitzman et al., 2011). Specific transmittable genetic mechanisms that may contribute to depression (e.g., chromosomes 3p25-26, 19p13) among multiple family members have been identified (Breen et al. 2011; Kang et al., 2020). There is also evidence of genetic connections between maternal depression and having an autistic child. Indeed, research indicates that mothers of autistic children are at increased risk for depression with the onset of symptoms beginning prior to having the autistic child (Hagberg et al., 2018; Vasa et al., 2012; Wiggins et al., 2019). Moreover, maternal depression prior to becoming a parent is associated with having an autistic child with a greater level of emotion dysregulation problems (Wiggins et al., 2019). Both autism and the broader autism phenotype (BAP), which involves sub-clinical autism traits such as flat affect and aloofness (Piven et al., 1997) and occurs at an elevated rate in biological relatives of autistic individuals (Bora et al., 2017; Rubenstein & Chawla, 2018), overlap in presentation with depression (Ingersoll & Hambrick, 2011; Pruitt et al., 2018). Thus, this phenotypic overlap has led some researchers to posit that autism, BAP, and depression share specific and novel mutations (Wenderski et al., 2020) or inherited genetic mechanisms (Bora et al., 2017), or that

risk of having an autistic child stem from exposure to maternal depression during gestation (Tirumalaraju et al., 2020).

In line with the developmental psychopathology framework, these genetic predispositions are likely to interact with modifiable environmental mechanisms. There is strong evidence to suggest that the family environment – and specifically through the parent-child subsystem – contributes to the connection between parent depression and child mental health problems across time within families (e.g., Cummings et al., 2000b). These connections appear to be bidirectional flowing from parent to child and vice versa. Indeed, frequent exposure to parent depression symptoms and recurrent and/or severe parent depressive episodes has been shown to predict increases in child mental health problems over time, especially internalizing problems such as depressed mood, for TD children (Kamis, 2021; Tirumalaraju et al., 2020; Schnabel et al., 2020) and autistic children (Cohen & Tsiouris, 2006; Wiggins et al., 2019). Parent-child relationship quality is posited to be one mechanism that drives (i.e., accounts for) these effects. Depressed parents often struggle to engage in sensitive and responsive parenting (Letourneau et al., 2010; Taraban & Shaw, 2018), instead resorting to harsher, more disengaged, and critical parenting (Wilson & Durbin, 2010). Depressed mothers report less positive engagement, less sensitivity, warmth, and responsiveness, and less consistency in their parenting strategies relative to non-depressed mothers (Bayer et al., 2006; Goodman et al., 2010; Rueger et al., 2011), and similar results have been found with depressed fathers (Wilson & Durbin, 2010). Consistent exposure to these maladaptive parenting behaviors has been shown to predict negative outcomes for the child, including mental health problems (Aktar & Bögels, 2017; Beardslee et al., 2011; Goodman & Gotlib, 1999; Goodman et al., 2020).

In a transactional manner, child mental health problems may, in turn, negatively impact the parent-child relationship quality and contribute to parent depression symptoms. In general population research, for example, child externalizing mental health problems have been found to foster a lower quality parent-child relationship through elevated parenting stress and parenting challenges (Mackler et al., 2015). Research on families with autistic children has revealed similar findings. McRae and colleagues (2018) assessed parent mood problems (using a combined measure of depression and anxiety), parenting behaviors, and child mental health problems in 67 parents of autistic children aged 6-12 years and found that parent mood problems positively predicted harsh and disengaged parenting as well as child internalizing mental health problems. In turn, disengaged parenting predicted a higher level of externalizing problems in the autistic child while more warm and supportive parenting predicted fewer child internalizing problems (McRae et al., 2018). Similarly, Hickey and colleagues (2020), using a sample of 150 parents with autistic children aged 5-12 years, found that mothers self-reporting fewer depression symptoms and less parenting stress exhibited more warmth and less criticism toward their child in a five-minute speech sample (FMSS). Father self-report of less parenting stress exhibited less criticism toward the child in a FMSS. These findings highlight the potentially important role of the parent-child relationship in being a modifiable conduit that connects parent and child mental health problems.

The goal of the current study was to understand how parent-child relationship quality (assessed by conveyed warmth and criticism toward the autistic child during a FMSS) impacts the association between parent depression and child mental health problems in families with autistic children. Analyses drew on a longitudinal study of families of autistic children (age 6-13 years) involving three time points of data collection, each spaced 12 months apart. Parents

completed questionnaires about both their own and their child's mental health, and a FMSS assessed warmth and criticism in the parent-child relationship. The study aims were to: 1) determine the association between parent-child relationship quality and parent depression and child mental health problems; and 2) evaluate whether parent-child relationship quality serves as a mediator of the association between parent depression and child mental health problems. Based on the developmental psychopathology and family systems frameworks, and previous research on TD populations, we hypothesized: 1) higher parent depression would predict lower warmth and higher criticism in the parent-child relationship 12 months later; 2) higher child mental health problems would predict lower warmth and higher criticism in the parent-child relationship 12 months later; 3) Warmth and criticism in the parent-child relationship would mediate the association between parent depression and child mental health problems across time. Example hypothesized mediation pathways: a) higher parent depression at T1 → lower warmth and higher criticism in the parent-child relationship at T2 → higher child mental health problems at T3; b) higher child mental health problems at T1 → lower warmth and higher criticism in parent-child relationship at T2 → higher parent depression at T3. Understanding how modifiable aspects of the family environment -such as parent-child relationship quality- contribute to the connection between parent depression and the mental health problems of autistic children may offer a meaningful intervention target.

Methods

The present study drew data from the second to fourth time point of data collection of the Family Outcomes in Autism Spectrum Disorder Study (R01MH199091; Hartley), which received IRB approval through the University of Wisconsin-Madison. For the present analyses, we have labeled these time points T1, T2, and T3. At T1, parent-couples (*n* =162 mothers and *n*

=156 fathers) with at least one autistic child participated in the study. To recruit participants, fliers were distributed throughout community settings, schools, and ASD clinics. Study information was also provided through research registries. The autistic child had to be between 5-12 years of age at the time of recruitment and had to have an educational or medical diagnosis of ASD based on a diagnostic evaluation that included the autism diagnostic observation schedule (ADOS-2nd edition; Lord et al., 2012). If a family had more than one autistic child, the oldest child within the 5-12 year range was considered the target child (i.e., focus of the study) as this marked the beginning of parenting an autistic child. Parents had to be at least 21 years of age or older, live in a cohabiting couple relationship for at least 3 years, and both parents had to agree to participate in the study. To assess the child's current ASD symptoms, parents completed the Social Responsiveness Scale- 2nd Edition (SRS-2; Constantino & Gruber, 2012). All but five children had an SRS-2 total t-score ≥60. However, after thorough review of medical/educational records and ADOS scores, these children were deemed to have met criteria for ASD, and included in analyses. Table 1 provides additional demographic information about the families.

Procedure

At each time point, parent-couples completed a 2.5-hour lab or home visit in which they answered sociodemographic questions and then separately completed questionnaires assessing parent depression symptoms and child mental health problems. Additionally, mothers and fathers separately completed a FMSS where they were asked to speak about their child and the parent-child relationship for five minutes. Each parent was compensated $50 this portion of the study. All parents provided informed consent.

Measures

Family sociodemographics. Parent-couples jointly reported on family sociodemographics, including both parent information (e.g., gender, parent age, household income), and child information (e.g., biological sex, and intellectual disability (ID) status). Parent gender was coded as mothers = 1, fathers = 2. Parent age was coded in years. Household income was coded on a scale of 1-14 (1 = $1-$9,999, 14 = $160,000+). Child ID status was coded as 0 = no ID, 1 = ID. ID status was determined by either the presence of a formal ID diagnosis or if the child met criteria for ID through IQ testing.

Parent depression symptoms. Mothers and fathers separately completed the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977). This 20-item questionnaire involved rating each item on a 4-point scale (e.g., 0 = rarely or none of the time to 3 = most or all of the time). A CES-D total score ≥ 16 suggests clinically significant depression symptoms (Radloff, 1977). Example items include "I was bothered by things that usually don't bother me" and "I felt that people disliked me." In the current sample, the CES-D indicated high internal consistency across time in mothers (Chronbach's α = .92-.93) and fathers (Chronbach's α = .91-.93). Table 2 provides the means and standard deviations for mother- and father-reported CES-D total scores across time as well as *t*-values for paired samples *t*-tests.

Child mental health problems. Child mental health problems were assessed via the preschool form (ages 1.5-5 years) and the school age form (ages 6-18 years) of the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2000, 2001). The CBCL is comprised of 113 items in which parents separately rate each item on a 3-point scale (0 = not true (as far as you know) to 2 = very true or often true). The item scores are then summed into a total problems score. The total problems score encompasses eight different syndrome scales: 1) anxious/depressed; 2) withdrawn depressed; 3) somatic complaints; 4) social problems; 5)

thought problems; 6) attention problems; 7) rule-breaking behavior; 8) aggressive behavior. In my dissertation, the CBCL Total *t* score will be used. Previous research has indicated that the CBCL has strong reliability in the ASD population (Pandolfi et al., 2014). In the present study, the CBCL had high internal consistency for both mothers (Chronbach's α = .94) and fathers (Chronbach's α = .94 - .95). Means and standard deviations for the CBCL total *t*-scores and *t*-scores comparing mother- and father-reports are in Table 2.

Parent-child relationship quality. At T1-T3, parents separately completed a FMSS (Magaña et al., 1986) in which they spoke about their autistic child for 5 minutes. Parents were in separate rooms and could not hear one another speak. They were asked to respond to the open-ended prompt: *"I'd like to hear your thoughts and feelings about (child's name), in your own words and without my interrupting with any questions or comments. When I ask you to begin, I'd like you to speak for 5 minutes, telling me what kind of person (child's name) is and how the two of you get along together. After you begin to speak, I prefer not to answer any questions until after the 5 minutes. Do you have any questions before we begin?"* Study personnel were not permitted to speak during the 5 minutes. The open-ended nature of the FMSS is meant to reduce response bias. All responses were recorded and a trained FMSS rater who was blind to the study questions coded the responses. The rater had high inter-rater reliability with 12 other FMSS trained raters (*M* = 93% [range = 80-100%]). Research has indicated that FMSS ratings of warmth and criticism are associated with observed behaviors within actual parent-child interactions (Weston et al., 2017). Additionally, the FMSS has been found to demonstrate high reliability and construct validity for families with autistic children (Greenberg et al., 2006).

Warmth. For the current study, warmth was measured as the number of positive remarks made during the FMSS per recommendations from Magaña and colleagues (1986). A positive

remark was defined as a statement that describes a positive characteristic of the child a statement that compliments or praises the child, or a statement that expresses something desirable or enjoyable about the child. An example quote high in positive remarks from a mother-report FMSS is, *"[child's name] is a great person. He's so much fun. He's so incredibly helpful …he's very agreeable…he's just really, really great… [he has] a funny sense of humor… I think we have a great relationship"*. An example quote from a father-report FMSS is, *"[child's name] is my guy…. Um I really like [child's name], he's as sweet as they come. He's a very good boy. He is very kind, very curious, um, very smart… and um yeah he's pretty awesome, I really love [child's name].* Table 3. provides an example quote and the coding process for positive remarks.

Criticism. Criticism was coded as the total number of critical comments made during the five minutes (Magaña et al., 1986). Examples of comments coded as critical include: 1) describing the parent-child relationship negatively; and 2) indication of dissatisfaction within the parent-child relationship. Critical comments express disapproving, criticizing, or disappointing thoughts toward the child. An example quote involving criticism from a mother-report FMSS is, *"Um I think our relationship could be better…Um, when [child's name] is in the picture, which is always, there's a lot of tension between everybody. He is very needy, and needs this and needs that, and he can't do anything on his own. He's afraid of everything…Um and I felt angry with him, and frustrated and trapped that sometimes I don't know what to do."* An example quote from a father-report FMSS is, *"uh she irritates me at the same time. She's disappointing in certain ways…um her inability to be regular or normal or intellectually anywhere is so disappointing…. I don't know, her tactile abilities and inabilities are annoying."* Table 3. provides an example quote and the coding process for critical comments.

Data Analysis Plan

Descriptive statistics and boxplots were used to examine the distribution of scores and screen for outliers. Attrition analyses were conducted to explore whether families who completed all time points differ in their ratings of parent depression, child mental health problems, and parent-child relationship quality from parents who had missing data at one or more time points. Bivariate Pearson correlations were conducted to assess the relatedness among the main study variables and with family sociodemographics. Family sociodemographic variables significantly correlated with main study variables were included as covariates in subsequent models. Specifically, we regressed the main study variables on the relevant family sociodemographic variables, and the unstandardized residual scores were entered in the model.

Two multi-group complete longitudinal mediation models using structural equation modeling (SEM) were then conducted. For a conceptual model, see Figure 1. The models were grouped by parent gender, providing separate results for mother- and father-reported measures and processes. Following the commonly implemented ratio of cases/observations to estimated parameters (10:1) and aligning with recommendations provided by Kline (2015) and Nunnally (1967), the minimum total sample size to detect acceptable power for each model is 240, and the minimum sample for each group (e.g., mothers and fathers) is 100. Model 1 investigated whether criticism (in FMSS) mediated the association between parent depression and child mental health problems. Model 2 explored whether warmth (in FMSS) mediated the association between parent depression and child mental health problems. A complete longitudinal mediation model, as opposed to a focused mediation model, allowed us to examine all longitudinal possibilities. Specifically, this model allowed all three variables of interest (e.g., parent depression, warmth/criticism, and child mental health problems) to be represented in the model at T1-T3 and test mediations for both parent and child-driven effects across the time points (Jose, 2016).

Models were run in Mplus statistical software (Muthen & Muthen, 2012), as it allows for the examination of multiple mediation pathways (Jose, 2016). To analyze model fit, the root mean square error of approximation (RMSEA), comparative fit index (CFI), Tucker-Lewis Index (TLI), and chi-square (χ^2) were examined. A RMSEA value between .05 and .08, CFI and TLI scores greater than .90, and a small χ^2 value were used to indicate good model fit (Hu & Bentler, 1999; Little, 2013). The full information maximum likelihood method was implemented to account for missing data (Little, 2013), which has been shown to be a better estimation method compared to deletion or imputation methods and is a robust estimator in SEM (Schlomer, Bauman, & Card, 2010). We analyzed confidence intervals for the indirect effects, utilizing bootstrapping with 5,000 iterations to determine if significant mediations are present within the models (Jose, 2013).

Results

Preliminary Analyses

Parent depression symptoms and child mental health problems were normally distributed without skew (kurtosis range for CES-D = .978 to 2.467; -.601 to -.414 for CBCL). Data was missing completely at random (MCAR: $X^2 = 2.611$, $p > .05$). At T1, data from both parents was available for 156 families. Six additional families provided mother-report only, for a total of 162 families (N = 318 parents). Of these families, at T2, data from both parents was available for 133 families. Four additional families provided mother-report only, for a total of 137 families (N = 270 parents). T3 included data from both parents for 122 families, with 3 additional families providing mother-report only, for a total of 125 families (N = 247 parents). Reasons for attrition included moving or not having time to participate at that time point. Attrition analyses explored possible differences between families who completed all three study visits (coded as 0 for

"completers" and those who had missing data at one or more time points (coded as 1 for "incompleters"), and independent t-tests revealed that no significant differences exist in terms of reported parent depression symptoms (mother: $t(160) = -1.265$, $p = .104$; father: $t(154) = .748$, $p = .228$) and child mental health problems (mother: $t(161) = -.472$, $p = .319$; father: $t(156) = -.398$, $p = .346$). No significant difference was found for mother-report of positive remarks ($t(145) = -1.540$, $p = .063$). However, in critical comments for mother-report ($t(145) = 1.817$, $p = .036$),"completers" ($M = .43$, $SD = .98$) used more critical comments than "incompleters" ($M = .13$, $SD = .41$). For father positive remarks ($t(146) = -2.258$, $p = .013$), "completers" ($M = 2.44$, $SD = 2.28$) used fewer positive remarks than "incompleters" ($M = 3.53$, $SD = 3.26$). There were no significant differences in critical comments ($t(146) = .972$, $p = .166$) for father-report. In terms of mother sociodemographic variables, there were significant differences between "completers" versus "incompleters" for parent age ($t(162) = -1.899$, $p = .030$), with "completers" ($M = 39.00$ years, $SD = 5.39$ years) being younger than "incompleters" ($M = 40.82$ years, $SD = 6.32$ years). Father-report revealed a significant difference in household income ($t(161) = 2.104$, $p = .018$) with "completers" ($M = 9.12$, $SD = 2.95$) having a higher income than "incompleters" ($M = 7.96$, $SD = 3.86$).

Table 2. provides the means and standard deviations for main study variables as well as within-couple differences in mother- versus father-reports. Paired sample t-tests revealed that mother- and father-reported CBCL scores did not differ significantly at any time point (t: 1.127-1.913, $p > .05$). Mother and father CES-D scores differed significantly at each time point (ts: 2.199-3.103, $p < .05$) with mothers reporting higher levels of depression symptoms than fathers. Mothers and fathers differed significantly on the number of critical comments at T1 ($t(145) = 2.261$, $p = .025$), with mothers reporting more critical comments than fathers, but no significant

difference was found for T1-T3 (ts: 1.654-1.679, p >.05). Mothers and father's differed significantly in their number of positive remarks for T1 and T3 (ts: 2.719-3.290, p <.05), with mothers reporting more positive remarks than fathers, but no differences were found at T2 (t(120) = .118, p = .906).

Table 4. shows Pearson correlations between parent depression symptoms, child mental health problems, critical comments, and positive remarks, as well as family sociodemographic variables. Associations between parent depression symptoms and child mental health problems were significantly positively correlated for both mother- (rs = .217-.460, p <.05) and father-report (rs = .167-.434, p <.05) across all time points. Additionally, mother-report of critical comments was significantly positively associated with child mental health problems at all time points (rs = .270-.329, p <.05). Mother-report of critical comments was also significantly associated with parent depression symptoms (T2 critical comments with T2 CES-D: r = .209, p = .011; T3 CES-D: r = .347, p <.001; T4 CES-D: r = .325, p <.001; T3 critical comments with T3 CES-D: r = .290, p <.001; T4 CES-D: r = .225, p = .017; T4 critical comments with T3 CES-D: r = .318, p <.001; T4 CES-D: r = .285, p = .002). Significant negative associations were also found between mother-report of positive remarks and child mental health problems (T1 positive remarks with T1 CBCL: r = -.228, p = .006; T3 CBCL: r = -.232, p = .009; T4 CBCL: r = -.243, p = .008; T2 positive remarks with T2 CBCL: r = -.204, p = .022; T3 CBCL: r = -.232, p = .0113; T4 positive remarks with T3 CBCL: r = -.223, p = .017). Additionally, significant negative associations were found for mother-report of positive remarks and parent depression (T2 positive remarks with T2 CES-D: r = -.250, p = .005; T3 positive remarks with T3 CES-D: r = -.225, p = .018).

Father-reports revealed significantly positive associations between critical comments and child mental health problems (T1 critical comments with T3 CBCL: r = .240, p = .009; T2 critical

comments with T3 CBCL: r = .207, p = .022; T4 CBCL: r = .225, p = .017; T3 critical comments with T1 CBCL: r = .248, p = .012; T2 CBCL: r = .204, p = .041; T3 CBCL r = .312, p = .001) and significantly negative correlations between positive remarks and child mental health problems at all time points (rs = -.355- -.220, p <.05). Neither critical comments nor positive remarks were significantly correlated with parent depression symptoms for father-reports.

Father age was correlated with child mental health problems (T1: r = .193, p = .016). Child ID status was significantly associated with father-report of child mental health problems (T1: r = .188, p = .018; T3: r = .201, p = .025) and father number of positive remarks (T1: r = -.242, p = .003). Household income was significantly correlated with parent depression symptoms for both mothers (T1: r = -.163, p = .038) and fathers (T1: r = -.157, p = .050; T3: r = -.283, p = .002). Given the significant associations between the main study variables and the sociodemographic variables, the complete-longitudinal mediation models controlled for parent age, child ID status, and household income. Specifically, we regressed parent depression symptoms, child mental health problems, number of critical comments, and number of positive remarks on parent age, child ID status, and household income at each time point. The unstandardized residual scores were then entered into the mediation models.

Complete-Longitudinal Mediation Models

Figures 2 and 3 depict the complete-longitudinal mediation models for mother- and father-report examining critical comments respectively, and figures 4 and 5 illustrate the complete-longitudinal mediation models for mother- and father-report examining positive remarks respectively. Tables 5 and 6 provide the path coefficients for the direct and indirect pathways for the critical comments model, respectively. Tables 7 and 8 provide the path coefficients for the direct and indirect pathways for the positive remarks model, respectively.

Critical Comments Model

The critical comments model revealed good model fit (X^2 (12) = 14.23, p = 0.2862; RMSEA = .034; TLI = 0.985; CFI = 0.997). Stability effects across time were present for both mother and father-report of parent depression (Mother: T1-T2 β = 0.521, p = .000; T2-T3 β = 0.288, p = .001; Father: T1-T2 β = 0.452, p = .001; T2-T3 β = 0.606, p = .000), child mental health problems (Mother: T1-T2 β = 0.699, p = .000; T2-T3 β = 0.787, p = .004; Father: T1-T2 β = 0.777, p = .000; T2-T3 β = 0.299, p = .001), and critical comments (Mother: T1-T2 β = 0.392, p = .005; T2-T3 β = 0.308, p = .001; Father: T1-T2 β = 0.414, p = .000; T2-T3 β = 0.309, p = .001).

Mother-report of child mental health problems directly predicted increased maternal depression symptoms 12 months later (T1 CBCL to T2 CES-D: β = .248, p = .000; T2 CBCL to T3 CES-D: β = .273, p = .000). Additionally, mother-report of child mental health problems at T1 directly predicted maternal critical comments at T2 (β = .237, p = .028). In the opposite direction, mother critical comments at T2 directly predicted child mental health problems at T3 (β = -.093, p = .000). In the father-report models, child mental health problems directly predicted the number of critical comments 12 months later (T1 CBCL to T2 Critical: β = .181, p = .032; T2 CBCL to T3 Critical: β = .159, p = .002). Further, father depression symptoms at T2 predicted critical comments (β = -.077, p = .005) as well as child mental health problems (β = .162, p = .000) at T3. Indirectly, father-report of child mental health problems at T1 predicted paternal depression symptoms at T2, which then predicted the number of father critical comments at T3 (β = -.018, p = .036; CI [-.025- -.011]). This finding suggests that parent depression partially mediated the connection between child mental health problems and father number of critical comments. Additionally, in the opposite direction, there was a trend-level

indirect effect in which paternal depression at T1 predicted child mental health problems at T2, which then predicted the number of father critical comments at T3 (β = .015, p = .053; CI [0.003-0.022]), suggesting that child mental health problems partially mediated the association between father depression and the number of critical comments about the child.

Secondary Analyses. Our critical comments model revealed two unexpected findings that were explored in secondary analyses. There was an unexpected direct negative effect of T2 mother critical comments on T3 child mental health problems (β = -.093, p = .000). However, further investigation highlighted a suppression effect (Maassen & Bakker, 2001), which can occur in longitudinal models when simultaneously evaluating multiple predictors rather than each pathway in isolation. Specifically, the highly correlated association between child mental health problems (CBCL) at all time points created a suppression effect (i.e., β value switches direction) (Maassen & Bakker, 2001). When multiple linear regressions were conducted to examine the effects of T1-T2 CBCL, T2 critical comments and T2 CES-D on T3 CBCL, the β of mother critical comments predicting T3 child mental health problems was weak and nonsignificant (β = -.006, p = .865). After removing T1 and T2 CBCL scores, the β value switched back to positive and became significant (β = .223, p < .001), indicating that T2 critical comments predicted *higher* T3 child mental health problems.

A similar suppression effect was found for the unexpected association between father-report of parent depression at T2 and fewer T3 critical comments. Multiple linear regression models examining the effects of T2 CES-D and T2 CBCL on T3 critical comments revealed that the β value for T2 CES-D became positive and significant (β = .145, p = .05), suggesting that parent depression at T2 predicts an *increase* in the number of critical comments. Due to this suppression effect being part of the significant father-reported *indirect* pathway from T1 child

mental health problems → T2 parent depression → T3 critical comments, we conducted the Sobel Test (Sobel, 1982) in SPSS. These results suggested that T2 parent depression partially mediated the association between T1 child mental health problems and T3 critical comments (Path A: β = .469, *SE* = .076; Path B: β = .009, *SE* = .004; Path C: β = .017, *SE* = .005; Sobel Test: *z* = 2.11, *SE* = .002, *p* = .035).

Positive Remarks Model

The positive remarks model also revealed good model fit (X^2 (12) = 9.048, *p* = 0.6988; RMSEA = .000; TLI = 1.000; CFI = 1.000). Stability effects were present for both mother and father depression (Mother: T1-T2 β = .546, *p* = .000; T2-T3 β = .301, *p* = .000; Father: T1-T2 β = .461, *p* = .000; T2-T3 β = .588, *p* = .000), child mental health problems (Mother: T1-T2 β = .755, *p* = .000; T2-T3 β = .516, *p* = .000; Father: T1-T2 β = .751, *p* = .000; T2-T3 β = .501, *p* = .000) and positive remarks (Mother: T1-T2 β = .563, *p* = .000; T2-T3 β = .415, *p* = .004; Father: T1-T2 β = .486, *p* = .000; T2-T3 β = .341, *p* = .001).

The complete-longitudinal mediation model revealed that both mother and father-report of child mental health problems predicted parent depression 12 months after (Mother: T1 CBCL to T2 CES-D: β = .270, *p* = .000; T2 CBCL to T3 CES-D: β = .191, *p* = .000; Father: T1 CBCL to T2 CES-D: β = .228, *p* = .033; T2 CBCL to T3 CES-D: β = .136, *p* = .000). Additionally, for fathers, there was a significant indirect effect in that father depression symptoms at T1 predicted father number of positive remarks at T2, which then predicted child mental health problems at T3 (β = .009, *p* = .049; CI [0.010 – 0.013]). This finding suggests that positive remarks mediated the association between higher father depression and higher child mental health problems.

Discussion

Families are interactive and reactive (Cridland et al., 2014), such that the emotions and behaviors of one person or one subsystem in the family impacts the lives of the other family members and subsystems. These connections mean that family relationships are a critical and modifiable mechanism which through parent mental health influences child mental health and vice versa. The present study sought to determine whether parent-child relationship quality mediates the association between parent depression and the mental health problems of autistic children at a within-family level across a two-year period.

We found evidence for robust connections between parent depression, child mental health problems, and criticism in the parent-child relationship. These pathways were in line with our hypotheses. Specifically, mother-report of child mental health problems predicted an increase in mother depression symptoms 12 months later (from T1 toT2 and T2 to T3). Both mother- and father-report of T1 child mental health problems predicted an increase in their critical comments about their autistic child and parent-child relationship at T2. These pathways align with prior research outside of autism (Chan et al., 2018; Crugnola et al., 2016; Mackler et al., 2015; Stone et al., 2016) and suggest that child mental health problems can alter parent mood and may contribute to critical parent-child interactions. In the opposite direction, both mother and father depression symptoms predicted an increase in the mental health problems of the autistic child across time (Mothers: T1-T2; Fathers: T2-T3). This is consistent with previous research that exposure to parent depression negatively affects child mental health (Cohen & Tsiouris, 2006; Wiggins et al., 2019).

We had hypothesized that criticism in the parent-child relationship would mediate the bidirectional associations between child mental health problems and parent depression. In partial support of this hypothesis, parent depression and child mental health problems served as partial

mediators. Specifically, higher T1 child mental health problems were related to higher T3 father critical comments, and this was partially mediated by higher T2 father depression. This pathway suggests that having an autistic child with high mental health problems may contribute to increases in father depression, which in turn, may contribute to a more critical parent-child relationship. Moreover, at a trend-level, there was a positive association between T1 father depression and T3 father critical comments that was partially mediated by higher T2 child mental health problems. Thus, on average, child exposure to fathers' depression symptoms may have taken a toll on the autistic child's mental health, and the increased child mental health problems may have, in turn, led the father to become more critical about the parent-child relationship. Overall, these pathways indicate that the mental health of parents and their autistic children are intertwined in mutually influential ways and that overtime, these challenges lead to an increasingly critical parent-child relationship.

We also found important associations between positivity in the parent-child relationship, parent depression, and child mental health in families of autistic children. Child mental health problems positively predicted parent depression for both mother- and father-report (from T1 to T2 and T2 to T3), in line with models of criticism in the parent-child relationship. Moreover, as hypothesized, positivity in the parent-child relationship mediated connections between parent and child mental health. Specifically, for fathers, there was a positive association between T1 parent depression and T3 child mental health problems that was mediated by decreased positive remarks about the child and parent-child relationship at T2. This pathway suggests that fathers with high depression symptoms had a less positive parent-child relationship overtime, which have subsequently added to the autistic child's mental health problems. Evidence for similar

pathways of effects has been found outside of autism (Aktar & Bögels, 2017; Rueger et al., 2011).

It is not clear why mediation pathways occurred for father-reported models but not mother-reported models of the parent-child relationship. Autistic children may be more sensitive to both criticism and positivity in the father-child relationship than in the mother-child relationship. Perhaps, this is because fathers, on average, take on fewer childcare responsibilities than mothers within families of autistic children (Hartley et al., 2014; Sharabi & Maron-Golan, 2018). Critical or warm interactions, even if infrequent, may take a greater toll on both children and parents without a context of frequent neutral interactions.

Strengths, Limitations, and Future Directions

The present study had several strengths including the use of complete longitudinal mediation modeling using structural equation modeling which allowed us to simultaneously explore all parent- and child-driven pathways of effects. This analytic approach provided a complete picture of multiple time-ordered associations between parent depression, parent-child relationship quality, and child mental health problems, mirroring theoretical models of complex and bidirectional family dynamics (e.g., Jose, 2016). Another study strength was the inclusion of both mother and father experiences within families and separating modeling but yoking these experiences through a group variable in the structural equation models. There is a paucity of father-perspectives in family research, and more specifically, in autism research and thus the current findings build on this literature and also highlight important differences in the experiences of mothers and fathers.

The current study also had limitations. The generalizability of findings is limited to the sociodemographics of the families included. Our sample largely consisted of White, non-

Hispanic parents and those of middle socio-economical class and heterosexual couples. Additionally, families who completed all three waves of the study and included in analyses were younger and had higher income, on average, than those who originally enrolled in the study but did not complete all three time points. While we did not find differences in reports of parent depression or child mental health problems between "completers" and "incompleters," the families who completed all three waves of the study had lower quality parent-child relationships (fewer maternal positive remarks and more paternal critical comments) than those who did not complete all three study cycles. It is possible that families who were satisfied with their parent-child relationship quality were not as motivated to engage in research to improve services and supports. Due to the lack of father perspectives in family research, the present study focused on mother and father perspectives because we were interested in exploring potential mediation patterns among the different parent genders, however we acknowledge that future research should include samples that represent a more diverse range of identities and family dynamics (e.g., same-sex couples, single parents, adoptive parents, low versus high socioeconomic status, etc.). Reporter bias is also possible in that depressed parents may have rated their parent-child relationship more negatively. Future studies should include observations of actual parent-child interactions to help avoid this potential bias.

Study Implications

When left untreated, mental health problems can negatively impact not just one's own life, but also the quality of one's relationships. Resources are needed to educate parents on how to recognize the overlap between autism symptoms and signs of a mental health problem for their child in hopes of identifying and addressing the mental health need in a timely manner. The present study's findings highlight the need for interventions aimed at promoting a high-quality

parent-child relationship in families with autistic children. These interventions should take a family-wide approach, focusing on methods that improve both the mental health of parents and children and the quality of the parent-child relationship. The Child First intervention, for example, strives to strengthen the parent-child relationship by providing psychoeducation to help parents better understand their child's mental health needs and the impact of their own mental health on the child's mental health (Child First, 2023). Additionally, in line with World Health Organization guidelines for parenting interventions to enhance parent-child relationships, child-led play interventions as well as empathy building exercises, and praise and reward strategies are all helpful methods for strengthening the parent-child bond (World Health Organization, 2022). Further, these guidelines also recommend interventions that promote parental self-management and emotion-regulation skills (World Health Organization, 2022). Strategies that intervene on parent and/or child mental health such as mindfulness training or cognitive behavioral therapy may also be beneficial in improving the parent-child relationship and positively impacting the mental health of the parent-child dyad. Future research should continue to investigate additional underlying environmental mechanisms within the parent-child subsystem that may influence or be influenced by parent depression and child mental health problems in families with autistic youth.

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Tables

Table 1.

Family Sociodemographics

Demographic	*M* (*SD*)
Mother (*n* = 162)	
Age in years (*M* [*SD*])	39.76 (5.61)
Race/Ethnicity (N [%])	
White, non-Hispanic	148 (91)
Other	14 (9)
Father (*n* = 156)	
Age in years (*M* [*SD*])	40.76 (6.19)
Race/Ethnicity (N [%])	
White, non-Hispanic	138 (89)
Other	18 (11)
Parent Couple	
Household Income (N [%])	
Less than $20,000	6 (4)
$20,000-$39,999	14 (9)
$40,000 and greater	142 (88)
Couple Relationship Length, years (*M* [*SD*])	14.55 (5.58)
Both Biological Parents (N [%])	144 (89)
Biological Mom, Stepdad (N [%])	10 (6)
Biological Dad, Stepmom (N [%])	4 (2)
Both Adoptive Parents (N [%])	4 (2)
Target Child (*n* =162)	
Male (N [%])	140 (86)
Age in years (*M* [*SD*])	9.03 (2.25)
ID (N [%])	57 (35)
SRS (*M* [*SD*])	77.03 (10.29)

Note. *M* = mean; *SD* = standard deviation; N = sample size; ID = intellectual disability; SRS = Social Responsiveness Scale-2nd edition (Constantino & Gruber, 2012).

Table 2.

Mother and father reported means, standard deviations, and t-values for main variables.

Measure	Mother $M^1(SD)^2$	Father $M(SD)$	*t*-value[3]	*df*[4]	*p*-value
Time 1	n = 162	n = 156			
CES-D[5] Total	18.40 (7.10)	16.44 (6.40)	3.029	154	.003**
CBCL[6] Total T	63.83 (8.71)	62.80 (9.33)	1.701	156	.091
Critical[7]	0.35 (0.88)	0.18 (0.52)	2.261	145	.025*
Positive[8]	3.55 (2.96)	2.72 (2.60)	3.290	145	.001**
Time 2	n = 137	n = 133			
CES-D Total	14.86 (11.29)	11.14 (9.51)	3.103	130	.002**
CBCL Total T	63.14 (9.01)	61.95 (8.92)	1.913	131	.058
Critical	0.35 (0.66)	0.23 (0.69)	1.679	120	.096
Positive	3.07 (3.05)	3.02 (3.12)	.118	120	.906
Time 3	n = 125	n = 122			
CES-D Total	14.68 (10.70)	12.40 (10.41)	2.199	116	.030*
CBCL Total T	62.79 (8.70)	62.06 (9.64)	1.127	120	.262
Critical	0.31 (0.69)	0.20 (0.47)	1.654	102	.101
Positive	3.24 (2.88)	2.44 (2.33)	2.719	102	.008**

Note. [1]mean; [2]standard deviation; [3]value for paired-samples *t*-test; [4]degrees of freedom; [5]Center for Epidemiological Studies-Depression Scale total score (Radloff, 1977); [6]Child Behavior Checklist Total Problems T-Score (Achenbach & Rescorla, 2001);[7]number of critical comments from the Five Minute Speech Sample (Magaña et al., 1986); [8]number of positive remarks from the Five Minute Speech Sample (Magaña et al., 1986); $^{*}p < .05$; $^{**}p < .01$.

Table 3.

Example Five Minute Speech Sample Quotes and Corresponding Scores.

Parent	Quote	Score
Mother: Highly Positive	*"[child's name] is* ***a great person***. ***He's so much fun. He's so incredibly helpful*** *...****he's very agreeable****...****he's*** *just* ***really, really great****... [he has] a* ***funny sense of humor****...* ***I think we have a great relationship****".*	Positive Remarks Score: 16 Critical Comments Score: 0
Father: Highly Positive	*"[child's name] is* ***my guy****....* *Um* ***I really like [child's name]****,* ***he's as sweet as they come. He's a very good boy. He is very kind****, very curious, um,* ***very smart****... and um yeah* ***he's pretty awesome****,* ***I really love [child's name]***	Positive Remarks Score: 23 Critical Comments Score: 0
Mother: Highly Critical	*"Um* ***I think our relationship could be better****...Um, when [child's name] is in the picture, which is always,* ***there's a lot of tension between everybody****.* ***He is very needy****, and needs this and needs that, and* ***he can't do anything on his own. He's afraid of everything****...Um and* ***I felt angry with him, and frustrated and trapped that sometimes I don't know what to do…"***	Positive Remarks Score: 0 Critical Comments Score: 7
Father: Highly Critical	*"uh* ***she irritates me*** *at the same time.* ***She's disappointing in certain ways****...um* ***her inability to be regular or normal*** *or intellectually anywhere* ***is so disappointing****.... I don't know,* ***her tactile abilities and inabilities are annoying****."*	Positive Remarks Score: 0 Critical Comments Score: 4

Note. Quotes are excerpts from larger Five Minute Speech Sample transcription.

Table 4.

Correlations between main study variables and sociodemographics.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. T1 P. Age[1]	---	-.025	.097	-.065	.010	-.157	.153	.087†	.084	-.040	-.006	.186†	-.020	-.025	-.022
2. T1 Income	-.011	---	-.183*	-.163*	-.045	-.051	.039	-.019	.052	-.094	-.148	-.097	.114	.033	-.050
3. Child ID[2]	.033	-.183*	---	.043	-.053	.063	.113	.161	.151	-.073	-.022	-.065	-.012	-.124	.009
4. T1 CES-D[3]	.063	-.157*	-.024	---	.589**	.691**	.267**	.246**	.217**	-.002	-.030	-.016	.209*	.086	.172
5. T2 CES-D	.073	-.124	-.010	.524**	---	.717**	.413**	.460**	.366**	-.160	-.250**	-.225**	.347**	.290**	.318**
6. T3. CES-D	.037	-.283**	-.130	.429**	.736**	---	.411**	.406**	.389**	-.046	-.153	-.174	.325**	.225*	.285**
7. T1 CBCL[4]	.193*	-.112	.188*	.167*	.273**	.247**	---	.783**	.792**	-.228**	-.165	-.126	.312**	.329**	.270**
8. T2. CBCL	.158	-.104	.048	.247**	.434**	.342**	.777**	---	.830**	-.232**	-.204*	-.142	.295**	.282**	.327**
9. T3 CBCL	.100	-.071	.201*	.193*	.354**	.315**	.809**	.833**	---	-.243**	-.232*	-.223*	.280**	.300**	.307**
10. T1 Pos.[5]	-.072	.083	-.242**	-.043	-.083	-.029	-.348**	-.310**	-.343**	---	.549**	.231*	-.298**	-.246**	-.191*
11. T2 Pos.	-.173	.050	-.015	-.113	-.106	-.039	-.220*	-.355**	-.326**	.434**	---	.400**	-.211*	-.296**	-.181
12. T3 Pos.	.003	.048	.001	-.085	-.101	-.180	-.328**	-.315**	-.334**	.479**	.458**	---	-.153	-.165	-.246**
13. T1 Crit. [6]	.025	.017	-.004	.099	.070	.081	.138	.149	.240**	-.155	-.190*	-.067	---	.469**	.458**
14. T2. Crit.	.008	-.103	-.048	.111	.013	.056	.178	.207*	.225*	-.271**	-.224*	-.212*	.604**	---	.308**
15. T3. Crit.	-.001	.015	-.122	-.043	.043	.134	.248*	.204*	.312**	-.147	-.246*	-.301**	.322**	.460**	---

Note. Pearson Correlations. Mother-report is shaded and above the diagonal. Father-report if unshaded and below the diagonal. [1]Parent age; [2]Intellectual Disability; [3]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [4]Child Behavior Checklist (Achenbach & Rescorla, 2001); [5]Positive Remarks from the Five Minute Speech Sample (Magaña et al., 1986); [6]Critical Comments from the Five Minute Speech Sample (Magaña et al., 1986). † = $p < .10$; *= $p < .05$; **= $p < .01$

Table 5.

Standardized and unstandardized path coefficients for mother- and father-reports of parent depression, parents' critical comments, and child mental health problems

Time Point	Mother-report *B*[1](*SE*[2]), Unstandardized	Mother-report *β*(*SE*), Standardized	Father-report *β*(*SE*), Unstandardized	Father-report *β*(*SE*), Standardized
Cross Effects	**CES-D[3] → Critical[4]**			
1→2	-0.012(0.009)	-0.126(0.085)	0.000(0.005)	-0.003(0.061)
2→3	-0.004(0.012)	-0.050(0.187)	-0.004(0.001)**	-0.077(0.028)**
	CES-D → CBCL[5]			
1→2	0.069(0.038)	0.049(0.022)*	0.136(0.093)	0.093(0.077)
2→3	-0.105(0.057)	-0.088(0.054)	0.253(0.057)**	0.162(0.025)**
	Critical → CES-D			
1→2	1.555(0.847)	0.119(0.073)	-0.061(2.788)	-0.003(0.154)
2→3	-0.315(0.373)	-0.019(0.030)	0.930(3.429)	0.056(0.116)
	Critical → CBCL			
1→2	-0.042(0.388)	-0.004(0.037)	-0.182(1.174)	-0.011(0.073)
2→3	-1.904(0.503)**	-0.093(0.024)**	0.462(.862)	0.018(0.015)
	CBCL→ Critical			
1→2	0.018(0.010)	0.237(0.108)*	0.012(0.005)*	0.181(0.085)*
2→3	0.050(0.028)	0.490(0.270)	0.009(0.002)**	0.159(0.050)**
	CBCL → CES-D			
1→2	0.333(0.048)**	0.248(0.055)**	0.245(0.144)	0.232(0.142)
2→3	0.299(0.059)**	0.273(0.041)**	0.089(0.103)	0.081(0.081)
Lagged Effects	**CESD**			
1→3	0.666(0.112)**	0.431(0.059)**	0.133(0.184)	0.082(0.101)
	Critical			
	0.224(0.044)**	0.198(0.067)**	0.120(0.040)**	0.128(0.049)**
	CBCL			
	0.194(0.281)	0.121(0.233)	0.367(0.177)*	0.222(0.110)*

Note.[1] Beta value; [2] Standard Error; [3]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [4]Critical comments from the Five Minute Speech Sample (Magaña et al., 1986); [5]Child Behavior Checklist (Achenbach & Rescorla, 2001); * p <.05; ** p < .01.

Table 6.

Estimates of Indirect Pathways for Critical Comments Model

Time Point	Pathway	Mother-Report β[1]-value of Indirect Effect	SE[2]	Lower Bound of 95% CI[3]	Upper Bound of 95% CI	Father-Report β-value of Indirect Effect	SE	Lower Bound of 95% CI	Upper Bound of 95% CI
1→2→3									
	CBCL[4] →Critical[5]→ CES-D[6]	-.005	.013	-.024	-.011	.010	.018	-.016	.023
	CBCL → CES-D → Critical	-.012	.048	-.039	.068	**-.018**[*]	.008	-.025	-.011
	Critical→CBCL→CES-D	-.001	.010	-.005	.004	-.001	.008	-.014	.000
	Critical →CES-D→CBCL	-.011	.006	-.013	-.008	-.001	.024	-.033	.000
	CES-D→Critical→CBCL	.012	.007	.004	.015	.000	.002	-.004	-.003
	CES-D→CBCL→Critical	.024	.023	-.003	.029	**.015**[†]	.008	.003	.022

Note. Estimates are standardized values. [1] Beta-value; [2]Standard Error; [3]Confidence Interval; [4]Child Behavior Checklist (Achenbach & Rescorla, 2001); [5]Critical comments from the Five Minute Speech Sample (Magaña et al., 1986); [6]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [*] $p < .05$; [†] $p < .10$

Table 7.

Standardized and unstandardized path coefficients for mother- and father-reports of parent depression, parents' positive remarks, and child mental health problems

Time Point	Mother-report *B*[1](*SE*[2]), Unstandardized	Mother-report *β*(*SE*), Standardized	Father-report *β*(*SE*), Unstandardized	Father-report *β*(*SE*), Standardized
Cross Effects	**CES-D[3] → Positive[4]**			
1→2	0.013(0.075)	0.029(0.161)	-0.061(0.031)	-0.119(0.076)
2→3	-0.037(0.028)	-0.149(0.102)	-0.001(0.022)	-0.005(0.075)
	CES-D → CBCL[5]			
1→2	0.062(0.077)	0.048(0.065)	0.150(0.063)*	0.104(0.054)
2→3	-0.013(0.035)	-0.017(0.045)	0.050(0.030)	0.050(0.032)
	Positive → CES-D			
1→2	-0.150(0.171)	-0.038(0.046)	-0.046(0.130)	-0.013(0.042)
2→3	-0.149(0.259)	-0.044(0.082)	0.232(0.166)	0.074(0.059)
	Positive → CBCL			
1→2	-0.011(0.142)	-0.004(0.050)	-0.222(0.145)	-0.065(0.047)
2→3	-0.050(0.149)	-0.018(0.059)	-0.236(0.105)*	-0.076(0.042)
	CBCL→ Positive			
1→2	-0.002(0.030)	-0.006(0.077)	-0.005(0.047)	-0.014(0.140)
2→3	0.013(0.029)	0.039(0.082)	-0.027(0.020)	-0.096(0.072)
	CBCL → CES-D			
1→2	0.365(0.055)**	0.270(0.049)**	0.243(0.141)	0.228(0.107)*
2→3	0.225(0.072)**	0.191(0.051)**	0.150(0.047)**	0.136(0.033)**
Lagged Effects	**CESD**			
1→3	0.687(0.148)**	0.454(0.096)**	0.140(0.162)	0.087(0.105)
	Positive			
	-0.055(0.102)	-0.057(0.110)	0.378(0.146)*	0.388(0.102)**
	CBCL			
	0.407(0.115)**	0.400(0.109)**	0.397(0.169)*	0.370(0.148)*

Note.[1] Beta value; [2] Standard Error; [3]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [4]Positive remarks from the Five Minute Speech Sample (Magaña et al., 1986); [5]Child Behavior Checklist (Achenbach & Rescorla, 2001); * p <.05; ** p < .01.

Table 8.

Estimates of Indirect Pathways for Positive Remarks Model

Time Point	Pathway	Mother-Report β[1]-value of Indirect Effect	SE[2]	Lower Bound of 95% CI[3]	Upper Bound of 95% CI	Father-Report β-value of Indirect Effect	SE	Lower Bound of 95% CI	Upper Bound of 95% CI
1→2→3									
	CBCL[4]→Positive[5]→CES-D[6]	.000	.005	-.001	.004	-.001	.010	-.018	.000
	CBCL→CES-D→Positive	-.040	.027	-.051	-.034	-.001	.023	-.035	.008
	Positive→CBCL→CES-D	-.001	.010	-.017	.006	-.009	.006	-.017	-.008
	Positive→CES-D→CBCL	.001	.005	-.004	.009	-.001	.002	-.004	-.001
	CES-D→Positive→CBCL	-.001	.009	-.008	.003	.**009**[*]	.005	.010	.013
	CES-D→CBCL→Positive	.002	.003	-.003	.005	-.010	.016	-.038	-.003

Note. Estimates are standardized values. [1] Beta-value; [2]Standard Error; [3] Confidence Interval; [4]Child Behavior Checklist (Achenbach & Rescorla, 2001); [5]Positive remarks from the Five Minute Speech Sample (Magaña et al., 1986); [6]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [*] $p < .05$

Figures

Figure 1.

Conceptual Model of Complete Longitudinal Mediation Model

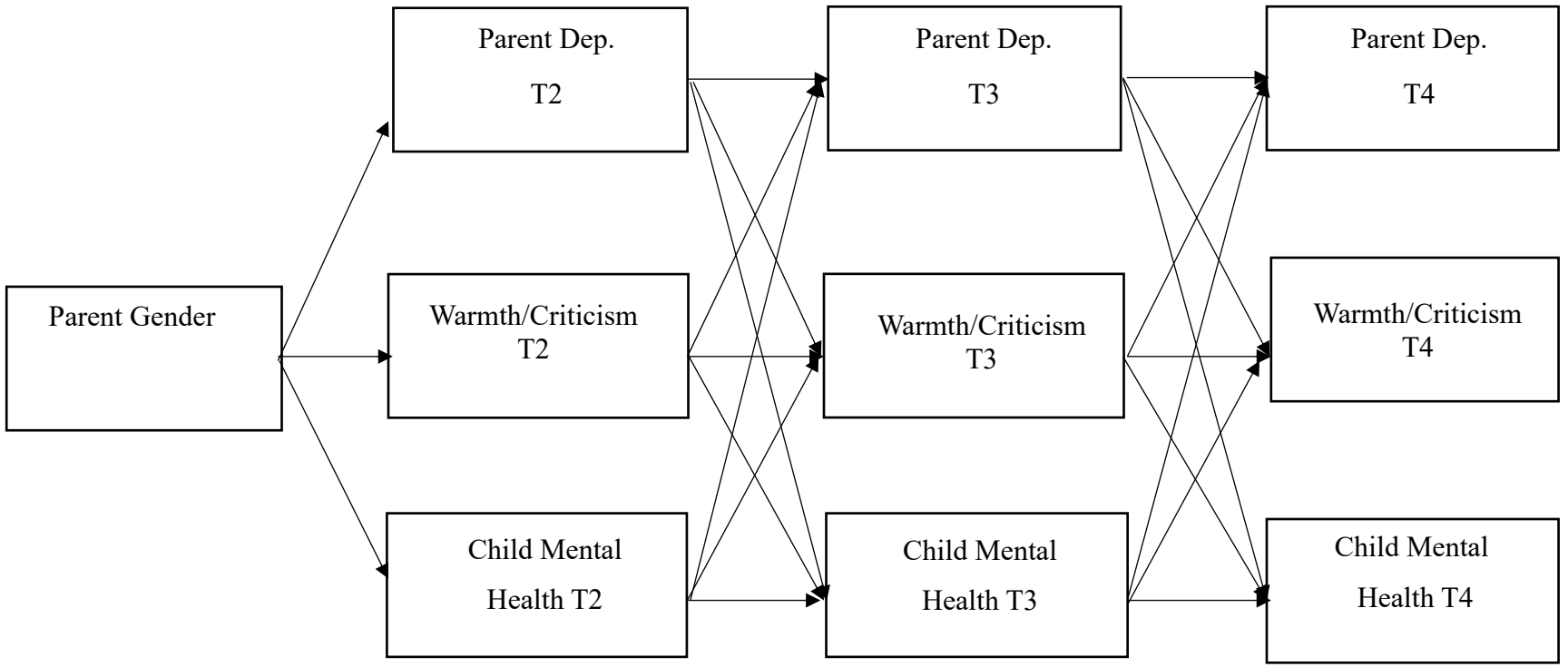
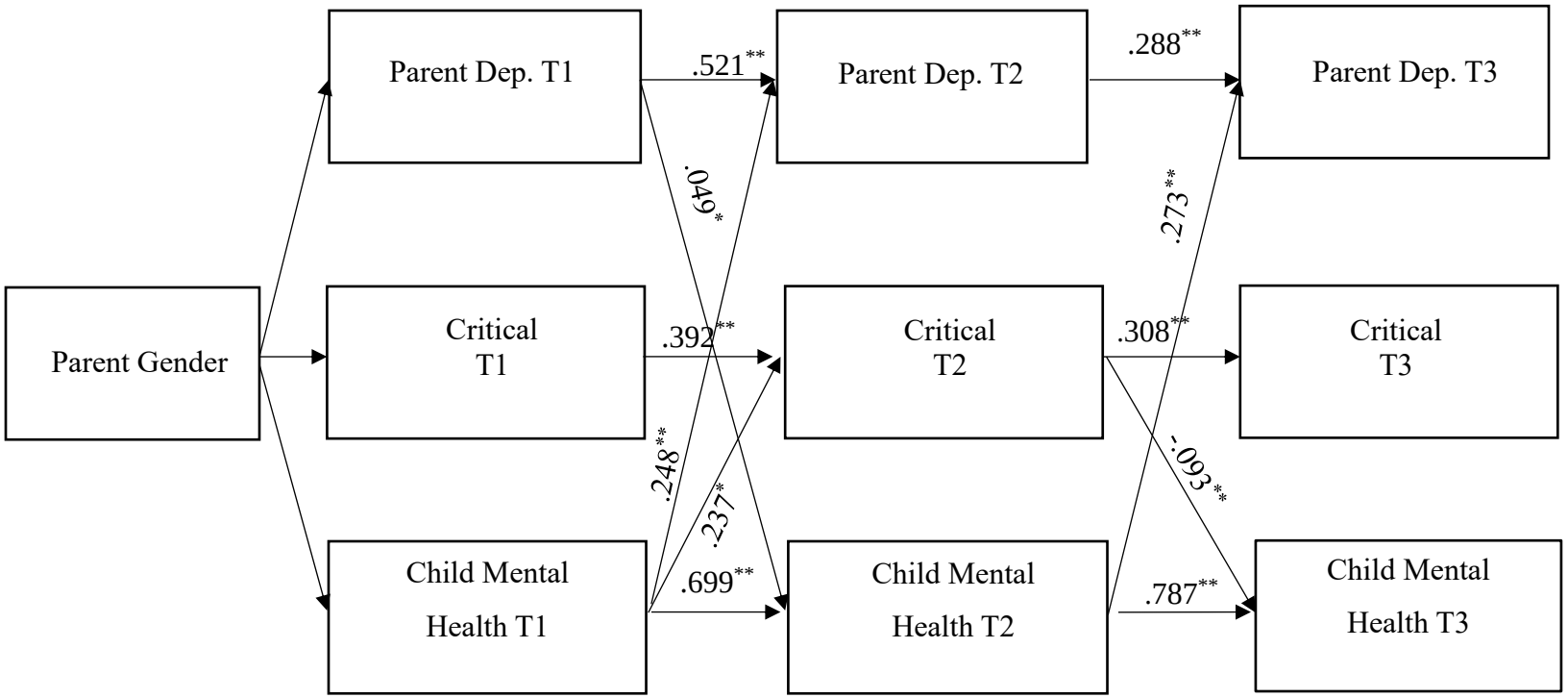


Figure 2.

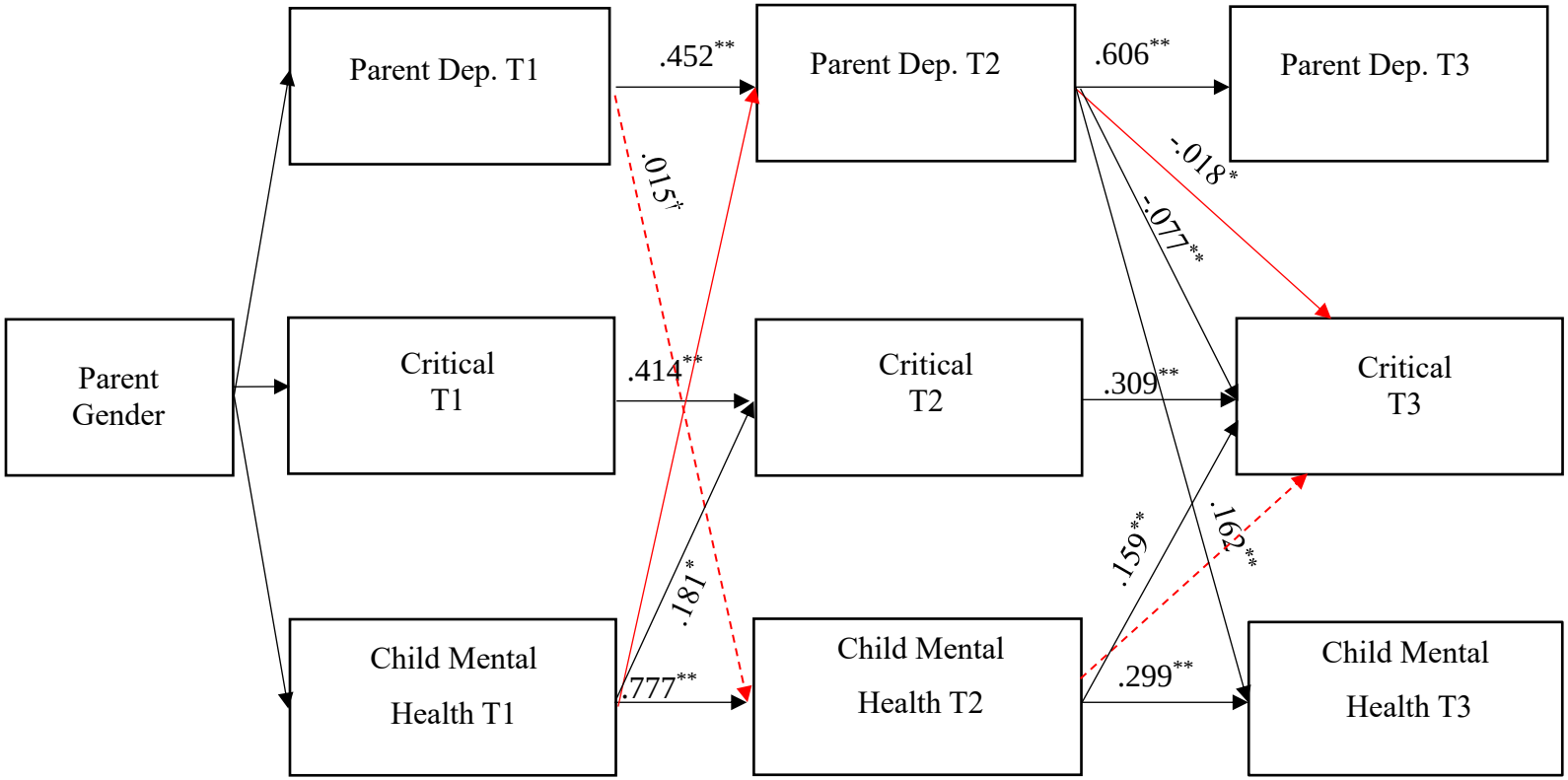
Complete longitudinal mediation model for mother-report of parent depression, parents' critical comments, and child mental health



Note. Results of the complete longitudinal mediation model for mother-report of parent depression symptoms, critical comments, and child mental health problems, controlling for parent age, household income, and child intellectual disability status. Values are standardized path estimates. Lagged paths are excluded from figure for simplicity. * p <.05; ** p < .01.

Figure 3.

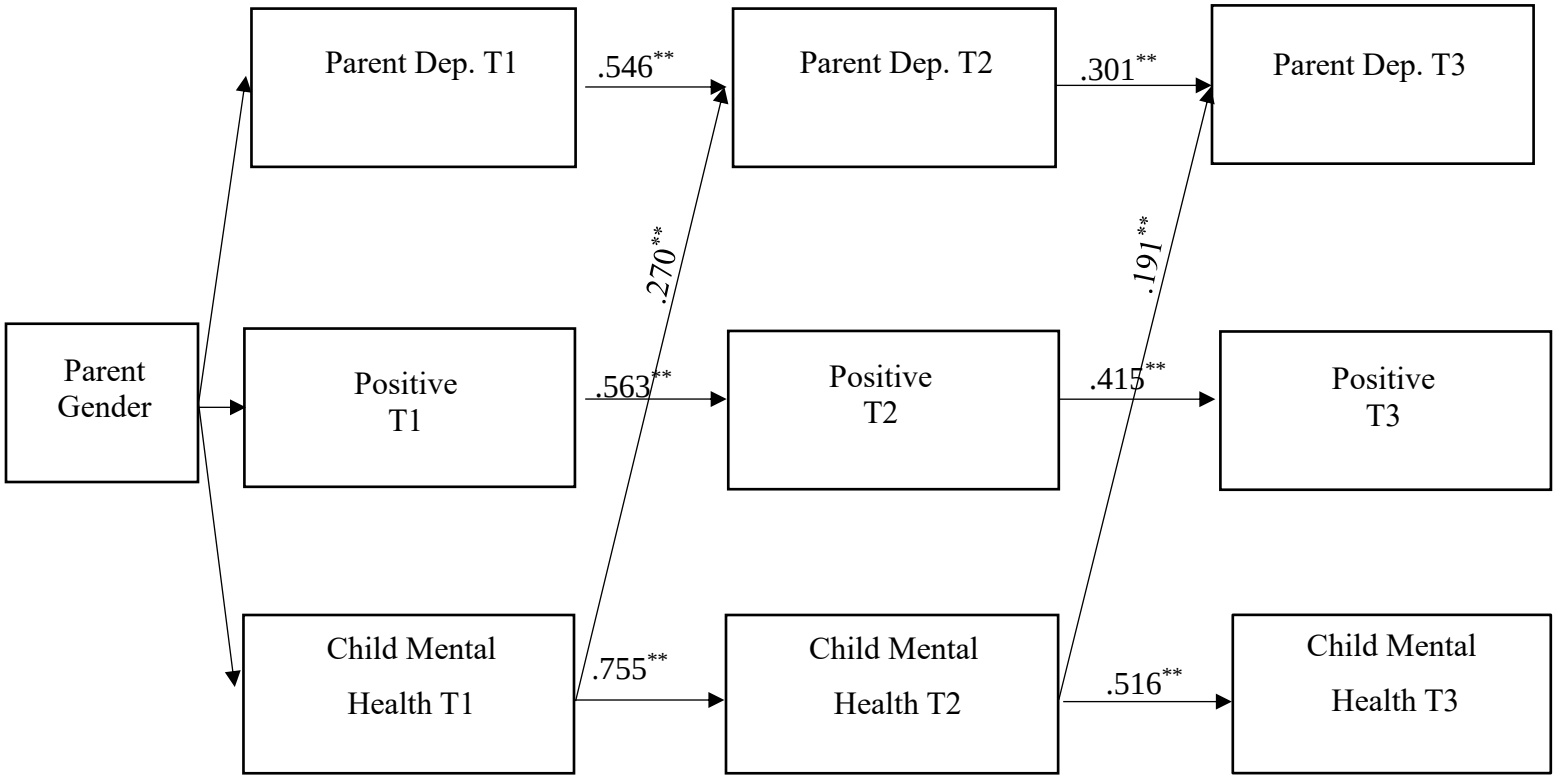
Complete longitudinal mediation model for father-report of parent depression, parents' critical comments, and child mental health



Note. Results of the complete longitudinal mediation model for father-report of parent depression symptoms, critical comments, and child mental health problems, controlling for parent age, household income, and child intellectual disability status. Values are standardized path estimates. Lagged paths are excluded from figure for simplicity. Red solid lines indicate significant *indirect* effects. Red dotted lines indicate trend level *indirect* effects. * p <.05; ** p < .01; $^{\dagger}p$ <.10.

Figure 4.

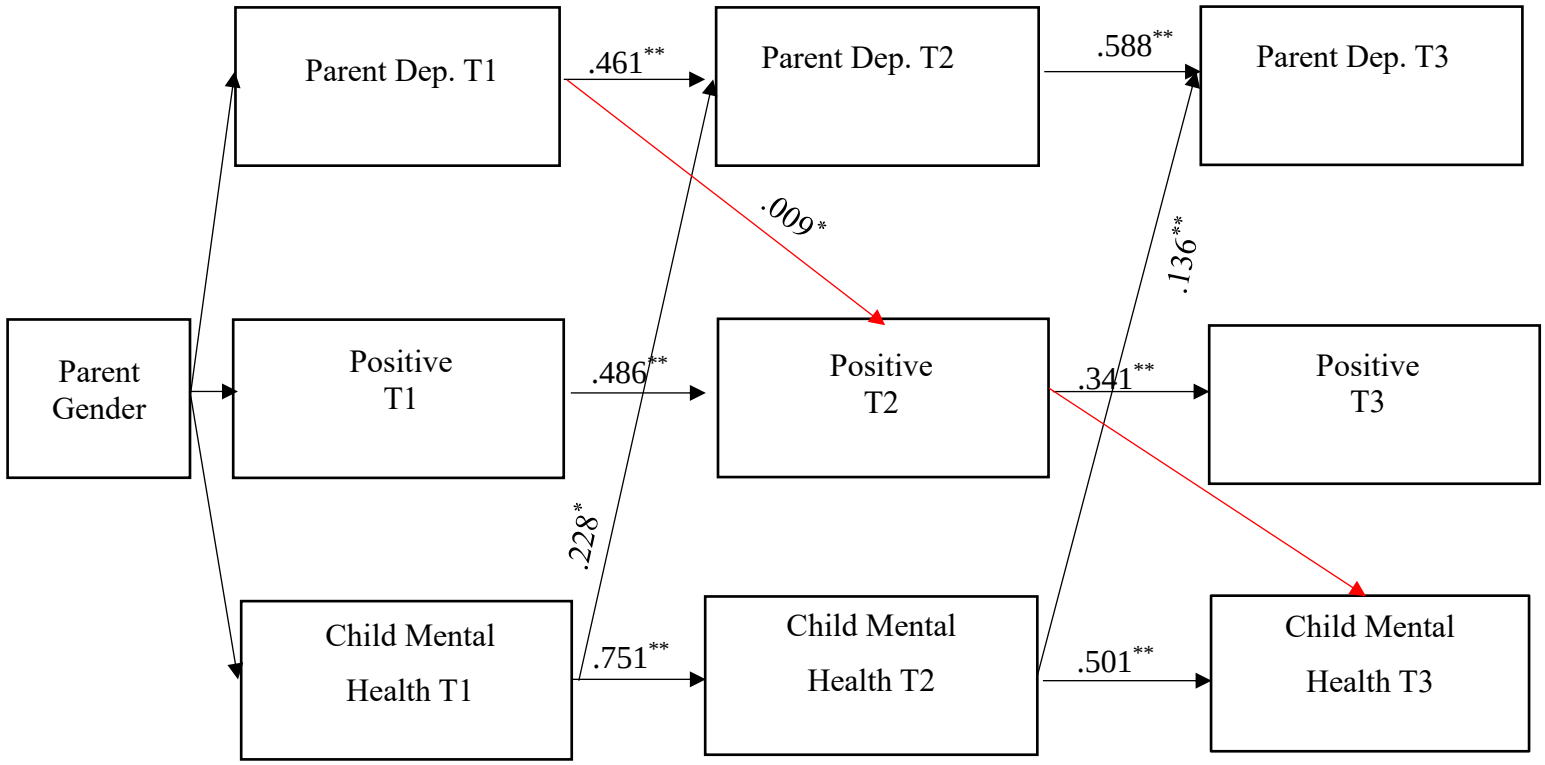
Complete longitudinal mediation model for mother-report of parent depression, parents' positive remarks, and child mental health



Note. Results of the complete longitudinal mediation model for mother-report of parent depression symptoms, positive remarks, and child mental health problems, controlling for parent age, household income, and child intellectual disability status. Values are standardized path estimates. Lagged paths are excluded from figure for simplicity. ** $p < .01$.

Figure 5.

Complete longitudinal mediation model for father-report of parent depression, parents' positive remarks, and child mental health



Note. Results of the complete longitudinal mediation model for father-report of parent depression symptoms, positive remarks, and child mental health problems, controlling for parent age, household income, and child intellectual disability status. Values are standardized path estimates. Lagged paths are excluded from figure for simplicity. Red lines indicate significant *indirect* pathway; * p <.05; ** p < .01.

Abstract: Manuscript 3

Within families, the parent-couple subsystem (marital or romantic partner relationship) is posited to shape the mental health of both parents and children. Both children and parents within families of autistic children are at-risk for poor mental health outcomes compared to families of typically developing children. The role of the parent-couple relationship in mediating within-family associations between parent and child mental health within families of autistic families remains unknown. Using four time points of data collected on a sample of 188 families of autistic children (aged 5-12), the present study investigated the time-ordered bidirectional associations between parent-couple relationship satisfaction, parent depression, and child internalizing and externalizing mental health problems. Two multi-group (grouped by parent gender) complete longitudinal mediation models in structural equation modeling were conducted. Results indicated that parent-couple relationship satisfaction mediated: 1) the association between parent depression and child internalizing mental health problems for both mothers and fathers, and 2) the association between child externalizing mental health problems and father depression. Father depression also mediated the link between parent-couple satisfaction and child internalizing mental health problems, and mother depression mediated the connection between child externalizing mental health problems and parent-couple satisfaction, across certain timepoints. Findings highlight the complex ways that parent and child mental health and parent-couple relationship are entwined in mutually influential ways across time in families of autistic children. Family-wide interventions that address the needs of multiple family members and family systems may be best suited to improve the mental health of parents and autistic children.

Keywords: autism, parent-couple relationship, marital satisfaction, child mental health problems, depression

Chapter 3: Manuscript 3

Parent-couple satisfaction and child and parent mental health in families with autistic children

Within the family systems framework (Cox and Paley, 1997), the quality of the parent-couple relationship (i.e., marital or romantic partner relationship) is theorized to be intertwined with the mental health of both parents (Shi & Whisman, 2023; Yang, Yang, & Yang, 2023) and children (Du et al., 2022; Matalon et al., 2021). Parents of autistic children are, on average, at risk for short-term and unsatisfying parent-couple relationships relative to other parents (Berg et al., 2016; Gau et al., 2012; Sim et al., 2016). Making up this average, however, is a wide range of experiences, including many parents of autistic children reporting highly satisfying and long-lasting couple relationships (Ekas et al., 2015). To-date, there are no published longitudinal studies on the role of the parent-couple relationship, a potentially modifiable intervention target, for shaping the mental health of parents and their autistic child at a within-family level across time. Yet, this information is of high public health importance given that 1 in 36 children in the U.S. meet criteria for autistic spectrum disorder (Centers for Disease Control and Prevention, 2023). To address this gap in research, the present study examined the bidirectional connections between parent-couple relationship satisfaction, parent depression, and the mental health problems of autistic children across 3 years.

The family systems framework posits that family members and subsystems within the family continuously influence one another (Cox & Paley, 1997; Cummings et al., 2000a; Kerig, 2016; Restifo & Bögels, 2009; Werner et al., 1992). The parent-couple relationship is often depicted at the center of the family system (e.g., Cummings & Davies, 1994; Cummings et al., 2000b). Findings from research have supported the posited bidirectional connections between the

quality of the parent-couple relationship and parent and child mental health and suggest that the parent-couple relationship may be a mediating conduit through which parent and child mental health influence one another. Indeed, parent-couple relationship dissatisfaction has been linked to greater levels of parent depression both cross-sectionally and three years later (Shi & Whisman, 2023), and a satisfying parent-couple relationship is thought to buffer parents from parenting stress by serving as a source that provides emotional support (Shi & Whisman, 2023; Thoits, 2011). In a bi-directional manner, mental health also impacts couple-satisfaction. For example, one's own and one's partner's level of depression and anxiety negatively predict level of parent-couple relationship satisfaction (e.g., Whisman, Uebelacker, & Weinstock, 2004; Yang, Yang, & Yang, 2023). A depressed parent often withdraws from their partner, leading to distance in the relationship, and/or becoming more irritable and hostile to their partner, engaging in destructive strategies such as defensiveness and personal insult which foster parent-couple conflict (Davies & Cummings, 1994; Keller et al., 2009) and ultimately, lower parent-couple relationship satisfaction (Johnson et al., 2018).

Parent couple satisfaction is also associated with child mental health in general population samples (e.g., Nagaraja et al., 2012; Du et al., 2022). Children who are exposed to maladaptive parent-couple behaviors (e.g., destructive parent-couple conflict) or otherwise perceive that their parents are unhappy in their parent-couple relationship are at risk for internalized mental health problems (e.g., feeling anxious and emotionally insecure) (Cummings et al., 2000b; Davies & Cummings, 1994; Du et al., 2022; Stutzman et al., 2011). For example, a recent study found that if mothers had higher parent-couple relationship satisfaction, their child perceived their family as functioning better, and in turn, had fewer depression symptoms (Du et al., 2022).

In the opposite direction, there is evidence that the parenting stressors, including from child mental health problems (e.g., Barroso et al., 2018; Matalon et al., 2021; Robinson & Neece, 2014), can contribute to a decrease in parent-couple relationship satisfaction (e.g., Jenkins et al., 2005; Keizer & Schenk, 2012; Vannier et al., 2018). Indeed, child mental health problems are theorized to affect the parent-couple subsystem in ways that make parents vulnerable to depressed affect if adaptive couple coping skills are not developed (Benson, 2014, 2018; Hastings et al., 2005). There is evidence from families of typically developing (TD) children that the unique difficulties related to parenting a child with mental health problems, and particularly externalizing mental health problems such as aggressive and disruptive behaviors, often contribute to feelings of parenting stress, fatigue, and depressed affect (e.g., Stone et al., 2016). In turn, parents may have limited capacity to engage with their partner in an engaged and affectionate manner (e.g., Crnic & Ross, 2017). Parent-couple relationship dissatisfaction that arises from this tension has been linked to parent depression (e.g., Choi & Marks, 2008).

Autism spectrum disorder is a lifelong neurodevelopmental disorder characterized by challenges related to speech and nonverbal communication, social interactions, and repetitive and restricted behaviors that interfere with everyday functioning (American Psychological Association, 2020). Autism is also associated with a plethora of co-occurring mental health problems including both internalizing (i.e., anxious or depressed mood) and externalizing (i.e., disruptive and aggressive behavior) problems (Bauminger et al., 2010; Hudson et al., 2019; Vasa & Mazurek, 2015). Indeed, 14-20% of autistic children experience at least one depressive episode before the age of 18 years (Upthegrove et al., 2018), 40% endure clinically elevated anxiety symptoms (van Steensel et al., 2011), 63% have co-occurring attention deficit hyperactivity disorder (Avni et al., 2018), and approximately 25% are reported to exhibit an

aggressive behavior problem (Hill et al., 2014). Other common co-occurring challenges include oppositional defiant disorder and conduct disorder (Kaat & Lecavalier, 2013). Parents of autistic children also face a higher risk for depression compared to parents of TD children (Cohrs & Leslie, 2017).

Parents of autistic children are at risk for negative parent-couple outcomes, including lower parent-couple relationship satisfaction (Gau et al., 2012; Sim et al., 2016), perceptions of decreased partner support and affection (Brobst et al., 2009), and higher rates of divorce or separation (Berg et al., 2016; Hartley et al., 2010) when compared to parents of TD children. Negative parent couple relationship experiences have been shown to have connections to both parent and child mental health in families of autistic children. Indeed, parents of autistic children with more mental health problems report lower parent-couple relationship satisfaction (Benson, 2006; Brown et al., 2020) and more negative and fewer positive parent-couple interactions (Hartley et al., 2018) than do parents of autistic children with fewer mental health problems. Moreover, higher parent-couple conflict and/or lower parent-couple relationship satisfaction predicts increased depression symptoms in mothers of autistic children (e.g., Lickenbrock et al., 2011). Recent evidence also suggests that autistic children exhibit greater levels of negative emotional, behavioral, and physiological responses in the face of parent-couple conflict than TD children (Ekas & Kouros, 2021), and thus may be especially sensitive to the negative impacts of maladaptive parent-couple relationships. However, to date, there are no published longitudinal studies examining the time-ordered role of the parent-couple relationship as conduit for the within-family connections between parent depression and the mental health of autistic children.

The goal of the current study was to understand whether parent-couple relationship satisfaction explains, in part, the relation between parent depression and child mental health

problems in families with autistic children. A total of 188 mothers and fathers of autistic children independently completed questionnaires assessing their own and their child's mental health, as well as their level of parent-couple relationship satisfaction. There were two study aims: 1) examine the association between parent-couple relationship satisfaction and parent depression and child mental health problems; and 2) determine whether parent-couple relationship satisfaction mediates the association between parent depression and child mental health problems. Drawing from research on TD populations and the family systems theoretical frameworks, we hypothesized that: 1) parent depression would predict decreased parent-couple relationship satisfaction 12 months later; 2) child mental health problems would also predict decreased parent-couple relationship satisfaction 12 months later; 3) parent-couple relationship satisfaction would mediate the association between parent depression and child mental health problems across time. Based on findings from families of TD children (e.g., Crnic & Ross, 2017; Du et al., 2022; Stone et al., 2016), child externalizing (vs. internalizing) mental health problems were hypothesized to have the strongest effects on parent depression and parent-couple relationship satisfaction, whereas parent depression and decreased parent-couple relationship satisfaction was expected to increase child internalizing (versus externalizing). Given the longitudinal nature of the data set (T1-T4), we focused on the mediation effect of parent-couple relationship satisfaction at T2 and T3 on associations between child mental health and parent depression from T1-T4. Thus, primary hypothesized pathways were: a) higher parent depression at T1 → decreased parent-couple relationship satisfaction at T2 → increased child mental health problems at T3; and b) higher child mental health problems at T1 → decreased parent-couple relationship satisfaction at T2 → increased parent depression at T3. Similar pathways are hypothesized from T2-T3-T4.

Methods

The current study used data from T1-T4 of the Family Outcomes in Autism Spectrum Disorder Study (R01MH199091; Hartley). IRB approval was obtained through the University of Wisconsin-Madison, and all parents provided informed consent before participating. At T1, 188 parent-couples participated in the study. In order to participate, parents had to have at least one autistic child (the target child) between the age of 5-12 years. If there was more than one autistic child within the family, the oldest child (within the 5-12 year age range) was deemed the target child because this represented when parents initially began parenting an autistic child. A variety of techniques were utilized in order to recruit participants, including research registries, information distributed within ASD clinics, and fliers placed throughout the community and schools. The autistic child had to have a medical or educational diagnosis of ASD which included the autistic diagnostic observation schedule (ADOS-2nd edition; Lord et al., 2012). The child's current level of ASD symptom severity was measured through parent-report of the Social Responsiveness Scale-2nd Edition (SRS-2; Constantino & Gruber, 2012). The SRS total t-score needed to be greater than 60 to participate in the present study. Five autistic children did not meet this threshold (received a t-score at or below 60), but a review of medical and educational records as well as ADOS scores revealed that these children did indeed meet criteria for ASD. As a result, they were included in the analyses. Additional inclusion criteria were as follows: 1) both parents had to agree to participate in the study; 2) parents had to be at least 21 years of age; 3) the parent-couple had to live in a cohabiting relationship for 3 years of greater. For further demographic information about the families, see Table 1.

Procedure

Mothers and fathers participated in a 2.5-hour study visit that took place either at their home or in a research lab at each time point, spaced approximately 12 months apart. Parents jointly answered sociodemographic questions and then independently reported on parent depression symptoms, child mental health problems, and level of parent-couple relationship satisfaction. Each parent was paid $50 for completing this portion of the study.

Measures

Family sociodemographics. Together, parent-couples answered questions regarding family sociodemographics. Parent information included: (A) gender (mothers = 1, fathers = 2); (B) parent age (years); (C) household income (1 = $1-$9,999 to 14 = $160,000+). Additionally, parents also reported on child presence or absence of an intellectual disability (ID) as determined through either IQ testing or a formal ID diagnosis (0 = no ID, 1 = ID).

Parent depression symptoms. The 20-item Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977) was separately completed by mothers and fathers. Parents rated each item on a 4-point scale with 0 indicating rarely or none of the time to 3 indicating most or all of the time. Example items from the CES-D include “I thought my life had been a failure” and “I felt that everything I did was an effort.” A total score greater than or equal to 16 indicates clinically significant depression symptoms (Radloff, 1977). The CES-D revealed high internal consistency in mothers (Chronbach’s α = .92-.93) and fathers (Chronbach’s α = .89-.93) across T1-T4. For the means, standard deviations, and *t*-values for mother- and father-reported CES-D total scores across time, see Table 2.

Child mental health problems. The Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2000, 2001) preschool form (ages 1.5-5 years) and school age form (ages 6-18 years) were utilized in order to assess child mental health problems. Parents complete this 113-item

questionnaire by separately rating each item on a 3-point scale (0 = not true to 2 = very or often true). The current study utilized the internalizing and externalizing *t*-score in model analyses. The CBCL internalizing scale consists of 32 items and is separated into three subscales: 1) anxious/depressed; 2) withdrawn/depressed; 3) somatic complaints. Example items include, "Feels worthless or inferior," "There is very little he/she enjoys," and "Overtired without good reason." The CBCL externalizing scale is composed of 35 items and is broken into two categories: 1) rule-breaking behavior; 2) Aggressive behavior. Example items include, "Doesn't seem to feel guilty after misbehaving," "Destroys things belonging to his/her family or others," and "Sudden changes in mood or feelings." The CBCL is highly reliable within the ASD population (Pandolfi et al., 2014), and had a high internal consistency across T1-T4 for both mothers (internalizing: Chronbach's α = .84 to .85; externalizing: Chronbach's α = .90 to .92) and fathers (internalizing: Chronbach's α = .82 to .86; externalizing: Chronbach's α = .89 to .90) in the present study. Table 2 provides means, standard deviations, and *t*- values for mother- and father-reports of the CBCL internalizing and externalizing t-scores across time.

Parent-couple relationship satisfaction. The Couple Satisfaction Index (CSI; Funk & Rogge, 2007) assessed parent-couple relationship satisfaction. This 32-item questionnaire is broken into a series of 6-point scales, with higher numbers representing greater parent-couple relationship satisfaction. An example item is, "In general, how often do you think that things between you and your partner are going well? [5 = all of the time to 0 = never]. A score of 104.5 or below indicates relationship dissatisfaction. For the present study, the number of mothers scoring below the CSI cutoff of 104.5 was as follows: T1) *n* = 59; T2) *n* = 56; T3) *n* = 45; T4) *n* = 30. The number of fathers scoring below the CSI cutoff was: T1) *n* = 62; T2) *n* = 44; T3) *n* = 33; T4) *n* = 30. The CSI had high internal consistency in the present study for both mothers

(Chronbach's α = .98 to .99) and fathers (Chronbach's α = .97 to .98) across T1-T4 and has been used in previous research for parents of autistic children (Sim et al., 2017).

Data Analysis Plan

Boxplots and descriptive statistics were used to understand the distribution of the data. An attrition analysis was used to determine if families who completed all time points differed from families with missing data on at least 1 time point. A series of bivariate Pearson correlations allowed us to examine the associations among the main study variables and with family sociodemographics. Family sociodemographics significantly associated with independent or dependent variables were included as covariates in primary analyses. Specifically, the independent and dependent variables were regressed on the relevant significant family sociodemographic variables and the unstandardized residual scores were saved and entered in the structural equation model (SEM).

For the present study, the primary model was a multi-group complete longitudinal mediation model conducted in SEM using Mplus statistical software (Muthen & Muthen, 2012); the recommended software for this type of mediation model (Jose, 2016). Data was from T1-T4 of the Family Outcomes in ASD study. For a conceptual model, see Figure 1. The model was grouped by parent gender, providing separate results for mother- and father reported measures, and investigated the impact of parent-couple satisfaction on the parent and child mental health connection. The recommended 10:1 ratio of cases/observations to estimated parameters (Kline, 2015; Nunnally, 1967) suggests that a minimum total sample size of 360 is needed to detect an acceptable sample size, or a minimum of 100 mothers and 100 fathers is needed for each group. By using a complete longitudinal mediation model rather than a focused model, we are able to analyze all potential longitudinal associations. In other words, the complete model provides for

the examination of a multitude of potential mediations, allowing us to explore both parent and child-driven pathways across the data collection time points (Jose, 2016). Bootstrapping with 5,000 iterations was included in the models, aligning with best practices for obtaining confidence intervals for indirect effects (Jose, 2013). Examining confidence intervals for the indirect effects allowed us to determine if significant mediations exist within the model. The Tucker-Lewis Index (TLI), comparative fit index (CFI), and the root mean square error of approximation (RMSEA) were examined in addition to the chi-square (χ^2). A good model fit includes CFI and TLI values greater than .90, an RMSEA value between .05 and .08, and a small χ^2 value (Hu & Bentler, 1999; Little, 2013). Missing data was accounted for via the full information maximum likelihood method, a robust estimator in SEM (Little, 2013; Schlomer, Bauman, & Card, 2010).

Results

Preliminary Analyses

Normality tests revealed that parent depression and child mental health problems were normally distributed (kurtosis range for CES-D = .978 to 2.467; CBCL internalizing = -.212 to 2.321; CBCL externalizing = -.550 to .096). MCAR tests revealed that data was missing completely at random (X^2 = 6.449, p > .05). At T1, 188 families provided data from both parents (N = 376 parents). Of these families, at T2, data from both parents was available from 155 families. Seven additional families provided mother-report only, and 1 family provided father-report only, for a total of 163 families (N = 318 parents). Data from both parents was available from 131 families at T3. Seven additional families provided mother-report only, and 2 families provided father-report only, for a total of 140 families (N = 271 parents). At T4, data from both parents was available for 117 families. Eight additional families provided mother-report only, and 5 additional families provided father-report only for a total of 130 families (N = 247

parents). Participants lost to attrition indicated moving or not having enough time at the present study cycle to participate as main reasons for leaving the study. Table 1 provides sociodemographic information for the sample.

Attrition analyses were conducted to determine whether families who completed the study at all four cycles (coded 1 for "completers") differed from families who had missing data at one or more study cycles (coded 2 for "incompleters"). Independent t-tests indicated that reports of parent depression (ts: -1.388 to .177, p >.05) and child mental health problems (internalizing ts: -.883 to .976, p >.05; externalizing ts: -1.442 to .422, p >.05) did not differ significantly for "completers" versus "incompleters." At T1 (t(373) = 2.266, p = .012) and T4 (t(227) = 1.789, p = .037), "completers" reported greater parent-couple satisfaction than "incompleters.". Additionally, parent age at T1 was significantly different for "completers" versus "incompleters" (t(372) = -2.089, p = .019), with "completers" being slightly younger (M = 39.19, SD = 5.60) than "incompleters" (M = 40.51, SD = 6.48). There were no significant differences between the two groups for household income.

Paired-sample t-tests indicated that mother- and father-reports of child internalizing mental health problems were statistically different at T3 (t (131) = 2.317, p = .022), with mothers reporting higher levels of child internalizing problems than fathers. There were no differences between mother- and father-report of child externalizing mental health problems (ts: -.468 to .629, p >.05). Mothers and fathers did, however, report statistically different parent depression scores (T1: t(184) = 2.289, p = .023; T2: t(154) = 3.029, p = .003; T3: t(130) = 3.103, p = .002; T4: t(116) = 2.199, p = .030), with mothers reporting higher levels of depression symptoms than fathers at each time point. Mothers and fathers did not report significantly different parent-couple

satisfaction scores (ts: -1.470 to .049, $p > .05$). Means, standard deviations, and within-couple differences between mother- and father-reports are provided in Table 2.

Results from bivariate Pearson correlations between child internalizing and externalizing mental health problems, parent depression, parent-couple satisfaction, and sociodemographics can be found in Table 3. Parent depression and child mental health problems were positively significantly associated for both mother- (T1-T4 CES-D with T1-T4 internalizing rs*:* .191 to .416, $p < .05$; T1 CES-D with T1-T4 externalizing rs: .277 to .371, $p < .001$; T2 CES-D with T1 externalizing: $r = .158$, $p = .045$; T2 externalizing: $r = .185$, $p = .019$; T3 externalizing: $r = .218$, $p = .012$; T3 CES-D with T1-T4 externalizing: rs: .311 to .419, $p < .001$; T4 CES-D with T1-T4 externalizing: rs: .241 to .320, $p < .05$) and father-report (T1 CES-D with T1-T4 internalizing: rs: .289 to .436, $p < .001$; T2 CES-D with T4 internalizing: $r = .198$, $p = .031$; T3 CES-D with T1-T4 internalizing: rs: .229 to .316, $p < .05$; T4 CES-D with T1-T4 internalizing: rs: .207 to .338, $p < .05$; T1 CES-D with T1-T4 externalizing: rs: .288 to .364, $p < .001$; T3 CES-D with T1-T4 externalizing: rs: .239 to .371, $p < .05$; T4 CES-D with T1-T4 externalizing: rs: .202 to .265, $p < .05$). Parent-couple satisfaction was significantly negatively associated with child mental health problems for both mother- (T1 CSI with T2-T4 externalizing: rs: -.257 to -.157, $p < .05$; T2 CSI with T2 internalizing: $r = -.189$, $p = .017$; T4 internalizing: $r = -.278$, $p = .002$; T1-T4 externalizing: rs: -.264 to -.183, $p < .05$; T3 CSI with T3 internalizing: $r = -.176$, $p = .042$; T4 internalizing: $r = -.248$, $p = .007$; T2-T4 externalizing: rs: -.301 to -.225, $p < .05$; T4 CSI with T2-T4 externalizing: rs: -.288 to -.204) and father-reports (T1 CSI with T3 internalizing: $r = -.259$, $p = .003$; T4 internalizing: $r = -.307$, $p < .001$; T1 externalizing: $r = -.169$, $p = .021$; T3 externalizing: $r = -.270$, $p = .002$; T4 externalizing: $r = -.199$, $p = .026$; T2 CSI with T1-T4 internalizing: rs: -.343 to -.177, $p < .05$; T1-T3 externalizing: rs: -.273 to -.204, $p < .05$; T3 CSI

with T2 internalizing: r = -.239, p = .007; T4 internalizing: r = -.329, p <.001; T1-T4 externalizing: rs: -.282 to -.190, p <.05; T4 CSI with T1 internalizing: r = -.184, p = .050; T3 internalizing: r = -.460, p <.001; T4 internalizing: r = -.253, p = .007; T1 externalizing: r = -.261, p = .005; T3 externalizing: r = -.311, p = .001; T4 externalizing: r = -.214, p = .022). Similarly, parent-couple satisfaction was significantly negatively correlated with parent depression symptoms for mother-report (rs: -.444 to -.218, p < .05) across all time points, and for father-reports (T1 CSI with T1 CES-D: r = -.344, p <.001; T3 CES-D: r = -.336, p <.001; T4 CES-D: r = -.267, p = .003; T2 CSI with T1-T4 CES-D: rs: -.522 to -.330, p <.05; T3 CSI with T1-T4 CES-D: rs: -.487 to -.250, p <.05; T4 CSI with T1 CES-D: r = -.312, p <.001; T3 CES-D: r = -.495, p <.001; T4 CES-D: r = -.459, p <.001).

Parent age was associated with father-report of child mental health problems (T1 internalizing: r = .147, p =.046; T1 externalizing: r = .162, p =.027; T3 externalizing: r = .209, p = .016). Household income was significantly associated with both mother-report (T2: r = -.172, p = .032) and father-report (T1: r = -.164, p = .028; T2: r = -.170, p = .036; T3: r = -.180 p = .041; T4: r = -.252, p = .006) of parent depression. Child ID status was associated with father-report of child externalizing mental health problems (T1 externalizing: r = .155, p =.033). Due to the significant correlations with the sociodemographic variables, the complete-longitudinal mediation models controlled for parent age, household income, and child ID status. We regressed the main study variables (parent depression, child internalizing mental health problems, child externalizing mental health problems, and parent-couple relationship satisfaction) on parent age, household income, and child ID at each time point, and the unstandardized residual scores were entered into the mediation models.

Complete-Longitudinal Mediation Models

Path coefficients of the direct and indirect pathways for the child internalizing mental health problems model can be found in Tables 4 and 5, respectively. Tables 6 and 7 provide the direct and indirect pathway coefficients for the child externalizing mental health problems model. Figure 1 provides a conceptual model illustrating all possible effects that were analyzed. Figures 2 and 3 illustrate the complete-longitudinal mediation models for exploring mother- and father-reported child internalizing mental health problems, respectively. Figures 4 and 5 depict the complete-longitudinal mediation models for mother- and father-reported child externalizing mental health problems, respectively.

Internalizing Mental Health Problems Model

The child internalizing mental health problems model indicated good model fit (X^2 (36) = 36.732, p = 0.4348; RMSEA = .011; TLI = 0.999; CFI = 1.000). Stability effects were present for mother- and father-report of parent depression (Mother: T1-T2 β = 0.561, p = .000; T2-T3 β = 0.599, p = .000; T3-T4 β = 0.302, p = .000; Father: T1-T2 β = 0.402, p = .000; T2-T3 β = 0.511, p = .000; T3-T4 β = 0.696, p = .000) across time. Stability effects were also present for parent-couple relationship satisfaction (Mother: T1-T2 β = 0.778, p = .000; T2-T3 β = 0.553, p = .001; T3-T4 β = 0.711, p = .000; Father: T1-T2 β = 0.713, p = .000; T2-T3 β = 0.506, p = .000; T3-T4 β = 0.545, p = .002) as well as for child internalizing mental health problems (Mother: T1-T2 β = 0.600, p = .000; T2-T3 β = 0.488, p = .000; T3-T4 β = 0.504, p = .000; Father: T1-T2 β = 0.560, p = .000; T2-T3 β = 0.494, p = .000; T3-T4 β = .535, p = .000).

Mother-report of parent depression symptoms significantly directly predicted parent-couple relationship satisfaction across the time points (T1 CES-D to T2 CSI: β = -.090, p = .002; T2 CES-D to T3 CSI: β = -.105, p = .018; T3 CES-D to T4 CSI: β = .124, p = .010). Mother-report of parent depression symptoms at T1 also directly predicted child internalizing mental

health problems at T2 (β = .187, p = .000). Mother-report of parent-couple relationship satisfaction at T3 directly predicted both parent depression symptoms (β = .145 p = .005) and child internalizing mental health problems (β = -.172, p = .000) at T4. Additionally, Mother-report of child internalizing mental health problems at T3 directly predicted parent depression symptoms at T4 (β = .148, p = .019). Mother-report revealed one significant indirect pathway (β = .018, p = .032; CI [.005, .027]), indicating that parent depression symptoms at T2 predicted parent-couple relationship satisfaction at T3, which then predicted child internalizing mental health problems at T4. This finding provides support that parent-couple relationship satisfaction may partially mediate the association between parent depression and child internalizing mental health problems.

Father-report of parent depression symptoms at T3 directly predicted child internalizing mental health problems at T4 (β = .092, p = .000). Father-report of parent-couple relationship satisfaction directly predicted parent depression (T2 CSI to T3 CES-D: β = -.319, p = .001; T3 CSI to T4 CES-D: β = -.075, p = .014) as well as child internalizing mental health problems (T2 CSI to T3 CBCL: β = -.151, p = .000). Two significant indirect pathways were found. First, parent depression symptoms at T2 predicted parent-couple relationship satisfaction at T3, which then predicted child internalizing mental health problems at T4 (β = .004, p = .020; CI [.000, .003]). This pathway suggests that parent-couple relationship satisfaction may serve as a mediator for the association between parent depression and child internalizing mental health problems. Second, father-report of parent-couple relationship satisfaction at T2 predicted parent depression at T3, which then predicted child internalizing mental health problems at T4 (β = -.029, p = .005; CI [-.039, -.018]), indicating that parent depression partially mediated the

association between parent-couple relationship satisfaction and child internalizing mental health problems.

Secondary Analyses. Mother-report revealed two unexpected findings which were examined in secondary analyses. There was a significant *positive* direct effect of T3 parent depression symptoms predicting T4 parent-couple relationship satisfaction (β = .124, p = .010). Further examination suggested a suppression effect (Maassen & Bakker, 2001). This statistical phenomenon can occur in longitudinal models that simultaneously include multiple predictors that are correlated. Parent-couple relationship satisfaction (CSI) ratings were highly correlated over time, which caused the β value to switch direction (i.e., from the expected negative to a positive direction; Maassen & Bakker, 2001). Multiple linear regression analysis examining the effects of T1-T3 CSI, T3 CES-D, and T3 CBCL internalizing on T4 CSI indicated that theβ-value for T3 CES-D was positive (β = .101, p = .093). After removing the highly correlated T1-T3 CSI, however, the β-value for T3 CES-D switched to negative (β = -.286, p = .008), indicating a suppression effect. When examined in isolation, higher T3 parent depression symptoms predicted a *decrease* in parent-couple relationship satisfaction at T4. Similarly, there was a significant positive direct effect of T3 parent-couple relationship satisfaction predicting T4 parent depression symptoms (β = .145, p = .005). Multiple linear regressions were again conducted to examine the effects of T1-T3 CES-D, T3 CSI, and T3 CBCL on T4 CES-D, and findings revealed that the β-value for T3 CSI was positive (β =.151, p = .029). After removing T1-T3 CES-D from the multiple linear regression, the β-value for T3 CES-D switched to *negative* (β = -.078, p = .383), but significance did not remain.

Externalizing Mental Health Problems Model

The child externalizing mental health problems model also revealed good model fit (X^2 (36) = 47.463, p = 0.0957; RMSEA = .042; TLI = 0.982; CFI = 0.995). Stability effects were present for parent depression symptoms (Mother: T1-T2 β = 0.574, p = .000; T2-T3 β = 0.352, p = .037; T3-T4 β = 0.308, p = .000; Father: T1-T2 β = 0.410, p = .000; T2-T3 β = 0.511, p = .000; T3-T4 β = 0.696, p = .000), parent-couple relationship satisfaction (Mother: T1-T2 β = 0.778, p = .000; T2-T3 β = 0.580, p = .008; T3-T4 β = 0.706, p = .000; Father: T1-T2 β = 0.705, p = .000; T2-T3 β = 0.489, p = .000; T3-T4 β = 0.560, p = .001), and child internalizing mental health problems (Mother: T1-T2 β = 0.727, p = .000; T2-T3 β = 0.538, p = .000; T3-T4 β = 0.544, p = .000; Father: T1-T2 β = 0.746, p = .000; T2-T3 β = 0.606, p = .000; T3-T4 β = 0.529, p = .000).

Mother-report of parent depression predicted parent-couple relationship satisfaction 12 months later (T1 CES-D to T2 CSI: β = -.077, p = .000; T2 CES-D to T3 CSI: β = -.086, p = .028; T3 CES-D to T4 CSI: β = 0.110, p = .048). Parent-depression also predicted child externalizing mental health problems (T1 CES-D to T2 CBCL externalizing: β = 0.129, p = .001; T2 CES-D to T3 CBCL externalizing: β = 0.123, p = .007). Mother-report of parent-couple relationship satisfaction at T3 predicted parent depression at T4 (β = 0.174, p = .000). Additionally, mother-report of child externalizing mental health problems predicted parent depression (T2 CBCL externalizing to T3 CES-D: β = 0.172, p = .000; T3 CBCL externalizing to T4 CES-D: β = 0.109, p = .000). One significant indirect effect was present for mother-report in that child externalizing mental health problems at T2 predicted parent depression at T3, which then predicted parent-couple relationship satisfaction at T4 (β = .019, p = .001; CI [.008, .017]) This finding suggests that parent depression may partially mediate the association between child externalizing mental health problems and parent-couple relationship satisfaction.

Father-report revealed one significant direct effect and one significant indirect effect. Directly, father-report of parent-couple relationship satisfaction at T2 predicted parent depression at T3 (β = -.313, *p* = .003). Indirectly, father-report of child externalizing mental health problems at T1 predicted parent-couple relationship satisfaction at T2, which then predicted parent depression symptoms at T3 (β = 0.018, *p* = .009; CI [.016, .028]). This suggests that parent-couple relationship satisfaction may serve as a partial mediator for the association between child externalizing mental health problems and parent depression symptoms.

Secondary Analyses. The child externalizing mental health problems model also revealed two suppression effects for mother-report. We conducted follow up multiple linear regressions to examine the unexpected positive effect between T3 parent depression and T4 parent-couple relationship satisfaction (β = 0.110, *p* = .048). The β-value for T3 CES-D was positive (β = 0.088, *p* = .132) for the original multiple linear regression examining effects for T1-T3 CSI, T3 CES-D, and T3 CBCL externalizing on T3 CES-D. After removing T1-T3 CSI, the β-value for T3 CES-D switched to negative (β = -.239, *p* = .022), suggesting that higher parent depression at T3 predicted *lower* parent-couple satisfaction at T4. Similarly, multiple linear regressions were conducted to investigate the positive effect between T3 parent-couple relationship satisfaction and T4 parent depression (β = 0.174, *p* = .000). The multiple linear regression investigating the effects of T1-T3 CES-D, T3 CSI, and T3 CBCL externalizing revealed a positive β value for T3 CSI (β = 0.172, *p* = .014). After removing T1-T3 CES-D, the β-value for T3 CES-D switched to negative, but was no longer significant (β = -.056, *p* = .563). Additionally, because the significant mother-reported indirect effect included a pathway impacted by a suppression effect (T2 CBCL externalizing → T3 CES-D → T4 CSI), secondary analyses were warranted. Specifically, we conducted a Sobel Test (Sobel, 1982) using SPSS

statistical software (Path A: β = .344, *SE* = .067; Path B: β = -1.048, *SE* = .189; Path C: β = -.039, *SE* = .194; Sobel Test: *z* = -3.77, *SE* = .096, *p* = .000), which indicated that the indirect pathway was indeed significant and in the expected direction in that greater child externalizing mental health problems predicted greater parent depression, which in turn, predicted lower parent-couple relationship satisfaction.

Discussion

The quality of the parent-couple relationship has important influences on both parent and child mental health within families from the general population (Du et al., 2022; Shi & Whisman, 2023). Parents of autistic children are at risk for poor parent-couple relationships, yet little is known about how this relationship is tied to the mental health of parents and autistic children across time. The present study examined whether parent-couple relationship satisfaction served as a mediator for the association between parent depression and child internalizing and externalizing mental health problems for families with autistic children.

We found that mother depression negatively predicted parent-couple relationship satisfaction 12 months later (T1 to T2, T2 to T3, and T3 to T4). In contrast, for fathers, parent-couple relationship satisfaction negatively predicted parent depression symptoms 12 months later (internalizing model: T2 to T3, T3 to T4; externalizing model: T2 to T3). These findings align with previous literature showing that parent mental health problems can alter one's perception of their parent-couple relationship, with depression and anxiety leading to more dissatisfaction in the relationship (e.g., Whisman, Uebelacker, & Weinstock, 2004). In a transactional manner, research has also found that parent-couple relationship dissatisfaction is associated with increased parent depression symptoms (e.g., Shi & Whisman, 2023; Yang et al., 2023). It is unclear why our findings indicate that mother's mental health drives her perceptions of parent-

couple relationship satisfaction, but for fathers, the satisfaction in the parent-couple relationship drives parent depression. In our sample, mothers were significantly more depressed than fathers at every time point. In line with previous research (e.g., Dong et al., 2022; Henderson et al.,2003; Yang et al., 2023) it is possible that mothers who are experiencing high depression employ a more negative approach and/or are less sensitive when resolving issues with their partner. Alternatively, low parent-couple relationship satisfaction has been linked to an increase in depression symptoms for fathers (Wang et al., 2021). It is possible that the low parent-couple relationship satisfaction takes a greater toll on father mental health than mother mental health. Indeed, research has indicated that men rely heavily on their wives for their primary social supports, and men who experience loneliness or tension within their intimate social connections (e.g.., parent-couple relationship), may be more likely to exhibit depression symptoms (Wang et al., 2020). Future research should examine this direction of effects more thoroughly for fathers.

Mother-report also revealed bidirectional effects between parent depression and both child internalizing (T1 CES-D to T2 CBCL; T3 CBCL to T4 CES-D) and externalizing (T1 CES-D to T2 CBCL; T2 CBCL to T3 CES-D; T3 CBCL to T4 CES-D) mental health problems. This finding aligns with previous ASD research (e.g., Benson, 2018; Ekas & Kouros, 2021). Mother depression may lead to greater levels of child internalizing and externalizing mental health problems via both genetic and environmental mechanisms such as a child's frequent exposure to parent depression symptoms (e.g., Kamis, 2021) or mother's altered parenting behaviors resulting in less positive engagement or responsiveness (e.g., Rueger et al., 2011; Tirumalaraju et al., 2020). In a transactional manner, greater child internalizing and externalizing mental health problems may leave mothers feeling overwhelmed and helpless and depressed (e.g., Benson, 2006, 2018). In contrast to mothers, in father-report models, higher parent

depression (T3 CES-D to T4 CBCL) predicted higher child internalizing mental health problems, but there was not a bidirectional effect of child mental health problems predicting later father depressive symptoms.

In support of our hypothesis, both mother- and father-report revealed a significant indirect pathway (T2 CES-D → T3 CSI → T4 CBCL) indicating that the parent-couple relationship satisfaction mediates the association between parent depression and later child internalizing mental health problems. This mediation model means that that parent depression symptoms led to a decrease in parent-couple relationship satisfaction, which in turn, led to an increase in internalizing mental health problems by the autistic child. Additionally, father-report yielded a second unexpected indirect effect (T2 CSI → T3 CES-D → T4 CBCL) such that greater parent-couple relationship satisfaction predicted decreased parent depression symptoms, which then predicted a decrease in child internalizing mental health problems.

The child externalizing mental health problems model revealed two significant indirect pathways. Unexpectedly, in the mother-report model, parent depression mediated the association between higher child externalizing mental health problems and later decreases in parent-couple relationship satisfaction (T2 CBCL → T3 CES-D → T4 CSI). Thus, greater child externalizing mental health problems predicted greater parent depression, which then predicted lower parent-couple satisfaction. In support of our hypothesis, father-reported models revealed an indirect pathway (T1 CBCL → T2 CSI → T3 CES-D), suggesting that having an autistic child with higher externalizing mental health problems contributed to lower parent-couple relationship satisfaction, which in turn, lad to an increase in parent depression symptoms. Relative to mothers, it is possible that fathers may be more sensitive to the challenges of parenting an autistic child with mental health problems, such that this stress “spills over” into the parent-

couple relationship, and in turn leads to depression. In contrast, for mothers, stress from parenting an autistic child with mental health problems has more direct effects on their mood.

It is interesting that our mediation pathways for the internalizing mental health problems model were parent driven (parent depression → child internalizing; parent-couple relationship satisfaction → child internalizing), whereas the mediations for the externalizing model were child driven (child externalizing → parent-couple relationship satisfaction; child externalizing → parent depression). This suggests that parent mental health and the parent-couple relationship are more sensitive to outwardly directed and/or disruptive externalizing behaviors than child internalizing mental health problems. In contrast, a family environment involving high parent depression and/or a dissatisfied parent-couple relationship creates internalized behaviors such as anxiety and depression in the autistic child.

In summary, in line with our main hypothesis, the parent-couple relationship is an important conduit through which parent depression shapes child mental health problems and vice versa across time. However, we also found and child mental health problems shape the parent-couple relationship through the mediated effect of mother depression and parent-couple relationship satisfaction effects child mental health problems through the mediated effect of father depression. Thus, mothers' mental health may be more sensitive to child mental health problems whereas fathers' mental health may be more sensitive to factors related to the parent-couple relationship. Thus, there are multidirectional feedback loops connecting parent depression, parent-couple relationship quality, and child mental health problems within families overtime.

Strengths, Limitations, and Future Directions

The current study had several strengths. It leveraged rich longitudinal data, that included both mother- and father- report, and examined multiple parent- and child- driven pathways simultaneously through the utilization of a complete longitudinal mediation model. The use of structural equation modeling allowed us to investigate our complex hypotheses and better understand the family system from multiple directions. Models also separately examined child internalizing and externalizing mental health problems, which were found to have different patterns of association with parent depression and the parent-child relationship.

There were also study limitations. The sample consisted primarily of White, non-Hispanic families with a mid-level socioeconomic status, which may limit the generalizability of our findings. Future research should utilize a more diverse sample that includes a wider range of sociodemographics as well as family characteristics (e.g., biological versus adoptive parents, low versus high socioeconomic status, same-sex parents, single parents, alternative caregivers, etc.). Additionally, parents who completed all four study cycles were younger and reported greater parent-couple relationship satisfaction, on average, than parents who did not complete one or more study cycles. While there were no significant differences between "completers" and "incompleters," on reports of parent depression or child mental health problems, it is possible that parents who were more dissatisfied with their parent-couple relationship did not have the motivation to engage in research, particularly in a study in which they both participate and answer questions about their relationship. Moreover, parents experiencing depression symptoms may be biased in their reporting of parent-couple relationship satisfaction and child mental health problems. Observational data capturing actual parent-couple interactions may help reduce this bias and provide a clearer picture of relationship satisfaction.

Study Implications

The parent-couple relationship is one of the most influential environmental factors in a child's life; therefore, when parents are struggling within their partner relationship, the whole family is struggling. Findings from the current study illuminate the need for family-wide intervention methods that encourage a high-quality parent-couple relationship (i.e., greater parent-couple relationship satisfaction) while also targeting both parent and child mental health. Our findings highlight a clear association between parent depression and parent-couple relationship satisfaction; thus interventions combining techniques of cognitive behavioral therapy and couple counseling may be beneficial. Further, Emotionally Focused Therapy (EFT; ICEEFT, 2023) helps couples recognize patterns of change that lead to feelings of dissatisfaction and disconnection, and therefore may also be helpful for improving the bond within the parent-couple dyad. Moreover, EFT can be provided at both the individual or family level, allowing families to strengthen attachment and bonds within their larger family system (i.e., parent-couple and parent-child) and address specific needs of each individual as well (ICEEFT, 2023). The Gottman Method (The Gottman Institute, 2023) is another intervention that focuses on relationship strengthening within the parent-couple dyad by addressing three main areas: 1) friendship; 2) conflict management; 3) creating shared meaning. The strategies associated with EFT and the Gottman Method may be best implemented in conjunction with mindfulness techniques as well as methods that also address mental health for both parents and children. It is imperative that future research continue exploring the influential nature of the parent-couple relationship on the mental health of both parents and children in families of autistic youth as well as how parent and child mental health problems impact the quality of the parent-couple relationship.

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Tables

Table 1.

Family Sociodemographics

Demographic	*M* (*SD*)
Mother (*n* = 188)	
Age in years (*M* [*SD*])	38.69 (5.62)
Race/Ethnicity (N [%])	
White, non-Hispanic	170 (90)
Other	18 (10)
Couple Satisfaction Index (*M* [*SD*])	115.23 (31.43)
Father (*n* = 188)	
Age in years (*M* [*SD*])	40.76 (6.19)
Race/Ethnicity (N [%])	
White, non-Hispanic	166 (88)
Other	22 (12)
Couple Satisfaction Index (*M* [*SD*])	117.34 (27.49)
Parent Couple	
Couple Relationship Length, years (*M* [*SD*])	14.55 (5.59)
Household Income (N [%])	
Less than $20,000	2 (1)
$20,000-$39,999	13 (7)
$40,000 and greater	166 (88)
Target Child (*n* =188)	
Male (N [%])	162 (86)
Age in years (*M* [*SD*])	7.88 (2.24)
ID (N [%])	65 (34)
SRS (*M* [*SD*])	77.03 (10.29)

Note. M = mean; *SD* = standard deviation; N = sample size; ID = intellectual disability; SRS = Social Responsiveness Scale-2nd edition (Constantino & Gruber, 2012); CBCL = Child Behavior Checklist (Achenbach & Rescorla, 2001)

Table 2.

Mother and father reported means, standard deviations, and t-values for main variables.

Measure	Mother *M*[1](*SD*)[2]	Father *M*(*SD*)	*t*-value[3]	*df*[4]	*p*-value
Time 1	**n = 188**	**n = 188**			
CES-D Total[5]	13.49 (10.33)	11.52 (8.90)	2.289	184	.023*
CBCL Int. T[6]	62.99 (9.55)	61.84 (9.66)	1.527	187	.128
CBCL Ext. T[7]	60.05 (11.12)	59.65 (10.30)	.629	187	.530
CSI Total[8]	115.23 (31.43)	117.34 (27.49)	-1.034	186	.303
Time 2	**n = 162**	**n = 156**			
CES-D Total	18.40 (7.10)	16.44 (6.40)	3.029	154	.003**
CBCL Int. T	61.37 (9.14)	60.66 (9.21)	1.103	156	.272
CBCL Ext. T	57.62 (9.96)	57.84 (9.90)	-.468	156	.640
CSI Total	114.13 (35.18)	117.51 (28.67)	-1.470	152	.144
Time 3	**n = 138**	**n = 133**			
CES-D Total	14.86 (11.29)	11.14 (9.51)	3.103	130	.002**
CBCL Int. T	61.42 (9.17)	59.37 (9.44)	2.317	131	.022*
CBCL Ext. T	56.96 (10.10)	56.93 (10.28)	.419	131	.676
CSI Total	115.78 (35.61)	118.26 (29.67)	-.693	128	.489
Time 4	**n = 125**	**n = 122**			
CES-D Total	14.68 (10.70)	12.40 (10.41)	2.199	116	.030*
CBCL Int. T	61.07 (9.30)	60.48 (9.05)	.741	120	.460
CBCL Ext. T	56.38 (10.56)	56.54 (10.98)	-.073	120	.942
CSI Total	117.61 (31.64)	117.53 (29.11)	.049	113	.961

Note. [1]mean; [2]standard deviation; [3]value for paired-samples *t*-test; [4]degrees of freedom; [5]Center for Epidemiological Studies-Depression Scale total score (Radloff, 1977); [6]Child Behavior Checklist Internalizing Problems T-Score (Achenbach & Rescorla, 2001); [7]Child Behavior Checklist Externalizing T-Score (Achenbach & Rescorla, 2001); [8]Couple Satisfaction Index total score (Funk & Rogge, 2007); $^{*}p < .05$; $^{**}p < .01$.

Table 3. *Correlations between main study variables and sociodemographics*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1.Age[1]	---	-.014	.080	.059	-.065	.021	-.150	-.086	-.017	-.085	.046	.077	.088	.002	-.015	.121	.121	.137	.065
2.Income	-.045	---	-.148*	-.136	.-172*	-.067	-.044	.074	.059	.028	.075	.058	.080	.089	.121	-.013	-.047	-.065	-.018
3. ID[2]	.024	-.148*	---	.024	.043	-.053	.063	-.031	-.023	-.087	-.091	-.032	-.055	-.045	-.074	.111	.099	.085	.100
4. Dep1[3]	.050	-.164*	.009	---	.553**	.699**	.704**	-.392**	-.380**	-.367**	-.221*	.287**	.318**	.396**	.389**	.277**	.326**	.371**	.349**
5. Dep2	.063	-.170*	-.024	.462**	---	.589**	.691**	-.238**	-.396**	-.351**	-.282**	.191*	.224**	.245**	.246**	.158*	.185*	.218*	.141
6. Dep3	.076	-.180*	-.010	.691**	.524**	---	.717**	-.357**	-.358**	-.444**	-.313**	.308**	.317**	.413**	.348**	.372**	.360**	.419**	.311**
7. Dep4	.041	-.252**	-.130	.671**	.429**	.736**	---	-.237**	-.282**	-.218*	-.278**	.232**	.354**	.440**	.416**	.241**	.320**	.303**	.309**
8. CSI1[4]	.059	.004	-.001	-.344**	-.098	-.336**	-.267**	---	.798**	.774**	.758**	-.040	-.084	-.102	-.175	-.089	-.157*	-.257**	-.220*
9. CSI2	-.055	.074	-.012	-.330**	-.382**	-.522**	-.366**	.745**	---	.789**	.800**	-.134	-.189*	-.140	-.278**	-.185*	-.264**	-.201*	-.183*
10.CSI3	-.028	.057	-.053	-.319**	-.250**	-.487**	-.436**	.741**	.794**	---	.875**	-.068	-.148	-.176*	-.248**	-.124	-.225*	-.301**	-.247**
11. CSI4	.068	.014	-.005	-.312**	-.159	-.495**	-.459**	.764**	.745**	.854**	---	-.018	-.070	-.108	-.118	-.143	-.204*	-.288**	-.236*
12. Int.1[5]	.147*	.000	-.017	.296**	.143	.229**	.209*	-.130	-.177*	-.134	-.184	---	.661**	.637**	.631**	.622**	.445**	.379**	.391**
13. Int.2	.127	-.041	.047	.315**	.133	.242**	.251**	-.156	-.236**	-.239**	-.156	.590**	---	.704**	.717**	.435**	.506**	.368**	.381**
14. Int.3	.026	-.017	-.032	.289**	.164	.275**	.207*	-.259**	-.309**	-.156	-.460**	.362**	.532**	---	.785**	.401**	.404**	.536**	.442**
15. Int.4	-.021	.092	-.010	.436**	.198*	.316**	.338**	-.307**	-.343**	-.329**	-.253**	.569**	.670**	.535**	---	.390**	.445**	.407**	.496**
16. Ext.1[6]	.162*	-.084	.155*	.308**	.113	.290**	.241**	-.169*	-.241**	-.282**	-.261**	.607**	.446**	.268**	.456**	---	.761**	.679**	.737**
17. Ext.2	.152	-.094	.112	.288**	.143	.239**	.202*	-.138	-.204*	-.190*	-.128	.411**	.562**	.298**	.486**	.747**	---	.767**	.749**
18. Ext.3	.209*	-.121	-.020	.364**	.165	.371**	.265**	-.270**	-.273**	-.229**	-.311**	.335**	.362**	.435**	.427**	.699**	.789**	---	.840**
19. Ext.4	.102	-.047	.147	.346**	.129	.275**	.243**	-.199*	-.158	-.205*	-.214*	.421**	.436**	.281**	.584**	.746**	.805**	.856**	---

Note. Pearson Correlations. Mother-report is shaded and above the diagonal. Father-report if unshaded and below the diagonal. [1]parent age; [2]Child Intellectual Disability; [3]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [4]Couple Satisfaction Index total score (Funk & Rogge, 2007); [5]Child Behavior Checklist Internalizing Problems T-Score (Achenbach & Rescorla, 2001); [6]Child Behavior Checklist Externalizing Problems T-Score (Achenbach & Rescorla, 2001); *= p <.05; **= p < .01

Table 4.

Path coefficients for mother- and father-reports of parent depression, parent-couple relationship satisfaction, and child internalizing mental health problems

Time Point	Mother-report $B^1(SE^2)$, Unstandardized	Mother-report $\beta(SE)$, Standardized	Father-report $\beta(SE)$, Unstandardized	Father-report $\beta(SE)$, Standardized
Cross Effects	**CES-D[3] → CSI[4]**			
1→2	-0.315(0.079)**	-0.090(0.029)**	-0.255(0.454)	-0.076(0.126)
2→3	-0.531(0.206)*	-0.105(0.044)*	-0.208(0.434)	-0.043(0.080)
3→4	0.347 (0.143)*	0.124(0.048)*	-0.252(0.425)	-0.081(0.131)
	CES-D → CBCL Int.[5]			
1→2	0.167(0.059)**	0.187(0.051)**	0.131(0.101)	0.120(0.082)
2→3	0.168(0.143)	0.125(0.127)	0.039(0.112)	0.025(0.078)
3→4	-0.016(0.028)	-0.020(0.037)	0.091(0.022)**	0.092(0.020)**
	CSI → CES-D			
1→2	-0.004(0.008)	-0.017(0.038)	0.015(0.040)	0.065(0.160)
2→3	-0.017(0.035)	-0.054(0.117)	-0.104(0.039)**	-0.319(0.096)**
3→4	0.042(0.018)*	0.145(0.051)**	-0.025(0.012)*	-0.075(0.031)*
	CSI → CBCL Int.			
1→2	0.011(0.025)	0.038(0.085)	-0.008(0.030)	-0.024(0.085)
2→3	0.014(0.015)	0.053(0.054)	-0.051(0.012)**	-0.151(0.042)**
3→4	-0.045(0.012)**	-0.172(0.035)**	-0.031(0.031)	-0.100(0.105)
	CBCL Int. → CSI			
1→2	-0.066(0.197)	-0.017(0.052)	-0.090(0.227)	-0.030(0.073)
2→3	0.061(0.182)	0.015(0.047)	-0.030(0.192)	-0.009(0.064)
3→4	-0.108(0.247)	-0.031(0.071)	-0.149(0.341)	-0.049(0.117)
	CBCL Int. → CES-D			
1→2	0.020(0.042)	0.026(0.060)	0.028(0.051)	0.044(0.074)
2→3	0.090(0.120)	0.071(0.094)	-0.035(0.072)	-0.035(0.076)
3→4	0.163(0.070)*	0.148(0.063)*	0.005(0.046)	0.005(0.042)
Lagged Effects	**CES-D**			
1→3	0.464(0.104)**	0.410(0.081)**	0.535(0.048)**	0.491(0.049)**
	CSI			
	0.278(0.160)	0.241(0.136)	0.382(0.095)**	0.339(0.110)**
	CBCL Int.			
	0.283(0.115)*	0.279(0.125)*	0.034(0.145)	0.033(0.151)
2→4	**CES-D**			
	0.496(0.096)**	0.336(0.053)**	0.032(0.148)	0.020(0.092)
	CSI			
	0.149(0.140)	0.164(0.164)	0.112(0.099)	0.110(0.098)
	CBCL Int.			
	0.211(0.067)**	0.207(0.072)**	0.400(0.147)**	0.403(0.152)**

Note.[1] Beta value; [2] Standard Error; [3]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [4]Couple Satisfaction Index total score (Funk & Rogge, 2007); [5]Child Behavior Checklist Internalizing Problems T-Score (Achenbach & Rescorla, 2001); * p <.05; ** p < .01.

Table 5.

Estimates of Indirect Pathways for Child Internalizing Mental Health Problems Model

Time Point	Pathway	Mother-Report β[1]-value of Indirect Effect	SE[2]	Lower Bound of 95% CI[3]	Upper Bound of 95% CI	Father-Report β-value of Indirect Effect	SE	Lower Bound of 95% CI	Upper Bound of 95% CI
1→2→3									
	CBCL[4]→CSI[5]→CES-D[6]	.001	.004	-.003	.006	.010	.021	-.016	.016
	CBCL→CES-D→CSI	-.003	.005	-.003	.003	-.002	.004	-.001	.000
	CSI→CBCL→CES-D	.003	.010	-.014	.013	.001	.007	-.010	.002
	CSI→CES-D→CBCL	-.002	.003	-.002	.002	.002	.005	.000	.010
	CES-D→CSI→CBCL	-.005	.009	-.015	-.003	.012	.014	.002	.030
	CES-D→CBCL→CSI	.003	.013	-.006	.007	-.001	.009	-.012	.008
2→3→4									
	CBCL→CSI→CES-D	.002	.005	-.001	.004	.001	.008	-.005	.015
	CBCL→CES-D→CSI	.009	.010	-.004	.021	.003	.015	-.017	.026
	CSI→CBCL→CES-D	.008	.010	.000	.009	-.001	.007	-.012	.000
	CSI→CES-D→CBCL	.001	.002	-.003	.000	**-.029****	.011	-.039	-.018
	CES-D→CSI→CBCL	**.018***	.008	.005	.027	**.004***	.002	.000	.003
	CES-D→CBCL→CSI	-.004	.014	-.027	.000	-.001	.019	-.040	.000

Note. Estimates are standardized values. [1] Beta-value; [2]Standard Error; [3] Confidence Interval; [4]Child Behavior Checklist Internalizing Sub-scale (Achenbach & Rescorla, 2001);[5] Couple Satisfaction Index (Funk & Rogge, 2007); [6]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); *p <.05

Table 6.

Path coefficients for mother- and father-reports of parent depression, parent-couple relationship satisfaction, and child externalizing mental health problems

Time Point	Mother-report *B*[1](*SE*[2]), Unstandardized	Mother-report *β*(*SE*), Standardized	Father-report *β*(*SE*), Unstandardized	Father-report *β*(*SE*), Standardized
Cross Effects	**CES-D**[3] **→ CSI**[4]			
1→2	-0.269(0.056)**	-0.077(0.020)**	-0.238(0.342)	-0.072(0.096)
2→3	-0.432(0.200)*	-0.086(0.039)*	-0.204(0.379)	-0.042(0.071)
3→4	0.305(0.158)†	0.110(0.056)*	-0.295(0.329)	-0.091(0.100)
	CES-D → CBCL Ext.[5]			
1→2	0.125(0.031)**	0.129(0.037)**	0.066(0.046)	0.058(0.045)
2→3	0.191(0.048)**	0.123(0.045)**	0.038(0.063)	0.024(0.044)
3→4	-0.041(0.025)	-0.045(0.029)	0.009(0.041)	0.008(0.034)
	CSI → CES-D			
1→2	-0.003(0.010)	-0.015(0.047)	0.016(0.041)	0.068(0.163)
2→3	-0.009(0.035)	-0.028(0.115)	-0.102(0.041)*	-0.313(0.107)**
3→4	0.051(0.016)**	0.174(0.036)**	-0.026(0.017)	-0.080(0.048)
	CSI → CBCL Ext.			
1→2	-0.012(0.015)	-0.037(0.048)	0.028(0.021)	0.075(0.060)
2→3	0.008(0.012)	0.026(0.041)	-0.019(0.018)	-0.056(0.058)
3→4	-0.011(0.011)	-0.036(0.035)	0.011(0.024)	0.030(0.061)
	CBCL Ext. → CSI			
1→2	-0.225(0.205)	-0.070(0.062)	-0.166(0.106)	-0.059(0.035)
2→3	-0.120(0.200)	-0.034(0.055)	0.004(0.180)	0.001(0.063)
3→4	-0.101(0.105)	-0.034(0.035)	0.089(0.137)	0.029(0.046)
	CBCL Ext. → CES-D			
1→2	-0.007(0.044)	-0.010(0.071)	0.011(0.043)	0.019(0.068)
2→3	0.200(0.053)**	0.172(0.039)**	0.041(0.059)	0.044(0.061)
3→4	0.103(0.022)**	0.109(0.029)**	-0.067(0.089)	-0.067(0.086)
Lagged Effects	**CES-D**			
1→3	0.452(0.118)**	0.399(0.090)**	0.478(0.063)**	0.444(0.076)**
	CSI			
	0.233(0.213)	0.205(0.190)	0.403(0.099)**	0.357(0.109)**
	CBCL Ext.			
	0.241(0.102)*	0.242(0.111)*	0.202(0.072)**	0.212(0.077)**
2→4	**CES-D**			
	0.505(0.091)**	0.341(0.049)**	0.019(0.151)	0.012(0.093)
	CSI			
	0.160(0.138)	0.177(0.164)	0.134(0.064)*	0.128(0.059)*
	CBCL Ext.			
	0.143(0.186)	0.135(0.176)	0.307(0.158)†	0.282(0.132)*

Note.[1] Beta value; [2] Standard Error; [3]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [4]Couple Satisfaction Index total score (Funk & Rogge, 2007); [5]Child Behavior Checklist Externalizing Problems T-Score (Achenbach & Rescorla, 2001); * p <.05; ** p < .01; † p < .10.

Table 7.

Estimates of Indirect Pathways for Child Externalizing Mental Health Problems Model

Time Point	Pathway	Mother-Report β[1]-value of Indirect Effect	SE[2]	Lower Bound of 95% CI[3]	Upper Bound of 95% CI	Father-Report β-value of Indirect Effect	SE	Lower Bound of 95% CI	Upper Bound of 95% CI
1→2→3									
	CBCL[4]→CSI[5]→CES-D[6]	.002	.014	-.012	.011	**.018**[*]	.007	.016	.028
	CBCL→CES-D→CSI	.001	.005	-.002	.005	-.001	.005	-.001	.000
	CSI→CBCL→CES-D	-.006	.005	-.008	-.005	.003	.008	.002	.006
	CSI→CES-D→CBCL	-.002	.004	-.004	-.001	.002	.018	-.001	.041
	CES-D→CSI→CBCL	-.002	.005	-.013	-.003	.004	.005	.000	.004
	CES-D→CBCL→CSI	-.004	.005	-.011	.002	.000	.004	-.009	.003
2→3→4									
	CBCL→CSI→CES-D	-.006	.006	-.013	.003	.000	.009	-.005	.017
	CBCL→CES-D→CSI	**.019**[**]	.006	.008	.017	-.004	.009	-.006	-.003
	CSI→CBCL→CES-D	.003	.003	.005	.006	.004	.002	-.003	.001
	CSI→CES-D→CBCL	.001	.004	-.006	.006	-.003	.012	-.019	.002
	CES-D→CSI→CBCL	.003	.003	-.001	.005	-.001	.003	-.004	.000
	CES-D→CBCL→CSI	-.004	.005	-.011	.000	.001	.001	.000	.001

Note. Estimates are standardized values. [1] Beta-value; [2]Standard Error; [3] Confidence Interval; [4]Child Behavior Checklist Externalizing Sub-scale (Achenbach & Rescorla, 2001);[5] Couple Satisfaction Index (Funk & Rogge, 2007); [6]Center for Epidemiological Studies-Depression Scale (Radloff, 1977); [*]p <.05

Figures

Figure 1.

Conceptual model of complete longitudinal mediation model for the associations between parent depression, parent-couple relationship satisfaction, and child mental health problems.

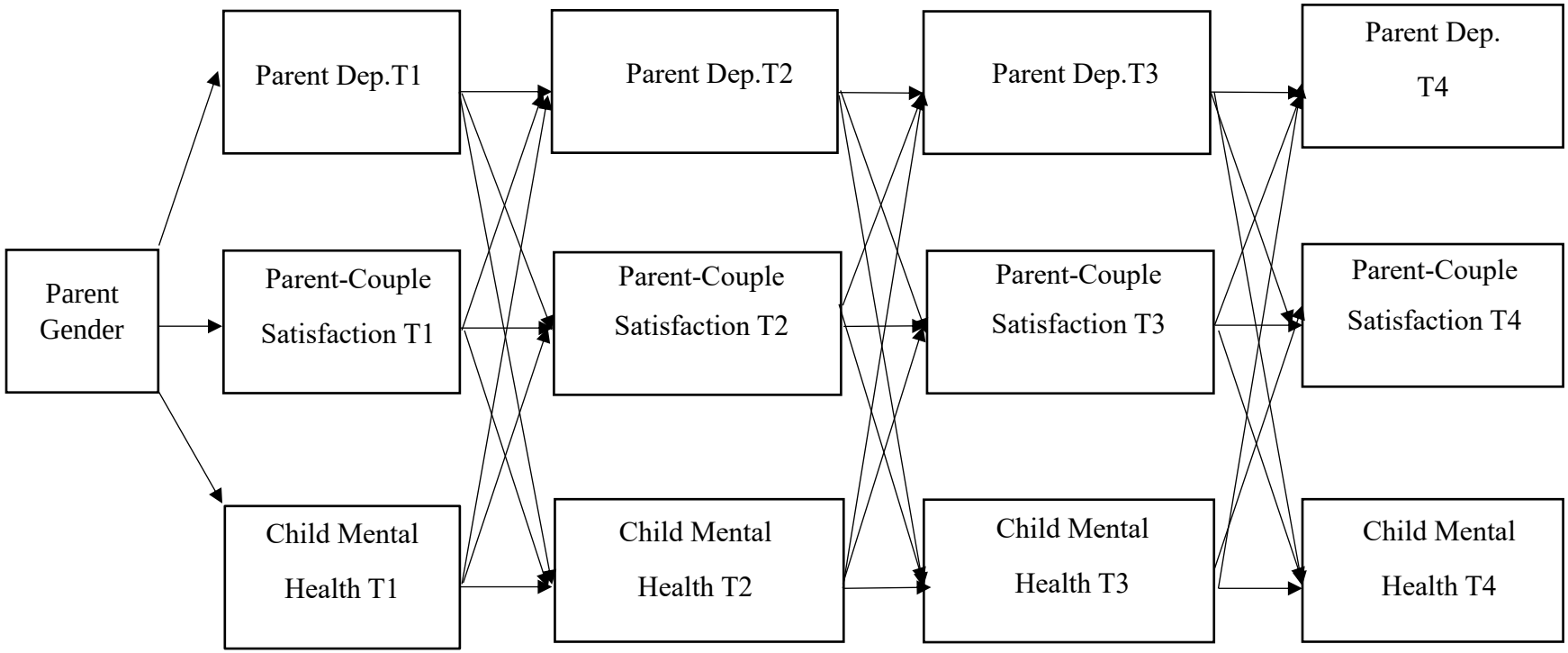
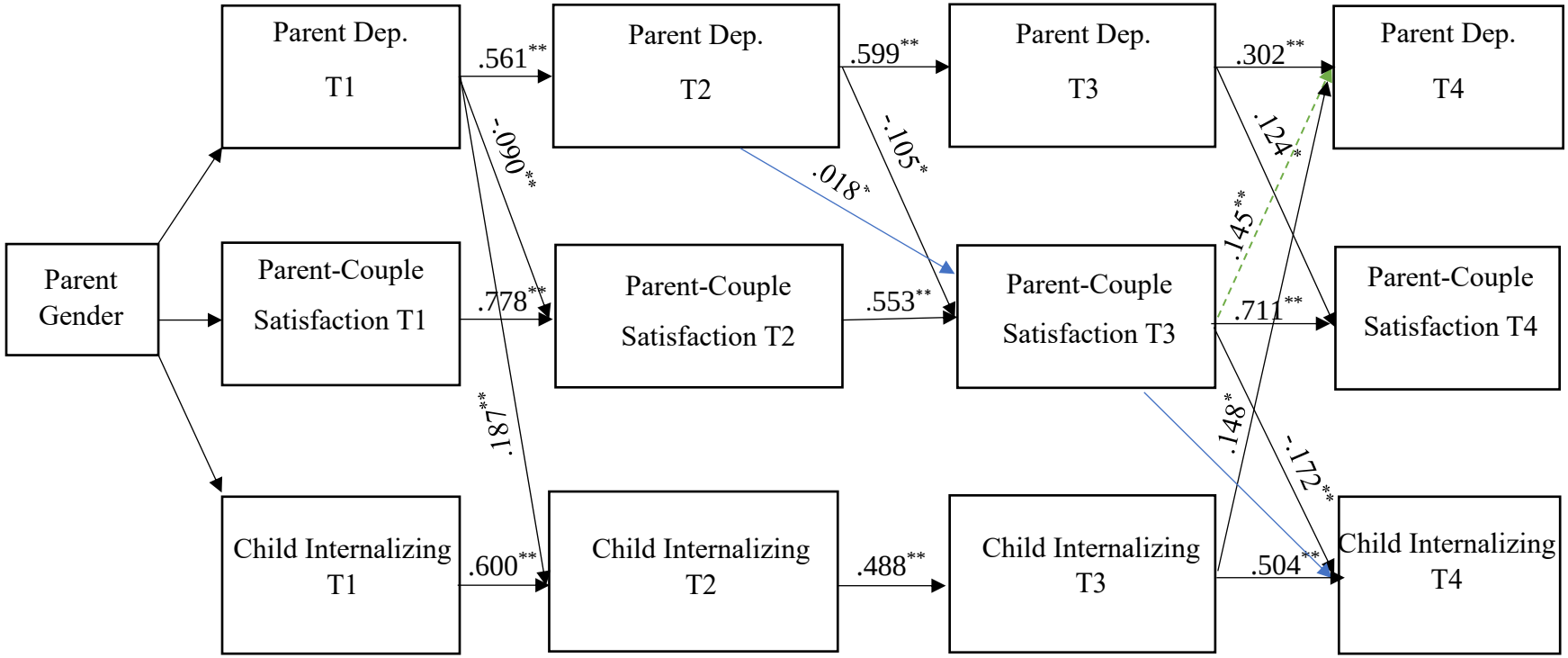


Figure 2.

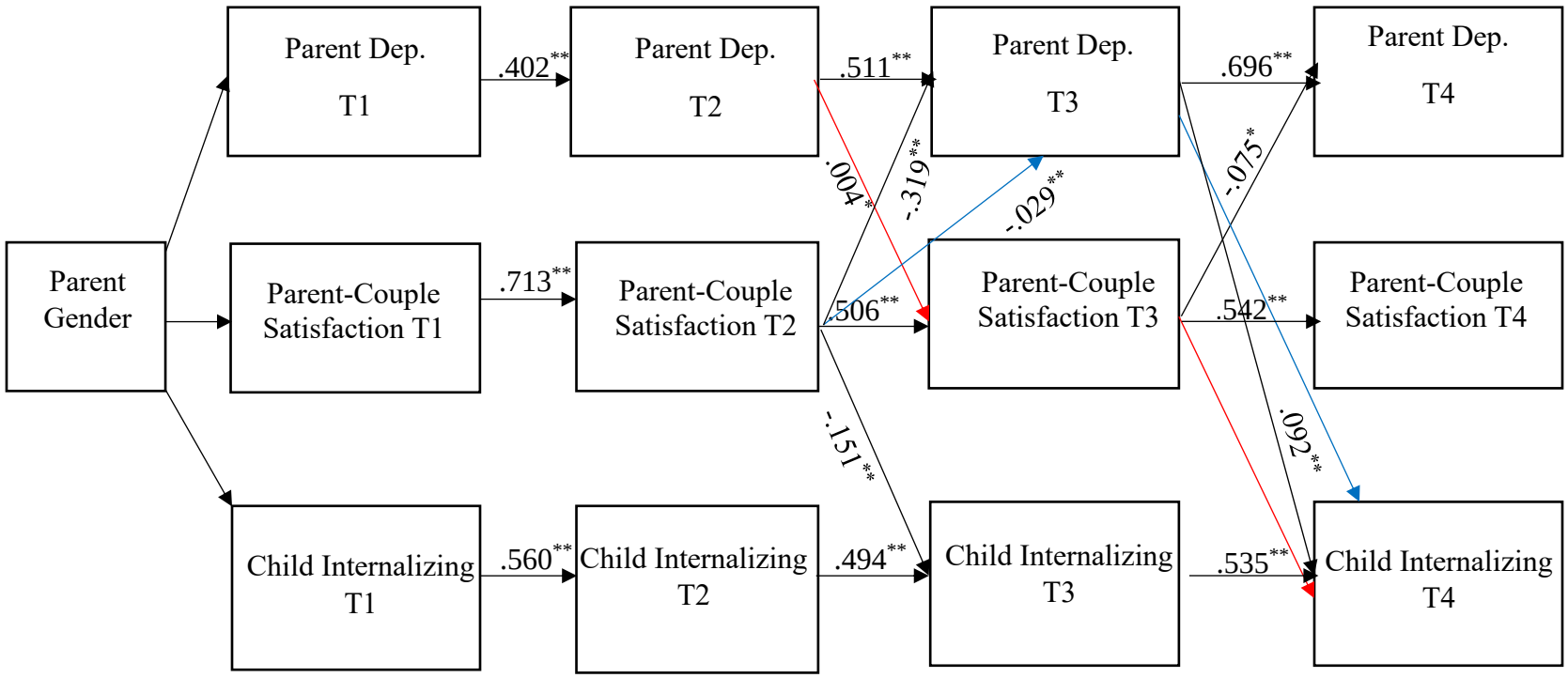
Complete longitudinal mediation model for associations between mother-report of parent depression, parent-couple relationship satisfaction, and child internalizing mental health problems.



Note. Results of the complete longitudinal mediation model for mother-report of parent depression symptoms, parent-couple relationship satisfaction, and child internalizing mental health problems, controlling for parent age, household income, and child intellectual disability status. Values are standardized path estimates. Blue lines indicate a significant *indirect* path. The green dotted line indicates loss of significance after secondary analyses were conducted. Lagged paths are excluded from figure for simplicity. * p <.05; ** p < .01.

Figure 3.

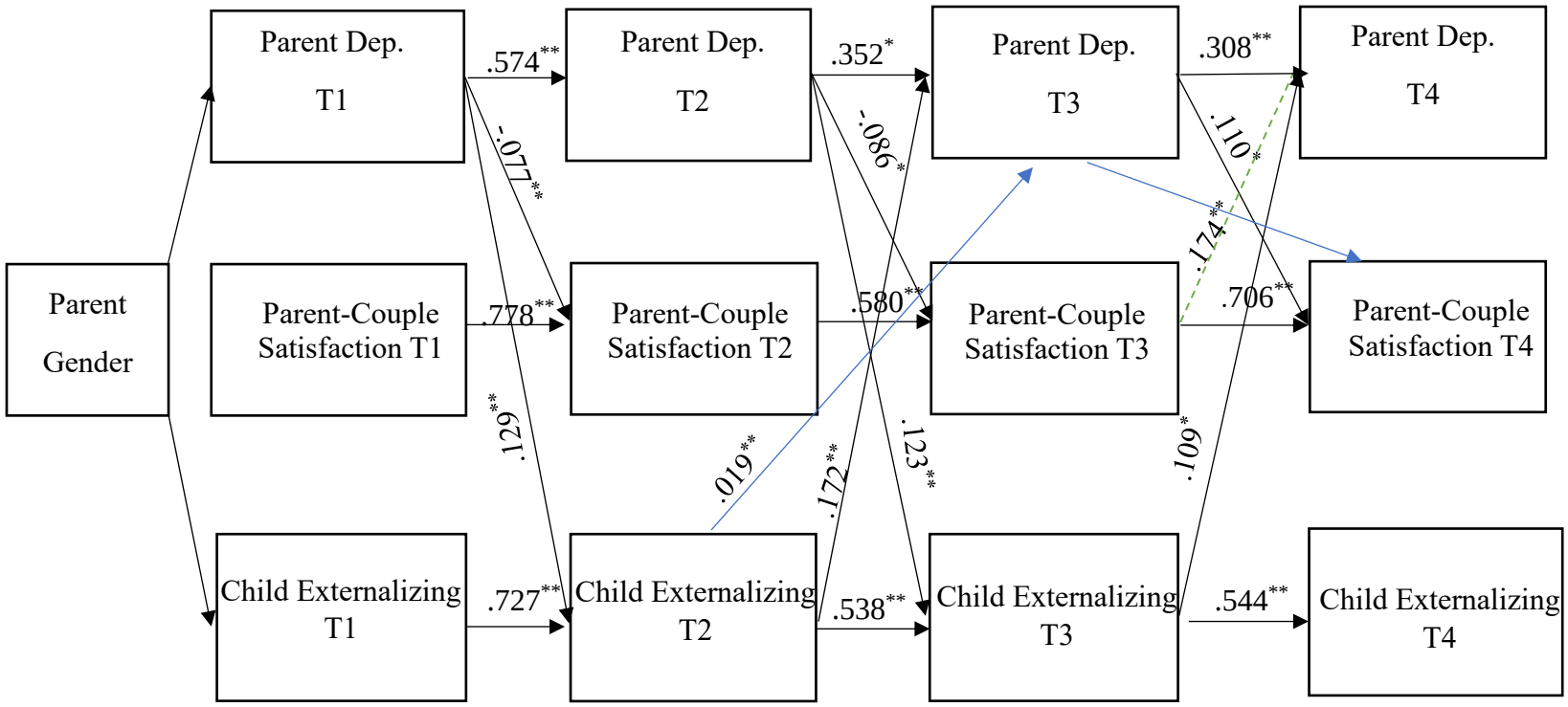
Complete longitudinal mediation model for associations between father-report of parent depression, parent-couple relationship satisfaction, and child internalizing mental health problems.



Note. Results of the complete longitudinal mediation model for father-report of parent depression symptoms, parent-couple relationship satisfaction, and child internalizing mental health problems, controlling for parent age, household income, and child intellectual disability status. Values are standardized path estimates. Red and blue lines indicate significant *indirect* pathways. Lagged paths are excluded from figure for simplicity. * p <.05; ** p < .01.

Figure 4.

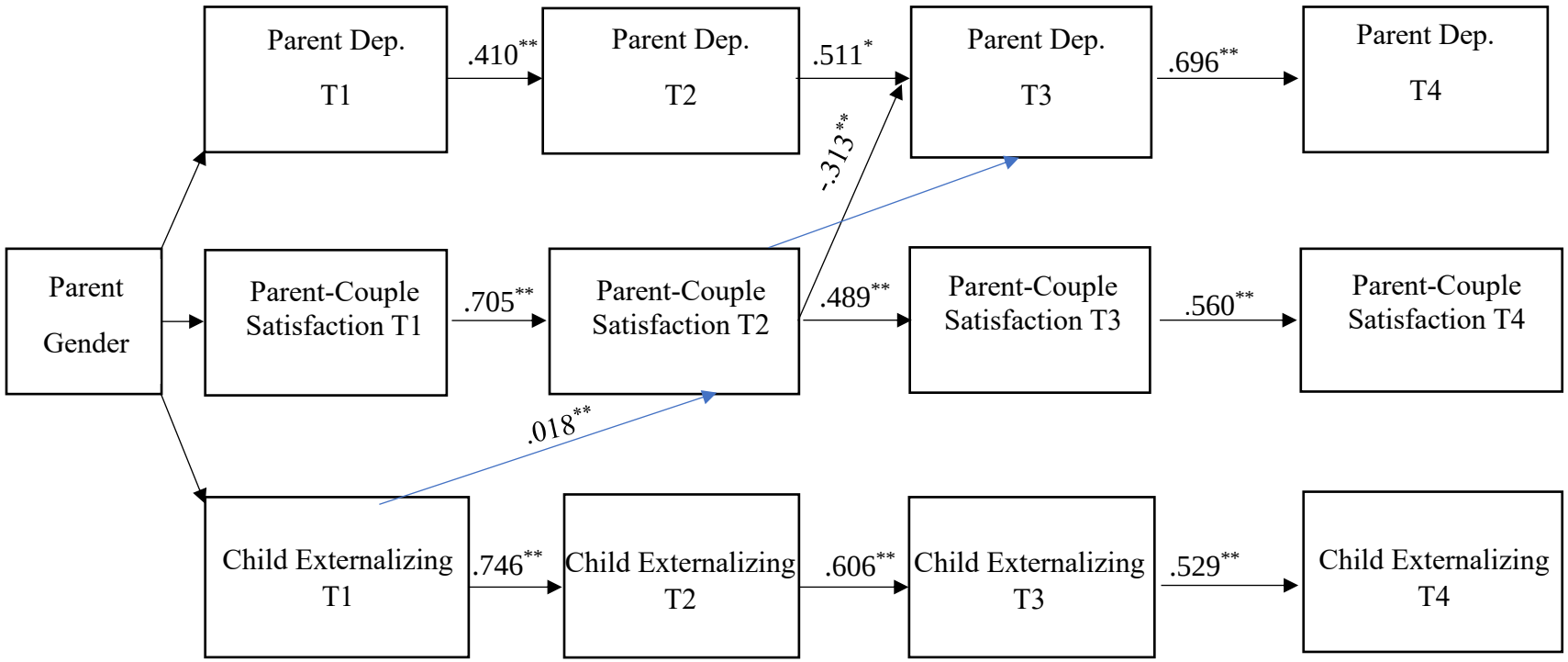
Complete longitudinal mediation model for associations between mother-report of parent depression, parent-couple relationship satisfaction, and child externalizing mental health problems.



Note. Results of the complete longitudinal mediation model for mother-report of parent depression symptoms, parent-couple relationship satisfaction, and child externalizing mental health problems, controlling for parent age, household income, and child intellectual disability status. Values are standardized path estimates. Blue lines indicate a significant *indirect* path. The green dotted line indicates loss of significance after secondary analyses were conducted Lagged paths are excluded from figure for simplicity. * p <.05; ** p < .01.

Figure 5.

Complete longitudinal mediation model for associations between father-report of parent depression, parent-couple relationship satisfaction, and child externalizing mental health problems.



Note. Results of the complete longitudinal mediation model for father-report of parent depression symptoms, parent-couple relationship satisfaction, and child externalizing mental health problems, controlling for parent age, household income, and child intellectual disability status. Values are standardized path estimates. Blue lines indicate a significant *indirect* path. Lagged paths are excluded from figure for simplicity. * p <.05; ** p < .01.

Chapter 4: Conclusions

Autistic children face an increased risk for co-occurring mental health problems compared to their typically developing (TD) peers (Hudson et al., 2019). Similarly, parents of autistic children experience higher levels of depression than parents of TD children (Schnabel et al., 2020). With a prevalence rate of 1 in 36 children meeting criteria for autism spectrum disorder in the United States (Centers for Disease Control and Prevention, 2023), the negative mental health outcomes for these families are a critical public health concern. While genetic mechanisms are likely to play a role in the links between parent and child mental health (e.g., Kang et al., 2020; Wiggins et al., 2019; Bora et al., 2017), theory and research also highlight the role of the family environment (e.g., Cummings et al., 2000a; Cox and Paley, 1997). Indeed, the mental health of family members is intertwined. The quality of the parent-child and parent-couple subsystems are thought to serve as important conduits that connect parent depression and child mental health problems. Rooted in the developmental psychopathology (Cummings et al., 2000b; Davies & Cicchetti, 2004) and family systems (Cox & Paley, 1997) theoretical lens, my dissertation aimed to better understand the impact of the parent-child relationship quality and parent-couple relationship satisfaction on the associations between parent depression and child mental health problems for families with autistic children. Across three manuscripts, I first explored bidirectional effects between parent depression and child mental health problems. I then investigated whether parent-child relationship quality (measured as critical comments and positive remarks during a five minute speech sample) and parent-couple relationship satisfaction, respectively, mediated the link between parent depression and child mental health problems.

Our findings revealed robust direct and bidirectional associations between parent depression, parent-child relationship quality, and child mental health problems. In line with our

central hypotheses, the parent-child and parent-couple subsystem served as important mediators that accounted for the connections between parent and child mental health. Specifically, positive remarks about the parent-child relationship mediated the connection between parent depression and child mental health problems for fathers. Parent-couple relationship satisfaction mediated the association between parent depression and child mental health problems for both mothers and fathers. In terms of directional flow, child externalizing (versus internalizing) mental health problems appeared to take a greater toll on the parent-couple relationship and parent depression, whereas the parent-couple relationship and parent depression had strongest effects on child internalizing (versus externalizing symptoms).

We also found unexpected mediation pathways: 1) father depression partially mediated the association between higher child mental health and more critical comments about the parent-child relationship; 2) at a trend level, child mental health problems partially mediated the link between higher father depression and more critical comments about the parent-child relationship; 3) father depression partially mediated the association between lower parent-couple relationship satisfaction and higher child internalizing mental health problems; and 4) mother depression partially mediated the link between higher child externalizing mental health problems and lower parent-couple satisfaction. These non-hypothesized pathways highlight the complexity of the family system and suggest that multiple feedback loops may be occurring between parent and child mental health, the parent-child relationship, and parent-couple relationship.

Implications and Next Steps

Parent depression and child mental health problems appear to be robustly connected to mechanisms within both parent-child and parent-couple relationships for families with autistic children, thus emphasizing the need for family-wide intervention strategies that address the needs of the subsystems in conjunction with individualized mental health treatment plans. Interventions should provide psychoeducation to assist parents in understanding the impact of their own mental health on their child's mental health, often through a negative parent-child relationship or negative parent-couple relationship. Moreover, interventions should work to strengthen the parent-child and parent-couple subsystems to improve the mental health of both parents and autistic children. For example, exercises that promote sensitive and positive child-led play may improve the quality of the parent-child relationship (e.g., World Health Organization, 2022). Additionally, couple counseling may be helpful in recognizing areas of the parent-couple relationship that create tension, dissatisfaction, or disconnection. Strategies that focus on healthy conflict resolution, emotion regulation, and the creation of shared meaning between the parent-couple may also assist with enhancing the parent-couple relationship. Finally, incorporating mindfulness and techniques associated with cognitive behavioral therapy into the family-wide intervention approach may further promote optimal outcomes for these families.

Future research should leverage longitudinal research designs to identify the specific aspects of the parent-child and parent-couple subsystems (e.g., parent responsiveness to child, child emotion dysregulation, parent-couple dyadic coping, parent-couple conflict resolution) that affect parent and child mental health problems. Further, additional informant reports of child mental health problems (e.g., teacher report), as well as an examination of additional family subsystems such as the child-sibling relationship would provide important perspectives regarding

the complex nature of the family system. Actor-Partner Interdependence Models may be a helpful analytic approach in determining specific differences between mental health and parent-child and/or parent-couple relationship mechanisms for mothers versus fathers. Moreover, observational data that captures parent-child and/or parent-couple interactions should be included in future research. Finally, researchers should also recruit diverse samples that comprise a wide variety of family characteristics (e.g., same sex parents, single parents, alternative caregivers) and sociodemographics (e.g., race, socioeconomic status).

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Appendix 1.

Please see Manuscript 1, published in the *Journal of Autism and Developmental Disorders*, below.

Journal of Autism and Developmental Disorders
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ORIGINAL PAPER

Parental Depression Symptoms and Internalizing Mental Health Problems in Autistic Children

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Abstract

Autistic youth are at risk for internalizing mental health problems such as depression and anxiety. Similarly, parents of autistic youth report higher levels of depression than parents of typically developing children. The goal of this study was to examine bidirectional associations between parent depression symptoms and the internalizing problems of autistic youth in 188 families across four time points (T1–T4; spaced 12 months apart). A cross-lagged panel model revealed that mother (T1 and T2) and father (T1) depression symptoms positively predicted the youth's internalizing problems 12 months later. The youth's internalizing problems at T3 positively predicted maternal depression symptoms at T4. Future research should explore genetic and environmental pathways that link parent depression and internalizing problems in autistic youth.

Keywords Autism · Depression · Family · Mental health · Parent · Internalizing

Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental condition that is estimated to occur in 1 in 54 children in the U.S. (Maenner et al., 2020). Hallmark characteristics of ASD include restricted and repetitive behaviors and difficulties in social communication that create challenges in everyday functioning (American Psychological Association [APA], 2020). Internalizing mental health problems, such as depression and anxiety, are common co-occurring conditions in autistic youth (Kaat & Lecavalier, 2013; Lopata, et al., 2010). It is estimated that 14–20% of autistic youth experience at least one depressive episode by the age of 18 years (Upthegrove et al., 2018), and nearly 40% experience clinically elevated anxiety symptoms or meet criteria for an anxiety disorder (van Steensel et al., 2011). According to the developmental psychopathology framework (e.g., Cicchetti & Howes, 1991; Davies & Cicchetti, 2004; Kerig, 2016), the high risk for depression and anxiety symptoms in autistic youth may be attributed to genetic and neurobiological vulnerabilities as well as ongoing reciprocal transactions between the individual and their environment. The present study sought to understand the bidirectional associations between the depression symptoms of parents and the internalizing mental health problems of autistic youth, as these outcomes may be linked in both genetic and environmental ways.

The developmental psychopathology framework posits that mental health is influenced by a complex interaction of biological, psychological, social, and contextual factors that accumulate and unfold over time (Cummings et al., 2000; Eme, 2017). This framework suggests that autistic youth both actively shape and are shaped by their environment (Masten & Cicchetti, 2010). One salient environmental factor for the mental health of autistic youth is the mental health of parents. Parents of autistic youth have an elevated risk for depression relative to adults in the general population (Cohrs & Leslie, 2017). For example, a recent systematic review and meta-analysis suggested that 31% of parents of autistic youth exhibit clinically-relevant depression symptoms (Schnabel et al., 2020), with individual studies reporting

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that up to 49% of parents report depression symptoms (Al-Farsi et al., 2016). In part, this elevated rate of depression has been linked to the high parenting stress reported by parents of autistic youth (e.g., Baker et al., 2011; Benson, 2018). Indeed, among parents of autistic youth, those who report higher parenting stress or more child-related challenges (e.g., severity of ASD symptoms or mental health problems) also report more depression symptoms (Benson, 2006). Thus, in part, elevated internalizing mental health problems in autistic youth may affect parents in ways that contribute to their own depression symptoms. However, biological parents of autistic youth have been shown to have an elevated rate of depression prior to having their child (e.g., Hagberg et al., 2018; Vasa et al., 2012; Wiggins et al., 2019). Indeed, parental mood disorders have been found to be associated with about a two-fold elevated risk of having an autistic child (e.g. Jokiranta et al., 2013). These findings suggest a potential genetic connection between lifetime risk of depression and having an autistic child. Moreover, maternal depression at earlier life stages (i.e., prior to becoming a parent) has been linked to having an autistic child with greater emotion dysregulation (Wiggins et al., 2019). This is similar to reports of an increased risk for emotion dysregulation in neurotypical children who have parents with a history of depression (Ghaziuddin & Greden, 1998; Weissman et al., 2006). Thus, the increased risk for depression in parents of autistic children could be driven by a combination of biological (including a genetic predisposition) and environmental (including child-related challenges) mechanisms that interact across time.

A family environment involving parent depression may, in turn, contribute to increased internalizing mental health problems in the autistic child. Neurotypical children with depressed parents are 2–3 times more likely to exhibit symptoms of depression and anxiety when compared to children without depressed parents (e.g., England & Sim, 2009; Gentile & Fusco, 2017; Kamis, 2021; Tirumalaraju et al., 2020; Weissman et al., 2006). Depressed mothers self-report that they are less responsive to their child, express less sensitivity, provide less positive engagement, and are less consistent or constructive in their parenting strategies (Bayer et al., 2006; Goodman et al., 2011; Murray et al., 2003; Rueger et al., 2011), all of which are parenting behaviors that can contribute to depressed and anxious affect in children (Aktar & Bögels, 2017; Cummings et al., 2000Goodman & Gotlib, 1999). Similar patterns have been found in parents of autistic children (e.g., McRae et al., 2018; Rodriguez et al., 2019; Zaidman-Zait et al., 2014). For example, in a sample of 150 families of children with ASD, mothers with less (versus more) depression symptoms were warmer and less critical of their autistic child in a five-minute speech sample (Hickey et al., 2020). In a cross-sectional study, McRae and colleagues (2018) found that parental adjustment, based on a combined measure of depression and anxiety, predicted both negative parenting behaviors (e.g., harsh or disengaged parenting practices) and higher child internalizing behavior problems in a sample of 67 parent–child dyads (primarily mothers). Thus, parental depression may lead to parenting behaviors (e.g., less warmth and more disengaged and harsh parenting) that contribute to internalizing mental health problems in autistic youth.

Current Study

The present study explored the bidirectional associations between parent depression symptoms and internalizing mental health problems in autistic youth in a sample of 188 families (n = 376 parents). Analyses drew from data collected at four time points, each spaced approximately 12 months apart. At Time 1 (T1), the autistic child was aged 5–12 years. At each time point, mothers and fathers separately reported on their own level of depression symptoms and rated their child's frequency and severity of internalizing mental health problems. Drawing on the developmental psychopathology framework and evidence of both genetic and environmental connections between parent depression and mood dysregulation in autistic youth, the following hypotheses were made: (1) at Time 1, parent depression would be positively associated with child internalizing mental health problems; (2) higher parental depression symptoms would predict increases in internalizing mental health problems in the autistic youth 12 months later, across time points; (3) higher internalizing mental health problems in the autistic youth would predict increased parental depression symptoms 12 months later, across time points. A priori hypotheses about mother-father differences were not made.

Method

Participants

Participants in the present study were mothers and fathers of autistic youth who were part of a longitudinal study investigating a variety of family experiences. Inclusion criteria included: (1) parent couple who had an autistic child between the ages of 5–12 years at the time of recruitment; (2) parents were 21 years of age or older; (3) child had an educational or medical diagnosis of ASD and the diagnostic evaluation included the autism diagnostic observation schedule (ADOS-2nd edition; Lord et al., 2012); (4) parents needed to be in a cohabiting couple relationship for 3 years or more; (5) both parents had to agree to participate. Recruitment occurred through research registries, and fliers mailed to schools and ASD clinics, as well as placed in community locations. Between 2013 and 2014, 188 parent couples who

met study requirements enrolled in the study. In order to assess the ASD symptom severity of the child, parents completed the social responsiveness scale (SRS-2; Constantino & Gruber, 2012). Five autistic children received an SRS-2 total t-score below or equal to 60; but, after reviewing relevant information such as medical/educational records and ADOS, these children were deemed to meet criteria for ASD, and thus were included in analyses.

For the present study, data was analyzed from the first four time points (T1, T2, T3, T4) of the study. Time points were spaced approximately 12 months apart ($M = 11.66$, $SD = 3.70$). The majority of the autistic youth were male (86%) and the breakdown of ethnicities/races were: White, non-Hispanic (83%); Latino (7%); Asian (4%); Black (3%); Native American (1%); and multiple racial/ethnic groups (3%). Approximately one-third (34%) of autistic youth had an intellectual disability (ID). Autistic children were, on average, 7.90 years old ($SD = 2.25$) at T1. Mothers were on average 38.69 years ($SD = 5.62$), and fathers had a mean age of 40.76 years ($SD = 6.19$). The majority of families were composed of both biological parents (n = 167; 89%). Parent couple relationship length was on average 14.55 years ($SD = 5.59$) at T1. Average household income at T1 was $80,000–$89,999. Table 1 provides demographic information.

Table 1 Family sociodemographics

Demographic	*M* (*SD*/%)
Mother ($n = 188$)	
Age in years [*M* (*SD*)]	38.69 (5.62)
Father ($n = 188$)	
Age in years [*M* (*SD*)]	40.76 (6.19)
Parent couple	
Couple relationship length, years [*M* (*SD*)]	14.55 (5.59)
Average household income	$80,000–$89,000
Both biological parents [N (%)]	167 (89)
Biological mom, stepdad [N (%)]	11 (5)
Biological dad, stepmom [N (%)]	5 (3)
Both adoptive parents [N (%)]	5 (3)
Target child ($n = 188$)	
Male [N (%)]	162 (86)
Age in years [*M* (*SD*)]	7.90 (2.25)
Child ethnicity [N (%)]	
White, non-Hispanic	156 (83)
Latino	13 (7)
Asian	8 (4)
Black	6 (3)
Native American	1 (1)
Multiple racial/ethnic groups	5 (3)
ID [N (%)]	65 (34.4)

M mean, *SD* standard deviation, *N* sample size, *ID* intellectual disability

Procedure

The Institutional Review Board at University of Wisconsin-Madison approved the present study. Parents provided informed consent prior to study participation. Mothers and fathers separately completed questionnaires about their own depression symptoms and the autistic youth's internalizing mental health problems and jointly reported on family demographics at each time point. Parents were paid $50 each for this portion of the study at every time point.

Measures

Family Sociodemographics

Parents reported on family sociodemographics including child age (years) and child intellectual disability status (0 = no ID, 1 = ID), based on the presence of an ID diagnosis or an indication that the child met criteria for ID via IQ testing. Parents also reported on their household income (Scale of 1–14; 1 = $1–$9,999, 14 = $160,000+).

Parent Depression Symptoms

Parental depression symptoms were assessed using the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977). The CES-D consists of 20 items that are summed into a total score. Mothers and fathers separately reported on how often they felt each item during the past week using a 4-point scale (e.g., 0 = rarely or none of the time to 3 = most or all of the time). Example items are: (1) I was bothered by things that don't usually bother me; (2) I felt that I could not shake off the blues even with help from my family or friends. A CES-D total score of ≥ 16 is indicative of clinically significant depression symptoms (Radloff, 1977). In the current sample, the CES-D had high internal consistency across time in mothers (Chronbach's $\alpha = 0.92–0.93$) and fathers (Chronbach's $\alpha = 0.89–0.93$). Table 2 provides the means and standard deviations for mother- and father-reported CES-D total scores.

Child Internalizing Mental Health Problems

Child internalizing mental health problems were assessed using the child behavior checklist internalizing subscale (CBCL; Achenbach & Rescorla, 2000, 2001) for ages 1.5–5 years (preschool form) and ages 6–18 years (school age form). The CBCL internalizing scale is composed of 32 items and is broken into three subscales: (1) anxious/depressed; (2) withdrawn/depressed; (3) somatic complaints. Items are scored based on a three-point Likert scale (*0 = not*

Table 2 Mother and father reported means, standard deviations for main variables

Measure	Mother *M* (*SD*)	Father *M* (*SD*)	*t*-value	*df*	*p*-value
Time 1	*n* = 187	*n* = 187			
CES-D Total	13.58 (10.35)	11.41 (8.79)	2.289	184	0.023*
CBCL Int. T	63.11 (9.44)	61.98 (9.40)	1.345	187	0.180
Time 2	*n* = 162	*n* = 157			
CES-D Total	14.85 (11.45)	12.59 (9.72)	2.291	154	0.023*
CBCL Int. T	61.41 (9.17)	60.68 (9.24)	1.103	156	0.272
Time 3	*n* = 137	*n* = 134			
CES-D Total	14.86 (11.29)	11.14 (9.51)	3.103	130	0.002**
CBCL Int. T	61.42 (9.28)	59.82 (8.46)	2.007	131	0.047*
Time 4	*n* = 127	*n* = 125			
CES-D Total	14.68 (10.70)	12.40 (10.41)	2.199	116	0.030*
CBCL Int. T	61.02 (9.28)	60.48 (9.05)	0.816	121	0.416

M mean, *SD* standard deviation, *t*-value value for paired-samples *t*-test, *df* degrees of freedom, *CES-D Total* center for epidemiological studies-depression scale total score (Radloff, 1977), *CBCL Int. T* child behavior checklist internalizing T-score (Achenbach & Rescorla, 2001)

*$p < 0.05$

**$p < 0.01$

true, 1 = somewhat or sometimes true, 2 = very true or often true). In the present study, analyses included the Total Internalizing T-score, which draws on all three Internalizing subscales. The CBCL has been shown to have strong reliability in the ASD population (Pandolfi et al., 2014). In the present sample, the CBCL Internalizing subscale had a high internal consistency across time as reported by both mothers (Cronbach's α = 0.84–0.85) and fathers (Cronbach's α = 0.82–85). The means and standard deviations for mother- and father-reported CBCL Total Internalizing T-scores are presented in Table 2.

Data Analysis Plan

Descriptive statistics and boxplots were used to identify potential outliers and examine the distribution of study variables. In order to examine relatedness among the variables, Pearson correlations between parental depression and child internalizing problems were examined. In addition, to determine covariates to include in primary analyses, Pearson correlations assessing the association between the main study variables (e.g., parental depression, child internalizing mental health problems) and family sociodemographics (e.g., child age, child ID status, and household income) were also conducted.

Structural equation modeling (SEM) was utilized to explore the bidirectional effects between parental depression symptoms and child internalizing mental health problems. Specifically, a multi-group cross-lagged panel model was conducted in MPlus7 (Muthen & Muthen, 2012), using maximum likelihood parameter estimators. Full information maximum likelihood (FIML) was used to account for missing data (Little, 2013). The cross-lagged panel model allows for the examination of bidirectional associations as well as controls for the stability of variables over time (Kearney, 2016). Lagged paths from T1 to T3 as well as from T2 and T4 were added for model stability. Parental depression symptoms and child internalizing mental health problems were entered as continuous variables, and mothers and fathers were entered as a dichotomous grouping variable (e.g., mothers = 1, fathers = 2). Based on recommendations from Little (2013), various model fit indices were examined including the chi-square statistic, root mean squared error of approximation (RMSEA), Tucker–Lewis index (TLI), and comparative fit index (CFI). Models included all 188 T1 families given the theorized genetic (in addition to environmental) pathways of effects between parent depression and child internalizing mental health problems, however, the above SEM models were re-run in follow-up analyses excluding the 21 families with a non-biological parent.

Results

Preliminary Analyses

The main study variables (parental depression symptoms and child internalizing mental health problems) were normally distributed without skew (kurtosis range for CES-D = 0.620–1.476; − 0.205 to 0.521 for CBCL). MCAR tests indicated that data were missing completely at random on the main study variables ($X^2 = 15.38$–29.06, $ps > 0.05$). At T1, 188 families completed the study. Of these families, 61 did not complete one or more of the subsequent time points (completed N: T1 = 188; T2 = 163; T3 = 137; T4 = 127). There were various reasons for study attrition, including no longer being interested in participating, being too busy, and unable to be contacted. Attrition analyses were conducted in order to examine whether families who completed all time points (coded as 0 for "completers") differed from families who had missing data on one or more time points (coded as 1 for "incompleters") on the main study variables of parental depression and child internalizing mental health problems. Independent t-tests revealed no significant differences between the two groups in parental depression [mother report: $t(183) = 0.358$, $p = 0.721$; father report: $t(186) = -1.102$, $p = 0.309$] and child internalizing mental health problems [mother report: $t(186) = 0.244$, $p = 0.808$; father report: $t(186) = -0.831$, $p = 0.407$]. Additional independent *t* tests also indicated no significant differences between the families who completed all four time points

and those who did not in regards to sociodemographics variables.

Means and standard deviations for the main study variables are provided in Table 2. Additionally, within-couple differences between mother and father reports were examined through paired sample *t*-tests, and these values can also be found in Table 2. Mothers and fathers reported significantly different scores on the CES-D at all four time points, with mothers reporting higher levels of depression than fathers. Table 3 displays the number and percentages of mothers and fathers with a CES-D score in the clinical range across the four time points and were as follows: T1 61(33%) of mothers and 46(25%) of fathers; T2 59(36%) of mothers and 47(30%) of fathers; T3 54(39%) of mothers and 29(22%) of fathers; T4 49(39%) of mothers and 29(23%) of fathers. Overall, 33–68 (26–36%) of youth with ASD had clinically elevated (i.e., *t*-score ≥ 70) internalizing scores as rated by mothers and fathers across the four time points (Table 3).

Pearson correlations between parental depression symptoms, child internalizing mental health problems, and sociodemographic variables (i.e., child ID status, child age, household income) can be found in Table 4. There

Table 3 Number and percent of mothers, fathers, and children exceeding cutoff for either parent depressive symptoms or child internalizing problems

Family member	Time 1 *n*(%)	Time 2 *n*(%)	Time 3 *n*(%)	Time 4 *n*(%)
Mom	61 (33)	59 (36)	54 (39)	49 (39)
Dad	46 (25)	47 (30)	29 (22)	29 (23)
Child	68 (36)	42 (26)	43 (31)	33 (26)
Mom, dad, and child	12 (6)	14 (9)	9 (7)	7 (6)

Cutoff for parent depressive symptoms is a total score ≥ 16 on the center for epidemiological studies-depression scale (CES-D; Radloff, 1977). Cutoff for child internalizing problems is a child behavior checklist (CBCL; Achenbach & Rescorla, 2001) internalizing *t*-score ≥ 70

Table 4 Correlations among study variables for mothers (shaded and above the diagonal) and fathers (unshaded and below the diagonal)

	1	2	3	4	5	6	7	8	9	10	11
1. Youth Age T1	---	-.072	.122	.030	.187*	.089	.092	-.046	-.108	-.047	-.022
2. Youth ID[1]	-.072	---	-.142	-.042	-.059	-.045	-.080	.017	.043	-.053	.063
3. Income T1	.122	.142	---	.081	.090	.089	.124	-.130	-.144	-.067	-.044
4.CBCL[2] T1	-.037	.008	.025	---	.662**	.637**	.627**	.282**	.235**	.308**	.232**
5.CBCL T2	.121	.045	-.037	.591**	---	.704**	.707**	.316**	.319**	.317**	.354**
6.CBCL T3	.005	-.074	-.021	.493**	.682**	---	.780**	.396**	.363*	.413**	.440**
7.CBCL T4	.086	-.010	.092	.569**	.670**	.763**	---	.388**	.352**	.342**	.410**
8.CES-D[3] T1	-.020	.019	-.164*	.295**	.315**	.386**	.436**	---	.727**	.699**	.704**
9.CES-D T2	-.045	.049	-.283**	.269**	.307**	.324**	.344**	.703**	---	.746**	.788**
10. CES-D T3	-.073	-.010	-.180*	.229**	.242**	.392**	.316**	.691**	.744**	---	.717**
11.CES-D T4	.062	-.130	-.252**	.209*	.251**	.333**	.338**	.671**	.673**	.736**	---

Pearson correlations

ID intellectual disability, *CBCL* child behavior checklist internalizing *t*-score (Achenbach & Rescorla, 2001), *CES-D* center for epidemiological studies-depression scale total score (Radloff, 1977)

*$p < 0.05$

**$p < 0.01$

were significant positive concurrent associations between mother and father reports of parental depression symptoms and child internalizing problems at all four time points. Additionally, mother report of child internalizing problems at T2 was positively associated with child age ($r = 0.187$, $p = 0.017$). Father report of depression symptoms was negatively associated with household income at all time points (T1: $r = -0.164$, $p = 0.028$; T2: $r = -0.283$, $p = 0.000$; T3: $r = -0.180$, $p = 0.041$; T4: $r = -0.252$, $p = 0.006$). There were no significant associations between child ID status and the main study variables for mother nor father reports. Given these findings, the cross-lagged panel models controlled for child age and household income. Specifically, we regressed parent depression and scores of child internalizing problems on child age and household income at each time point and saved the unstandardized residual scores. These residual scores were then entered into the cross-lagged panel model.

Cross-Lagged Panel Models

Table 5 displays both the standardized and unstandardized path coefficients for the panel model. The model indicated good fit [X^2 (16) = 18.603, $p = 0.2898$; TLI = 0.993; CFI = 0.998; RMSEA = 0.030]. Stability effects in variables across time were present for both mother and father reports. Specifically, mother reports indicated stability in scores of parent depression symptoms (T1–T2: β = 0.737, $p = 0.000$;

Table 5 Path coefficients and standard errors for mother and father reports of parent depressive symptoms and youth internalizing mental health problems

Time point	Mother report *β*(*SE*), standardized	Mother report *β*(*SE*), unstandardized	Father report *β*(*SE*), standardized	Father report *β*(*SE*), unstandardized
Cross effects	Depression → CBCL			
1 → 2	0.181 (0.061)**	0.157 (0.054)**	0.136 (0.064)*	0.145 (0.069)*
2 → 3	0.163 (0.064)**	0.130 (0.051)**	0.061 (0.063)	0.065 (0.067)
3 → 4	0.051 (0.060)	0.041 (0.049)	−0.008 (0.055)	−0.011 (0.078)
	CBCL → Depression			
1 → 2	0.032 (0.057)	0.039 (0.070)	0.079 (0.064)	0.078 (0.063)
2 → 3	0.049 (0.058)	0.063 (0.073)	0.004 (0.068)	0.004 (0.068)
3 → 4	0.166 (0.059)**	0.189 (0.068)**	0.116 (0.089)	0.116 (0.090)
Lagged effects				
CBCL				
1 → 3	0.476 (0.077)***	0.243 (0.080)**	0.035 (0.078)	0.036 (0.081)
2 → 4	0.291 (0.073)***	0.300 (0.077)***	0.031 (0.070)	0.044 (0.100)
Depression				
1 → 3	0.281 (0.084)**	0.311 (0.093)**	0.321 (0.090)***	0.344 (0.098)***
2 → 4	0.587 (0.075)***	0.533 (0.074)***	0.212 (0.093)*	0.222 (0.098)*

CBCL child behavior checklist (Achenbach & Rescorla, 2001)

*$p < 0.05$

**$p < 0.01$

***$p < 0.001$

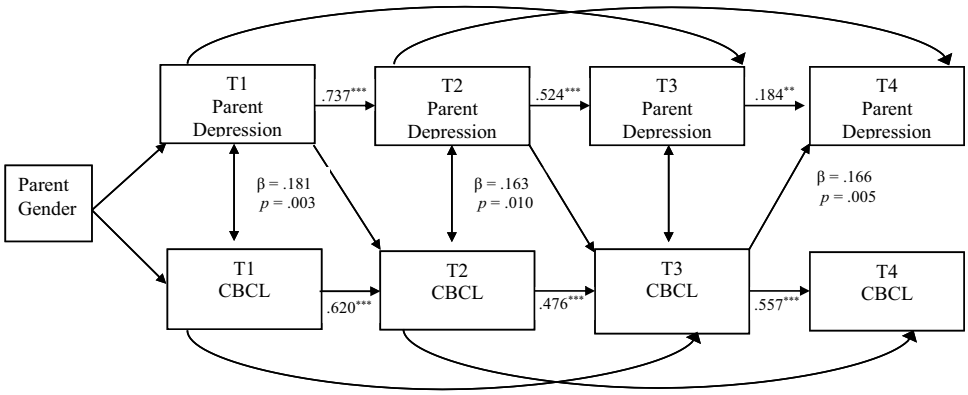

Fig. 1 Results of the cross-lagged panel model for mother-reported parent depression and youth internalizing mental health problems, controlling for child age and household income. Values are standardized path estimates. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

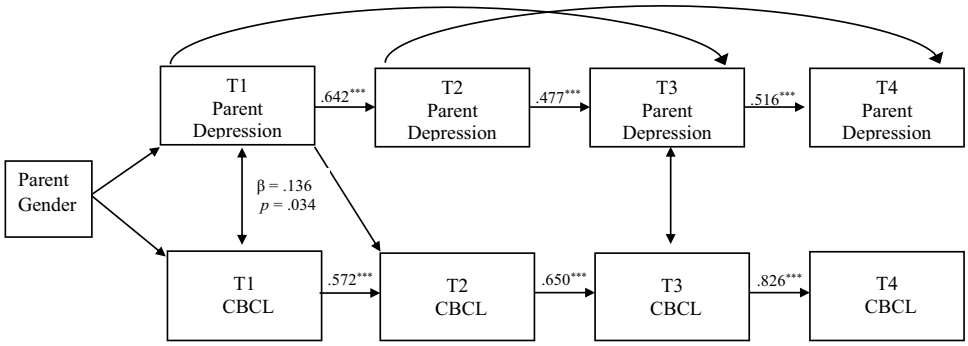

Fig. 2 Results of the cross-lagged panel model for father-reported parent depression and youth internalizing mental health problems, controlling for child age and household income. Values are standardized path estimates. $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

T2–T3: β = 0.524, $p = 0.000$; T3–T4: β = 0.184, $p = 0.027$) as well as in scores of child internalizing problems (T1–T2: β = 0.620, $p = 0.000$; T2–T3: β = 0.476, $p = 0.000$; T3–T4: β = 0.557, $p = 0.000$). Father reports also indicated stability in scores of parent depression (T1–T2: β = 0.642, $p = 0.000$; T2–T3: β = 0.477, $p = 0.000$; T3–T4: β = 0.516, $p = 0.000$) and in scores of child internalizing problems (T1–T2: β = 0.572, $p = 0.000$; T2–T3: β = 0.650, $p = 0.000$; T3–T4: β = 0.826, $p = 0.000$). After controlling for child age and household income, the cross-lagged panel model revealed that maternal depression symptoms at T1 predicted child internalizing problems at T2 (β = 0.181, $p = 0.003$) and maternal depression symptoms at T2 predicted child internalizing problems at T3 (β = 0.163, $p = 0.010$). In the opposite direction, child internalizing problems at T3 predicted maternal depression symptoms at T4 (β = 0.166, $p = 0.005$). Additionally, father's depression symptoms at T1 predicted child internalizing problems at T2 (β = 0.136, $p = 0.034$). Figures 1 and 2 depict the significant pathways in the cross-lagged panel model for mothers and fathers, respectively. Given the potential role of genetic mechanisms in linking parent depression to youth internalizing problems, we reran the above model after excluding the 21 families that included a non-biological parent(s). All significant pathways remained and thus models with all 188 families are reported on.

Discussion

The developmental psychopathology framework suggests that mental health problems arise from a dynamic interplay between an individual's genetic and neurobiological vulnerabilities and an ongoing interplay with the environment (Cummings et al., 2000; Eme, 2017). Within this framework, parental depression may serve as both a genetic and environmental factor that both shapes and is shaped by the internalizing mental health of autistic youth. Consistent with previous reports (e.g., Cohrs & Leslie, 2017; Schnabel et al., 2020), 33–39% of mothers and 22–30% of fathers of autistic youth in the current study reported clinically-elevated depression symptoms, and approximately one-third (26–31%) of autistic youth had clinically-elevated internalizing mental health problems across the three year study. Results also indicate important associations between the internalizing mental health problems of autistic youth and the depression symptoms of parents across time.

There was evidence to suggest that parental depression may contribute to greater child internalizing mental health problems. Specifically, from T1 to T2 (using both mother and father report) and from T2 to T3 (using mother report), higher levels of parent depression symptoms predicted increased internalizing mental health problems in the autistic youth 12 months later. It is important to note that this association could be driven by genes, the environment, or possibly both, consistent with prior work in non-ASD populations (e.g., England & Sim, 2009; Kamis, 2021; Taraban & Shaw, 2018; Tirumalaraju et al., 2020). In the other direction, from T3 to T4, mother-report of child internalizing mental health problems predicted increased maternal depression symptoms 12 months later. Thus, parenting an autistic youth with high internalizing mental health problems may be taxing and contribute to the risk for maternal depression. It is important to note that this direction of effects was present from T3 to T4, but not for earlier time points. Early adolescence is well known as a period of increased risk for internalizing symptoms in neurotypical children, with the incidence of depression doubling from childhood to adolescence (McLaughlin & King, 2014) with a change point around 12 years old (Cohen et al., 2018). Similar patterns have also been found in autistic samples (e.g., Gotham et al., 2015), with evidence of a slightly earlier change point for the increase in internalizing mental symptoms in ASD as compared to neurotypical samples (e.g., Schwartzman & Corbett, 2020). In the present study, at T3 autistic youth were aged 8–15 years ($M = 10$ years). It is also possible that internalizing mental health problems may present in different ways across development and/or it may be more difficult for mothers to manage these problems in older autistic children and autistic adolescents as compared to in younger autistic children. Moreover, overtime, the internalizing mental health

problems of the autistic youth may take a greater toll on mothers as they are increasingly seen as chronic problems.

The connections between parent depression and child internalizing problems were most pronounced for mothers. It is unclear why we only found one effect for father report (e.g., father depression at T1 predicted child internalizing at T2). Some research has found that mothers report higher levels of youth internalizing mental health problems, suggesting that mothers could be more aware of these problems and/or observe more of them than fathers, albeit findings are mixed (e.g., van der Veen-Mulders et al., 2017). In part, this could be related to findings that mothers tend to report taking on more parenting responsibilities, on average, than fathers in families of autistic youth (e.g., Hartley et al., 2014; May et al., 2017). As a result, maternal depression symptoms may be more strongly influenced by and be a stronger influencer of internalizing mental health problems in autistic youth than paternal depression symptoms. However, it is important to note that mothers reported more depressive symptoms than fathers, on average. The higher level of depressive symptoms may have biased mothers' rating of the son/daughter/s internal mental health problems to be more in line with mother's own mood. In addition to environmental pathways, it is possible that genetic pathways linking parental depression symptoms and the internalizing mental health problems of autistic youth are stronger in mothers than fathers. Indeed, in previous epidemiological research studies showing a link between depression at earlier life stages and later risk of having an autistic child, effects were stronger in mothers than fathers (Cohen & Tsiouris, 2006; Goodman & Gotlib, 1999; Tirumalaraju et al., 2020). More research is needed to explore these and other potential mechanisms.

Study Strengths, Limitations, and Future Directions

The present study had many strengths including the cross-lagged panel modeling approach which allowed for the examination of bidirectional pathways between parent depression symptoms and child internalizing mental health problems across time in both mothers and fathers of autistic youth. That said, our sample primarily consisted of White, non-Hispanic parents, which limits the generalizability of findings. Future research is needed to determine whether the pattern of effects would remain in larger and more diverse samples. Our sample also only included coupled parents; thus, findings may not be applicable to the experiences of families in single-parent households. Although attrition analyses revealed no significant differences at T1 between families who did and did not complete all time points, families may have differed in other areas not examined. The present study also focused on a single reporter (e.g., mother report of parent depression predicting mother report of child internalizing mental health problems) because we wanted to examine perspectives unique to each parent. In other words, each parent has experiences within their parent–child relationship separate from the other parent's experiences. Given what we know of the effects of parental mental health on perceptions of child behavior (e.g., Gartstein et al., 2009), we acknowledge that this may have inflated associations. Nevertheless, we believe the single-reporter method is important to understand as the transactional nature of the parent–child relationship may perpetuate both parental depression as well as child internalizing symptoms. It is also important to note that 11% of our families were composed of at least one non-biological parent. These families were removed in follow-up analyses and significant pathways of effects did not change. However, future research should leverage larger samples of non-biological parents to isolate the effects of environmental, relative to genetic, pathways between parental depression and internalizing mental health problems in autistic youth. The present study is also limited in that it did not assess lifetime parent mental health history in biological parents; however, this will also be important for determining genetic pathways of effects. Future research should also examine links between the broader autism phenotype (BAP), which involves sub-clinical autism traits and has an increased prevalence in genetic relatives of autistic youth (Piven et al., 1997). BAP has been posited to overlap to some degree with depression (e.g., Asano et al., 2014; Ingersoll & Hambrick, 2011; Pruitt et al., 2018), and thus parent BAP may also have genetic links to internalizing mental health problems in autistic youth.

Study Implications

Findings emphasize a need for interventions focused on improving the mental health of both parents and autistic youth. Specifically, it is important to screen for both parent and child mental health conditions in families of autistic youth. It may also be important to offer family-wide, not just individual-level, supports and interventions given evidence that the mental health of parents affects the autistic youth and vice versa. As a result, the mental health of both the parent and autistic youth may need to be targeted for optimal gains. Indeed, there is evidence from non-ASD populations that if parents are depressed, treatment-related gains for the children are reduced (e.g., Eckshtain, et al., 2019). Examples of family-wide programs could include mindfulness training and psychoeducation on child emotion and behavior management for parents combined with cognitive behavioral therapy for the autistic youth. Future research should explore the mechanisms that underlie the potentially genetic and environmental pathways (and specific parenting and child behaviors that may mediate the environmental

effects) between parent depression and internalizing mental health problems in autistic youth.

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Author Contributions BPG: conceptualization, formal analysis, writing (original draft); JG: writing (review and editing); EJH: writing (review and editing); JMP: writing (review and editing); EL: writing (review and editing); SLH: conceptualization, writing (review and editing), supervision, funding acquisition. All authors have read and approved the final version of the manuscript.

Declarations

Conflict of interest The authors reported no conflict of interest.

Ethical Approval The study was approved by the University of Wisconsin-Madison IRB. All procedures followed the ethical standards of the institutional and national responsible committee on human experimentation.

Consent to Participate Written informed consent was obtained from all parents participating in this study prior to data collection.

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