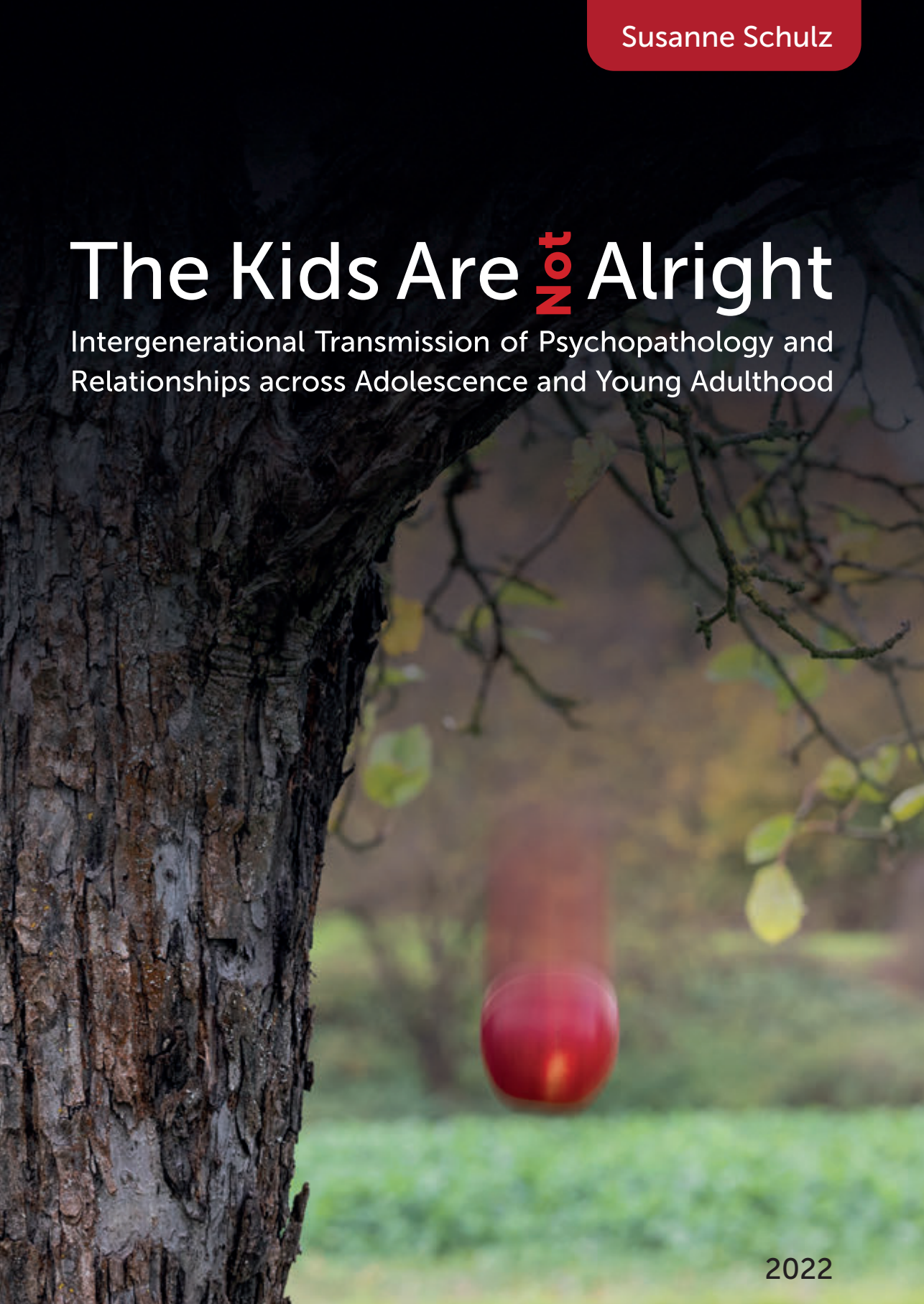
Susanne Schulz

The Kids Are Not Alright

Intergenerational Transmission of Psychopathology and Relationships across Adolescence and Young Adulthood



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The Kids Are Not Alright

Intergenerational Transmission of Psychopathology and Relationships across Adolescence and Young Adulthood

De Kinderen Zijn Niet Oké

Intergenerationele Overdracht van Psychopathologie en Relaties in de Adolescentie en Jongvolwassenheid

(met een samenvatting in het Nederlands)

Die Kinder Sind Nicht Okay

Intergenerationelle Weitergabe von Psychopathologie und Beziehungen im Jugend- und Erwachsenenalter

(mit einer Zusammenfassung in deutscher Sprache)

Proefschrift

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One year
Twenty years
Forty years
Fifty years
Down the road in your life
You'll look in the mirror
And say, "My parents are still alive."
– Modest Mouse

For Yuna Lou
'Cause if you stay with us you're gonna be pretty kookie, too. – David Bowie

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CHAPTER 1

General Introduction

Adolescence is a crucial developmental period that is characterized by important contextual, biological, and interpersonal changes. The onset of puberty and management of new developmental demands call for refined emotional and interaction behaviors (Dahl, Allen, Wilbrecht, & Suleiman, 2018). Adolescents experience more extreme and variable emotions than children (Arnett, 1999; Larson, Moneta, Richards, & Wilson, 2002; Reitsema, Jeronimus, Dijk, & Jonge, 2022), which they need to learn to regulate to be able to flexibly respond to environmental demands. At the same time, adolescents' increasing social competence and independence stimulate changes in existing social relationships (Branje, Mastrotheodoros, & Laursen, 2021; Laursen & Bukowski, 1997) and motivate the need to form healthy, positive relationships with peers and romantic partners. These developmental tasks and demands provide many opportunities, but also leave adolescents vulnerable to a variety of social-emotional problems, such as psychopathology and disrupted relationships, which can have detrimental consequences for health and adjustment in adulthood (Cohen, 2004; Korhonen, Luoma, Salmelin, Siirtola, & Puura, 2018; Schwartz-Mette, Shankman, Dueweke, Borowski, & Rose, 2020).

Parents play an important role in adolescents' social-emotional development. They help adolescents to manage their emotions and guide positive relationship behaviors (Eisenberg, Cumberland, & Spinrad, 1998). At the same time, parents are also among the most salient risk factors for adolescents' adjustment problems. Parental problem behaviors are often observed to inadvertently continue from one generation to the next via genetic and environmental pathways (Branje et al., 2020; Kerr & Bowen, 1988). At the same time, parental behaviors might not only shape children's behaviors, but children are also highly likely to influence their parents (Bell, 1968; Sameroff, 2009). Despite a long history of research on intergenerational transmission, much remains unknown about bidirectional associations between parental and adolescent problem behaviors as well as the mechanisms that explain how and for whom intergenerational continuity is maintained across time.

To address these questions, this dissertation employs a transactional, longitudinal perspective to provide novel insights into intergenerational transmission processes across adolescence and young adulthood. Specifically, the present dissertation investigates how parental and adolescent problem behaviors are reciprocally associated, focusing on the transmission of characteristics that are salient in adolescence and develop in the context parent-child transactions: Psychopathological symptoms and relationship behaviors.

A Theoretical Framework on Intergenerational Transmission

Intergenerational transmission refers to the way in which traits, behaviors, or characteristics transfer from one generation to the next. Particularly psychopathology, which comprises internalizing (e.g., depression or anxiety) and externalizing (e.g., aggression or delinquency) symptoms, has been

observed to be transmitted between parents and their children (e.g., Connell & Goodman, 2002; Goodman et al., 2011; Meeus, 2016; Schneider, Atkinson, & Tardif, 2001). Genetic similarities play a role in intergenerational transmission processes (e.g., Rutter, Moffitt, & Caspi, 2006), but they cannot fully explain the associations between parental and adolescent problem behaviors (Eley et al., 2015; Natsuaki et al., 2014). Among additional mechanisms that have been proposed to explain transmission effects, including altered neuroregulatory systems through prenatal experiences and the shared family environment, such as socioeconomic stressors or cultural factors, parenting may be particularly relevant (Deater-Deckard, 2001; Goodman & Gotlib, 1999; Goodman, Simon, Shamblaw, & Kim, 2020; Serbin & Karp, 2004). Indeed, aspects of parenting, including parental (and child) cognitions, behaviors, and affect, are the most frequently proposed and studied pathway in the intergenerational transmission of psychopathology, as parental behaviors directly link to parents' and children's emotions and are not just easily observable, but also modifiable. One way through which psychopathological symptoms are transmitted from parents to adolescents is parental modeling of affect and behavior (Bandura, 1977). At the same time, psychopathological symptoms of parents also affect their children as they can decrease the parent's emotional availability towards their children (Goodman et al., 2020) and thus disrupt interactions with them (Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Rueger, Katz, Risser, & Lovejoy, 2011). Disrupted parent-child interactions in turn increase adolescents' risk for psychopathological symptoms, as they might feel emotionally insecure or rejected (Cummings & Davies, 1996; Garber & Flynn, 2001; Stocker, Richmond, Rhoades, & Kiang, 2007).

Interaction behaviors between parents and adolescents may not only underlie intergenerational transmission processes; they can be transmitted as well. Similar to the transmission of psychopathological symptoms, parental modeling plays an important role, as adolescents observe and imitate behaviors that they experience in interactions with their parents (Bandura, 1977). These observed behaviors are translated into internal working models or schemas of relationships that guide adolescents' future behaviors (Dodge & Pettit, 2003; Furman & Collibee, 2018). Constant exchange among the different interrelated social systems in which adolescents are embedded (e.g., Hartup, 1979; Kerr & Bowen, 1988) further facilitate transmission processes between parents and children through modified emotions and behaviors. In this way, positive and negative aspects of parent-child relationships can be transferred to relationships with other social partners, such as peers and romantic partners.

Aims of this Dissertation

Despite a long tradition of studying intergenerational transmission, important questions still remain in understanding the complex processes involved in parent-child transactions. Using multi-

method longitudinal data and state-of-the art methodological designs, this dissertation pursues three overarching aims to address existing gaps in research and provide innovative insights into the intergenerational transmission of psychopathology and relationships: 1) to investigate how psychopathological symptoms and relationship behaviors are transmitted across adolescence and whether there are changes in parental influences across time, 2) to shed light on the direction of transmission effects between parents and adolescents, and 3) to provide insights into potential mechanisms involved in intergenerational transmission processes. The following section elaborates on these aims and describes how they are addressed in this dissertation. A schematic overview of the associations between all studies and concepts is depicted in Figure 1.

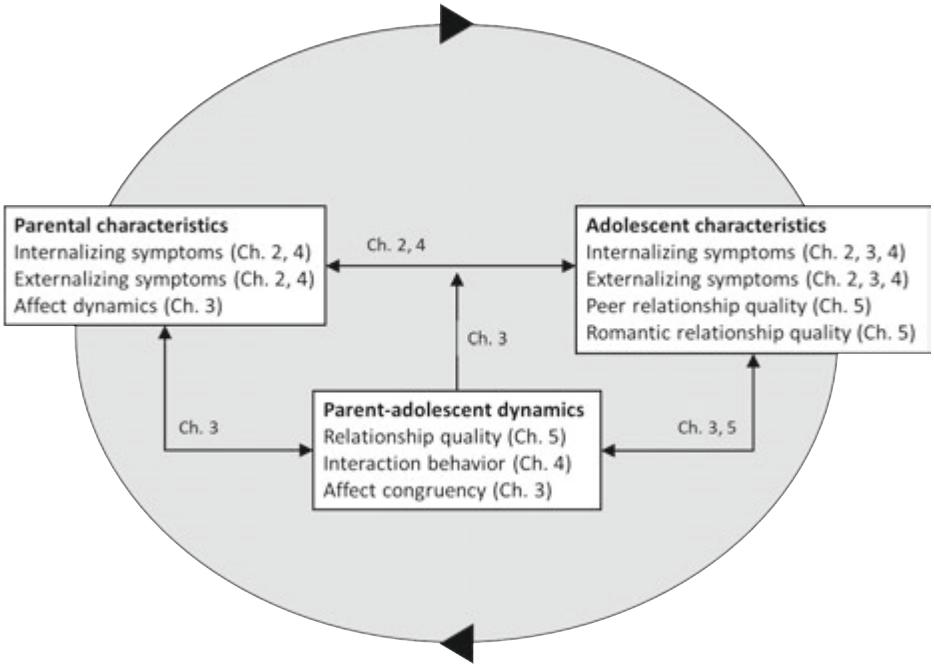

Figure 1. Overview of intergenerational transmission processes examined in this dissertation.

Aim 1: Intergenerational transmission processes across adolescence and young adulthood

The first and overarching aim of this dissertation was to investigate how intergenerational transmission processes unfold across adolescence and young adulthood, focusing on the transmission of psychopathology (Chapter 2, 3, 4) and relationship quality (Chapter 5). Driven by profound changes in youth's emotional experiences and their relationships, adolescence is often considered a sensitive period for social learning (Dahl et al., 2018; Laursen & Veenstra, 2021). Transmission effects between parents and children may therefore be particularly critical

in this period. Besides parents and adolescents exhibiting similar symptoms and behaviors (i.e., intergenerational similarity), much remains unknown about how symptoms and behaviors are transmitted across adolescence and whether these transmission processes change over time.

Psychopathological symptoms most often emerge and increase during adolescent years (Kim-Cohen et al., 2003; Nivard et al., 2017). Adolescent psychopathological problems are highly prevalent and impose 10-times the health costs of mental health problems that develop in adulthood (WHO, 2007). Emotional challenges during this time, such as regulating more variable, intense emotions or increased negative interactions with parents (Branje, 2018; Hadiwijaya, Klimstra, Vermunt, Branje, & Meeus, 2017), leave adolescents particularly insecure and thus sensitive to parental symptoms and behaviors. Not just their emotional development, but also social changes argue for adolescence as an important period for intergenerational transmission. As children strive towards increased independence from their parents, parent-adolescent relationships also undergo major changes that are accompanied by modified interaction patterns (Branje et al., 2021). While parent-adolescent relationships become more reciprocal and align towards horizontality, relationships with peers and romantic partners emerge and become other important sources of support (Bagwell & Bukowski, 2018). Interaction patterns in relationships with parents are likely to provide prototypes or working models for these developing relationships.

From a traditional, purely generational, perspective, examining how symptoms and behaviors are transmitted from one generation to the next (i.e., from parents to children) requires assessing parents and children at the same ages or developmental stages (Thornberry, 2016). Specifically, do parental symptoms or behaviors at a specific point in time, such as during their adolescent years, affect their children's symptoms or behaviors at the same age in adolescence? While such assessments are important as they reflect similar experiences or expressions of behaviors in both generations (Cairn et al., 1998; Patterson, 1998), transmission processes also occur in proximity. To understand how children respond to being exposed to parental feelings and behaviors, it is therefore important to also assess both parental and adolescents' behaviors simultaneously as they co-occur. Studies examining proximal processes between parents and children mainly used cross-sectional designs that provide insights into intergenerational similarity, but cannot shed light on the consistency and change of transmission effects over time. Evidence on transmission effects from longitudinal studies that span multiple years across adolescent development remain scarce. The studies in this dissertation aimed to unravel transmission processes within the formative period of adolescence and young adulthood by assessing parents and adolescents repeatedly at the same moment in time.

Proximal longitudinal processes between parents and children can further complement our understanding of intergenerational transmission by identifying patterns of transmission. The influence of parents on adolescents' development is likely to change across time – both with regard to the timing of parental influence (i.e., influence in early vs. late adolescence) as well as the timing of adolescent outcomes (i.e., short-term vs. long-term effects). For example, changes in early adolescence might be particularly influential for children's development, but the strengths of these associations might depend on short- or long-term outcomes. As early experiences provide a foundation for later experiences that are likely to shape development more directly, theories on transient effects (Fraley & Roisman, 2015; Rutter, 1996; Schulenberg, Sameroff, & Cicchetti, 2004) propose that current experiences weaken the effects of earlier experiences. Based on these perspectives, parental effects on adolescents' development are expected to fade across time. Theories on enduring effects (Fraley & Roisman, 2015; Sroufe, Egeland, & Kreutzer, 1990), on the other hand, suggest that previous experiences, particularly those during sensitive developmental periods, continue to shape development across time by scaffolding current experiences and thus providing long-lasting working models. Similarly, transmission effects might even become stronger over time as adolescents resemble their parents more closely as they get older (Halberstadt & Eaton, 2002). According to these perspectives, the transmission processes between parents and adolescents are expected to remain stable, or even increase, over time.

The present dissertation examined how intergenerational transmission processes between parents and adolescents unfold across adolescence, using a long-term longitudinal design from early adolescence to young adulthood. Specifically, how parental symptoms were associated with adolescents' internalizing and externalizing symptoms was investigated from early to late adolescence (Chapter 2) and into young adulthood (Chapter 3). In a meta-analytic study, existing evidence on the longitudinal associations between parent-adolescent and peer or romantic relationships across time was quantified (Chapter 5). Moderation analyses further demonstrated how these associations and parental influences change across adolescence and young adulthood.

Aim 2: Transactional processes in the intergenerational transmission of psychopathology and relationships

Traditional transmission models (e.g., transmission of psychopathology model, Goodman & Gotlib, 1999; Goodman et al., 2020; social mold models, Furman & Collins, 2009; Hartup, 1978) typically assume unidirectional transmission from parents to adolescents, and thus do not provide conclusions about reciprocal effects from adolescents to parents. Ignoring potential adolescent-to-parent effects might result in spuriously inflated estimates regarding intergenerational transmission processes. The second aim of this dissertation was therefore to examine potential bidirectional

associations in the intergenerational transmission between parents and adolescents, to disentangle whether transmission in adolescence is driven by effects from parents to adolescents (i.e., parent effects) or from adolescents to parents (i.e., adolescent effects). Specifically, children's symptoms and behaviors can prompt evocative processes by eliciting changes in parental feelings or behaviors. Adolescents' anxious feelings or aggressive behaviors, for example, may evoke nonoptimal parental responses, such as increased stress or adverse parenting behaviors (Berg-Nielsen, Vikan, & Dahl, 2002; Serbin, Kingdon, Ruttle, & Stack, 2015; Sheeber, Hops, & Davis, 2001), which may in turn negatively affect parental as well as adolescent adjustment. Repeated negative transactions between parents and adolescents can ultimately give rise to coercive processes, in which parents and adolescents inadvertently reinforce each other's adverse emotions or behaviors (Patterson, 1982). In that way, specific symptoms or behaviors often accumulate in families.

Initial studies provide some support for bidirectional associations between parental and adolescent symptoms and behaviors (e.g., de Goede, de Branje, de Delsing, & Meeus, 2009; Ge, Conger, Lorenz, Shanahan, & Elder, 1995; Hughes & Gullone, 2010), but findings remain inconclusive. This dissertation therefore investigated bidirectional associations between parental and adolescent psychopathological symptoms (Chapter 2, 4) as well as relationship behaviors (Chapter 5) to provide insights into the direction of transmission processes in adolescence and young adulthood.

Aim 3: Understanding how and for whom – Mechanisms and moderators in the intergenerational transmission of psychopathology and relationships

Although several potential pathways and moderators that may explain intergenerational transmission have been theoretically proposed, few have been tested empirically. The third aim of this dissertation was to (1) examine parent-child dynamics as mechanisms that play a role in the transmission of psychopathological symptoms and (2) examine which children are at particular risk for, or resilient against, intergenerational transmission processes.

The emotional climate in the family, characterized by parental affect and parent-adolescent interaction behavior, plays a crucial role in how behaviors are transmitted between parents and adolescents (e.g., Goodman, Simon, Shamblaw, & Kim, 2020; Morris, Silk, Steinberg, Myers, & Robinson, 2007). As gender inequality and traditional family roles still persist despite significant improvements in the past decades (Mize, Kaufman, & Petts, 2021; Scarborough, Sin, & Risman, 2019), children usually spend more time with their mothers than other caregivers. Mothers have not only been found to use more emotion coaching than fathers (Stocker et al., 2007), but their emotional expressions and socialization practices also seem most relevant for children's emotional development (Aznar & Tenenbaum, 2013; McNeil & Repetti, 2021). Maternal affect and behaviors

are thus particularly influential in children's lives and central in shaping the emotional climate in the family. Psychopathological symptoms are likely to generate a maladaptive emotional climate, as they render mothers to be less sensitive to their children and to express more negative and less positive affect (Morris et al., 2007). Additionally, maternal symptoms not only influence how mothers interact with their children, but can also elicit negative emotions or behaviors from adolescents (e.g., Goodman et al., 2011), which leaves them similarly vulnerable to psychopathological symptoms.

The emotional climate that mothers create constitutes repeated expressions and dynamic interactions within the family that unfold across days or even moments. Understanding mother-adolescent dynamics and their role in intergenerational transmission therefore requires examining them in repeated intensive assessments in, or close to, real time (e.g., moment-to-moment, day-to-day). This dissertation used novel methodological and statistical approaches, such as experience sampling methods and Dynamic Structural Equation Modelling (DSEM; Asparouhov, Hamaker, & Muthén, 2018) to shed light on the emotional (i.e., maternal affect; Chapter 3) and behavioral (i.e., mother-adolescent interaction behavior; Chapter 4) family dynamics underlying intergenerational transmission. Studies investigating momentary or daily parent-child dynamics as mediators in the transmission of psychopathology, particularly in prospective longitudinal designs, are lacking. Most evidence is based on studies that investigated some pathways of this mediation, such as associations between parental symptoms and parental behaviors (e.g., McCabe, 2014) or between parental behaviors and adolescent symptoms (e.g., Pinquart, 2017b, 2017a). Despite ample support for associations between parental behaviors and adolescent symptoms, these studies provide little information about how maternal affect contributes to adolescent psychopathological symptoms. For that, it is important to not only consider affect levels but also changes in affect across time (i.e., affect dynamics). Chapter 3 aimed to better understand how maternal affect functioned as a potential mechanism in the intergenerational transmission of psychopathology, by examining the associations of daily levels and dynamics of maternal affect with children's internalizing and externalizing symptoms across adolescence and young adulthood.

To further provide insights into longitudinal mediation effects in the transmission of psychopathology, Chapter 4 examined whether moment-to-moment mother-adolescent interaction behavior underlies the associations between maternal and adolescent internalizing symptoms from early to mid-adolescence. Using Bayesian analyses, the study incorporated previous findings on mediation pathways and updated them with new information. Theoretical and empirical findings on the associations between parental and adolescent behaviors that have accumulated in decades of research were used to not only build hypotheses, but also to conduct novel empirical

analyses with new data. By generating cumulative knowledge, this novel approach increases the robustness and precision of the conclusions.

Not all families are the same, and there are likely individual differences in transmission effects. To understand individual risk and resilience, we need to not only focus on intergenerational continuity but also on discontinuity. In other words, we need to understand why certain behaviors are transmitted in some families, but not in others. Risk or protective factors are often attributed to characteristics of the family, such as diagnosed mental health disorders, socioeconomic status, and marital relationship quality, or characteristics of the child, such as gender and age (Connell & Goodman, 2002; Goodman et al., 2011, 2020; Jeon & Neppl, 2019). Additionally, children's sensitivity to their environmental context has been proposed as one source for individual differences (e.g., Pluess, 2015), indicating that children are more or less susceptible to environmental influences. Individual differences in how congruent adolescents are with their mothers' affect may represent such environmental sensitivity. Mother-adolescent affect congruency describes the degree to which adolescents and mothers experience or share the same affect. For high-congruency adolescents, transmission effects from parents to adolescents might be stronger (i.e., more continuity) than for low-congruency adolescents (i.e., more discontinuity). Whether the outcomes are favorable or adverse depends on the emotional environment, such as adaptive or maladaptive maternal behaviors or affect. Previous studies found that affect congruency between close relationship partners was indeed differently related to adjustment, with some studies emphasizing favorable outcomes (e.g., Anderson, Keltner, & John, 2003; Bruder, Dosmukhambetova, Nerb, & Manstead, 2012) and others emphasizing adverse outcomes (Anderson & Keltner, 2004; Mercado, Kim, Gonzales, & Fuligni, 2019). Chapter 3 therefore investigated whether mother-adolescent affect congruency moderated the associations of maternal daily positive and negative affect with adolescent and young adult psychopathological symptoms.

Sample and Study Design

This dissertation combines longitudinal cohort and meta-analytical data. An overview of study characteristics and measures of each chapter can be found in Table 1.

Table 1. Overview of study characteristics and measures per chapter

Chapter	Sample	Design	Parental characteristics	Adolescent characteristics	Mechanism/Moderator	Age
2	RADAR Young (*N* = 497)	Questionnaires (multi-informant) 6 waves	Internalizing symptoms Externalizing symptoms	Internalizing symptoms Externalizing symptoms	-	13-18
3	RADAR Young (*N* = 497)	Questionnaires 7 waves Daily diaries (multi-informant) 75 assessments across 5 waves	Positive affect intensity Positive affect inertia Positive affect variability Negative affect intensity Negative affect inertia Negative affect variability	Internalizing symptoms Externalizing symptoms	Mother-adolescent affect congruency (moderation)	13-27
4	Meta-analysis (*k* = 9) RADAR Young observation sample (*N* = 102)	Questionnaires (multi-informant) 2 waves Observation (multi-directed) 1 wave	Internalizing symptoms	Internalizing symptoms	Positive interaction Negative interaction (mediation)	13-15
5	Meta-analysis (*k* = 87)		Parent-adolescent support Parent-adolescent negative interaction Parent-adolescent control	Peer support Peer negative interaction Peer control Romantic support Romantic negative interaction Romantic control	Age (moderation)	10-27

Longitudinal cohort sample. First, data from the ongoing Research on Adolescent Development And Relationships Young (RADAR-Young) study was used to examine how psychopathological symptoms are associated between parents and adolescents (Chapter 2, 3, and 4). The sample consists of 497 Dutch adolescents (43.1% girls) and their parents (495 mothers and 446 fathers) from mainly intact two-parent families with medium-to-high socio-economic status. Adolescents and their parents were followed across 11 annual or biennial assessments from early adolescence (age 13) to young adulthood (age 27). In the first five assessment waves, adolescents and their mothers additionally participated in 75 additional daily assessments across 15 measurement weeks, and a subsample of 102 mother-adolescent dyads also participated in annual observational assessments.

Meta-analytical samples. Second, two meta-analytical samples from synthesized longitudinal studies were used to examine whether parent-adolescent interaction behaviors underlie the transmission of internalizing psychopathology (Chapter 4) and whether they are also transmitted to other social relationships (Chapter 5). The first meta-analytical sample was used for Bayesian analyses and included 47 effect sizes from four meta-analyses and five longitudinal studies examining associations between parent-adolescent interactions and parent or adolescent internalizing symptoms (Chapter 4). The second meta-analytical sample included 559 effect sizes from 87 independent longitudinal studies (Chapter 5), of which 53 studies involved associations between parent-adolescent interactions and peer outcomes and 38 studies involved associations between parent-adolescent interactions and romantic outcomes.

Fully understanding how parents and adolescents affect each other requires a multidimensional view on intergenerational transmission that considers different time scales as well as perspectives and experiences of the involved individuals. The studies in this dissertation therefore examined intergenerational transmission from a multi-informant, multi-method perspective, combining different time scales from momentary (Chapter 3, 4, 5) to long-term assessments (Chapter 2, 3, 4, 5) as well as different informants, including parental and adolescent self-reports (Chapter 2, 3, 4, 5) and independent raters (Chapter 4, 5). As adolescents are affected by both parents, but most research on intergenerational transmission has focused on mothers, this dissertation also examined whether associations between parental and adolescent psychopathological symptoms differ for fathers and mothers (Chapter 2).

Additionally, this dissertation considered both how families differ from each other and how parents and children affect each other within families. Based on various theoretical models, intergenerational transmission is a transactional process between parents and adolescents that unfolds *within* individual families over time (Berry & Willoughby, 2017). Most studies to date examined processes

related to intergenerational transmission using between-family models or models in which between- and within-family estimates are intertwined. It therefore remains unclear how symptoms or behaviors are transmitted within families, and whether these processes differ from associations between families. Chapter 2 used random-intercept cross-lagged panel models to examine how parental and adolescent psychopathological symptoms are associated within families. Findings from within-family studies are particularly useful to identify which factors could be targeted in programs to break negative (or potentially maintain positive) transmission. At the same time, it is important to consider how interindividual differences between families change over time. Chapters 3, 4, 5 examined how relative changes in parental symptoms or behaviors are associated with relative changes in adolescent symptoms or behaviors. Chapter 3 and 4 further combined both approaches by investigating the role of within-family dynamics in explaining the associations between maternal affect and adolescent psychopathology.

Outline of this Dissertation

Chapter 2 investigated the intergenerational transmission of psychopathological symptoms from early to late adolescence. Specifically, the chapter disentangled within-family processes from between-family associations to examine how changes in parental internalizing and externalizing symptoms predict changes in adolescent internalizing and externalizing symptoms (and vice versa), using a 6-year multi-informant longitudinal design. Zooming in on day-to-day parental effects, Chapter 3 addressed how maternal daily affect levels and dynamics across adolescence contribute to youth internalizing and externalizing symptoms in adolescence and young adulthood. Individual differences in risk for psychopathological symptoms were further identified by investigating the moderating role of mother-adolescent affect congruency. Chapter 4 provided insights into mechanisms in intergenerational transmission processes. Building on existing information from previous studies, the chapter examined whether observed parent-adolescent interaction behaviors underlie the associations between maternal and adolescent internalizing symptoms. Finally, Chapter 5 examined the intergenerational transmission of relationship quality by synthesizing evidence on the longitudinal associations of parent-adolescent relationships with peer and romantic relationships. To further understand when and for whom these transmission processes might be most relevant, moderators in these associations were examined, such as the role of time and adolescent age.

CHAPTER 2

Examining Intergenerational Transmission of Psychopathology: Associations between Parental and Adolescent Internalizing and Externalizing Symptoms Across Adolescence

This chapter is published as:

Schulz, S., Nelemans, S. A., Oldehinkel, A. J., Meeus, W., & Branje, S. (2021). Examining intergenerational transmission of psychopathology: Associations between parental and adolescent internalizing and externalizing symptoms across adolescence. Developmental Psychology, 57(2), 269–283. https://doi.org/10.1037/dev0001144

ABSTRACT

Adolescent psychopathological (i.e., internalizing and externalizing) symptoms are quite prevalent and decrease well-being in adulthood. Parental symptoms can put adolescents at risk for developing psychopathological symptoms. This study examined the reciprocal, longitudinal associations between parental and adolescent internalizing and externalizing symptoms between and within families, using random-intercept cross-lagged panel models (RI-CLPMs). Participants were 497 Dutch adolescents (43.1% girls; M_{age} T_1 =13.0 years; mostly medium to high socioeconomic backgrounds) and their parents from the general population. Across six years, adolescents and their mothers and fathers reported annually on their internalizing and externalizing symptoms. Between families, maternal, but not paternal internalizing and externalizing symptoms were consistently associated with adolescent internalizing and externalizing symptoms, while within families, only increases in adolescent internalizing symptoms predicted subsequent increases in maternal internalizing symptoms. These findings suggest that associations within families differ from associations between families, and that within-family processes in the transmission of internalizing symptoms are particularly driven by adolescent-to-mother effects.

Keywords: intergenerational transmission, psychopathology, adolescence, within-family associations

Author contributions

SuS, SN, SB, and WM conceptualized the study. SuS performed the statistical analyses and drafted the manuscript. All authors provided feedback on the manuscript.

EXAMINING INTERGENERATIONAL TRANSMISSION OF PSYCHOPATHOLOGY: ASSOCIATIONS BETWEEN PARENTAL AND ADOLESCENT INTERNALIZING AND EXTERNALIZING SYMPTOMS ACROSS ADOLESCENCE

Adolescence is a crucial time for the development of psychopathological symptoms (Kim-Cohen et al., 2003). Estimated prevalence rates for adolescent internalizing symptoms, such as depression or anxiety, and externalizing symptoms, such as conduct or delinquent problems, are 5-25% (Kessler et al., 2012), which impose ten times the health costs of problem behaviors that develop in adulthood (WHO, 2007). Although adolescent internalizing and externalizing symptoms sometimes co-occur, they represent two distinct liabilities within a hierarchical structure of psychopathology (Achenbach, 1966; Lahey, Krueger, Rathouz, Waldman, & Zald, 2017), and predict different health trajectories in adulthood (Korhonen, Luoma, Salmelin, Siirtola, & Puura, 2018). It is therefore important to identify how risk factors predict both internalizing and externalizing symptoms throughout adolescence. Parental psychopathology is among the most prominent risk factors for internalizing and externalizing symptoms. Whereas theoretical models on how parental psychopathological problems are passed on to children concern effects from parents to their own children within individual families, empirical support has mainly been derived from between-family or group-level findings, which only inform us on how families differ from each other and do not allow conclusions about within-family effects (e.g., Hamaker, Kuiper, & Grasman, 2015). Therefore, this study examined the longitudinal associations between parental and adolescent internalizing and externalizing symptoms from early to late adolescence from a within-family perspective, to provide innovative and comprehensive insights into the transmission of psychopathology.

Parental Psychopathology as Risk Factor for Adolescent Psychopathology

The transmission of psychopathological symptoms between parents and adolescents is a transactional process that unfolds *within* one individual over time through several social interactions (Berry & Willoughby, 2017). However, to date, associations between parental and adolescent psychopathology have been typically examined with designs that focus on how families differ from each other in terms of psychopathological symptoms (i.e., between-family differences) that cannot distinguish these differences from changes in psychopathological symptoms within families (i.e., within-family processes). As between-family associations and within-family associations reflect very different processes, these designs do not match the theories we aim to test (Berry & Willoughby, 2017; Branje, Keijsers, Van Doorn, & Meeus, 2012; Hamaker et al., 2015; Keijsers, 2016). At the between-family level, we investigate whether parents who experience more internalizing and externalizing symptoms than other parents have adolescents who experience more psychopathological

symptoms than other adolescents. At the within-family level, we examine fluctuations from symptom levels that are considered typical for a parent or adolescent (i.e., average symptom levels), and how these fluctuations are associated with fluctuations in the symptoms of other members in their own family. In other words, within-family associations concern changes across time and the processes through which parents and adolescents influence each other. As such, they reflect transactional associations between parental and adolescent symptoms within one family (Papp, 2004). What occurs at the between-family level may therefore not always reflect what occurs at the within-family level. For example, that the level of aggressive behavior is higher for all family members in some families than in others (between-family association) does not necessarily imply that when one family member in a specific family becomes less aggressive, the other family member will also become less aggressive (within-family association).

It is well established that psychopathological symptoms are associated between generations; thus parental psychopathological symptoms appear to put adolescents at increased risk of developing internalizing and externalizing symptoms (see Connell & Goodman, 2002; Goodman et al., 2011 for reviews). Apart from transmitting risk genetically (e.g., Kendler, Ohlsson, Sundquist, & Sundquist, 2018; Rutter, Moffitt, & Caspi, 2006; Sullivan, Neale, & Kendler, 2000), parental internalizing and externalizing symptoms have been proposed to elevate stress levels, strain the parent-adolescent relationship, and prompt more hostile, inconsistent, or rejecting parenting, which might in turn elicit adolescent psychopathological symptoms (Coyne, 1976; Hammen, Shih, & Brennan, 2004; Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Granic & Patterson, 2006; Patterson, 1982). Through these pathways, parental internalizing and externalizing symptoms might not just elicit similar problem behaviors, but also a variety of other problem behaviors (i.e., multifinality; Cicchetti & Rogosch, 1996).

Most previous studies on the transmission of psychopathology provide evidence for multifinality, suggesting that parental and adolescent psychopathological symptoms, such as internalizing symptoms, are associated generically (i.e., not only with internalizing, but also with externalizing symptoms), rather than domain-specifically. Cross-sectional meta-analytic evidence even indicates that parental internalizing and externalizing symptoms are equally strongly associated with both adolescent internalizing and externalizing problems (Connell & Goodman, 2002). Longitudinal studies that tested whether parental symptoms precede adolescent symptoms are less conclusive. Whereas parental internalizing symptoms have been shown to predict later adolescent internalizing and externalizing symptoms (e.g., Goodman et al., 2011), parental externalizing symptoms seem to predict later adolescent externalizing symptoms (Salvatore et al., 2015; Smith & Farrington, 2004) but not internalizing symptoms (Kim, Capaldi, Pears, Kerr, & Owen, 2009; Kim, Capaldi, & Stoolmiller, 2003). The current study will extend previous findings by disentangling between-family processes from

within-family processes. We will examine how changes in parental psychopathological symptoms predict changes in adolescent symptoms within families over time.

Adolescent Psychopathology as Risk Factor for Parental Psychopathology

Transactional theories indicate that adolescents are not only shaped by, but also actively shape their environment (Bell, 1968; Patterson, 1982; Sameroff, 2009). Specifically, adolescent internalizing or externalizing symptoms may increase parental stress or elicit nonoptimal parental responses, which may in turn elicit or exacerbate parental psychopathological symptoms (Belsky, 1984; Hammen et al., 2004; Raposa, Hammen, & Brennan, 2011; Serbin, Kingdon, Ruttle, & Stack, 2015). It is therefore likely that adolescent symptoms are not only predicted by parental symptoms, but also predict subsequent parental symptoms. Specifically, fluctuations in adolescent internalizing and externalizing symptoms compared to their own average might predict similar fluctuations in parental symptoms compared to their own average. This may result in a vicious cycle in which parents and adolescents reinforce each other's psychopathological symptoms within one family.

Similar to the line of research examining intergenerational transmission from parents to adolescents, the few longitudinal studies that examined potential bidirectional associations between parental and adolescent internalizing and externalizing symptoms mainly relied on designs that assessed associations at the group level, without distinguishing what happens within a family (i.e., within-family processes) from what differentiates families from each other (i.e., between-family associations). These studies suggest that longitudinal associations of parental psychopathology with adolescent psychopathological problems are indeed bidirectional (Ge, Conger, Lorenz, Shanahan, & Elder, 1995). Specifically, bidirectional associations have been found between parental internalizing symptoms and adolescent internalizing (Hughes & Gullone, 2010; Wilkinson, Harris, Kelvin, Dubicka, & Goodyer, 2013) as well as adolescent externalizing symptoms (Gross, Shaw, & Moilanen, 2008). Another study examining bidirectional effects in childhood contrasts with these previous bidirectional findings and only found unidirectional associations, that is, from child externalizing to subsequent parental internalizing symptoms (McAdams et al., 2015). Concerning parental externalizing symptoms, the evidence is only indirect, that is, based on parenting measures, such as harsh punishment. While one study found bidirectional associations between harsh punishment and adolescent externalizing, but not internalizing symptoms (Wang & Kenny, 2014), other studies found that harsh punishment predicted adolescent internalizing or externalizing symptoms, but no reverse associations were found (Hipwell et al., 2008; Lansford et al., 2011). This study will build on existing preliminary bidirectional evidence and thus also include reversed associations on how changes in adolescent internalizing and externalizing symptoms predict changes in parental internalizing and externalizing symptoms within families over time.

Disentangling Between-Family Associations from Within-Family Associations

As theoretical assumptions concerning intergenerational transmission of psychopathological symptoms typically refer to processes that occur within families, intergenerational transmission needs to be tested with methods that distinguish between-family from within-family processes. Bidirectional associations between parental and adolescent psychopathology have been typically examined with cross-lagged panel models (CLPMs) in which between- and within-family estimates are interwoven. However, between-family associations can differ from, and even contrast with within-family associations (Hamaker et al., 2015; Keijsers, 2016). For example, a recent study found that higher levels of adolescent social anxiety symptoms were associated with higher levels of maternal psychological control and lower levels of parental autonomy support *between* families, while higher levels of adolescent social anxiety symptoms predicted lower levels of psychological control and higher levels of autonomy support *within* families over time (Nelemans et al., 2019). It is thus critical to match the statistical analysis to the (theoretical) research question at hand.

Random-intercept cross-lagged panel models (RI-CLPMs; Hamaker et al., 2015) allow researchers to disentangle how fluctuations in one's symptoms above or below that person's average are associated with fluctuations in other's symptoms above or below their own average (i.e., within-family) from how families differ in their average symptom levels (i.e., between-family). As an extension of CLPMs, RI-CLPMs include two latent factors (i.e., random intercepts) that capture characteristics that make individuals different from each other (in this case average symptom levels across time): one for parents and one for adolescents. In that way, differences among parental and adolescent symptom scores at some point in time are separated into average differences among persons and fluctuations around a person's average symptoms. Likewise, associations between parental and adolescent average psychopathological symptoms captured with the latent factors reflect between-family associations, while the other associations in the model reflect within-family processes (i.e., how fluctuations in parental symptoms are associated with fluctuations in adolescent symptoms).

To understand whether parents contribute to adolescent psychopathology, it is crucial to identify whether and how changes in parental symptoms are associated with changes in their own children's symptoms. In this study, we therefore disentangled within-family associations from between-family associations and examined how within-family fluctuations in parental symptoms predicted within-family fluctuations in adolescent symptoms across adolescence, and vice versa.

The Present Study

To our knowledge, this is the first study to investigate within-family processes in the transmission of internalizing and externalizing symptoms across adolescence, using a six-year multi-informant longitudinal design. We aimed to extend previous findings on between-family associations by examining within-family associations between parental and adolescent internalizing and externalizing symptoms, using RI-CLPMs (Hamaker et al., 2015) that disentangle within-family from between-family associations. We expected bidirectional associations between fluctuations in parental and fluctuations in adolescent internalizing and externalizing symptoms within families across adolescence. To date, no previous studies have investigated the transmission of psychopathological symptoms both between and within families. Therefore, we did not propose specific hypotheses about how between-family findings might differ from within-family findings. However, based on previous research that disentangled between-family from within-family associations (e.g., Keijsers, 2016; Nelemans et al., 2019), we expected that parental symptoms would be stronger associated with adolescent symptoms between families than within families. By examining whether fluctuations in adolescents' symptoms predict fluctuations in their parents' symptoms over time and vice versa, RI-CLPMs allow researchers to identify processes that occur within the parent-child dyad while accounting for inter-individual differences.

Previous studies further suggest that the associations between parental and adolescent psychopathological symptoms might differ for parent and adolescent gender (e.g., Connell & Goodman, 2002; Ge et al., 1995; Kim et al., 2009). For example, females appear to be at increased risk of developing internalizing symptoms while males are at increased risk of developing externalizing symptoms (Kessler et al., 2012; Kramer, Krueger, & Hicks, 2008; Zahn-Waxler, Shirtcliff, & Marceau, 2008). Moreover, maternal psychopathological symptoms seem to contribute more strongly to adolescent internalizing symptoms than paternal psychopathological symptoms (Connell & Goodman, 2002), although longitudinal evidence is inconclusive (e.g., Ge et al., 1995; Hughes & Gallone, 2010; Reeb et al., 2015; Wilkinson et al., 2013). Therefore, we conducted all analyses separately for mothers and fathers to examine the role of parent gender in the associations between parental and adolescent internalizing and externalizing symptoms. Additionally, we conducted sensitivity analyses to examine the role of adolescent gender.

METHODS

Participants

The sample consisted of 497 adolescents (43.1% girls, M_{age} T_1 = 13.03, SD_{age} = .46) and their parents (495 mothers, M_{age} T_1 = 44.41, SD_{age} = 4.45, and 446 fathers, M_{age} T_1 = 46.74, SD_{age} = 5.10) who participated in the Research on Adolescent Development And Relationships Young (RADAR-Y) study. All participants attended the 1st grade of secondary school at the onset of the study and were annually assessed for six years. All adolescents, and most of their mothers (92.8%) and fathers (82.5%), were of Dutch origin and lived in mainly intact two-parent families (84.7%) with medium to high socioeconomic status (87.7%), based on parents' occupation level.

Average sample attrition per year across all measurement occasions was low (3.2-3.4%), with 426 adolescents, 420 mothers, and 375 fathers still remaining in the study at the sixth measurement occasion. Adolescents and their mothers and fathers who remained in the study until the sixth year did not significantly differ from those who dropped out of the study on any of the study outcomes ($ps \geq .060$), except that fathers who remained in the study reported significantly lower baseline levels of internalizing symptoms compared to fathers who dropped out of the study, $F(1, 434) = 5.11$, $p = .024$.

Procedure

The present study used data from the first to the sixth measurement occasion, which followed participants from age 13 to 18. Participants were recruited through 230 randomly selected elementary schools in the central and western regions of the Netherlands. Of all selected participants (N = 1,544), families were excluded if they did not fulfil the full family requirements (n = 364), could not be contacted or withdrew their participation (n = 569), or failed to provide written consent of all family members (n = 114). This resulted in 497 families who participated at the first measurement occasion. Adolescents and their parents provided active written informed consent before the onset of the study and at all measurement occasions. Trained interviewers conducted the annual assessments at participants' homes, during which adolescents and their parents completed the questionnaires. All participants received monetary reimbursements for their participation. The study procedure was approved by the Medical Research Ethics Committee of the University Medical Center Utrecht (RADAR: Research on Adolescent Development and Relationships, 05/159-K).

Measures

Adolescent internalizing symptoms. We assessed internalizing symptoms as a combined score of adolescent self-reported anxiety symptoms, using the 38-item Screen for Child Anxiety Related

Emotional Disorders (SCARED; Birmaher et al., 1997), and depression symptoms, using the 23-item 2nd edition of the Reynolds Adolescent Depression Scale (RADS-2; Reynolds, 2000). Adolescents rated how anxious they felt on a 3-point scale and how depressed they felt on a 4-point scale. We created an internalizing symptom score for each participant by standardizing and then averaging their total anxiety and total depression scores. Scores were only averaged when both anxiety and depression scores were non-missing (99.8-100%, except 54.1% at T_1) to ensure that the internalizing construct was comparable across participants. The anxiety, depression, and final internalizing scales showed high internal consistency across all waves (α = .96.-91). Higher scores reflected higher mean levels of internalizing symptoms.

Adolescent externalizing symptoms. We assessed adolescent externalizing symptoms using the 11-item delinquent and the 19-item aggressive behavior scales of the Youth Self Report (YSR; Achenbach, 1991). Adolescents rated their externalizing behaviors on a 3-point scale. The scale showed high internal consistency across all waves (α = .91.-87). Higher scores reflected higher mean levels of externalizing symptoms.

Parental internalizing symptoms. We assessed parental internalizing symptoms using the 18-item anxious/depressed, the 9-item withdrawn, and the 12-item somatic complaints syndrome scales of the Adult Self Report (ASR; Achenbach & Rescorla, 2003). Mothers and fathers rated their internalizing behaviors in the past six months on a 3-point scale. Maternal and paternal symptoms showed high internal consistency across all waves (α = .93.-87). Higher scores reflected higher mean levels of internalizing symptoms.

Parental externalizing symptoms. We assessed parental externalizing symptoms using the 14-item rule-breaking behavior and the 15-item aggressive behavior subscales of the Adult Self Report (Achenbach & Rescorla, 2003). Mothers and fathers rated their externalizing behaviors in the past six months on a 3-point scale. Maternal and paternal symptoms showed adequate internal consistency across all waves (α = .85.-74). Higher scores reflected higher mean levels of externalizing symptoms.

Statistical Analyses

Missing data ranged from 0.4-25.2% for most variables, with overall 14.1% of all values missing. Six adolescents were excluded from the analyses involving internalizing symptoms, because their data was missing on all measurement occasions. Little's missing completely at random (MCAR) test detected no systematic patterns of missingness, normed $\chi^2/df = 1.11$, indicating that missing data was not likely to bias the analyses.

We calculated intra-class correlations (ICCs) to ensure that there was sufficient variance at both the between-family and the within-family level to partition the variance into between-family average symptom levels and within-family fluctuations (Keijsers, 2016). As in multi-level models, the ICC describes the proportion of the variance that is explained by the group level (here: between-family level) and the individual level (here: within-family level). We then constructed eight RI-CLPMs in M*plus* 8.1 (Muthén & Muthén, 1998–2015) to model the associations between parental and adolescent internalizing and externalizing symptoms across time for mothers and fathers, respectively. All RI-CLPMs included one-year autoregressive paths for parental and adolescent symptoms, within-time associations between parental and adolescent symptoms at the same time point, and one-year longitudinal cross-lagged paths from parental to adolescent symptoms and vice versa across all six years.

In contrast to CLPMs, in which all paths are specified between the observed scores, in RI-CLPMs, these paths are specified between the latent constructs. To partition between-family from within-family associations in parental and adolescent symptoms, RI-CLPMs include two random intercepts that capture all between-family variance (see between-family latent factors in Figures 1-4). Correlations between the two random intercepts reflect differences between families and describe how average differences in psychopathological symptoms between parents are associated with average differences in psychopathological symptoms between adolescents (see between-family associations in Figures 1-4). The repeated measurements of parental and adolescent psychopathological symptoms are centered per person; all within-family associations are based on these within-person centered variables. The auto-regressive and cross-lagged paths thus describe whether higher or lower levels than one family member's average psychopathological symptoms at one point in time predict higher or lower levels than that family member's average symptoms (within-person auto-regressive paths, see Figures 1-4) or higher or lower levels than another family member's average symptoms (within-family cross-lagged paths; see Figures 1-4) at a later point in time. Within-family correlations between parental and adolescent symptoms at the same time reflect correlated change and indicate whether higher or lower levels of one parent's own symptoms are associated with higher or lower levels of their child's own symptoms at a specific moment in time (see within-time associations in Figures 1-4).

We estimated all models using maximum likelihood estimation with robust standard errors and chi-square robust to nonnormality (MLR estimator; Muthén & Muthén, 1998-2015). We handled missing values for all variables with Full Information Maximum Likelihood estimation, and assessed model fit with the comparative fit index (CFI), the root mean squared error of approximation (RMSEA) and its 90% confidence interval, and the standardized root mean square residual (SRMR). CFI values ≥ 0.95,

RMSEA values ≤ 0.06, and SRMR values ≤ 0.08 indicate acceptable fit (Hu & Bentler, 1999). To estimate adequate power, we used the sample size-to-parameters ratio rule that defines a minimum *N:q* ratio of 10:1 and an ideal ratio of 20:1 (Jackson, 2003; Kline, 2011). With a *N:q* ratio of 15:1, our sample size was appropriate to test the proposed models. As parsimony was preferred, we constrained all longitudinal parameters to be time invariant in all baseline models (Kline, 2011). This means, for example, that the autoregressive and cross-lagged paths from Wave 1 to Wave 2 were equal to the autoregressive and cross-lagged paths, respectively, from Wave 2 to Wave 3, from Wave 3 to Wave 4, from Wave 4 to Wave 5 and from Wave 5 to Wave 6. We examined potential changes in associations over time (e.g., whether paths from Wave 1 to Wave 2 differ from paths from Wave 2 to Wave 3) by stepwise testing whether freeing within-time associations and cross-lagged parameters significantly improved the model fit, using Satorra–Bentler scaled chi-square difference tests ($\Delta\chi^2_{SB}$; Satorra & Bentler, 2001). If the freely estimated model did not fit significantly better than the fully constrained model, we chose the most parsimonious model (i.e., the constrained time invariant model). Parameter estimates were standardized on their own respective variance (STDYX; Muthén & Muthén, 1998-2015). Statistical significance was evaluated at $\alpha = .05$.

RESULTS

Descriptive Statistics

Table 1 displays all means and standard deviations among all study variables. Parental and adolescent internalizing and externalizing symptoms correlated modestly for mothers, $r = .06$-$.30$, but weakly for fathers, $r = -.01$-$.15$, both concurrently and across time (see Table S1 in the supplementary material).

RI-CLPMs on Parental and Adolescent Psychopathological Symptoms

Intraclass correlation coefficients (ICCs) for parental and adolescent symptoms indicated that there was sufficient variance at both the between-family level and the within-family level. Specifically, for adolescent internalizing and externalizing symptoms, the ICCs were .681 and .575, respectively, which indicates that 68.1% of the variance in adolescent internalizing symptoms and 57.5% in adolescent externalizing symptoms were explained by differences between adolescents, and 31.9% and 42.5%, respectively, were explained by fluctuations within adolescents. The ICCs for parental internalizing and externalizing symptoms were .734 and .674, respectively, for mothers, and .721 and .672, respectively, for fathers. Sufficient variance at both levels for parental and adolescent symptoms implies that we should indeed separate the within-family variance from the between-family variance.

Table 1. Range of Descriptives of All Study Variables Across Time

	M	*SD*
1 Adolescent anxiety symptoms (SCARED)[a]	1.26 - 1.34	0.25 - 0.28
2 Adolescent depressive symptoms (RADS)[b]	1.50 - 1.63	0.49 - 0.54
3 Adolescent internalizing symptoms (combined)[c]	0.00 - 0.04	0.86 - 0.94
4 Adolescent externalizing symptoms[d]	0.31 - 0.35	0.23 - 0.27
5 Maternal internalizing symptoms[d]	0.17 - 0.20	0.16 - 0.19
6 Maternal externalizing symptoms[d]	0.08 - 0.12	0.09 - 0.13
7 Paternal internalizing symptoms[d]	0.17 - 0.19	0.16 - 0.19
8 Paternal externalizing symptoms[d]	0.09 - 0.13	0.12 - 0.13

Note. [a] Possible scores range from 1 to 3, [b] possible scores range from 1 to 4, [c] average based on standardized scores, [d] possible scores range from 0 to 2.

The fully constrained baseline RI-CLPMs showed good fit for all associations of maternal and paternal internalizing and externalizing symptoms with adolescent internalizing and externalizing symptoms (CFIs ≥ .958; RMSEAs ≤ .059, SRMRs ≤ .057; see Table S2 in the supplementary material). Freeing the within-family correlated change did not significantly improve the fit for any model (*p*s ≥ .118, see Table S2), which indicates that how fluctuations in maternal and paternal symptoms were associated with fluctuations in adolescent symptoms at the same time did not change across adolescence. Freeing the cross-lagged paths from adolescents to mothers improved the fit for the associations between maternal and adolescent externalizing symptoms ($\Delta\chi^2_{SB}(4) = 12.62$, $p = .013$). The significantly better fit of the model including freely estimated paths from adolescent to maternal symptoms indicates that how fluctuations in adolescent externalizing symptoms predict fluctuations in maternal symptoms one year later was not stable, but differed across adolescence. In this model, the cross-lagged paths from adolescent to maternal externalizing symptoms were therefore freely estimated. For all other models, cross-lagged associations between maternal or paternal symptoms and adolescent symptoms did not vary across time (*p*s ≥ .096).

Table 2. Overview of All Parameter Estimates of the Random-Intercept Cross-Lagged Panel Models

		Maternal models			Paternal models		
Model		B	*p*	β/*r*	B	*p*	β/*r*
Parental internalizing symptoms							
A	Between-family correlation	.03	<.001	.26	.01	.143	.09
	Parent int → Adolescent int	.27	.109	.04-.05	.13	.462	.02-.03
	Adolescent int → Parent int	.02	.001	.10-.12	.01	.201	.04-.06
	T1 correlation	.01	.419	.06	.01	.277	.10
	Correlated change T2-6	.01	.001	.10-.13	.00	.035	.05-.08
B	Between-family correlation	.01	<.001	.22	.00	.066	.12
	Parent int → Adolescent ext	.08	.185	.04	−.02	.663	−.01
	Adolescent ext → Parent int	.03	.139	.05-.06	−.01	.724	−.01-−.02
	T1 correlation	.00	.054	.12	−.00	.583	−.04
	Correlated change T2-6	.00	.135	.04-.06	.00	.360	−.02-−.03
Parental externalizing symptoms							
A	Between-family correlation	.02	<.001	.32	.01	.181	.08
	Parent ext → Adolescent int	.15	.557	.02	−.01	.980	−.00
	Adolescent int → Parent ext	.00	.741	.01	.00	.828	.01
	T1 correlation	.00	.856	.01	.01	.140	.12
	Correlated change T2-T6	.00	.297	.02-.03	.00	.170	.03-.05

Table 2. Continued.

Model		Maternal models			Paternal models		
		B	*p*	β/*r*	B	*p*	β/*r*
B	Between-family correlation	.00	<.001	.30	.00	.032	.13
	Parent ext → Adolescent ext	.05	.603	.01-.02	.09	.230	.03-.04
	Adolescent ext → Parent ext	−.06-.03	.016-.361	−.19-.09	.02	.170	.04-.07
	T1 correlation	.00	.014	.16	.00	.794	−.02
	Correlated change T2-T6	.00	.713	−.01	.00	.161	.03-.06
Autoregressive paths in all models							
	Parent int → parent int	.20-.21	.001	.19-.26	.13	.052-.056	.10-.17
	Parent ext → parent ext	.15-.16	<.001	.14-.21	.12	.016	.11-.16
	Adolescent int → adolescent int	.35-.36	<.001	.34-.39	.36	<.001	.35-.39
	Adolescent ext → adolescent ext	.45-.46	<.001	.37-.53	.45	<.001	.36-.53

Note. All models are listed separately involving maternal and paternal internalizing (int) and externalizing (ext) symptoms with adolescent internalizing (A) and externalizing symptoms (B); between-family correlations indicate associations between parental and adolescent symptoms between families (i.e., between the random intercepts), arrows indicate within-family longitudinal paths, T1 correlations indicate unconstrained within-time associations between fluctuations in parental and adolescent symptoms at T1, correlated change indicate constrained within-time associations between fluctuations in parental and adolescent symptoms from T2-T6.

Figure 1. Random-intercept cross-lagged panel models on the associations between maternal internalizing and adolescent internalizing (1A) and externalizing symptoms (1B), m = mother, a = adolescent. *** $p < .001$, ** $p < .01$.

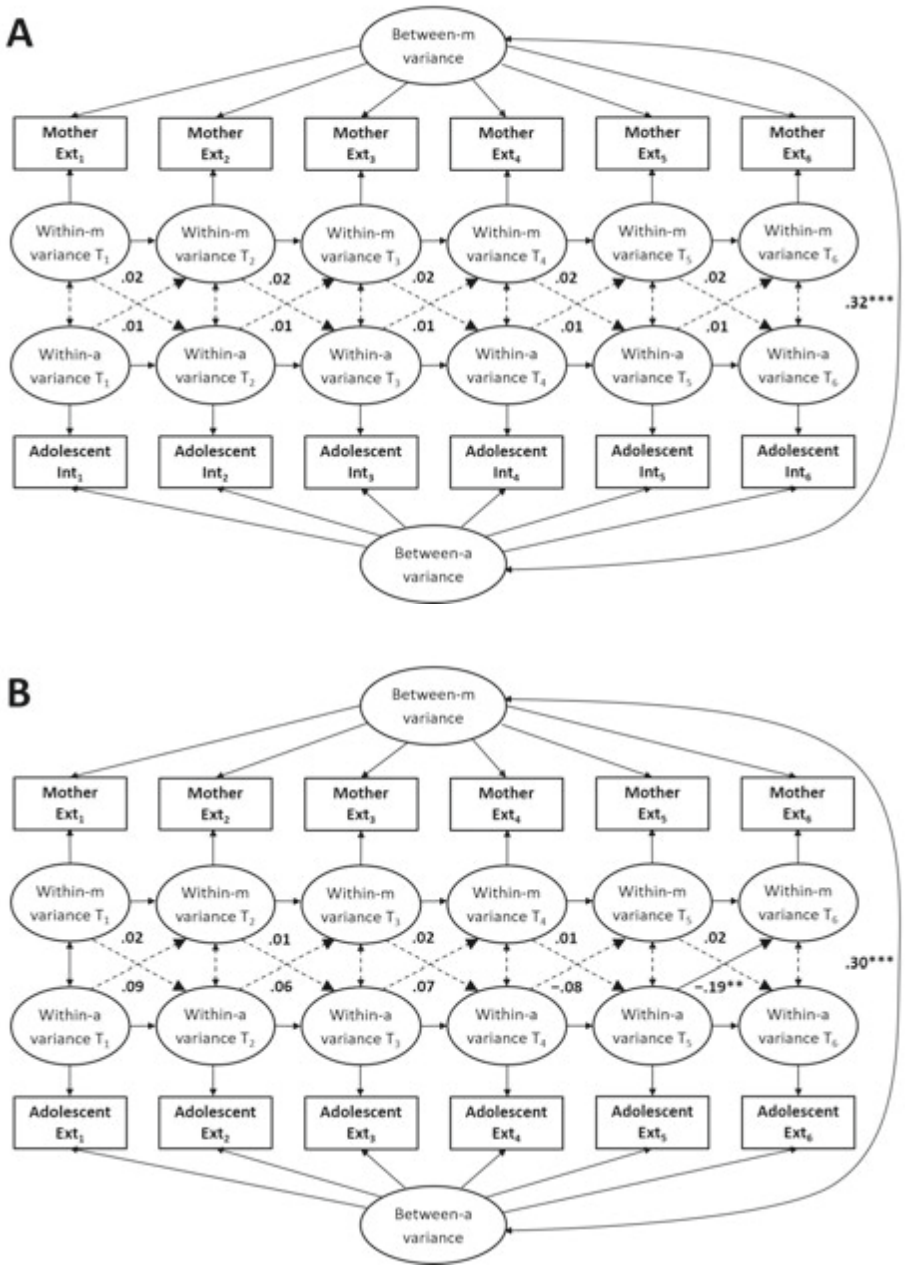

Figure 2. Random-intercept cross-lagged panel models on the associations between maternal externalizing and adolescent internalizing (2A) and externalizing symptoms (2B), m = mother, a = adolescent. *** $p < .001$, ** $p < .01$.

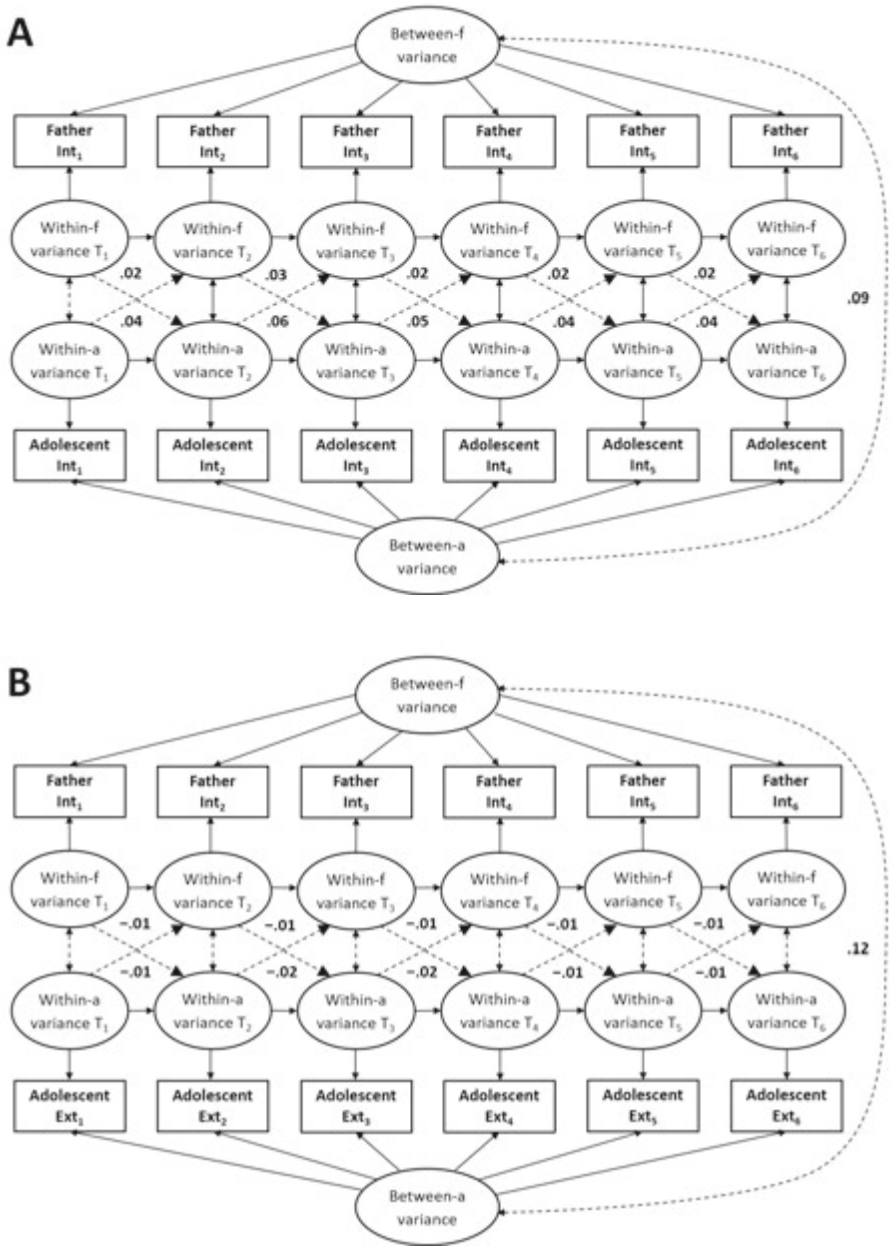

Figure 3. Random-intercept cross-lagged panel models on the associations between paternal internalizing and adolescent internalizing (3A) and externalizing symptoms (3B), f = father, a = adolescent.

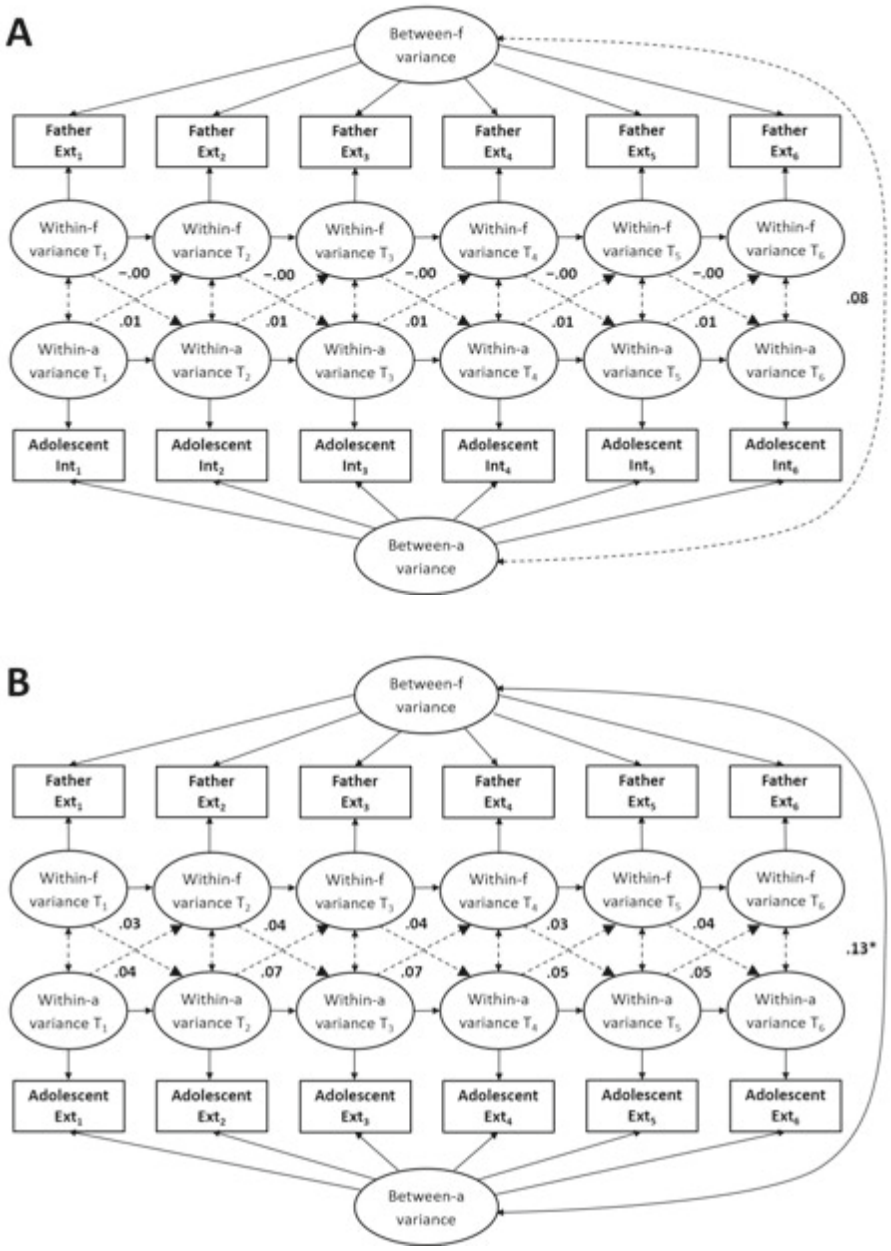

Figure 4. Random-intercept cross-lagged panel models on the associations between paternal externalizing and adolescent internalizing (4A) and externalizing symptoms (4B), f = father, a = adolescent. * $p < .05$.

Within-family associations with maternal and paternal psychopathological symptoms. The within-family parameter estimates of all RI-CLPMs are presented in Table 2. Controlling for differences in symptom levels between families, when mothers reported higher levels of internalizing symptoms than their own average, their children did not report higher levels of internalizing (β = .04-.05, *p*s ≥ .095; see cross-lagged paths in Figure 1A) or externalizing symptoms (β = .04, *p*s ≥ .182; see Figure 1B) than their own average one year later. Mothers did report significantly higher levels of internalizing symptoms than their own average one year after adolescents reported higher levels of internalizing symptoms than their own average (β = .10-.12, *p*s = .001; see Figure 1A), but not after adolescents reported higher levels of externalizing symptoms than their own average (β = .05-.06, *p*s = .126; see Figure 1B). No significant longitudinal associations were found from fluctuations in maternal externalizing symptoms to subsequent fluctuations in adolescents' internalizing (*p*s ≥ .553; see Figure 2A) or externalizing symptoms (*p*s ≥ .601; see Figure 2B). The longitudinal associations from adolescent to maternal externalizing symptoms differed across adolescence (see Figure 2B): While in early to mid-adolescence, mothers did not report higher levels of externalizing symptoms than their own average one year after adolescents reported higher levels of externalizing symptoms than their own average ($\beta_{T1\text{-}T5}$ = –.08-.09, *p*s ≥ .194), in late adolescence, mothers reported significantly higher levels of externalizing symptoms than their own average one year after adolescents reported higher levels of externalizing symptoms than their own average ($\beta_{T5\text{-}T6}$ = –.19, *p* = .008). This indicates that only adolescent and maternal internalizing symptoms were consistently associated across adolescence in that increases in adolescent internalizing symptoms predicted subsequent increases in maternal symptoms.

Within-time associations were mostly found for increases in maternal and adolescent internalizing symptoms compared to their own average ($\beta_{T2\text{-}T6}$ = .10-.13, *p*s ≤ .002). When mothers reported higher levels of internalizing symptoms than their own average, their children also reported higher levels of internalizing symptoms at that time point. Other within-time associations between maternal and adolescent symptoms were generally not significant across adolescence. Furthermore, for both mothers and adolescents, higher levels of internalizing and externalizing symptoms than their own average predicted higher levels of their own symptoms than their average in the following year.

For fathers, no significant longitudinal associations were found between higher levels of paternal internalizing (*p*s ≥ .207; see Figure 3) or externalizing symptoms than their own average (*p*s ≥ .153; see Figure 4) and higher levels of adolescent internalizing or externalizing symptoms than their own average. However, similarly to mothers, fluctuations in paternal internalizing symptoms were generally associated with fluctuations in adolescent internalizing symptoms at the same time point ($\beta_{T2\text{-}T6}$ = .05-.08, *p*s ≤ .039; see Figure 3A). When fathers reported higher levels of internalizing

symptoms than their own average, their children also reported higher levels of internalizing symptoms at that time point. Other within-time associations between paternal and adolescent symptoms were generally not significant across adolescence. Furthermore, higher levels of paternal externalizing, but not internalizing symptoms than their own average were predicted by higher levels of their own symptoms than their average in the previous year.

Sensitivity Analysis

Outlier analyses. Multivariate outlier analyses based on Mahalanobis distance (> 51.18) detected 16 influential cases for maternal models and 15 influential cases for paternal models. Excluding these cases from the analyses did not affect the general pattern of findings (see Figures S1-S4 in the supplementary material).

Associations for adolescent boys and girls. We conducted multigroup analyses to investigate gender differences between adolescent boys and girls (see supplementary material for more detailed results). Results indicated bidirectional associations between higher levels of maternal internalizing symptoms and higher levels of adolescent girls' internalizing symptoms within families, but only unidirectional associations from higher levels of adolescent boys' internalizing symptoms to higher levels of maternal internalizing symptoms. While we detected some other differences between adolescent boys and girls, all results pointed in the same direction as the main analyses, indicating that other associations with maternal symptoms only differed to a small extent between adolescent boys and girls. For models involving paternal symptoms, no differences between adolescent girls and boys were found.

DISCUSSION

The present study examined transactional processes in the transmission of parental and adolescent internalizing and externalizing symptoms across six years from early to late adolescence. As opposed to previous studies examining the longitudinal associations between parental and adolescent internalizing and externalizing symptoms, we used RI-CLPMs that disentangle processes that occur within families from differences between families to investigate how parental and adolescent symptoms bidirectionally predict each other. We detected consistent between-family associations of parental with adolescent psychopathological symptoms, as well as consistent lagged effects for internalizing, but not externalizing symptoms. Specifically, maternal, but not paternal internalizing and externalizing symptoms were generally associated with adolescent internalizing and externalizing symptoms across families, while only changes in adolescent internalizing symptoms predicted subsequent changes in maternal internalizing symptoms. Our findings suggest that

processes within families differ from associations between families, particularly for parental, that is maternal and paternal, and adolescent externalizing symptoms.

Between-Family Associations between Parental and Adolescent Psychopathology

Consistent with our hypotheses, maternal internalizing and externalizing symptoms were modestly associated with adolescent internalizing and externalizing symptoms between families. Specifically, adolescents who reported higher internalizing and externalizing symptoms often had mothers who reported higher internalizing and externalizing symptoms as well. These findings were consistent across adolescence and, thus, confirm and extend previous evidence for intergenerational associations of psychopathological symptoms (e.g., Connell & Goodman, 2002; Goodman et al., 2011; Smith & Farrington, 2004). Hence, in families with more, compared to less maternal psychopathological symptoms, adolescents might display more problem behaviors as well. Furthermore, they suggest that particularly generic associations between maternal and adolescent symptoms may be due to individual differences in average levels of psychopathological symptoms across time, rather than processes that occur within a family.

While maternal psychopathological symptoms were consistently associated with adolescents' psychopathological symptoms between families, only paternal externalizing symptoms were associated with adolescent externalizing symptoms. As mothers are more involved in daily activities with their children (see Pleck, 1997), their psychopathological symptoms may be more likely than paternal symptoms to converge with adolescent psychopathological symptoms. Furthermore, the associations with maternal and paternal symptoms were generally equally strong for adolescent boys and girls, except that domain-specific associations between maternal and adolescent psychopathological symptoms were stronger for girls than boys.

Longitudinal Associations between Parental and Adolescent Psychopathology Within Families

Partially in line with our hypotheses, we consistently found that increases in adolescent internalizing symptoms predicted subsequent increases in maternal internalizing symptoms within families over time. This suggests that associations between maternal and adolescent internalizing symptoms within families seem to be particularly driven by adolescent-to-mother effects, which were twice as large as the mother-to-adolescent effects. These reverse associations are in line with theoretical (Bell, 1968; Sameroff, 2009) and empirical evidence (Hughes & Gullone, 2010) regarding the role of child-to-parent effects. It is likely that increases in an adolescent's internalizing symptoms stress or worry the mother, undermine optimal parenting practices, and strain the mother-adolescent relationship, which in turn elicit maternal internalizing symptoms in these families (Belsky, 1984;

Coyne et al., 1976; Hammen et al., 2004; Raposa et al., 2011; Serbin et al., 2015). Indeed, an array of studies confounding between-and within family associations indicates that particularly adolescent psychopathology is related to changes in the quality of relationships with mothers even more so than vice versa (for a review, see Meeus, 2016). As adolescents start to spend more time outside the family, it is possible that maternal influence decreases as well, while adolescent emotional problems still exert influence on their mothers. Such negative, stressful experiences throughout adolescence are likely to negatively influence the family climate. Within-family difficulties might further accumulate in these families and consequently decrease adolescents' developmental chances (for a discussion, see Meeus, 2016).

Similar to our findings between families, the longitudinal associations found within families were only significant for maternal, not paternal psychopathological symptoms. This is in line with meta-analytic and longitudinal evidence indicating that maternal psychopathological symptoms are more strongly associated with adolescent internalizing symptoms than paternal psychopathological symptoms (Connell & Goodman, 2002; Hughes & Gallone, 2010). As mothers are often the primary caregiver (for a review, see Pleck, 1997), they are more involved in daily situations with their children and have more intimate relationships with their children than fathers (Steinberg & Silk, 2002). Mothers are thus more likely to be affected by strains in the parent-adolescent relationships elicited by adolescent internalizing symptoms (Coyne et al., 1976) than fathers. Furthermore, women tend to be more emotionally expressive with their children (Fivush, Brotman, Buckner, & Goodman, 2000; Van der Giessen & Bögels, 2017), report to be more empathic towards others (Eisenberg & Lennon, 1983), and have more conflictual interactions with their children than men (Branje, Laursen, & Collins, 2012). It is thus possible that mothers are more emotionally distressed by their children's internalizing symptoms than fathers, which consequently affects their own well-being. Relatedly, we detected bidirectional associations between maternal and adolescent girls', but not boys' internalizing symptoms over time. As females are more likely than males to express internalizing emotions (Kramer et al., 2008) and extensively discuss or dwell on the same problems, including its negative feelings (i.e., co-rumination; Rose, 2002), mother-girl dyads might reciprocate, and thus reinforce each other's emotional problems over time.

Contrary to our expectations, neither maternal nor paternal externalizing symptoms were longitudinally associated with adolescent psychopathological symptoms within families. This suggests that for parental and adolescent externalizing symptoms, processes within families differ from associations between families across generations. Indeed, what renders families different from each other does not always accurately reflect what occurs within one average family (Hamaker et al., 2015). It is possible that parent and child externalizing problems influence each other more in (earlier)

childhood than adolescence through modified and coercive parenting practices that reinforce externalizing behaviors (Patterson, 1982). As adolescents become more independent and spend more time outside the parental home, they may be more likely to display externalizing behaviors in peer groups or at school, which might affect parental behaviors less directly. Alternatively, the transmission of externalizing problems might be bound to situational circumstances and thus operate at shorter time intervals. Coercion theory (Granic & Patterson, 2006; Patterson, 1982), which emphasizes that adolescent externalizing symptoms determine parental symptoms and vice versa, is based on action-reaction principles, in which one event (immediately) follows the other. When an adolescent displays externalizing behaviors, for example, the parent might immediately react to this behavior through cycles of negative reinforcement, while internalizing symptoms might elicit symptomatic reactions only after prolonged, cumulated experiences. Annual assessments might thus be less suitable to capture situational changes in the transmission of externalizing behaviors within parent-adolescent dyads.

Strengths, Limitations, and Future Directions

This is the first study to examine within-family associations between parental and adolescent psychopathological symptoms from early to late adolescence over time. By disentangling between-family from within-family associations, our findings provide insight into the unique transactional processes that occur within families. This study thus allowed us to more directly test theoretical assumptions that focus on how changes in psychopathological symptoms are transmitted from one family member to the other, rather than how families differ from each other in their psychopathological symptoms. Assessing self-reported parental and adolescent symptoms several times throughout adolescence further showed that adolescent and maternal internalizing symptoms were consistently associated over time, above and beyond concurrent associations.

Despite these strengths, this study has some limitations. First, our community sample included mainly highly educated, ethnic majority, intact families with relatively low levels of internalizing and externalizing symptoms, which warrants caution in overgeneralizing the results. Second, annually measured mood and behavior render it difficult to unravel dynamic interactional processes that occur at the micro-level. Examining moment-to-moment or day-to-day interactions between parents and adolescents might better capture how parents and adolescents determine and reinforce each other's mood and behaviors. Further research is needed to detect accurate time intervals that combine more micro assessments with longer term assessments to comprehensively determine how psychopathological symptoms are associated within families. Third, although RI-CLPMs capture all inter-individual differences, such as individual differences in genetic make-up, in the between-person latent factors, this study does not examine genetic moderation, which

may also play a role in the intergenerational transmission of psychopathology (Kendler et al., 2018; Natsuaki et al., 2014; Rutter et al., 2006). Initial findings indeed suggest that gene-environment interactions involving parental psychopathological symptoms predict adolescent development (Leve et al., 2010; Thapar, Harold, Rice, Langley, & O'Donovan, 2007). Future longitudinal studies might examine how genetic processes moderate the transmission of psychopathology to help identify underlying environmental mechanisms that can be targeted in programs to preempt or reduce adolescent symptoms. Finally, while our findings established magnitude and consistency of the associations between parental and adolescent psychopathological symptoms, we cannot draw causal inferences from these results. Future experimental designs within prospective longitudinal studies might provide further insights into causal mechanisms through which parental symptoms elicit and exacerbate adolescent psychopathological symptoms. These mechanisms can inform future clinical efforts to break the negative cycle of psychopathological transmission and reduce both parental and adolescent psychopathological symptoms. Furthermore, future studies might investigate triadic models including adolescent, mother, and father as how strongly increases in parental psychopathological symptoms predict increases in adolescent symptoms may depend on the cumulative effect of maternal and paternal psychopathological symptoms. While examining such effects was beyond the scope of this study, it may be possible that maternal or paternal symptoms buffer or exacerbate the effects of one parent's symptoms on the adolescent's symptoms.

CONCLUSION

The findings of this study emphasize that within families, associations between parental and adolescent internalizing symptoms are mainly driven by adolescent-to-mother effects. While the results further suggest potential transactional processes between internalizing symptoms in mother-girl dyads, longitudinal associations between parental externalizing and subsequent adolescent psychopathological symptoms may be less evident within families across adolescence. Across families, on the other hand, maternal psychopathological symptoms consistently coexisted with adolescent psychopathological symptoms. These differences highlight the importance of distinguishing between-family associations from within-family processes. Within-family models that allow separating this between-family component from within-family associations might thus be particularly suitable to investigate how parental and adolescent psychopathological symptoms are associated over time. Such insights can contribute to breaking the vicious cycle of parent and adolescent psychopathology, particularly for internalizing symptoms, which might in turn increase well-being throughout and beyond adolescence and consequently lower the enormous health costs associated with adolescent psychopathology.

SUPPLEMENTAL MATERIAL

Results of Sensitivity Analyses

Multigroup models for differences between adolescent boys and girls

All models showed good fit for the associations of maternal and paternal symptoms with adolescent girls' as well as adolescent boys' internalizing and externalizing symptoms (CFIs ≥ .960; RMSEAs ≤ .060, SRMRs ≤ .057).

Between-family associations for adolescent boys and girls. Multigroup models indicated that the association between maternal externalizing symptoms and adolescent externalizing symptoms differed for boys and girls ($\Delta\chi^2_{SB}(1) = 4.19$, $p = .041$). Specifically, if mothers reported higher levels of externalizing symptoms than other mothers, adolescent girls reported higher levels of externalizing symptoms ($b = .01$, $\beta = .39$, $p < .001$) than adolescent boys ($b = .00$, $\beta = .25$, $p = .002$). All other associations between maternal and adolescent psychopathological symptoms were equally strong for boys and girls. No gender differences were detected for paternal models.

Within-family associations for adolescent boys and girls. The results indicated significant gender differences in the longitudinal associations from maternal internalizing symptoms to subsequent adolescent internalizing symptoms ($\Delta\chi^2_{SB}(1) = 6.30$, $p = .012$) and from maternal externalizing symptoms to subsequent adolescent internalizing ($\Delta\chi^2_{SB}(1) = 4.51$, $p = .034$) and externalizing symptoms ($\Delta\chi^2_{SB}(1) = 3.92$, $p = .048$). Specifically, fluctuations in maternal internalizing symptoms were followed by similar fluctuations in adolescent girls' ($b = .64$, βs = .09-.10, ps ≤ .036), but not boys' internalizing symptoms ($b = -.01$, $\beta = -.00$, ps = .942) one year later. Fluctuations in maternal externalizing symptoms were also followed by stronger fluctuations in girls' internalizing ($b = .70$, βs = .06-.09, ps ≥ .069) and externalizing symptoms ($b = .15$, βs = .05-.07, ps ≥ .138) compared to boys' internalizing symptoms ($b = -.23$, βs = –.02-–.04, ps ≥ .386) and externalizing symptoms ($b = -.09$, βs = –.02-–.03, ps ≥ .478), but these associations did not reach statistical significance. Furthermore, we detected gender differences in the within-family associations between maternal internalizing and adolescent externalizing symptoms ($\Delta\chi^2_{SB}(2) = 8.71$, $p = .013$). Specifically, at times when mothers reported higher or lower internalizing symptoms compared to their own average, their adolescent sons also reported higher (or lower) internalizing symptoms than expected ($b = .00$, βs = .07-.11, ps ≤ .022), but their adolescent daughters did not ($b = .00$, βs = –.02-–.03, ps ≥ .622). All other associations between maternal and adolescent psychopathological symptoms were equally strong for boys and girls. No gender differences were detected for paternal models.

Table S1. Correlations Between all Study Variables.

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
1 Adolescent int T_1		.651	.656	.578	.564	.512	.459	.303	.296	.218	.173	.295	.090	.029	.074	.143	.086	.039	.100	.056	.136	.133	.081	.089
2 Adolescent int T_2	.644		.729	.684	.607	.556	.255	.308	.236	.226	.198	.227	.034	.028	.068	.058	.055	.058	.07	.066	.107	.096	.087	.091
3 Adolescent int T_3	.645	.731		.762	.701	.657	.301	.296	.352	.279	.238	.261	.032	.049	.122	.106	.097	.078	−.005	.056	.093	.069	.060	.067
4 Adolescent int T_4	.579	.686	.764		.808	.722	.207	.194	.230	.251	.212	.264	.033	.042	.071	.063	.042	.061	.040	.066	.073	.054	.034	.053
5 Adolescent int T_5	.566	.611	.707	.809		.783	.185	.226	.248	.226	.291	.344	.064	.050	.084	.081	.077	.109	.022	.025	.048	.042	.048	.057
6 Adolescent int T_6	.513	.559	.664	.722	.783		.212	.291	.269	.238	.281	.448	.102	.054	.113	.073	.110	.146	.020	.054	.086	.064	.060	.094
7 Adolescent ext T_1	.444	.254	.300	.208	.187	.222		.609	.599	.513	.420	.406	.017	.001	.036	.047	.047	.027	.069	.009	.106	.107	.098	.060
8 Adolescent ext T_2	.279	.309	.299	.197	.229	.299	.608		.599	.538	.485	.402	-.003	−.006	.034	.025	.075	.035	.061	.001	.116	.104	.105	.074
9 Adolescent ext T_3	.288	.238	.354	.229	.248	.278	.597	.598		.777	.667	.574	.105	.017	.098	.076	.105	.077	.075	.080	.126	.125	.131	.098
10 Adolescent ext T_4	.226	.228	.283	.252	.228	.244	.513	.539	.777		.741	.634	.063	.031	.097	.065	.070	.076	.073	.073	.138	.138	.098	.110
11 Adolescent ext T_5	.180	.200	.241	.210	.290	.284	.422	.487	.668	.741		.713	.066	−.008	.071	.059	.023	.088	.044	.089	.090	.099	.084	.113
12 Adolescent ext T_6	.302	.232	.267	.266	.346	.454	.404	.401	.572	.633	.714		.121	.048	.121	.102	.103	.125	.067	.097	.102	.086	.096	.100
13 Parent int T_1	.161	.220	.203	.196	.220	.173	.195	.164	.124	.146	.184	.144		.676	.770	.698	.676	.673	.696	.025	.538	.540	.466	.503
14 Parent int T_2	.179	.212	.198	.165	.172	.112	.144	.151	.125	.115	.144	.095	.747		.724	.673	.677	.655	.491	.003	.465	.478	.439	.465
15 Parent int T_3	.180	.237	.284	.205	.218	.194	.173	.197	.176	.182	.171	.136	.674	.772		.791	.779	.786	.578	.490	.667	.565	.509	.587
16 Parent int T_4	.123	.230	.258	.231	.256	.205	.140	.160	.152	.149	.173	.179	.682	.739	.790		.808	.759	.537	.690	.584	.696	.596	.577
17 Parent int T_5	.146	.197	.219	.234	.252	.205	.128	.145	.112	.122	.144	.143	.685	.722	.770	.813		.758	.553	.497	.632	.659	.726	.602
18 Parent int T_6	.095	.204	.189	.179	.202	.178	.127	.141	.113	.098	.136	.162	.617	.693	.757	.760	.797		.510	.464	.570	.554	.529	.740
19 Parent ext T_1	.139	.224	.194	.194	.190	.211	.239	.249	.160	.170	.195	.156	.697	.524	.471	.489	.500	.454		.519	.702	.672	.613	.609
20 Parent ext T_2	.185	.235	.231	.221	.199	.175	.198	.198	.159	.147	.151	.131	.536	.675	.570	.535	.563	.502	.730		.669	.652	.623	.588
21 Parent ext T_3	.186	.242	.260	.189	.207	.210	.216	.246	.204	.217	.197	.181	.489	.582	.721	.593	.600	.565	.634	.739		.755	.727	.697
22 Parent ext T_4	.192	.260	.295	.264	.279	.247	.196	.220	.212	.193	.212	.224	.472	.530	.550	.690	.615	.552	.632	.708	.699		.806	.729
23 Parent ext T_5	.119	.219	.233	.186	.198	.201	.151	.228	.124	.124	.138	.106	.478	.541	.576	.610	.702	.576	.638	.720	.738	.756		.731
24 Parent ext T_6	.061	.202	.208	.156	.152	.165	.154	.221	.135	.113	.089	.122	.458	.508	.559	.588	.573	.699	.562	.667	.675	.680	.732	

Note. int = internalizing, ext = externalizing, correlations for maternal symptoms are below the diagonal and for paternal symptoms above the diagonal.

Table S2A. Fit Indices and Model Comparison for all maternal RI-CLPMs

	Model fit					Model comparison	
	χ^2(df)	*p*	CFI	RMSEA [CI]	SRMR	$\Delta\chi^2_{SB}$(df)	*p*
$Mother_{int}$ → $Adolescent_{int}$							
1 Baseline	64.97 (57)	.219	.997	.017 [.000;.034]	.038		
2 Free correlated change	58.31 (53)	.287	.998	.014 [.000;.033]	.037	6.80 (4)	.147
3 Free cross-lagged$_{P\rightarrow A}$	59.58 (53)	.249	.997	.016 [.000;.034]	.037	5.79 (4)	.215
4 Free cross-lagged$_{A\rightarrow P}$	60.50 (53)	.224	.997	.017 [.000;.034]	.038	4.45 (4)	.348
$Mother_{int}$ → $Adolescent_{ext}$							
1 Baseline	88.40 (57)	.005	.986	.033 [.019; .046]	.037		
2 Free correlated change	84.99 (53)	.004	.986	.035 [.020; .048]	.037	2.79 (4)	.593
3 Free cross-lagged$_{P\rightarrow A}$	84.70 (53)	.004	.986	.035 [.020; .048]	.037	2.90 (4)	.576
4 Free cross-lagged$_{A\rightarrow P}$	85.07 (53)	.003	.986	.035 [.020;.048]	.035	2.76 (4)	.599
$Mother_{ext}$ → $Adolescent_{int}$							
1 Baseline	79.25 (57)	.027	.990	.028 [.010; .042]	.051		
2 Free correlated change	76.39 (53)	.019	.990	.030 [.012; .044]	.050	1.65 (4)	.800
3 Free cross-lagged$_{P\rightarrow A}$	72.40 (53)	.040	.991	.027 [.006; .042]	.051	7.49 (4)	.112
4 Free cross-lagged$_{A\rightarrow P}$	74.16 (53)	.029	.991	.028 [.009; .043]	.049	4.97 (4)	.291
$Mother_{ext}$ → $Adolescent_{ext}$							
1 Baseline	113.19 (57)	<.001	.973	.045 [.032; .056]	.055		
2 Free correlated change	108.71 (53)	<.001	.974	.046 [.034; .058]	.055	2.48 (4)	.649
3 Free cross-lagged$_{P\rightarrow A}$	105.29 (53)	<.001	.975	.045 [.032; .057]	.053	7.89 (4)	.096
4 Free cross-lagged$_{A\rightarrow P}$	102.14 (53)	<.001	.977	.043 [.030; .056]	.052	12.62 (4)	.013

Note. All models are compared to their fully-constrained baseline model; int = internalizing, ext = externalizing, correlated change = freely estimated within-time associations, cross-lagged$_{P\rightarrow A}$ = freely estimated associations between maternal and subsequent adolescent symptoms, cross-lagged$_{A\rightarrow P}$ = freely estimated associations between adolescent and subsequent maternal symptoms.

Table S2B. Fit Indices and Model Comparison for all paternal RI-CLPMs

	Model fit					Model comparison	
	χ^2(df)	*p*	CFI	RMSEA [CI]	SRMR	$\Delta\chi^2_{SB}$ (df)	*p*
$Father_{int}$ → $Adolescent_{int}$							
1 Baseline	119.81 (57)	<.001	.975	.047 [.035;.059]	.054		
2 Free correlated change	112.90 (53)	<.001	.976	.048 [.036; .060]	.053	6.38 (4)	.173
3 Free cross-$lagged_{P→A}$	114.51 (53)	<.001	.976	.049 [.036; .061]	.053	3.00 (4)	.557
4 Free cross-$lagged_{A→P}$	112.49 (53)	<.001	.977	.048 [.035; .060]	.052	6.93 (4)	.139
$Father_{int}$ → $Adolescent_{ext}$							
1 Baseline	155.36 (57)	<.001	.958	.059 [.048; .070]	.057		
2 Free correlated change	147.04 (53)	<.001	.960	.060 [.048; .071]	.057	7.36 (4)	.118
3 Free cross-$lagged_{P→A}$	147.06 (53)	<.001	.960	.060 [.048; .071]	.057	7.48 (4)	.113
4 Free cross-$lagged_{A→P}$	151.88 (53)	<.001	.958	.061 [.050;.073]	.057	3.12 (4)	.538
$Father_{ext}$ → $Adolescent_{int}$							
1 Baseline	67.65 (57)	.158	.995	.020 [.000; .036]	.040		
2 Free correlated change	63.72 (53)	.149	.995	.020 [.000; .037]	.040	3.35 (4)	.501
3 Free cross-$lagged_{P→A}$	65.15 (53)	.122	.994	.022 [.000; .038]	.040	0.43 (4)	.980
4 Free cross-$lagged_{A→P}$	64.62 (53)	.132	.995	.021 [.000; .037]	.040	1.98 (4)	.739
$Father_{ext}$ → $Adolescent_{ext}$							
1 Baseline	94.33 (57)	.001	.981	.036 [.023; .049]	.039		
2 Free correlated change	91.62 (53)	.001	.981	.038 [.025; .051]	.039	0.91 (4)	.924
3 Free cross-$lagged_{P→A}$	90.34 (53)	.001	.981	.038 [.024; .051]	.039	2.34 (4)	.674
4 Free cross-$lagged_{A→P}$	90.41 (53)	.001	.981	.038 [.024; .051]	.040	3.20 (4)	.524

Note. All models are compared to their fully-constrained baseline model; int = internalizing, ext = externalizing, correlated change = freely estimated within-time associations, cross-$lagged_{P→A}$ = freely estimated associations between paternal and subsequent adolescent symptoms, cross-$lagged_{A→P}$ = freely estimated associations between adolescent and subsequent paternal symptoms.

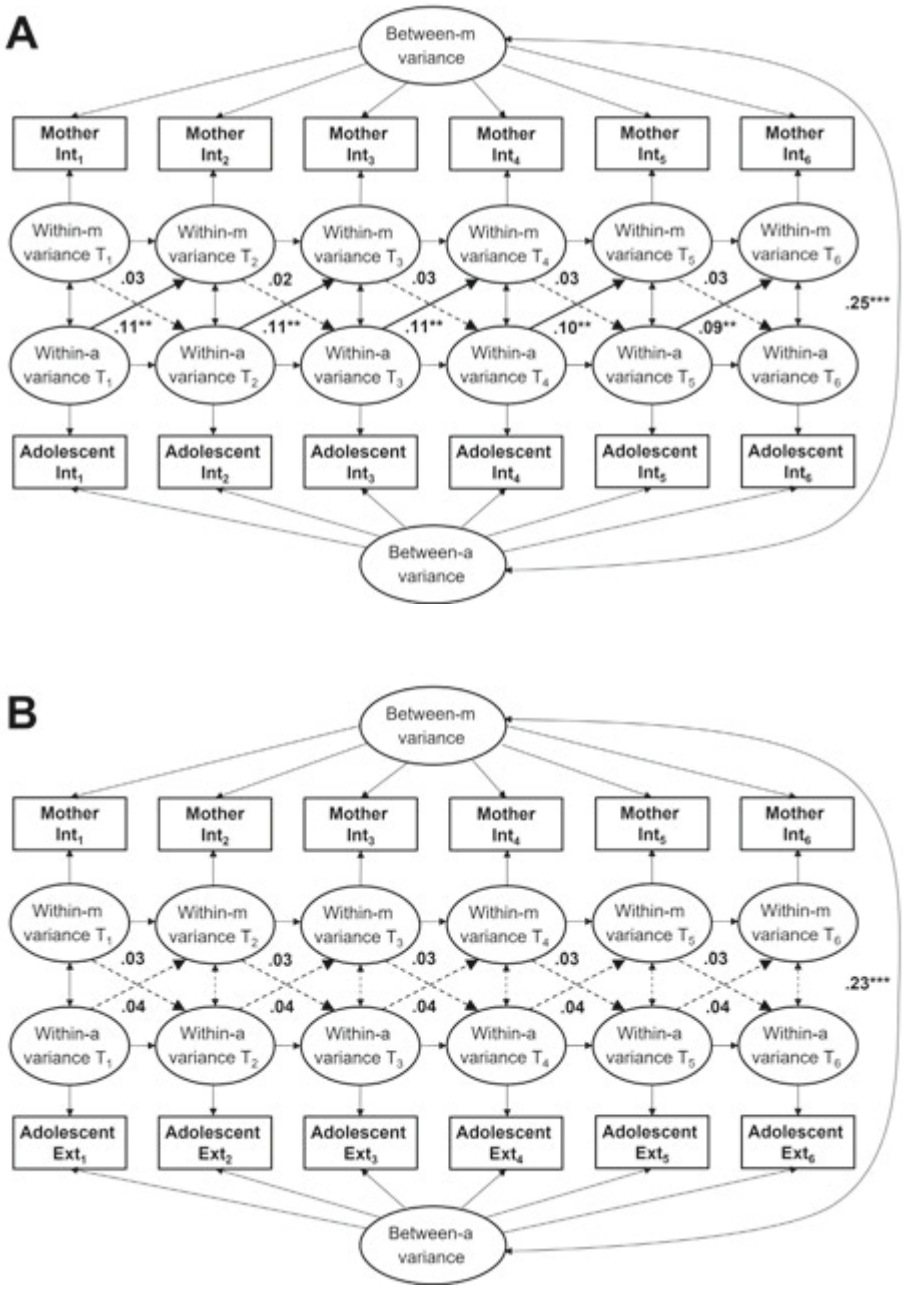

Figure S1. Random-intercept cross-lagged panel models excluding multivariate outliers on the associations between maternal internalizing and adolescent internalizing (1A) and externalizing symptoms (1B), m = mother, a = adolescent. *** $p < .001$, ** $p < .01$.

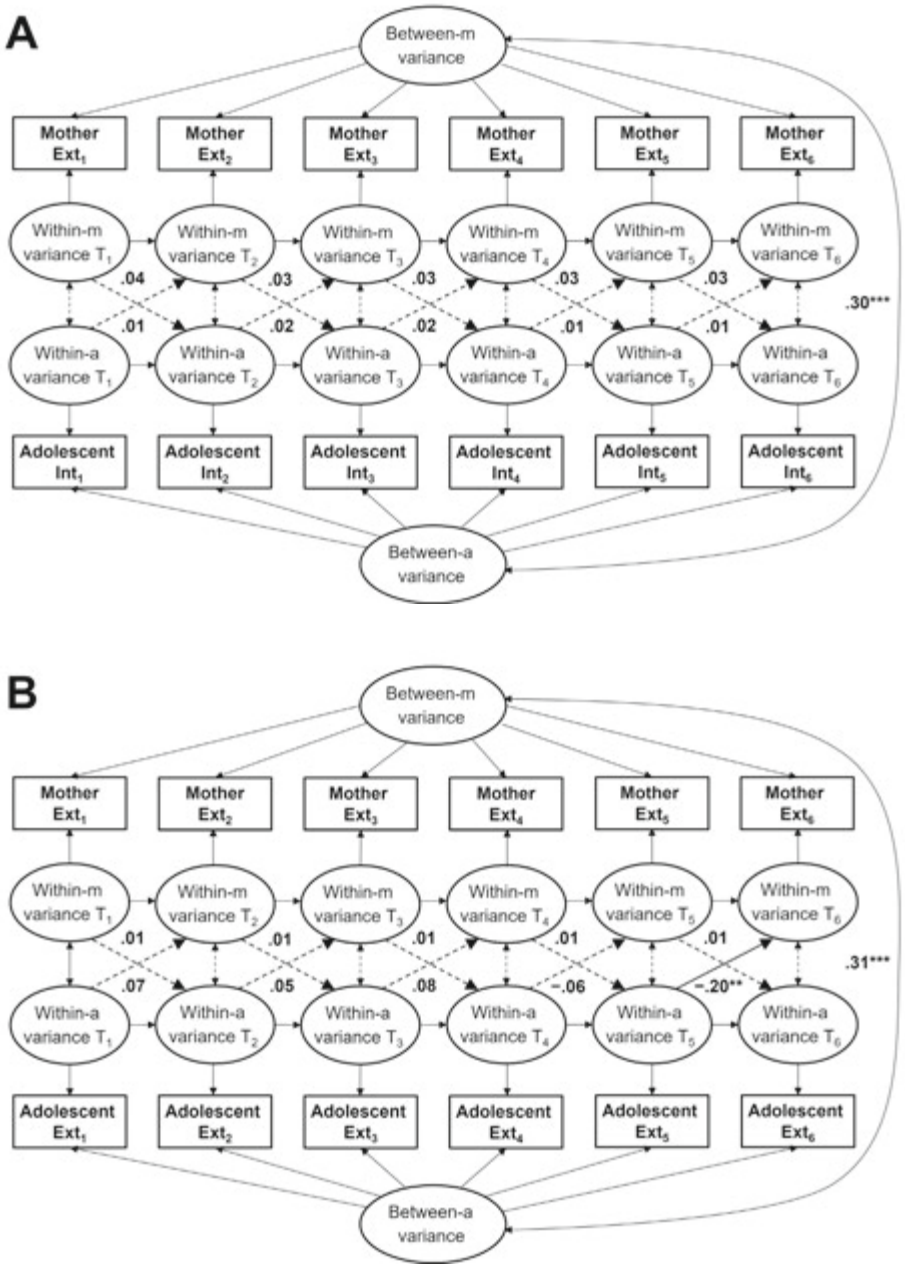

Figure S2. Random-intercept cross-lagged panel models excluding multivariate outliers on the associations between maternal externalizing and adolescent internalizing (2A) and externalizing symptoms (2B), m = mother, a = adolescent. *** $p < .001$, ** $p < .01$.

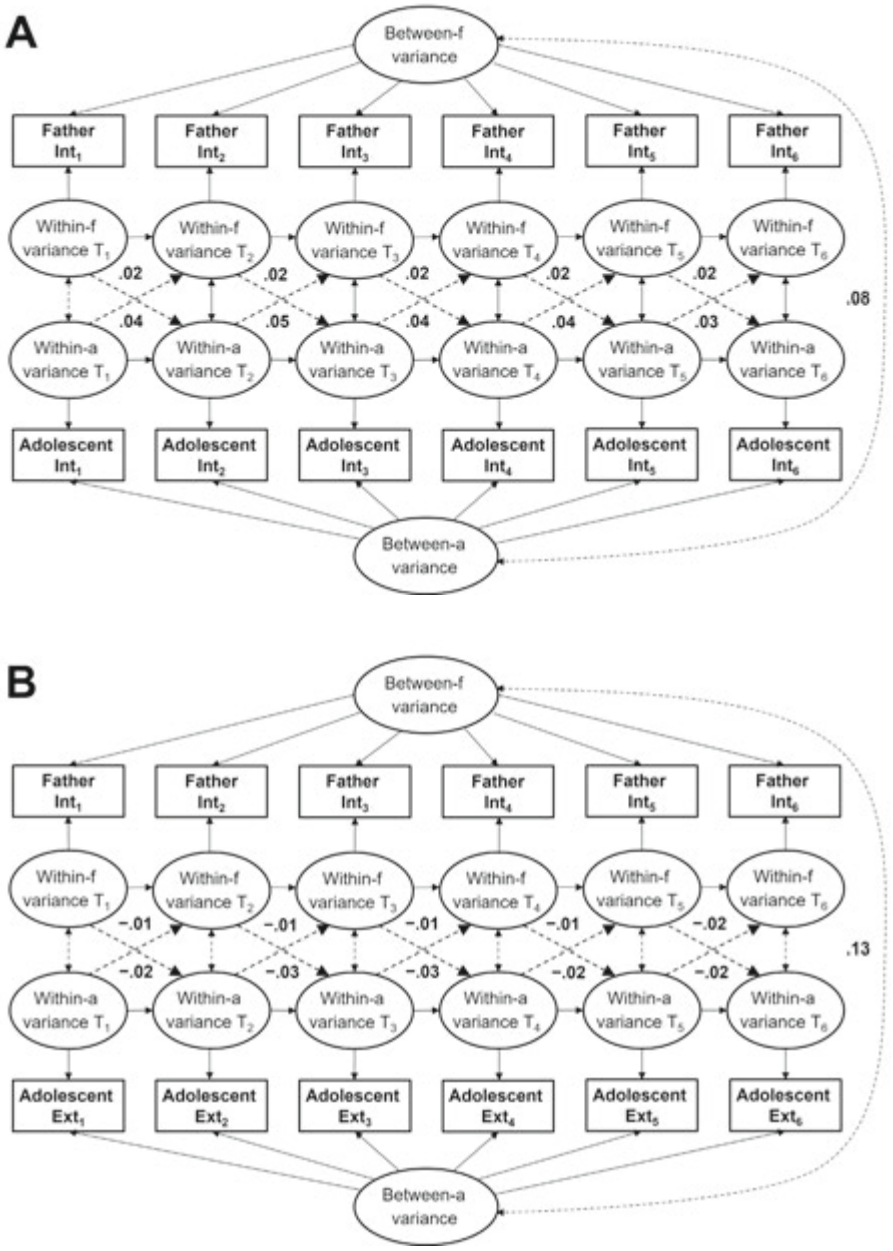

Figure S3. Random-intercept cross-lagged panel models excluding multivariate outliers on the associations between paternal internalizing and adolescent internalizing (3A) and externalizing symptoms (3B), f = father, a = adolescent.

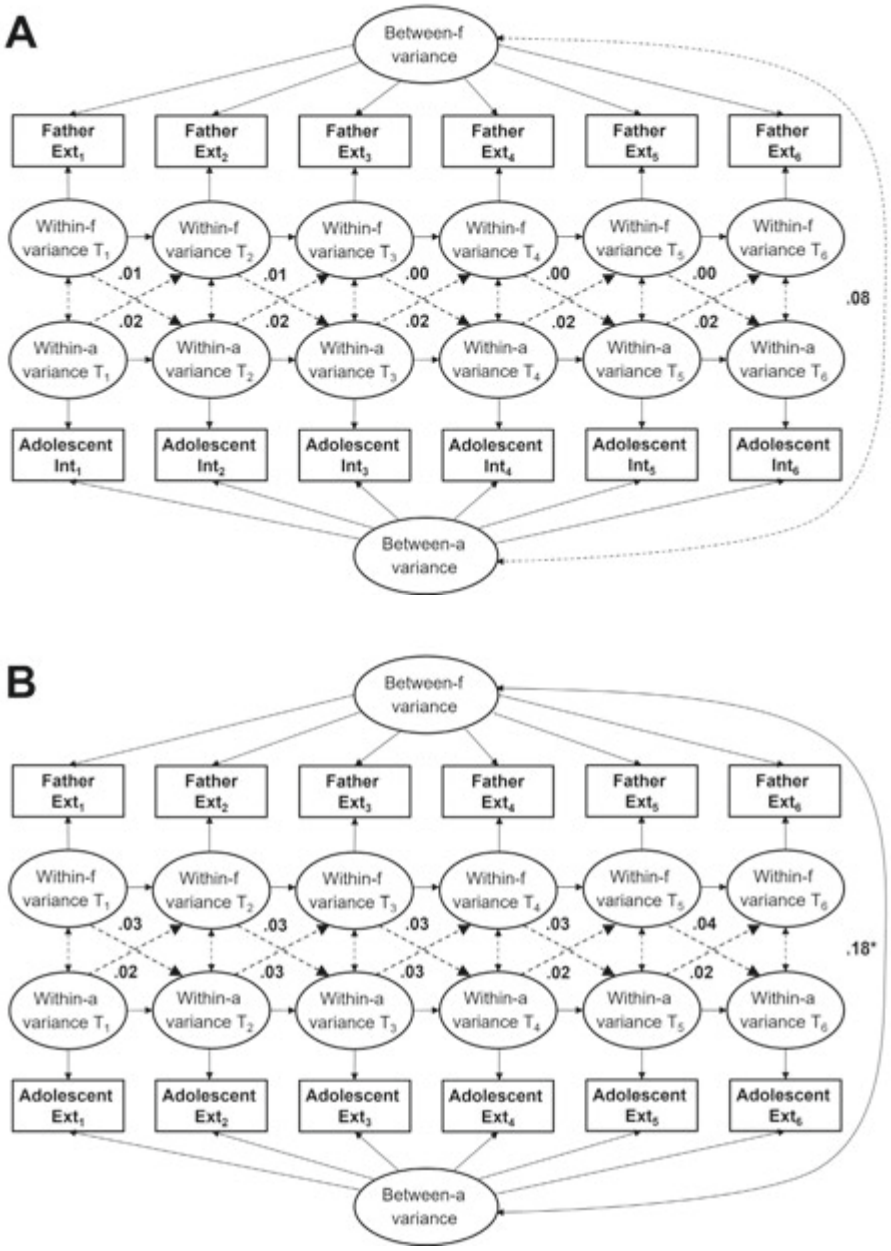

Figure S4. Random-intercept cross-lagged panel models excluding multivariate outliers on the associations between paternal externalizing and adolescent internalizing (4A) and externalizing symptoms (4B), f = father, a = adolescent. * $p < .05$.

CHAPTER 3

Maternal Affect and Youth Psychopathology: The Role of Mother-Adolescent Affect Congruency

This chapter is currently under review:

Schulz, S., Nelemans, S.A., Oldehinkel, A.J., Meeus, W., & Branje, S. (2022). Maternal affect and youth psychopathology: The role of mother-adolescent affect congruency

(preregistered at OSF: https://bit.ly/3htH7nD).

ABSTRACT

Maternal affect contributes to children's psychosocial adjustment. How maternal daily affect intensity and dynamics (i.e., inertia and variability) are associated with adolescents' concurrent and later psychopathological symptoms, however, remains unclear. This preregistered study examined (1) associations of maternal day-to-day positive and negative affect intensity, inertia, and variability with adolescent and young adult psychopathological symptoms, and (2) moderating effects of mother-adolescent affect congruency on these associations. Mother-adolescent dyads (*N*=488) reported positive and negative affect in 75 daily assessments across ages 13-17 years. Adolescents rated their psychopathological symptoms at ages 14-18, 20, and 27 years. Maternal daily affect intensity and inertia, but not variability, were associated with adolescent psychopathological symptoms. Higher mother-adolescent affect congruency was associated with fewer psychopathological symptoms and reflected resilience against adverse effects of maternal affect inertia. While both maternal affect intensity and dynamics play a role in adolescents' psychopathology, promoting mother-adolescent affect congruency might benefit adolescents' adjustment.

Keywords: developmental psychopathology, maternal affect, affect dynamics, mother-adolescent affect congruency, adolescence

Author contributions

SuS conceptualized the study, which was further refined by SN, AO, WM, and SB. SuS performed the statistical analyses and drafted the manuscript. All authors provided feedback on the manuscript.

MATERNAL AFFECT AND YOUTH PSYCHOPATHOLOGY: THE ROLE OF MOTHER-ADOLESCENT AFFECT CONGRUENCY

The emotional climate in the family plays a crucial role in adolescents' functioning and their emotional development. Mothers' psychopathological symptoms that involve difficulties regulating positive and negative affect are among the most salient predictors of adolescent psychopathological symptoms (McLaughlin et al., 2012; Weissman et al., 2016). One mechanism through which psychopathological symptoms are transmitted is mothers' affect, which influences their ability to meet the child's social and emotional needs, and thus has an impact on adolescents' risk for later psychopathological symptoms (Goodman & Gotlib, 1999). To thoroughly understand how maternal affect constitutes adolescent psychopathological risk, we need to consider not only mean levels of affect (i.e., affect intensity), but also temporal changes in affect (i.e., affect dynamics). As affective experiences are dynamic and therefore difficult to recollect (Scherer, 2009; Schwarz, 2007), they are best captured on a day-to-day level when they unfold. However, adolescents may vary in their susceptibility to maternal affect as not all adolescents are equally sensitive to their environment (Belsky, 1997; Pluess, 2015). The effects of maternal affect on adolescent functioning could depend on how congruent adolescents' affect is with their mothers' affect, that is, how strongly adolescent and mother affect are associated. Using five years of measurement bursts of maternal and adolescent affect in a 14-year longitudinal study, we examined (1) how intensity, inertia, and variability of maternal daily positive and negative affect contribute to offspring psychopathological symptoms across adolescence and in young adulthood, and (2) whether mother-adolescent affect congruency moderated these associations.

Maternal Affect and Adolescent Psychopathology

Adolescence is an important developmental period, in which individuals are confronted with new environmental challenges, experience more extreme, fluctuating emotions than in childhood (Arnett, 1999; Larson et al., 2002; Reitsema et al., 2022), and become more susceptible to changes in their social environment (e.g., Dahl et al., 2018). The emotional development of adolescents occurs largely in the context of the family. Apart from genetic transmission, parental daily positive and negative affect constitutes an emotional climate in which children learn how to understand, express, and regulate emotions (Eisenberg et al., 1998; Thompson & Meyer, 2007). If maladaptive, this emotional climate contributes to adolescents' mental health problems directly or indirectly through emotion regulation difficulties (Morris et al., 2007; Stoop & Cole, 2022). Particularly maternal affect may play a crucial role in adolescents' adjustment. Not only do mothers spend a considerable amount of time with their children (e.g., Pleck, 1997), but they are also strongly involved in managing and supporting adolescents' emotions (Stocker et al., 2007). If mothers cannot provide a safe emotional

environment, children might feel emotionally insecure, rejected, or intimidated (Cummings & Davies, 1996; Stocker et al., 2007), which puts them at risk for social and psychopathological problems (Goodman & Gotlib, 1999; Halberstadt, Crisp, & Eaton, 1999). While mothers' affective experiences have been found to impact their ability to meet the social and emotional needs of their children (e.g., Goodman et al., 2020) and their interaction behaviors towards their children (e.g., Rueger et al., 2011), it remains unclear whether maternal affect intensity or other affect dynamics render children vulnerable to psychopathological problems.

Most psychopathological symptoms are characterized by aberrant affect intensity, such as high negative affect or low positive affect (American Psychiatric Association, 2013; Gross & Barrett, 2013). High negative and low positive affect intensity can impede mothers' responsiveness, attentiveness, or use of effective problem-solving (e.g., Dix, 1991) and thereby contribute to adolescent internalizing and externalizing symptoms. Albeit scarce, initial research indeed found that mothers' daily negative affect was related to both concurrent and future child disruptive behaviors (Elgar et al., 2004) as well as concurrent adolescent depressive symptoms (Leung et al., 2009). Similarly, adolescent-reported maternal negative affect was related to concurrent adolescent internalizing and externalizing symptoms (Stocker et al., 2007). Previous studies further indicated that maternal positive affect was associated with positive functioning, including prosocial behavior (Michalik et al., 2007) and social competence (Eisenberg et al., 2001), in (early) childhood as well as fewer problem behaviors in childhood (Cumberland-Li et al., 2003; Eisenberg et al., 2001; McCoy & Raver, 2011) and adolescence (Milan & Carlone, 2018). Generally, these studies suggest that higher levels of maternal negative affect and lower levels of maternal positive affect contribute to adolescent psychopathological symptoms. However, previous evidence is typically based on one-time assessments, which provide only limited conclusions about how maternal daily affect levels across time and contexts contribute to both concurrent and later adolescent psychopathological symptoms.

Maladaptive affect does not only constitute the intensity of emotions, but also the patterns in which affective experiences change or persist across moments or days – characterized as 'affect dynamics' (Koval et al., 2013). Maladaptive affect dynamics have been found to be involved in psychopathological problems of adults and adolescents (Houben et al., 2015; Scott et al., 2020), as they make it difficult for individuals to flexibly and consistently respond to contextual demands. Maternal maladaptive affect patterns might constitute a particularly detrimental emotional environment for adolescents. Prolonged durations of affective states, such as affect inertia, reflect difficulties to recover from affective experiences. Such a rigid emotional environment might increase mothers' preoccupation with themselves and reduce their sensitivity as well as engagement towards their children (Rottenberg et al., 2005), thereby impacting adolescents' emotional adjustment. Highly

fluctuating maternal affect, or affect variability, might similarly elicit adolescent symptoms through modified parental behaviors. Mothers who frequently shift between extreme levels of affect have difficulties maintaining stable emotional states (Gruber et al., 2013), which creates an unpredictable emotional environment that increases children's emotional insecurity.

The Moderating Role of Mother-Adolescent Congruency

Although maladaptive maternal positive and negative affect can signal risk for adolescent psychopathological symptoms, not all children exposed to such a negative affective environment ultimately develop psychopathological symptoms (Kemp et al., 2022). Individual differences in environmental sensitivity (e.g., Belsky, 1997; Ellis et al., 2011; Pluess, 2015) suggest that children are generally more or less sensitive to maternal influences. Sensitivity to maternal affect may be reflected in how congruent adolescents' affect is with maternal affect, in that high mother-adolescent affect congruency indicates high sensitivity and low congruency indicates low sensitivity. As emotions are easily transmitted between social partners (see theories on emotion contagion; Hatfield et al., 1994; Joiner & Katz, 1999, emotion convergence; Anderson & Keltner, 2004, emotion coregulation; Butler & Randall, 2013; Sbarra & Hazan, 2008, and interpersonal emotion dynamics; Butler, 2011), maternal and adolescent affect are associated to some degree. Congruency between maternal and adolescent affect serves adaptive functions as it facilitates understanding, closeness, and social cohesion (Anderson et al., 2003; Bruder et al., 2012). Applied in the context of Goodness of Fit theories (Thomas & Chess, 1977; Lerner et al., 1986), mother-adolescent affect congruency may further promote adolescent psychosocial adjustment, as consonance between the environmental context that parents create and the characteristics that adolescents display yields optimal developmental outcomes.

Although theories generally argue for favorable effects of mother-adolescent congruency, such congruency may be less beneficial when partners adopt maladaptive emotional patterns (Anderson & Keltner, 2004). For example, reciprocity of negative affect in couples or mother-child dyads has been associated with adverse outcomes (e.g., Gottman, 1994), including higher levels of internalizing symptoms (Mercado et al., 2019) and risk for depression (McMakin et al., 2011). Whether congruency effects are positive or negative thus likely depends on whether maternal affect intensity and dynamics are adaptive or maladaptive. Specifically, high mother-adolescent affect congruency may put adolescents at decreased risk for psychopathology when maternal affect is adaptive, but at increased risk when maternal affect is maladaptive.

The Present Study

The present study aimed to investigate how maternal daily positive and negative affect levels (i.e., affect intensity) and affect dynamics (i.e., affect inertia and variability) contribute to adolescent internalizing and externalizing symptoms, and whether mother-adolescent affect congruency moderated these associations. As negative emotions are distinct from positive emotions (Carl et al., 2013) and have been shown to exert stronger effects than positive ones (Baumeister et al., 2001; Houben et al., 2015), we examined the associations of maternal affect with adolescent psychopathological symptoms separately for maternal positive and negative affect. By examining affect intensity, inertia, and variability jointly, we provided insights into the unique contributions of different aspects of maternal affect, which have been mostly studied in isolation. Examining associations in maternal affect and mother-adolescent congruency on a day-to-day level repeatedly across adolescence further allowed for a better understanding of how these micro-processes drive long-term development.

Based on the salient role of maternal affect in adolescent psychopathology, we expected that lower maternal daily positive affect intensity and higher maternal daily negative affect intensity were associated with more adolescent and young adult internalizing and externalizing symptoms. Based on the role of affect dynamics in psychopathology, we further expected that higher positive and negative affect inertia and variability were associated with more adolescent and young adult internalizing and externalizing symptoms.

Furthermore, there might be differences in how maternal daily affect patterns predict adolescent symptoms in the short-term and in the long-term. Theories on transient effects (Fraley & Roisman, 2015; Rutter, 1996; Schulenberg et al., 2004) propose that early experiences provide a foundation for later experiences, which shape adjustment more directly and strongly. The enduring effects model (Fraley & Roisman, 2015; Sroufe et al., 1990) proposes that previous experiences not only generate, but also scaffold current experiences and shape adjustment. As such, they provide long-lasting working models that continue to influence development across life stages. Furthermore, it has been suggested that parental influence develops over time and that adolescents start to resemble their parents more closely as they get older (Halberstadt & Eaton, 2002). To examine whether maternal affect has transient or enduring effects on offspring functioning, we investigated psychopathological outcomes during adolescence, during a transitional period between late adolescence and young adulthood, and during young adulthood. Based on inconsistent previous findings, it is possible that the associations between maternal daily affect and adolescent psychopathological symptoms either decrease, or even disappear, from adolescence to young adulthood, or that they remain similarly strong across the three time points.

Finally, based on the differential susceptibility hypothesis, we expected that mother-adolescent affect congruency moderated the associations between maternal affect and adolescent internalizing and externalizing symptoms. Compared to adolescents with low congruency scores, adolescents who were strongly congruent with their mothers' daily affect were expected to report more internalizing and externalizing symptoms when maternal daily affect was maladaptive (i.e., low positive and high negative affect intensity as well as high positive and negative inertia and variability), but fewer symptoms when maternal daily affect was adaptive. Adolescents who were less congruent with their mothers' affect were expected to remain at the same risk for psychopathology, irrespective of maternal adaptive or maladaptive affect. As psychopathological symptoms differ for females and males (Kessler et al., 2012), we used adolescent gender as covariate in all analyses.

Transparency and openness

Preregistration. All hypotheses, study design, and analyses of this study were preregistered (https://osf.io/y9s76).

Data, materials, code, and online resources. All code and online resources will be published on OSF after acceptance (https://osf.io/d2jna/). The data is publicly available and can be accessed via https://doi.org/10.17026/dans-zrb-v5wp.

Reporting. This study involved an analysis of existing data. We report all data exclusions and all measures in the study.

Ethical approval. The study procedure was approved by the Medical Research Ethics Committee of the University Medical Center Utrecht (RADAR: Research on Adolescent Development and Relationships, 05/159-K) and conducted in accordance with the Declaration of Helsinki.

METHODS

Participants

The sample consisted of 488 adolescents (43.2% adolescent girls, M_{age} T_1 = 13.04, SD_{age} = .46) and their mothers (M_{age} T_1 = 44.40, SD_{age} = 4.41) who participated in the ongoing Research on Adolescent Development And Relationships Young (RADAR-Y; Branje & Meeus, 2018) study that followed adolescents from age 13 onwards. Participants were annually assessed from age 13 to 18 and biennially assessed from age 20 to age 27. All adolescents and most of their mothers (92.8%) were of Dutch origin, identified as racial-ethnic majority (98.8%), and lived in mainly intact two-parent

families (85.9%) with medium to high socioeconomic status (88.3%), based on parents' occupation level.

Sample attrition was 3.0-11.5% across all waves, with 339 adolescents remaining in the study at age 27. Participants who remained in the study at age 27 were significantly younger at the first measurement occasion, $F(486) = 14.33$, $p < .001$, and had significantly older mothers, $F(486) = 5.59$, $p = .018$, than adolescents who dropped out of the study. They were also more likely to be girls, $\chi^2(1) = 8.19$, $p = .004$, and to be from medium to high socioeconomic status families, $\chi^2(1) = 8.65$, $p = .003$, but did not differ from adolescents who dropped out on their baseline psychopathological symptoms.

Procedure

The present study used daily diary data from age 13 to 17 and annual self-report data from age 14 to 27. Adolescents attended the 1st grade of secondary school at the onset of the study and were recruited through 230 randomly selected elementary schools in the central and western regions of the Netherlands. Of all selected participants ($N = 1,544$), families were excluded if they did not fulfil the full family requirements ($n = 364$), could not be contacted or withdrew their participation ($n = 569$), or failed to provide written consent of all family members ($n = 114$). Out of 497 families who participated at the first measurement occasion, $n = 6$ mother-adolescent dyads were excluded from the analyses because their data was missing for all waves and $n = 3$ mother-adolescent dyads were excluded due to non-valid data at any assessment of daily affect. This resulted in a final sample size of $n = 488$. Adolescents and mothers provided active written informed consent at all waves and were compensated monetarily for their participation. They completed daily assessments online and annual assessments at their homes (age 14-18) or online (age 20 and 27).

Measures

Adolescent psychopathological symptoms. Self-reported internalizing symptoms were assessed as a combined score of adolescent anxiety symptoms, using the 38-item Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1997), and depression symptoms, using the 23-item 2nd edition of the Reynolds Adolescent Depression Scale (RADS-2; Reynolds, 2000). At age 27, the adult version of the SCARED was used (i.e., SCARED-A; van Steensel & Bögels, 2014), which consists of 71 items. To keep the adolescent and adult versions consistent, we excluded the school phobia scale from the SCARED and the obsessive-compulsive disorder (9 items), post-traumatic stress disorder (4 items), animal phobia (3 items), blood-injection-injury phobia (7 items), and situational-environmental phobia (5 items) scales from the SCARED-A. Adolescents rated how anxious they felt on a 3-point scale (1 = almost never, 3 = often) and how depressed they felt on a

4-point scale (1 = almost never, 4 = often). All scales showed high internal consistencies across time points (α = .92-.95). Items for the SCARED and the RADS-2 were averaged, respectively, to create one depression and one anxiety score per participant per wave. The total anxiety and depression scores were then standardized and averaged to create an internalizing symptom score per participant per wave. Scores were averaged only when both anxiety and depression scores were non-missing to ensure that the internalizing construct was comparable across participants.

Self-reported externalizing symptoms were assessed using the 11-item delinquent and the 19-item aggressive behavior scales of the Youth Self Report (YSR; Achenbach, 1991). At age 27, the 14-item rule-breaking behavior and the 15-item aggressive behavior subscales of the Adult Self Report were used (Achenbach & Rescorla, 2003). Adolescents rated their externalizing behaviors on a 3-point scale (0 = never, 2 = often). The scales showed high internal consistencies across time points (α = .88 - .91). If data were available for ≥ 90% of all items, items were averaged to create one externalizing score per participant per wave. To reflect adolescent internalizing and externalizing symptoms in one total score across adolescence, respectively, the scores from age 14 to age 18 were averaged.

Daily positive and negative affect. Maternal and adolescent daily positive and negative affect were assessed yearly from adolescent age 13 to 17 using the Daily Mood Device, an adapted internet version of the Electronic Mood Device (DMD; Hoeksma et al., 2000). Participants rated their primary emotions (happy, angry, anxious, sad) on a 9-point scale on five consecutive days at three time points per year, resulting in 75 assessments across adolescence. Each emotion was measured with three items. The items on the happy scale were averaged to form a total score of positive affect intensity per day. The items on the angry, afraid, and sad scales were averaged, respectively, and the mean scores from each scale were averaged to form a total score of negative affect intensity per day. Positive affect scores were formed if participants reported on at least one item of the happy scale per day; negative affect scores were formed if participants reported on at least one item of the angry, anxious, and sad scales, respectively. These affect scores were then used to calculate maternal affect inertia and variability with Dynamic Structural Equation Modelling (DSEM) techniques discussed in the following section.

Data analysis

Missing data ranged from 1.0 - 30.7% across all outcome variables and from 20.5 - 23.9% across maternal and adolescent positive and negative affect (average days with missing data per participant ranged from 14.8 – 17.2 days). Little's missing completely at random (MCAR) test detected no systematic patterns of missingness, normed $\chi^2/df = 1.28$ across study variables for the positive affect model and normed $\chi^2/df = 1.52$ for the negative affect model (Bollen, 1989).

Data preparation using DSEM. All analyses were conducted using M*plus* version 8.5 (Muthén & Muthén, 1998–2017). To extract measures of maternal affect intensity, inertia, and variability for positive and negative affect, respectively, we constructed two bivariate multilevel path models using dynamic structural equation modeling (DSEM; see Figure 1). In DSEM, all intensive affect measures were split into a within-person and a between-person component using latent mean centering. The between-person component reflects an individuals' trait affect scores, while the within-person component reflects temporal deviations from that individual's means, characterized as affective states (Hamaker et al., 2018). These models were used to obtain individual scores for maternal positive affect intensity, inertia, variability, and mother-adolescent positive affect congruency (Figure 1A) and individual scores for maternal negative affect intensity, inertia, variability, and mother-adolescent negative affect congruency (Figure 1B).

We followed the general approach suggested by Hamaker and colleagues (2018): We modeled two first-order autoregressive VAR(1) models using Bayesian Markov chain Monte Carlo (MCMC) estimation (two chains, 50,000 iterations) with default priors. PSR values for each parameter were very close to 1 (≤ 1.014 for positive affect and ≤ 1.039 for negative affect) and trace plots were dense, which indicates model convergence. Covariance coverage was > .175 for both models. Missing data were handled using MCMC sampling. Due to nonequidistant measurements (i.e., no assessments on the weekend and during the remaining weeks of the year) and missing data (e.g., participants who failed to complete all daily assessments of the week), not all intervals between measurements were equal. As the effect sizes of the lagged associations depend on the time interval (see Gollob & Reichardt, 1987; Hamaker et al., 2018), it is important to account for its unequal structure. To best reflect our day-to-day dynamics of interest and the structure of our data, in which most assessments were obtained with an interval of one day, we specified the time interval of interest to a value of 1 (unit of days) and inserted missing values (10 days) for the days that were not consecutive but included longer time intervals between measurements.

Maternal positive and negative affect levels at Day *t* were constructed as outcome variables that were regressed on maternal positive and negative affect levels at the previous Day *t*-1, respectively. All autoregressive and cross-lagged effects were randomly estimated at the within-person level, indicating that the scores of mothers and adolescents within one family were fixed across time but varied across families. Each model included four random effects: The random mean μ for maternal positive or negative affect constitutes positive or negative affect intensity, or average affect levels across days. The autoregression path Φ between maternal positive or negative affect at *t*-1 and maternal positive or negative affect at *t* constitutes maternal positive or negative affect inertia, or the tendency for an emotion to carry over from one day to the next. The random residual variance

log(π) of maternal positive or negative affect (i.e., innovation variance) constitutes maternal positive or negative affect variability, or the range of daily fluctuations, irrespective of temporal order. This measure describes the novel aspects of positive or negative affect on a specific day that cannot be explained by the autoregressive effect, such as individual differences in sensitivity to other factors that influence an individual's daily affect (Jongerling et al., 2015). The standardized regression path between maternal positive or negative affect at Day *t* and adolescent positive or negative affect at Day *t* constitutes mother-adolescent positive and negative affect congruency, respectively[1]. This approach reflects congruency both statistically as well as conceptually as adolescents' sensitivity to their environment. All random effects were allowed to covary with each other at the between-person level. Results of the DSEM models and correlations between all random effects can be found in Table S1 in the Supplemental Material.

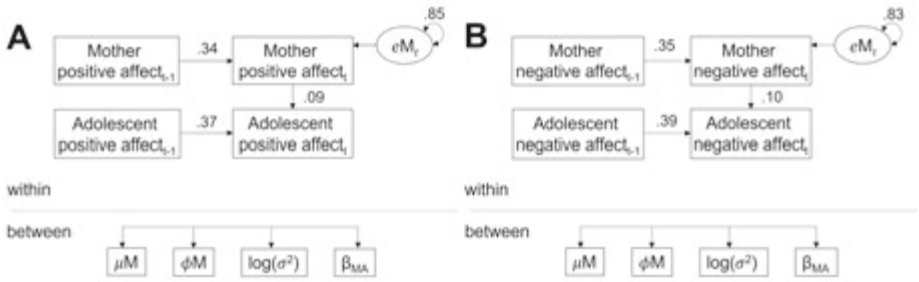

Figure 1. Final DSEMs for positive (A) and negative affect (B), depicting the within-person estimates averaged across participants. All estimates were significant based on the 95% Bayesian credibility intervals that did not include zero; μ = individual mean (intensity level), ϕ = autoregressive paths (inertia), log(σ2) = individual differences in innovation variance (variability), β_{MA} = regression path from mother affect to adolescent affect at the same time point (mother-adolescent affect congruency).

Path models. After obtaining the predictor scores from DSEM, our analytical procedure to answer our research questions was twofold: First, we constructed six path models - three path models for

1 In constructing mother-adolescent affect congruency scores, we deviated from our preregistration. In setting up the models, we encountered difficulties with our preferred and preregistered approach as we were not able to extract a meaningful covariance between maternal and adolescent affect from a separate multilevel model, and in DSEM it is not (yet) possible to estimate and extract individual differences using the correlation at T_0. Our alternative approach, using the covariance of innovations, however, was not suitable either, as (1) all covariances in DSEM are restricted to be either positive or negative and we could not assume that maternal and adolescent affect are positively associated for all dyads, and (2) the covariance of innovations corrects for strong autoregression and within-time associations, and thus depends strongly on the (chosen) time interval between assessments. Therefore, we consulted with an independent DSEM expert who suggested the described approach.

positive affect and three for negative affect - in which maternal positive or negative affect intensity, inertia, and variability extracted from the DSEM analyses predicted internalizing and externalizing symptoms in adolescence (Model 1), a transitional period in young adulthood (Model 2), and young adulthood (Model 3). To examine whether the effects of maternal affect on adolescent internalizing and externalizing symptoms are transient or enduring, we tested whether the standardized regression coefficients for the three models were equal using z-tests.

Second, we added mother-adolescent positive and negative affect congruency as direct effects and moderators to each respective model. We evaluated three interaction effects for positive and negative affect, respectively: Mother-adolescent affect congruency*maternal affect intensity, mother-adolescent affect congruency*maternal affect inertia, and mother-adolescent affect congruency*maternal affect variability. To further examine significant interactions, we estimated the association between maternal affect and adolescent psychopathological symptoms at ± 1 *SD* from the mean of mother-adolescent affect congruency (Cohen, Cohen, West, & Aiken., 2003). As recommended practice for differential susceptibility (Preacher et al., 2006; Roisman et al., 2012), we calculated the region of significance (1) with respect to the moderator to examine the range of values of mother-adolescent affect congruency for which maternal affect and adolescent symptoms were significantly associated, and (2) with respect to the predictor to examine the range of values of maternal affect for which mother-adolescent affect congruency and adolescent symptoms were significantly associated. To determine differential susceptibility, the regression lines for high and low mother-adolescent affect congruency should differ significantly when maternal affect is adaptive (i.e., low negative or high positive affect, low positive or negative affect inertia, low positive or negative affect variability) as well as when maternal affect is maladaptive (i.e., high negative or low positive affect, high positive or negative affect inertia, high positive or negative affect variability), as indicated by ± 2 *SD* boundaries from the mean. Regions of significance were calculated and illustrated using a web-based program (Roisman et al., 2012).

Sensitivity analyses. Multivariate outlier analyses using Mahalanobis Distance identified a few outliers (*n*s = 5-8) for each analysis. We therefore conducted the path models without their respective outliers as sensitivity analyses. As DSEM models were used to generate the predictor estimates for each individual family (within-person estimates), sensitivity analyses were only conducted for the path models. To avoid discussing potential artifacts, we only interpreted results that were consistent across analyses with and without outliers. The full results for all analyses can be found in the Supplemental Material.

RESULTS

Daily maternal affect predicting adolescent psychopathological symptoms

Correlations between all variables in the positive affect models (A) and negative affect models (B) are presented in Table S2 in the Supplemental Material.

Maternal positive affect. All associations were corrected for multiple comparisons using False Discovery Rate (FDR; $p_{adj} < .021$ for positive affect and $p_{adj} < .015$ for negative affect). The full results for all main analyses can be found in Table S3 in the Supplemental Material. Partially consistent with our hypotheses, higher maternal positive affect intensity during adolescence was significantly associated with fewer adolescent internalizing and externalizing symptoms concurrently at age 14-18 (see Figure 2A) and during young adulthood at age 20 (see Figure 2B), but not at age 27 (see Figure 2C). As partially expected, higher maternal positive affect inertia during adolescence was associated with more adolescent and young adult externalizing symptoms at age 27, but not with internalizing symptoms at any age or young adult externalizing symptoms at age 20. As partially expected, higher maternal positive affect variability during adolescence was associated with more young adult externalizing symptoms at age 27, but not with adolescent symptoms or young adult symptoms at age 20.

Sensitivity analyses without outliers generally resulted in similar conclusions (see Table S4). Using z-tests, we further examined whether the effects of maternal positive affect on adolescent symptoms differed across time points. The associations of maternal daily positive affect intensity with internalizing symptoms were significantly stronger at age 14-18 than at age 27 ($p = .027$). None of the other associations differed significantly across time points ($ps > .059$).

Maternal negative affect. Partially consistent with our hypotheses, higher maternal negative affect intensity during adolescence was associated with more adolescent internalizing and externalizing symptoms concurrently at age 14-18 (see Figure 2A; see Table S3), but not with young adult internalizing or externalizing symptoms at age 20 (see Figure 2B) or at age 27 (see Figure 2C). As partially expected, higher maternal negative affect inertia during adolescence was associated with more adolescent internalizing and externalizing symptoms concurrently at age 14-18, but not with young adult internalizing or externalizing symptoms at age 20 or at age 27. Contrary to our expectations, maternal negative affect variability during adolescence was not associated with adolescent internalizing or externalizing symptoms at any age.

Sensitivity analyses without outliers resulted in the same conclusions, except that higher negative affect variability was also significantly associated with young adult externalizing symptoms at age 20 (see Table S4). None of the associations between maternal negative affect and internalizing or externalizing symptoms differed significantly across age (*p*s > .154).

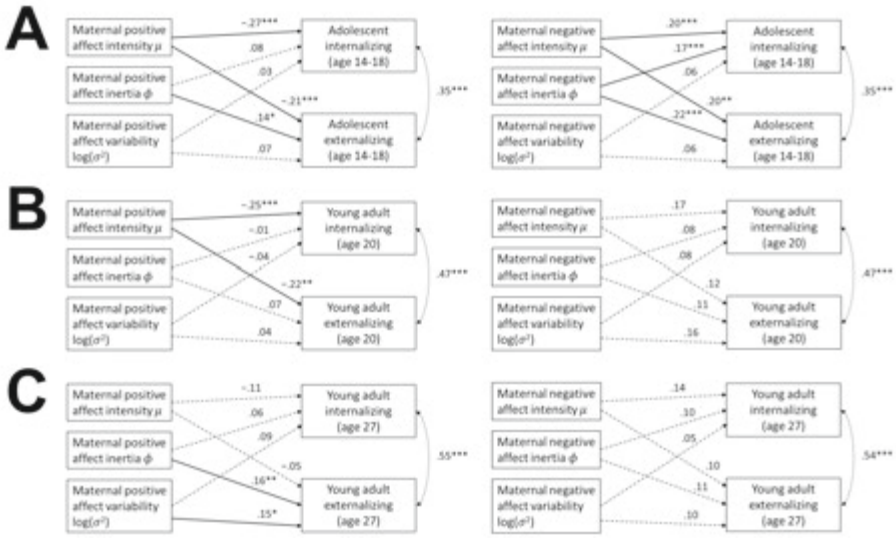

Figure 2. Path models predicting internalizing and externalizing symptoms from maternal daily positive affect at age 14-18 (A), at age 20 (B), and at age 27 (C).

Moderation by mother-adolescent daily affect congruency

The results of all moderation analyses are depicted in Table 1. All associations were corrected for multiple comparisons using False Discovery Rate (FDR; $p_{adj} < .030$ for positive affect and $p_{adj} < .028$ for negative affect). Adding mother-adolescent positive and negative affect congruency revealed significant moderately strong associations of mother-adolescent positive and negative affect congruency with adolescent symptoms across the moderation analyses (see Table 1). Adolescents who were more congruent with their mother's positive or negative affect consistently reported fewer internalizing and externalizing symptoms at every age. The association of mother-adolescent affect congruency with adolescent internalizing and externalizing symptoms was also evident in posthoc exploratory analyses including only main effects of maternal affect measures and mother-adolescent affect congruency, but excluding the interaction between maternal affect measure and mother-adolescent affect congruency as predictors of adolescent adjustment (βs = –.20 - –.30, *p*s < .017, except for externalizing symptoms at age 20, β = –.13 , *p* = .060; see Table S5).

Table 1. Results of the moderation analyses.

	Adolescence (age 14-18)						Transitional period (age 20)						Young adulthood (age 27)					
	Internalizing symptoms			Externalizing symptoms			Internalizing symptoms			Externalizing symptoms			Internalizing symptoms			Externalizing symptoms		
Positive affect	β	*p*	95%CI	β	*p*	95%CI	β	*p*	95%CI	β	*p*	95%CI	β	*p*	95%CI	β	*p*	95%CI
Sex	.30	<.001	.233, .371	-.12	.009	-.200, -.029	.27	<.001	.176, .358	-.04	.410	-.136, .056	.18	.001	.091, .278	-.09	.113	-.192, .020
Intensity	-.25	<.001	-.335, -.166	-.19	.001	-.229, -.081	-.24	<.001	-.337, -.140	-.20	.002	-.317, -.074	-.08	.135	-.180, .024	-.02	.713	-.145, .100
Inertia	.33	<.001	.201, .454	.35	<.001	.204, .504	.22	.004	.070, .368	.23	.033	.019, .445	.27	.001	.116, .425	.37	<.001	.213, .534
Variability	.19	<.001	.085, .300	.23	<.001	.103, .361	.10	.106	-.021, .218	.20	.020	.032, .375	.29	<.001	.141, .434	.35	<.001	.185, .508
Congruency	-.31	<.001	-.420, -.208	-.29	<.001	-.416, -.168	-.26	<.001	-.384, -.143	-.22	.017	-.397, -.039	-.27	<.001	-.397, -.140	-.26	<.001	-.404, -.121
Intensity x congruency	.08	.021	.012, .150	.04	.421	-.055, .132	.01	.832	-.071, .088	.03	.614	-.085, .143	-.01	.900	-.088, .077	-.00	.941	-.104, .096
Inertia x congruency	-.09	.036	-.170, -.006	-.07	.214	-.182, .041	-.07	.120	-.153, .018	-.11	.019	-.210, -.019	-.15	<.001	-.239, -.068	-.17	<.001	-.256, -.080
Variability x congruency	.07	.083	-.009, .147	.11	.035	.008, .202	.01	.917	-.087, .097	.10	.087	-.014, .209	.04	.442	-.056, .128	.01	.826	-.088, .110
Negative affect																		
Sex	.28	<.001	.212,.349	-.14	.001	-.222, -.057	.26	<.001	.174, .350	-.04	.455	-.128, .057	.17	.001	.066, .269	-.08	.131	-.186, .024
Intensity	.17	<.001	.077,.270	.17	.002	.061, .281	.14	.048	.001, .279	.09	.279	-.070, .242	.11	.101	-.021, .234	.07	.291	-.060, .201
Inertia	.34	<.001	.246,.427	.38	<.001	.272, .480	.21	<.001	.092, .326	.18	.013	.038, .321	.26	<.001	.134, .391	.23	<.001	.103, .364
Variability	.22	<.001	.113,.320	.20	.001	.086, .323	.19	.006	.053, .324	.27	.002	.099, .438	.18	.007	.048, .303	.25	<.001	.123, .369
Congruency	-.30	<.001	-.384,-.211	-.26	<.001	-.349, -.168	-.21	<.001	-.320, -.106	-.15	.039	-.298, -.008	-.24	<.001	-.362, -.116	-.22	<.001	-.343, -.096
Intensity x congruency	-.10	.014	-.176,-.019	-.08	.113	-,187, .020	-.06	.349	-.177, .063	-.05	.573	-.208, .115	-.06	.221	-.149, .034	-.01	.913	-.124, .111
Inertia x congruency	-.12	.001	-.183,-.051	-.16	<.001	-.243, -.069	-.06	.201	-.145, .031	-.08	.146	-.190, .028	-.07	.086	-.156, .010	-.13	.013	-.226, -.026
Variability x congruency	.06	.170	-.025,.142	.02	.635	-.075, .123	.05	.429	-.069, .162	.09	.286	-.077, .260	.01	.873	-.093, .110	.06	.247	-.044, .170

Note. All associations were corrected for multiple comparisons using FDR, $p_{adj} < .030$ for positive affect and $p_{adj} < .028$ for negative affect.

Maternal positive affect. Out of 18 potential interaction effects for maternal positive affect, we detected one significant interaction involving positive affect intensity and three significant interactions involving positive affect inertia. Sensitivity analyses without outliers resulted in similar conclusions for the findings involving positive affect inertia, yet these analyses detected no significant interactions involving positive affect intensity and one additional significant interaction involving positive affect inertia (see Table S4). Contrary to what we expected, mother-adolescent affect congruency *weakened* the associations of maternal positive affect inertia with externalizing symptoms at age 20 and 27 and internalizing symptoms at age 27.

The regions of significance for mother-adolescent affect congruency revealed that associations of maternal affect inertia with youth symptoms were only significant for low-congruent, and not for high-congruent adolescents (see Table S6). At adolescent age 27, maternal positive affect inertia and externalizing symptoms were also significantly associated for high-congruent adolescents, but only for congruency scores up to roughly 1 ½ *SD*s. Combined with the extreme upper bound significance values for mother-adolescent affect congruency scores that do not hold practical relevance, these results suggest that maternal positive affect inertia and adolescent symptoms were mainly associated for low-congruent adolescents.

The regions of significance for maternal positive affect inertia revealed that high-congruent adolescents reported *fewer* internalizing and externalizing symptoms in young adulthood than low-congruent adolescents only when maternal positive affect inertia was high (see Figure 3A). The extreme lower bound significance values for maternal positive affect inertia scores that do not hold practical relevance further indicated that mother-adolescent affect congruency and adolescent symptoms are unlikely to be significantly associated at low levels of maternal positive affect inertia.

Maternal negative affect. Out of 18 potential interaction effects for maternal negative affect, we detected one significant interaction involving negative affect intensity and three significant interactions involving negative affect inertia (see Table 1). Sensitivity analyses without outliers resulted in the same conclusions for negative affect inertia, but detected no significant interactions involving negative affect intensity. Contrary to our expectations, mother-adolescent negative affect congruency *weakened* the associations of maternal negative affect inertia with internalizing at age 14-18 and externalizing symptoms at age 14-18 and age 27. The regions of significance for mother-adolescent affect congruency revealed that associations of maternal affect inertia with youth symptoms were significant and positive for both low-congruent and high-congruent adolescents, but stronger for low-congruent adolescents (see Table S6). The regions of significance for maternal negative affect inertia revealed that high-congruent adolescents reported fewer externalizing

symptoms than low-congruent adolescents only when maternal negative affect inertia was high (see Figure 3B). One exception was the association between maternal negative affect inertia and age 14-18 internalizing symptoms, which only significantly differed between high- and low-congruent adolescents at values close to 2 *SD*s of maternal negative affect inertia. Based on the extreme lower bound significance values for maternal negative affect inertia scores, mother-adolescent affect congruency and adolescent symptoms are unlikely to be significantly associated at lower levels of maternal negative affect inertia.

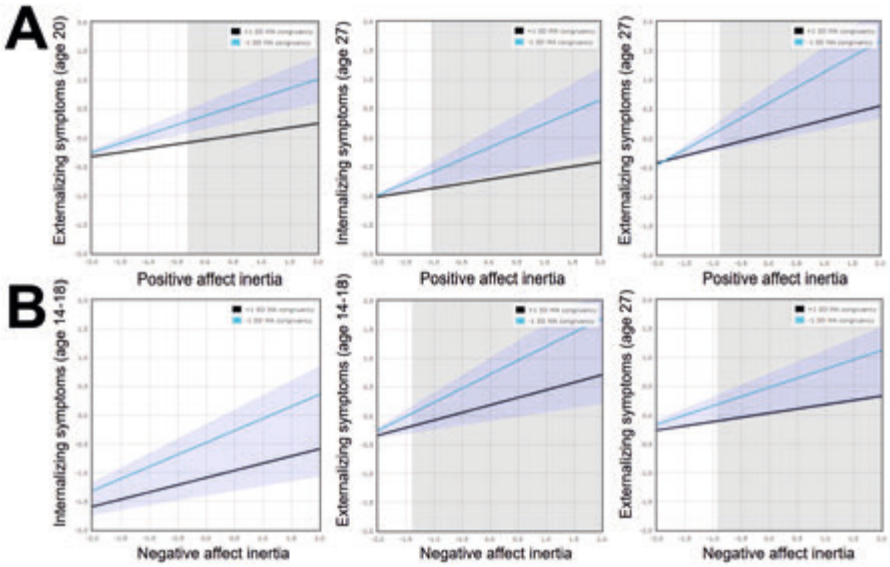

Figure 3. Significant interactions of positive (A) and negative affect inertia with mother-adolescent (MA) affect congruency predicting internalizing and externalizing symptoms in adolescence and young adulthood; shaded areas depict regions of significance.

Exploratory analyses. Due to the complexity and novelty of the moderation analyses, we performed additional exploratory analyses for all moderators separately as a robustness check to better understand the results. These analyses are presented in Table S7 in the Supplemental Material. The interaction effects involving inertia remained quite robust across different analyses (see Table S7 for two exceptions), but the interaction effects involving positive and negative affect intensity, which were also inconsistent in the analyses with and without moderators, did not replicate. Furthermore, compared to the analyses including all moderators, the exploratory analyses also identified five significant interactions out of 12 possible involving positive and negative affect variability. As partially hypothesized, but in contrast to the results involving affect inertia, mother-adolescent positive affect congruency exacerbated the associations of maternal positive

affect variability with internalizing symptoms at age 27 and externalizing symptoms at age 14-18 and age 20 for high-congruent, but not low-congruent adolescents. Similarly, mother-adolescent negative affect congruency exacerbated the associations of maternal negative affect variability with internalizing symptoms at age 14-18 and externalizing symptoms at age 27 for high-congruent, but not low-congruent adolescents. High-congruent adolescents reported fewer symptoms than low-congruent adolescents when maternal positive and negative affect variability was low (see Table S8; Figure S1). When maternal positive, but not negative affect variability was high, on the other hand, high-congruent adolescents reported more externalizing symptoms at age 20 than low-congruent adolescents. High- and low-congruent adolescents did not differ as a function of maternal affect variability at any other age.

DISCUSSION

This study examined the role of maternal affect intensity, inertia, and variability during adolescence in adolescent and young adult internalizing and externalizing symptoms and how mother-adolescent affect congruency may moderate these associations. As partially expected, lower maternal positive affect intensity was associated with more internalizing and externalizing symptoms in adolescence and the transition to young adulthood, whereas positive affect inertia mainly contributed to more adolescent externalizing symptoms in adolescence and young adulthood. For maternal negative affect, we found that higher affect intensity and inertia were associated with more internalizing and externalizing symptoms during adolescence, but not beyond adolescence. The interaction models further suggested that mother-adolescent affect congruency buffered the effects of maternal maladaptive affect, that is, adolescents who were more congruent with their mother's positive and negative affect were at lower risk for particularly externalizing symptoms than adolescents who were less congruent with their mother's affect.

The Role of Maternal Affect in Adolescent Psychopathology

Controlling for affect dynamics and adolescent gender, maternal affect intensity most consistently predicted offspring internalizing and externalizing symptoms in adolescence and during a transitional period to young adulthood, albeit not in young adulthood. This predominant role of affect intensity is in line with studies indicating associations of maternal positive and negative affect with child psychopathological symptoms (Elgar et al., 2004; Leung et al., 2009) and studies highlighting the dominant role of mean-level intensity over affect dynamics in predicting internalizing symptoms (e.g., Dejonckheere et al., 2019). Mothers' higher levels of negative affect and lower levels of positive affect can undermine their ability to be responsive and attentive or to use effective problem-solving strategies in interactions with their children (e.g., Dix, 1991), thereby

increasing their children's risk for psychopathological symptoms in their development towards adulthood. The effects of maternal affect intensity seemed to be enduring, rather than transient, as the associations with internalizing and externalizing symptoms were generally similarly strong across all time points, albeit only statistically significant in adolescence and during a transitional period to young adulthood. This suggests that mothers' affect levels play an important role in children's functioning both across and beyond adolescence.

Albeit less consistently, maternal affect dynamics, mainly inertia, still contributed to particularly adolescent externalizing symptoms above and beyond maternal affect intensity. Other studies also emphasize the unique role of inertia in well-being above and beyond mean-levels (Koval et al., 2016). High positive or negative affect inertia decreases mothers' abilities to flexibly respond to immediate environmental demands and to engage with their children (e.g., Rottenberg et al., 2005). These mothers hence may have difficulties to meet their children's social and emotional needs, which increases adolescents' risk for psychopathological problems (Goodman et al., 2020; Goodman & Gotlib, 1999).

Stronger fluctuations in maternal positive or negative affect appear to be less detrimental than affect inertia according to our findings. One reason may be that mothers with more variable affect show a wide range of emotional expressions, which has been found to promote children's emotional development (Halberstadt et al., 1999; Morris et al., 2007). Although higher variability, particularly with regard to negative affect, has been associated with lower psychological adjustment in some studies (Houben et al., 2015; Scott et al., 2020), another line of research suggests that affect variability rather reflects flexibility, which promotes well-being (Kashdan & Rottenberg, 2010). Based on these contrasting propositions and findings with regard to affect variability, it is possible that maternal affect variability is not linearly associated with adolescent symptoms. Instead, a balanced amount of variability might reflect most adaptive functioning and may allow mothers to flexibly respond to their children's needs, thus predicting best outcomes in adolescents.

Our findings provided more evidence for enduring than transient effects, suggesting that the affective maternal environment continues to play a role in children's development across time. While we generally found similarly strong associations for maternal positive and negative affect with adolescent psychopathological symptoms, the effects of maternal positive affect seemed to be longer lasting. This is contrary to what we would expect given that negative experiences and emotions are often more influential than positive ones (Baumeister et al., 2001). According to the broaden-and-build theory (Fredrickson, 1998, 2018), positive emotions broaden individuals' cognitive and social resources, resulting in long-term effects on functioning and well-being. Also,

positive emotions are associated with goal-oriented behavior (Carl et al., 2013) and function to buffer the detrimental effects of stress and negative emotions (Garland et al., 2010). As such, maternal positive affect may influence children in ways that have long-term effects on adolescent functioning more so than maternal negative affect.

The Moderating Role of Mother-Adolescent Affect Congruency

Contrary to our hypotheses, we found limited robust evidence for the moderating role of mother-adolescent positive and negative affect congruency. High-congruent and low-congruent adolescents only consistently differed on their psychopathological symptoms in the context of high maternal affect inertia. The unexpected finding that adolescents whose daily affect was more congruent with their mother's daily affect fared *better* than low-congruent adolescents when their mothers showed higher levels of affect inertia (and, less consistently, when maternal affect variability was lower) suggests a *buffering* effect of high mother-adolescent congruency on adolescent psychopathological symptoms, particularly when the emotional environment provided by mothers is highly inert or invariable. Being familiar with maternal affect patterns might help adolescents to predict these patterns and feel emotionally secure (Eisenberg et al., 1998). Being connected and congruent with their mothers may then help adolescents to better apprehend their mothers' lack of changes in affect, and thus be less affected by them. Alternatively, it is possible that if mothers' and adolescents' affect is more congruent, inert maternal affect reflects less turbulent maternal emotions, or to some extent less emotionally turbulent mother-adolescent interactions, which contribute to adolescents' overall positive adjustment (e.g., Branje et al., 2010).

The importance of mother-adolescent congruency in the context of maternal affect inertia and variability depended on the spectrum of maternal affect that was moderated. If both higher levels of affect inertia and higher levels of affect variability are considered maladaptive for well-being and psychopathology (Houben et al., 2015), *lower* inertia, or variability, on the other end of the spectrum might reflect a more adaptive environment. Considering this distinction, the findings in the context of higher maternal affect inertia are in line with diathesis-stress theory (Monroe & Simons, 1991; Pluess, 2015), which proposes that vulnerable adolescents are at increased risk for problematic outcomes in maladaptive environments. In contrast, the findings in the context of lower maternal affect variability are in line with vantage sensitivity (Pluess, 2015; Pluess & Belsky, 2013), which suggests that adolescents respond differentially to adaptive environments. However, while inertia and variability are distinct affective constructs as reflected in the results of the main analyses, they are also mathematically and empirically associated (e.g., Koval et al., 2013). The patterns of the moderation findings could thus also suggest that maternal affect inertia and variability reflect opposite ends of a similar concept. Alternatively, it is possible that neither affect inertia nor

affect variability are adequate measures to cover the whole range from adaptive to maladaptive environments. Particularly in nonclinical samples, in which mothers are not likely to show extreme levels of inertia or variability, lower or higher inertia and variability might not necessarily reflect more positive or negative environments.

That no consistent moderation effects were found in the context of maternal affect intensity is in line with other studies highlighting the prominent role of affect levels in psychopathology. It is possible that the intensity of mothers' positive and negative affect poses a particularly salient risk for youth psychopathology that influences adolescents irrespective of whether they display similar affect as their mothers. At the same time, being congruent with mothers' affect was as 'main effect' consistently associated with lower internalizing and externalizing symptoms at all ages. Previous research suggests that similarity between close relationship partners promotes understanding and social cohesion (Anderson et al., 2004; Bruder et al., 2012), and that a good fit between the maternal and adolescent characteristics promotes psychosocial adjustment in general (Thomas & Chess, 1977; Lerner et al., 1986). For example, synchrony between mothers' and children' positive affect predicted reduced feelings of sadness, for children of mothers with and without a history of depression (Kudinova et al., 2019). More congruent mother-adolescent dyads might thus have more positive mother-adolescent relationships than less congruent dyads, which contributes to adolescent positive adjustment (e.g., Branje et al., 2010; McLeod, Weisz, et al., 2007; McLeod, Wood, et al., 2007). Rather than a sensitivity marker to the environment, mother-adolescent affect congruency seemed to function as a protective factor in adolescent psychological adjustment, irrespective of the emotional climate in the family.

Limitations and Future Directions

This is the first study to examine how maternal day-to-day affect dynamics and their interactions with adolescent affect across time contribute to internalizing and externalizing symptoms in adolescence and young adulthood. Our unique design predicting outcomes on the macro-level from daily affect on the micro-level provided valuable insights into the daily processes that shape adolescent adjustment and explain differences in intraindividual trajectories to psychopathological symptoms. Despite these strengths, this study is not without limitations. First, as we focused on day-to-day affect associations, we could not investigate context-dependent maternal affect in different situations. Therefore, it is not clear whether maladaptive maternal affect dynamics indeed reflect difficulties to flexibly respond to environmental demands or whether mothers just did not encounter situations that would elicit different emotions. Future studies might record several affect assessments throughout the day in relation to the specific situations in which they occur to identify adaptive or maladaptive responses and their effects on adolescent adjustment. Furthermore, it is

possible that interactions between different aspects of affect drive individual risk trajectories in adolescent adjustment. For example, one study found that for individuals with high negative affect intensity, high levels of variability seem to be adaptive (Jenkins et al., 2019). Future studies should further investigate which maternal affect profiles best contribute to positive youth outcomes.

Second, instead of using a correlation between mother and adolescent affect as we had originally planned to, we measured mother-adolescent affect congruency as a direct effect from maternal affect to adolescent affect on the same day. Although this resulted in a deviation from our preregistration, the direct effect best reflects how sensitive adolescents are to their mothers' affect within the realm of possibilities in the DSEM model. However, mothers and adolescents are likely to influence and coregulate each other's emotions (e.g., Saxbe & Repetti, 2010). While mothers have been found to coregulate adolescents' negative affect towards positivity (e.g., Van Bommel et al., 2019), reinforcing each other's negative mood could accumulate negativity and ultimately psychopathological symptoms in these families. Future studies should investigate how parents and children coregulate each other's affective behaviors across time and contexts and how this drives the development and course of adolescent psychopathological symptoms.

Finally, our sample included mainly well-functioning mothers who reported relatively high levels of positive affect and low levels of negative affect across days. High negative affect inertia does therefore not necessarily indicate prolonged negative emotions, but could also indicate a prolonged absence of negative emotions. Although low negative affect inertia could be maladaptive, particularly in situations that warrant negative emotions, high negative affect is expected to have the most adverse effects on maternal interaction behaviors. Affect patterns involving high negative emotions may thus be most strongly associated with adolescent adjustment. Future research is necessary to investigate whether our conclusions, particularly with regard to maternal affect inertia, generalize to populations who exhibit more daily negative affect, such as mothers (and adolescents) with clinically relevant levels of psychopathology or who experience multiple stressors in daily life. Relatedly, this study included mother-adolescent dyads only, due to the prominent role of maternal influence in adolescent development. The emotional climate of the family, however, also includes other nuclear family members such as additional caregivers or siblings. As paternal behaviors can moderate the effects maternal depression and behaviors on child functioning (e.g., McKee et al., 2007; Vakrat et al., 2018), it is possible that different family members' affect exacerbate or counter the effects of maternal affect on adolescent adjustment.

CONCLUSION

The results of this study provide novel insights into the affective processes that contribute to adolescent adjustment. How maternal affect unfolds at a daily level plays a role in adolescent, and to some extent young adult adjustment, highlighting the importance of short-term processes in shaping long-term development. While a recent study found that affect dynamics contribute little to the prediction of psychopathological symptoms above and beyond mean levels of positive and negative affect (Dejonckherre et al., 2020), our results suggest that maternal affect dynamics might be an important contributing factor to externalizing behaviors in youth. High mother-adolescent affect congruency, in contrast, reflects resilience, also against maladaptive maternal affect dynamics. Adopting similar affective patterns as mothers might serve adaptive functions by fostering understanding and empathic connections between mothers and adolescents.

Table S1. Results and correlations for all random effects from the DSEM models

	Unstandardized estimates				Correlations (standardized covariance estimates) between-level		
			95% CI				
	M	*SD*	Lower 2.5%	Upper 2.5%	μM	ϕM	log(σ^2)
Positive affect							
Maternal affect intensity μM	6.81***	0.06	6.71	6.92			
Maternal affect inertia ϕM	0.34***	0.01	0.32	0.36	−0.02		
Maternal affect variability log(σ^2)	0.11*	0.05	0.02	0.20	−0.46***	0.04	
Mother-adolescent affect congruency β_{MA}	0.10***	0.01	0.08	0.11	0.04	−0.07	−0.30**
Negative affect							
Maternal affect intensity μM	2.05***	0.04	1.96	2.13			
Maternal affect inertia ϕM	0.35***	0.01	0.32	0.37	0.35***		
Maternal affect variability log(σ^2)	−0.86***	0.06	−0.99	−0.75	0.60***	0.20**	
Mother-adolescent affect congruency β_{MA}	0.11***	0.01	0.10	0.13	−0.18*	−0.16*	−0.19*

Note. Credibility intervals (CI) that do not include zero indicate statistical significance.
*** $p < .001$ ** $p < .01$ * $p < .05$

Table S2A. Correlations between all study variables, involving maternal positive affect (PA)

	M (SD)	1	2	3	4	5	6	7	8
1 PA intensity	6.81 (1.13)								
2 PA inertia	0.10 (0.07)	.067							
3 PA variability	0.11 (0.94)	−.481***	−.438***						
4 Internalizing (age 14-18)	0.00 (0.83)	−.274***	.111*	.097*					
5 Internalizing (age 20)	-0.01 (0.92)	−.245***	.050	.051	.779***				
6 Internalizing (age 27)	0.00 (0.95)	−.138*	.049	.085	.621***	.605***			
7 Externalizing (age 14-18)	0.33 (0.22)	−.228***	.089	.113*	.349***	.303***	.226***		
8 Externalizing (age 20)	0.22 (0.22)	−.230***	.033	.109*	.361***	.473***	.283***	.610***	
9 Externalizing (age 27)	0.18 (0.19)	−.099	.087	.099	.294***	.304***	.538***	.474***	.430***

*** $p < .001$ ** $p < .01$ * $p < .05$

Table S2B. Correlations between all study variables, involving maternal negative affect (NA)

	M (SD)	1	2	3	4	5	6	7	8
1 NA intensity	2.05 (0.88)								
2 NA inertia	0.11 (0.07)	–.254**							
3 NA variability	–0.86 (1.27)	.616**	–.264**						
4 Internalizing (age 14-18)	0.00 (0.83)	.212**	.165**	.136**					
5 Internalizing (age 20)	-0.01 (0.92)	.209**	.060	.152**	.779**				
6 Internalizing (age 27)	0.00 (0.95)	.138*	.084	.090	.621**	.605**			
7 Externalizing (age 14-18)	0.33 (0.22)	.179**	.133**	.120**	.349**	.303**	.226**		
8 Externalizing (age 20)	0.22 (0.22)	.190**	.032	.206**	.361**	.473**	.283**	.610**	
9 Externalizing (age 27)	0.18 (0.19)	.121*	.057	.128*	.294**	.304**	.538**	.474**	.430**

*** $p < .001$ ** $p < .01$ * $p < .05$

Table S3. Results of all main analyses without moderators

	Adolescence (age 14-18)				Transitional period (age 20)				Young adulthood (age 27)			
	Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms	
Positive affect	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	B	*p*
Sex	.33	<.001	-.09	.052	.29	<.001	-.02	.766	.21	<.001	-.05	.398
Intensity	-.27	<.001	-.21	<.001	-.25	<.001	-.22	.001	-.11	.044	-.05	.444
Inertia	.08	.042	.14	.012	-.01	.824	.07	.282	.06	.268	.16	.004
Variability	.03	.652	.07	.300	-.04	.498	.04	.611	.09	.134	.15	.014
Negative affect												
Sex	.32	<.001	-.10	.017	.28	<.001	-.02	.688	.20	<.001	-.05	.395
Intensity	.20	<.001	.20	.002	.17	.028	.12	.208	.14	.033	.10	.131
Inertia	.17	<.001	.22	<.001	.08	.114	.11	.088	.10	.064	.11	.044
Variability	.06	.341	.06	.390	.08	.300	.16	.079	.05	.373	.10	.071

Note. All associations were corrected for multiple comparisons using FDR, $p_{adj} < .021$ for positive affect and $p_{adj} < .015$ for negative affect.

Table S4. Sensitivity analyses excluding outliers

	Adolescence (age 14-18)				Transitional period (age 20)				Young adulthood (age 27)			
	Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms	
Main effects	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*
Positive affect												
Sex	.33	<.001	-.09	.051	.29	<.001	-.04	.473	.20	<.001	-.09	.106
Intensity	-.25	<.001	-.18	.002	-.21	<.001	-.17	.015	-.06	.322	-.02	.809
Inertia	.07	.087	.14	.009	-.02	.695	.02	.682	.05	.352	.14	.012
Variability	.03	.558	.11	.047	.01	.828	.12	.085	.13	.038	.19	.002
Negative affect												
Sex	.32	<.001	-.11	.016	.28	<.001	-.05	.288	.21	<.001	-.07	.167
Intensity	.20	<.001	.16	.011	.13	.059	.03	.702	.17	.019	.09	.141
Inertia	.17	<.001	.22	<.001	.06	.274	.06	.297	.11	.046	.09	.094
Variability	.08	.208	.11	.072	.12	.079	.24	.001	.02	.753	.23	.203
Interaction effects												
Positive affect												
Sex	.31	<.001	-.11	.013	.27	<.001	-.06	.238	.17	.002	-.12	.022
Intensity	-.26	<.001	-.19	.001	-.21	<.001	-.17	.012	-.06	.329	-.01	.840
Inertia	.31	<.001	.35	<.001	.19	.017	.17	.087	.28	.001	.34	<.001
Variability	.13	.012	.20	.001	.10	.088	.21	.004	.25	<.001	.30	<.001
Congruency	-.31	<.001	-.28	<.001	-.24	.001	-.19	.034	-.28	<.001	-.23	.004
Intensity x congruency	**.07**	**.070**	.01	.792	-.01	.801	.04	.469	-.00	.964	-.02	.777
Inertia x congruency	**-.09**	**.026**	-.06	.283	-.07	.123	-.13	.009	-.16	<.001	-.16	<.001
Variability x congruency	.04	.407	.08	.174	-.02	.642	.06	.245	-.01	.932	-.04	.532

Table S4. Continued.

	Adolescence (age 14-18)				Transitional period (age 20)				Young adulthood (age 27)			
	Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms	
	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*
Negative affect												
Sex	.28	<.001	-.14	.001	.26	<.001	-.07	.177	.17	.001	-.11	.046
Intensity	.18	.001	.14	.017	.11	.116	.02	.780	.13	.065	.09	.218
Inertia	.32	<.001	.36	<.001	.18	.002	.11	.090	.27	<.001	.21	.002
Variability	.21	<.001	.23	<.001	.23	.001	.30	<.001	.15	.030	.21	.003
Congruency	-.27	<.001	-.23	<.001	-.20	<.001	-.10	.177	-.24	<.001	-.19	.004
Intensity x congruency	**-.07**	**.128**	-.03	.604	-.03	.606	.05	.545	-.09	.127	-.00	.949
Inertia x congruency	-.10	.007	-.12	.011	-.10	.035	-.07	.270	-.09	.042	-.13	.019
Variability x congruency	.03	.551	-.04	.425	-.00	.959	-.01	.905	.03	.618	.02	.672

Note. All associations are FDR-corrected, $p_{adj} < .021$ and $p_{adj} < .015$ for positive and negative affect in the main models, and $p_{adj} < .030$ and $p_{adj} < .028$ in the interaction models. Estimates in bold indicate differences in conclusions from the main analyses.

Table S5. Results of main analyses including mother-adolescent congruency as predictor

	Adolescence (age 14-18)				Transitional period (age 20)				Young adulthood (age 27)			
	Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms	
Positive affect	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	B	*p*
Sex	.31	<.001	-.11	.015	.28	<.001	-.02	.550	.19	<.001	-.07	.185
Intensity	-.27	<.001	-.21	<.001	-.24	<.001	-.21	.001	-.10	.062	-.04	.544
Inertia	.34	<.001	.37	<.001	.22	.003	.25	.020	.28	.001	.38	<.001
Variability	.15	.006	.18	.006	.08	.222	.13	.137	.21	.002	.28	<.001
Congruency	-.30	<.001	-.28	<.001	-.27	<.001	-.21	.017	-.25	<.001	-.25	<.001
Negative affect												
Sex	.29	<.001	-.13	.002	.27	<.001	-.03	.542	.17	.001	-.07	.189
Intensity	.20	<.001	.19	.002	.16	.045	.11	.251	.12	.052	.09	.189
Inertia	.34	<.001	.37	<.001	.20	.001	.18	.010	.26	<.001	.25	<.001
Variability	.17	.004	.16	.015	.16	.043	.22	.030	.15	.008	.19	.001
Congruency	-.29	<.001	-.27	<.001	-.20	<.001	-.13	.060	-.25	<.001	-.22	<.001

Note. All associations were corrected for multiple comparisons using FDR, $p_{adj} < .021$ for positive affect and $p_{adj} < .015$ for negative affect.

Table S6. Regions of significance (RoS) with respect to the moderator and predictors

	RoS on mother-adolescent congruency		RoS on maternal affect	
Moderated associations	lower bound (SDs)	upper bound (SDs)	lower bound (SDs)	upper bound (SDs)
PA intensity → adolescent internalizing	< 1.344	> 18.721	< 1.772	> 22.016
PA inertia → age 20 externalizing	< 0.078	> 16.760	< –14.414	> –0.311
PA inertia → age 27 internalizing	< 0.701	> 6.340	< –4.858	> –1.036
PA inertia → age 27 externalizing	< 1.368	> 5.591	< –3.825	> –0.879
NA intensity → adolescent internalizing	< 0.569	> 7.331	< –11.828	> –1.432
NA inertia → age 14-18 internalizing	< 2.394	> 8.350	< –7.745	> –2.165
NA inertia → age 14-18 externalizing	< 2.010	> 6.834	< –4.824	> –1.387
NA inertia → age 27 externalizing	< 1.072	> 9.898	< –10.578	> –0.925

Note. All values outside the denoted regions are significant; PA = positive affect, NA = negative affect.

Table S7. Sensitivity analyses for all interactions separately

	Adolescence (age 14-18)				Transitional period (age 20)				Young adulthood (age 27)			
	Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms		Internalizing symptoms		Externalizing symptoms	
Model	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*
Positive affect												
Intensity	-.28	<.001	-.23	<.001	-.24	<.001	-.23	<.001	-.15	.004	-.10	.060
Intensity x congruency	**.06**	**.162**	.00	.999	-.02	.687	-.02	.755	-.03	.447	-.03	.535
Inertia	.10	.107	.13	.051	.08	.270	.05	.611	.04	.479	.11	.120
Inertia x congruency	**-.06**	**.015**	-.05	.136	-.03	.306	-.08	.023	-.09	.010	-.08	.014
Variability	.16	.001	.16	<.001	.11	.037	.19	.001	.18	.002	.16	.004
Variability x congruency	.12	.057	**.16**	**.012**	.08	.214	**.19**	**.006**	**.13**	**.013**	.11	.054
Negative affect												
Intensity	.20	<.001	.18	<.001	.20	<.001	.19	<.001	.14	.009	.15	.012
Intensity x congruency	**.-00**	**.964**	.02	.784	.00	.955	.04	.572	-.00	.982	.11	.120
Inertia	.17	<.001	.21	<.001	.06	.287	.03	.675	.12	.043	.09	.151
Inertia x congruency	**-.04**	**.068**	-.07	.007	-.01	.805	-.03	.446	-.01	.682	-.07	.006
Variability	.20	<.001	.17	.001	.21	<.001	.27	<.001	.15	.007	.20	<.001
Variability x congruency	**.12**	**.026**	.11	.071	.08	.226	.14	.093	.05	.380	**.16**	**.016**

Note. All associations are corrected for multiple comparisons using FDR, $p_{adj} < .030$ for positive affect and $p_{adj} < .028$ for negative affect in the interaction models. Estimates in bold indicate differences in conclusions from the main analyses.

Table S8. Regions of significance (RoS) with respect to the moderator and predictors for additional significant interactions in post-hoc exploratory analyses, separately for all aspects of affect

	RoS on mother-adolescent congruency		RoS on maternal affect	
Moderated associations	lower bound (SDs)	upper bound (SDs)	lower bound (SDs)	upper bound (SDs)
PA inertia → age 14-18 internalizing	< −0.351	> 10.089	< −10.052	> 0.090
PA variability → age 14-18 externalizing	< −8.293	> 0.987	< −0.560	> 2.178
PA variability → age 20 externalizing	< −3.623	> −0.369	< −0.191	> 1.431
PA variability → age 27 internalizing	< −4.292	> −0.680	< 0.120	> 3.801
NA variability → age 14-18 internalizing	< −13.740	> −0.658	< 0.641	> 10.077
NA variability → age 27 externalizing	< −5.472	> −0.642	< 0.326	> 4.173

Note. All values outside the denoted regions are significant; PA = positive affect, NA = negative affect; only interactions are presented that indicate differences in conclusions from the main analyses.

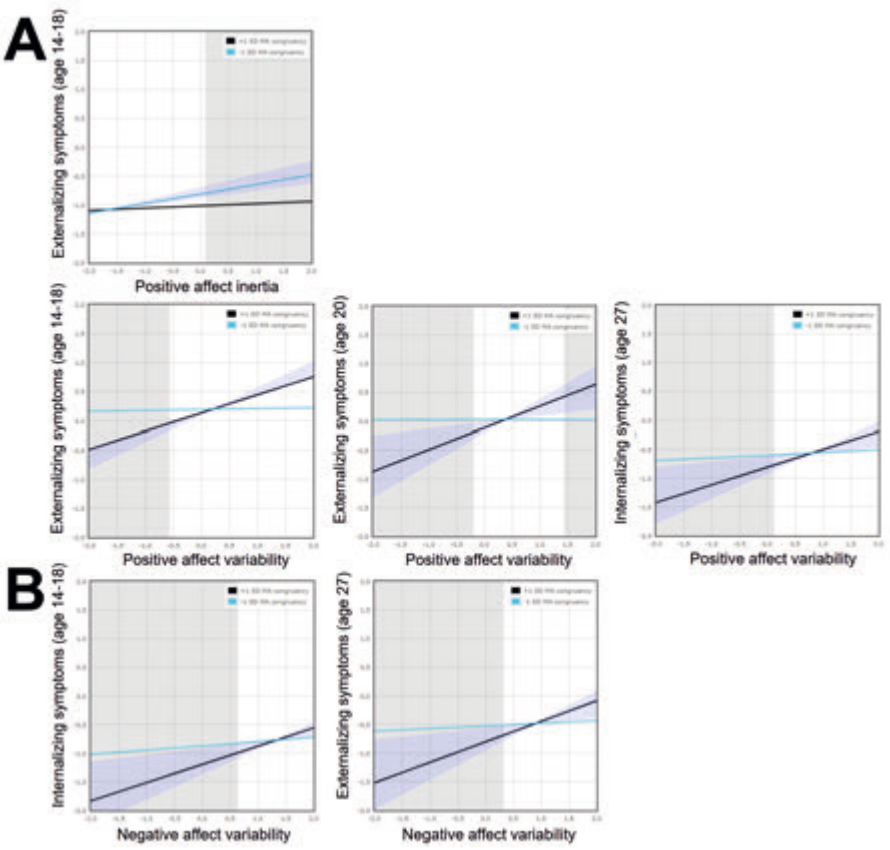

Figure S1. Additional significant interactions of positive (A) and negative aspects of affect (B) with mother-adolescent (MA) affect congruency from exploratory sensitivity analyses examining each aspect of affect separately; shaded areas depict regions of significance.

CHAPTER 4

Systematically Defined Informative Priors in Bayesian Estimation: An Empirical Application on the Transmission of Internalizing Symptoms through Mother-Adolescent Interaction Behavior

This chapter is published as:

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ABSTRACT

Background: Bayesian estimation with informative priors permits updating previous findings with new data, thus generating cumulative knowledge. To reduce subjectivity in the process, the present study emphasizes how to systematically weigh and specify informative priors and highlights the use of different aggregation methods using an empirical example that examined whether observed mother-adolescent positive and negative interaction behavior mediate the associations between maternal and adolescent internalizing symptoms across early to mid-adolescence in a 3-year longitudinal multi-method design. **Methods:** The sample consisted of 102 mother-adolescent dyads (39.2% girls, M_{age} T1 = 13.0). Mothers and adolescents reported on their internalizing symptoms and their interaction behaviors were observed during a conflict task. We systematically searched for previous studies and used an expert-informed weighting system to weigh the relevance of the previous results. Subsequently, we aggregated the (power) priors using three methods: linear pooling, logarithmic pooling, and fitting a normal distribution to the linear pool by means of maximum likelihood estimation. We compared the impact of the three differently specified informative priors and default priors on the prior predictive distribution, shrinkage, and the posterior estimates. **Results:** The prior predictive distributions for the three informative priors were quite similar and centered around the observed data mean. The shrinkage results showed that the logarithmic pooled priors were least affected by the data. Most posterior estimates were similar across the different priors. Some previous studies contained extremely specific information, resulting in bimodal posterior distributions for the analyses with linear pooled prior distributions. The posteriors following the fitted normal priors and default priors were very similar. Overall, we found that maternal, but not adolescent, internalizing symptoms predicted subsequent mother-adolescent interaction behavior, whereas negative interaction behavior seemed to predict subsequent internalizing symptoms. Evidence regarding mediation effects remained limited. **Conclusion**: A systematic search for previous information and an expert-built weighting system contribute to a clear specification of power priors. How information from multiple previous studies should be included in the prior depends on theoretical considerations (e.g., the prior is an updated Bayesian distribution), and may also be affected by pragmatic consequences of the previous results at hand (e.g., extremely specific previous results).

Keywords: intergenerational transmission, internalizing psychopathology, mother-adolescent interaction, informative priors, power priors, Bayesian estimation, longitudinal mediation analysis

Author contributions

SuS conceptualized the study, which was further refined by SN, AO, SB, and WM. SuS drafted the manuscript, MZ drafted the statistical sections. MZ and DV verified the analytical methods and performed the statistical analyses. All authors discussed the results and critically revised the manuscript.

SYSTEMATICALLY DEFINED INFORMATIVE PRIORS IN BAYESIAN ESTIMATION: AN EMPIRICAL APPLICATION ON THE TRANSMISSION OF INTERNALIZING SYMPTOMS THROUGH MOTHER-ADOLESCENT INTERACTION BEHAVIOR

New studies and analyses in social sciences are theoretically and empirically grounded in previous knowledge that has often accumulated in decades of research. While there is overall agreement that this process is essential to generate strong hypotheses, findings from previous studies are rarely integrated into new analyses. Accounting for such previous findings in subsequent analyses by means of informative priors in Bayesian estimation allows to draw more precise conclusions and obtain insight into the relation between previous knowledge and the current data.

Bayesian estimation with informative priors increases the precision of the posterior distributions by updating previous information with new data and thus gradually accumulating knowledge. While the frequentist approach regards parameters of interests as unknown, but assumes that there is only one true parameter value in the population, the Bayesian approach regards parameters of interest as uncertain and describes them with a probability distribution (van de Schoot et al., 2014). By combining previous information with new data from the analyses, Bayesian estimation allows researchers to make assumptions about model parameters, such as curtailing or excluding certain parameter values (Zondervan-Zwijnenburg, Peeters, Depaoli, & Van de Schoot, 2017). To date, most empirical studies rely on diffuse or naive prior distributions, such as default software settings, that do not account for the available previous knowledge (e.g. van de Schoot, Winter, Ryan, Zondervan-Zwijnenburg, & Depaoli, 2017). Simulation studies and mathematical demonstrations indicated that using informative priors that are derived from previous studies, meta-analyses, or experts, outperformed frequentist approaches and approaches using diffuse priors in terms of decreased relative bias, improved estimation accuracy (e.g., decreased RMSE values), and increased power when samples were too small for complex analyses (Smid, McNeish, Miočević, & van de Schoot, 2019; Zitzmann, Lüdtke, Robitzsch, & Hecht, 2020). However, if informative priors are not chosen carefully or are weakly defined, Bayesian estimation methods may perform poorly and result in biased estimates (Depaoli, 2013; Holtmann, Koch, Lochner, & Eid, 2016). Therefore, a systematic and

transparent approach is essential when specifying informative priors (van de Schoot et al., 2021; Zondervan-Zwijnenburg et al., 2017). The present study highlights the use of different approaches to systematically define informative priors and the integration of new data to answer novel research questions.

Weighting Previous Studies

If previous designs are not consistent with the new study, for example due to different populations or different assessments, this can raise potential bias and inflated type I errors (Hobbs, Carlin, Mandrekar, & Sargent, 2011; Viele et al., 2014). Previous findings should therefore strongly inform the posterior distributions when they are based on designs that are comparable to the present study, and weakly when they are not. To ensure that previous findings do not outweigh the current data and dominate the posterior distributions, power priors that downweigh previous data by determining the amount of relevant information have been recommended (Ibrahim & Chen, 2000). Specifically, a power prior takes the likelihood of the information from the previous study to the power δ, where δ is a value between 0 (ignore the previous data completely) and 1 (treat the data as equal to the current data and fully include the evidence). For normal distributions, when delta $\delta \neq 0$, raising the likelihood to the power δ is equal to dividing the variance from the previous study by δ and using it as the prior variance σ_0^2. Traditionally, power priors include the use of unknown weights, which have been criticized to over-attenuate the influence of previous data (Neelon & O'Malley, 2010) as they do not capture the extent to which previous findings are applicable to the present design and data.

Previous studies can be more or less similar to a specific study's design and thus provide stronger or weaker input for priors than other studies. Meta-analyses, for example, quantify existing information, and thus provide accumulated, more robust evidence than single studies. However, they also include a wide range of different methodological designs, such as different participant age ranges or assessment methods, and thus cannot provide strong input for specific parameter estimates. Empirical studies that closely reflect the research questions and design of the new study that is to be conducted provide the strongest input for informative priors, but are more susceptible to potential estimation errors, biases, or chance findings than meta-analyses. How much weight a particular study receives, should therefore depend on a range of aspects that correspond to the study's design at hand. Longitudinal studies, for example, involve different considerations than cross-sectional studies, such as temporal ordering and the lengths of intervals between time points. If the study at hand employs a longitudinal design, findings from studies with repeated measurements would receive more weight than studies that solely include measurements at the same time point. Only a previous study with data that can be considered exchangeable with the new data should

receive a weight of 1. To determine how much an individual study deviates from the new data, we therefore propose to determine each study's individual weight for the construction of power priors. Studies with lower relevance obtain lower scores for δ, which means that their variance will be inflated. The larger the variance (i.e., uncertainty), the smaller the impact of a previous study on the posterior distribution.

A carefully constructed and justified weighting scheme that is tailored to the specific research question is essential when specifying informative priors. To avoid arbitrary and subjective decisions, expert knowledge can inform this process (Bolsinova, Hoijtink, Vermeulen, & Béguin, 2017; van de Schoot et al., 2018; Veen, Egberts, van Loey, & van de Schoot, 2020). Expert knowledge as input for prior distributions has been previously used to estimate the size of parameters for which no data was available (e.g., Hald et al., 2016) or to complement existing data (e.g., van de Schoot et al., 2018). A systematic approach to collecting and specifying informative priors in its strictest form therefore resembles, but exceeds that of a meta-analysis. It includes quantifying and weighing previous information, systematically collecting and justifying all decisions, visualizing informative priors, and conducting sensitivity analyses to compare the impact of different priors on the posterior estimates (Zondervan-Zwijnenburg et al., 2017). This can be beneficial beyond a pure meta-analytical approach that solely quantifies previous information. As such, Bayesian estimation with informative priors allows researchers to update previous information by combining it with new data.This cumulative process gradually decreases the uncertainty of parameter estimates (König & van de Schoot, 2018). In the current study, we used expert knowledge to define inclusion criteria and create an appropriate weighting scheme for all included previous studies.

Aggregating Previous Studies

If multiple studies contain information on one parameter, the previous information needs to be aggregated into one distribution. Three aggregation methods are: 1) linear pooling, 2) logarithmic pooling, and 3) a normal distribution fitted to the linear pool.

Linear Pooling. The linear pool of distributions sums the densities provided by the different studies, resulting in a mixture prior (Genest & Zidek, 1986). The linear pool directly represents the previous studies by combining them without any modifications to the initial information. One way to obtain the linear pool is to run multiple Bayesian analyses: one for each prior specification. Subsequently, the posterior samples can be aggregated (see Zondervan-Zwijnenburg, Van de Schoot-Hubeek, Lek, Hoijtink, & Van de Schoot, 2017). This method can be applied in any software package that allows for Bayesian estimation with customizable prior specifications. However, as parameter estimates within a model are not independent, this method becomes impractical in

a model in which multiple parameters have multiple sources of previous information. In more advanced Bayesian software such as Stan (Stan Development Team, 2011-2019), the linear pool of previous studies can be programmed at once. A difficulty that remains is that a linear pool becomes multimodal when the different prior likelihoods diverge. Multimodality is complex for estimation and interpretation. It may cause non-convergence, and it can be odd to consider, for example, .2 and .5 equally plausible values, but .35 a value with low probability. There is the possibility that this scenario occurs when local maxima have previously been found.

Logarithmic Pooling. Whereas the linear pool sums distributions, the logarithmic (a.k.a. geometric) pool multiplies them. In practice this means that extreme modes originating from only one study can be compensated by their multiplication with other studies that allocate less probability to this area. In this manner, the logarithmic pool emphasizes the common range of parameter values. Logarithmic pools are typically unimodal and less dispersed than linear pools (Genest & Zidek, 1986). The logarithmic pool can also be considered a Bayesian updating procedure, in which the first[2] study is the initial prior. A potential disadvantage of the logarithmic pool, however, is that if one previous study places near-zero probability to a range of values, the multiplication by near-zero probability will predominate in the pooled distribution. De Carvalho, Villela, Coelho & Bastos (2020) define pooled distribution and their parameters for sets of common distributions. When the pooled distribution is a common distribution as well, the prior can be easily specified in software packages that allow for Bayesian estimation with custom prior distributions.

Normal distribution fitted to the linear pool. Another alternative to including a potentially bimodal linear pool, is to obtain the normal distribution best fitting to this pooled distribution. In this method, the previous studies are considered to be samples from an underlying normal distribution. By fitting a normal distribution to the results of the previous studies, we aim to retrieve the underlying normal distribution of the parameter. When the underlying previous studies have different means, the fitted normal distribution will have a variance larger than that of the underlying studies. Once the hyperparameters of the fitted normal distribution are obtained, the normal prior distribution can be specified in any software package that allows for Bayesian estimation with custom prior distributions.

Conducting sensitivity analyses with different priors, including diffuse default priors, allows us to compare findings and highlight the robustness of our model results if priors are modified (van de Schoot et al., 2017). The current study will compare the results of these three pooling methods and diffuse default priors on the posterior distributions in an empirical example that examined

2 Note that just as in multiplication in general, the order of updating is irrelevant for the final outcome.

whether mother-adolescent interaction behavior meditates the associations between maternal and adolescent internalizing behavior.

Empirical Application: Mother-Adolescent Interaction Behavior as Mediator in the Transmission of Internalizing Symptoms

Adolescence is a crucial period for the development of internalizing problems, such as symptoms of anxiety or depression, which increase adolescents' risk for psychopathological disorders, school dropout, and unemployment in later life (Clayborne, Varin, & Colman, 2019; Kessler et al., 2012). Maternal internalizing symptoms are among the most salient predictors of adolescent internalizing symptoms (e.g., Connell & Goodman, 2002; Goodman & Gotlib, 1999). Genetic similarities cannot fully explain these associations (Eley et al., 2015; Natsuaki et al., 2014) and specific patterns of how mothers and adolescents interact may be another mechanism through which maternal internalizing symptoms are associated with adolescent internalizing symptoms (Goodman & Gotlib, 1999). Specifically, internalizing symptoms might render mothers less sensitive to their children's needs, more emotionally unavailable, and more irritated, which can suppress mothers' expression of positive interaction behavior and increase their expression of negative, hostile and angry interaction behavior toward the adolescent (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Such diminished positive and heightened negative interaction behavior may in turn undermine the adolescents' self-esteem and emotion-regulation, make them feel helpless, and prompt negative self-evaluations, which render them more sensitive to internalizing symptoms (Garber & Flynn, 2001; Gottman, Katz, & Hooven, 1997). Hence, it is likely that maternal interaction behavior underlies the transmission of internalizing symptoms from mothers to adolescents.

Transactional theories (e.g., Sameroff, 2009) suggest that adolescents are not only influenced by their parents, but also influence their parents. Hence, associations between maternal and adolescent internalizing symptoms are likely to be bidirectional (Hughes & Gullone, 2010; Wilkinson, Harris, Kelvin, Dubicka, & Goodyer, 2013). Adolescent internalizing symptoms can disrupt interactional processes in the family (Berg-Nielsen, Vikan, & Dahl, 2002; Sheeber, Hops, & Davis, 2001) and thus similarly predict changes in mother-adolescent interaction behavior (e.g., Nelemans, Hale, Branje, Hawk, & Meeus, 2014), which in turn prompt maternal internalizing symptoms. It is thus important to include bidirectional associations between maternal and adolescent internalizing symptoms when investigating the mediating role of mother-adolescent interaction behavior. Similarly, as social interactions include two partners who continuously regulate and react to each other's behaviors (Fogel, 1993), it is essential to examine not only maternal interaction behavior toward adolescents, but also adolescents' interaction behavior toward mothers. However, most studies to date are based on the assumption that associations between maternal and adolescent internalizing symptoms

are unidirectional from mothers to adolescents and only driven by maternal interaction behavior toward adolescents. If potential effects from adolescents to mothers are ignored, alleged mediation effects may be spurious. Fully understanding the mediating role of mother-adolescent positive and negative interaction behavior in the transmission of internalizing symptoms thus requires a model that reflects reciprocal associations between mothers and adolescents. In this study, we will investigate whether mother-adolescent interaction behavior underlies the intergenerational transmission of internalizing symptoms, including associations from both mothers to adolescents and from adolescents to mothers.

Several studies have been conducted to support each pathway in the theoretically proposed mediation model (see supplemental material for a systematic and critical review of previous literature). Findings from meta-analyses on mother-child interactions indeed indicated associations of maternal interaction behavior with maternal internalizing symptoms (Lovejoy et al., 2000; McCabe, 2014) and child internalizing symptoms (McLeod, Weisz, & Wood, 2007; McLeod, Wood, & Weisz, 2007; Pinquart, 2017; Yap, Pilkington, Ryan, & Jorm, 2014). Observational, longitudinal assessments in adolescence best reflect our study's design and thus provide strong specific information. The few studies that meet these criteria, however, remain inconsistent regarding whether maternal internalizing symptoms predict both subsequent positive (Feng, Shaw, Skuban, & Lane, 2007; Simons, Lorenz, Wu, & Conger, 1993) and negative interaction behavior (Feng et al., 1997) as well as whether interaction behavior predict subsequent adolescent internalizing symptoms (Hofer et al., 2013; Milan & Carlone, 2018) or not (Feinberg, Kan, & Hetherington, 2007; Schwartz et al., 2012). Studies on reversed associations from adolescents to mothers remain scarce and the one available study found that adolescent interaction behavior did not predict maternal internalizing symptoms (Milan & Carlone, 2018).

The Present Study

This study applied a systematic approach to defining informative priors in Bayesian estimation to highlight the role of Bayesian estimation in integrating and cumulating empirical knowledge. We compared the effects of three different kinds of informative priors on the posterior distribution using an empirical illustration: Specifically, we examined whether observed mother-adolescent positive and negative interaction behavior mediate associations between maternal and adolescent internalizing symptoms, using a multi-method longitudinal design (see Figure 1). To increase the precision of our results, we systematically searched and weighed findings from previous studies, using an expert-designed weighting and scoring system, and synthesized the information into linear pool, logarithmic pool, and fitted normal prior distributions. Furthermore, we conducted sensitivity analyses to compare the impact of informative and diffuse priors on the mediating

effects of mother-adolescent interaction behavior in the transmission of internalizing symptoms. This allowed us to identify the role of different priors and the robustness of our results.

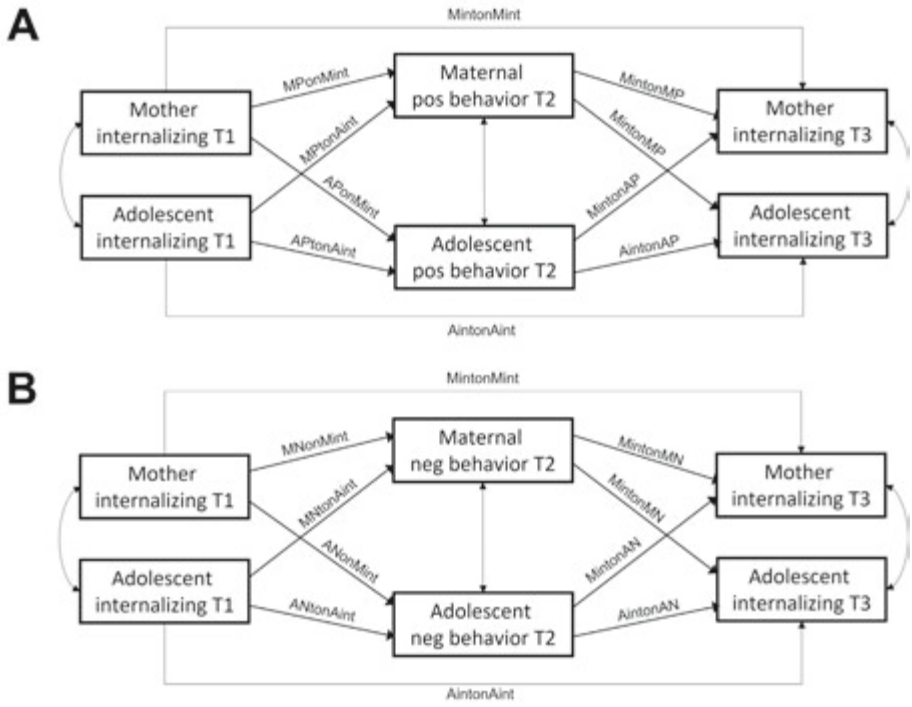


Figure 1. Conceptual SEM models examining the mediating effects of positive (model A) and negative interaction behavior (model B) in the associations between maternal and adolescent internalizing symptoms. M = maternal, A = adolescent, pos = positive, neg = negative.

MATERIALS AND METHODS

All relevant materials, documents, and syntax files are available at https://osf.io/c37mv.

Participants

The sample consisted of 102 mother-adolescent dyads (39% girls, M_{age} T_1 = 13.0, SD_{age} = .51) who were part of a larger sample of families participating in the ongoing Research on Adolescent Development And Relationships Young (RADAR-Y) study. All participants were assessed in annual home visits. Most adolescents (95%) and their mothers (91%) were of Dutch origin. They predominantly lived with both biological parents (86%) in medium to high socioeconomic status households (91%), based on parents' occupation level.

Sample attrition was low across all time points (1-7%), with 94 mother-adolescent dyads who participated at the first time point remaining in the study at the third time point. Mothers and adolescents who dropped out of the study did not significantly differ from those who remained in the study on most of the study or background measures (ANOVA *p*-values ≥ .056). However, mothers who dropped out of the study showed more negative interaction behavior at the second time point, $F(1, 87) = 4.67$, $p = .033$, than mothers who remained in the study.

Procedure

The present study used three time points from early to mid-adolescence, when adolescents were on average approximately 13, 14, and 15 years of age. Families were recruited through 230 randomly selected elementary schools in the central and western regions of the Netherlands. Of those initially selected ($N = 1,544$), families who did not fulfil the full family requirements ($n = 364$), could not be contacted or withdrew their participation ($n = 569$), or did not provide written consent for all family members ($n = 114$) were excluded. Of those 497 families who participated at the first time point, a subsample of 102 randomly selected mother-adolescent dyads participated in an interaction task.

During annual home assessments, adolescents and their mothers completed a series of questionnaires and subsequently participated in a conflict interaction task. The conflict task consisted of a 10-minute videotaped interaction between adolescents and their mothers, during which they discussed a topic of frequent disagreement, explained their individual thoughts, and presented a solution to the conflict. Prior to the task, adolescents and their mothers agreed upon a topic, chosen out of a series of suggested subjects or an own subject. The interviewer ensured that a topic was chosen, but was otherwise absent during the topic selection and the actual conflict task. Adolescents and mothers were compensated for their participation at each time point. The study procedure was approved by the Medical Research Ethics Committee of the University Medical Center Utrecht.

Measures

Adolescent internalizing symptoms. We assessed adolescent internalizing symptoms as a combined score of self-reported anxiety and depression symptoms. Anxiety symptoms were measured with the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1997), which consists of 38 items (e.g., "I get really frightened for no reason at all") on a 3-point scale (1 = almost never, 3 = often). Depression symptoms were measured with 2nd edition of the Reynolds Adolescent Depression Scale (RADS-2; Reynolds, 2000), which consists of 23 items (e.g., "I feel that no one cares about me") on a 4-point scale (1 = almost never, 4 = often). As anxiety and depression symptoms correspond to the same higher-order latent factor of internalizing symptoms within a

hierarchical structure of psychopathology (Achenbach, 1966; Lahey et al., 2017), total anxiety and depression scores were averaged after a multiple imputation procedure to form a total internalizing symptom score for each participant. The anxiety, depression, and total internalizing scales showed high internal consistency across all time points (α = .91-.96). Higher scores indicated higher levels of adolescent internalizing symptoms.

Maternal internalizing symptoms. We assessed maternal internalizing symptoms with the anxious/depressed, withdrawn, and somatic complaints syndrome scales of the Adult Self Report (ASR; Achenbach & Rescorla, 2003). The syndrome scales consist of 18 items (e.g., "I feel lonely"), 9 items (e.g., "I keep from getting involved with others"), and 12 items (e.g., "I feel tired without good reason), respectively, that are measured on a 3-point scale (0 = not true, 2 = very true or often true). The total internalizing scale showed high internal consistency across all time points (α = .90-.91). Higher scores indicated higher levels of maternal internalizing symptoms.

Maternal and adolescent interaction behavior. Rating scales were adapted from the *Family Interaction Task* coding system (Weinfield, Ogawa, & Egeland, 2002; Weinfield et al., 1999). We observed maternal and adolescent positive interaction behavior toward the other by coding verbal and nonverbal expressions/displays of maternal emotional involvement during the conflict task. Verbal expressions include showing interest, listening, responding, and understanding. Nonverbal expressions included smiling, interested attitude, nodding, maintained eye contact. We observed maternal and adolescent negative interaction behavior toward the interaction partner by coding how hostile and angry the mother or adolescent behaved during the conflict task. Maternal negative behaviors included blaming, rejecting, mocking, and exerting negative facial expressions or physical reactions. Adolescent negative behaviors include sighing and groaning, pouting, refusing to cooperate, criticizing, and exerting negative facial expressions or physical reactions.

Three independent raters coded maternal and adolescent interaction behavior toward the other on a 5-point scale (1 = low score on the relevant interaction behavior, 5 = high score on the relevant interaction behavior). All raters underwent extensive training before coding a random selection of the sample. Higher scores of positive interaction behavior indicated more common, appropriate, and consistent use of these verbal and nonverbal expressions, while higher scores of negative interaction behavior indicated higher levels of negative, hostile behaviors. Interrater agreements using interclass correlations (ICC) based on 15% of the sample showed acceptable agreement for maternal interaction behavior (ICCs = .80-.89) and adolescent interaction behavior (ICCs = .86-.87).

Prior Distributions from Previous Knowledge

For the regression paths in our models, we implemented two search strategies (see Figure 2 for a flowchart on study inclusion): a search for meta-analyses and reviews, and a search for empirical studies.

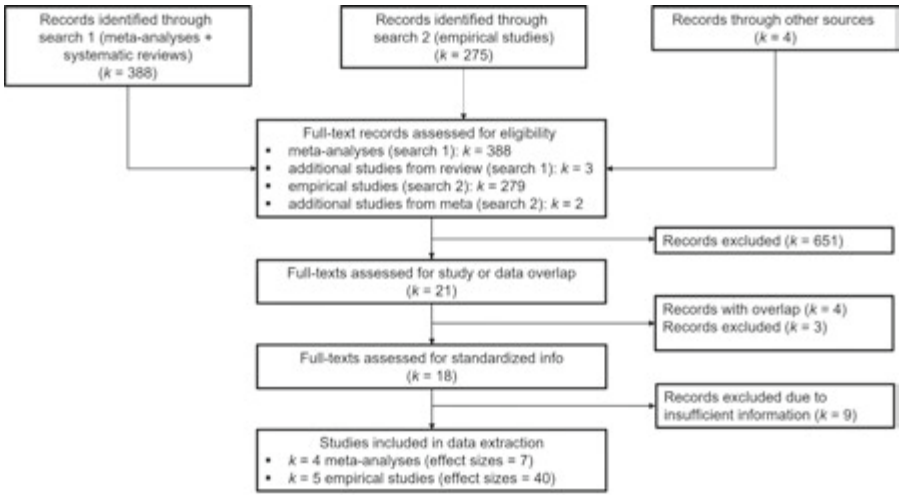


Figure 2. Flow chart for study inclusion from search 1 (meta-analyses and systematic reviews) and search 2 (empirical studies) based on the PRISMA guidelines.

Meta-analyses and systematic reviews. We conducted a literature search in Web of Science for all meta-analyses and systematic reviews published until December 2019, based on a combination of key words that reflected the target sample (child, adolescent) and their parents (parent*, maternal, mother), internalizing symptoms (anxi*, depress*, internalizing), as well as positive and negative behaviors (positive, negative, affect, warmth, hostil*, rejection) during the interaction (interaction*, relation*, parenting). Meta-analyses were selected if they a) included studies on adolescence, and b) assessed positive and/or negative interaction behavior, as defined for our sample, from mother or parent toward adolescent and/or from adolescent toward mother or parent. This search strategy identified 388 studies, of which 7 meta-analyses and 1 systematic review were included in this study. Some meta-analyses showed substantial overlap in studies. In these cases, we only included the meta-analysis that scored highest on the scoring scheme (i.e., most comparable to our design) to avoid biasing the results. This led to a final inclusion of 4 meta-analyses, of which 2 focused on the associations between maternal internalizing symptoms and mother-adolescent interaction behavior and 2 focused on the associations between mother-adolescent interaction behavior and adolescent internalizing symptoms. The systematic review that was included identified 3 additional empirical studies that were not included in the meta-analyses and mainly focused on associations that were not investigated in any meta-analysis (e.g., associations between adolescent internalizing symptoms

and adolescent interaction behavior). One of these empirical studies did not provide standardized information and was thus excluded.

Empirical studies. Our second search strategy to identify relevant studies was twofold: First, we conducted a literature search in Web of Science for all empirical studies that were not included in the meta-analyses published from January 2012[3] until March 2020 using the same search string as for the meta-analyses, but only for adolescent samples (adolescen*, youth, teen*, youngst*, student*, emerging adult*, early adult*, young adult*) and observational studies (observ*, code*, rater, tape*, task*, record*). Studies were selected if they a) included an adolescent sample, but did not include participants younger than 7 years or older than 25 years at the first measurement, b) included longitudinal estimates for the cross-lagged parameters, and c) assessed positive and/or negative interaction behavior from mother toward adolescent and/or from adolescent toward mother using observations. This search identified 275 studies, of which 11 were included (see Figure 2). Second, we searched all cross-sectional meta-analyses for studies that met the inclusion criteria and had estimates that were not included in the meta-analytic effect sizes. This resulted in an additional inclusion of 2 studies. Studies that failed to provide any or only partial standardized information were excluded (k = 8). The final inclusion yielded 47 effect sizes from 4 meta-analyses and 5 independent empirical studies (see Table S1 in the supplemental material for all included studies per parameter and model).

Power prior weighting scheme. To evaluate each previous study's contribution to our research question, we designed a scoring system that reflects each study's weight in the specification of prior distributions. Four experts on adolescent relationships and mental health (third, fifth, sixth, and seventh author) discussed and evaluated the importance of several methodological aspects, which were further quantified to represent one score (see Table 1A). For example, a longitudinal measurement most closely reflected our study design, and therefore received a higher score than a cross-sectional measurement. The final weighting scheme included ten categories: longitudinal associations, same time lag, controlling for earlier internalizing symptoms, mother-adolescent interaction behavior assessed solely observational, age range from early to mid-adolescence (12-16), included symptoms of depression and anxiety, or anxiety only, controlling for other partner's symptoms, controlling for other partner's interaction behavior, community sample, and meta-analysis. The ten categories were associated with 5-20 points depending on the importance of the criterion. Each included study received the allocated number of points per category depending on whether or not they fulfilled the criteria (see Table 1B). The final score for each study determined its associated weight, δ, in the power prior.

3 As starting year, we chose the date of the last updated search of the meta-analyses.

Table 1A. Weighting scheme for informative priors.

Category	Points	Details
T1-T2 (longitudinal)	10	The estimates of longitudinal studies are usually smaller. As our parameter are longitudinal estimates as well, I think longitudinal designs should receive most weight in relation other categories.
- controlling for symptoms at T1	20	Longitudinal studies that do not control for symptoms at T1 might still have quite large estimates and cannot indicate change. As this is the most crucial aspect of longitudinal research, studies that also control for T1 symptoms should receive more weight. *Not applicable for T1 → T2 associations (deleted from final score)!*
- Same time lag (1 year)	5	Studies that use the same time lag as we do are closer to our study design and thus deserve more weight.
Observation	15	The study list only includes empirical studies with observational assessments of the parent-adolescent interaction as these (multi-method) estimates are usually smaller than self-reports. However, the meta-analyses often include a combination of observations and self-reports, which is difficult to disentangle. Therefore, estimates from "pure" observations should receive more points than mixed studies (and most weight in relation to other categories as this is another main aspect of our study).
Early adolescence (12-16)	10	Some studies, and particularly the meta-analyses, used a broader age range than our study or even just adolescence (but all studies include adolescence). As our study focuses on early-mid adolescence, studies that included a similar age group should receive some more points.
Internalizing symptoms include both anxiety and depression, or anxiety only	10	Most studies do not focus on a combination of depression and anxiety symptoms, but only include one of those symptoms (mostly depression). As we will use a combination of both, studies that include measures on internalizing symptoms or both depression and anxiety symptoms should receive more points. *Most studies focus on mother or adolescent depression (rather than anxiety). To counterbalance that, we will also award 5 points if the study only focused on anxiety (i.e., either combined or anxiety only).*
Including covariates *- parental symptoms* *- other interaction behaviors*	5 5	If studies include other relevant covariates that might better reflect our study associations, such as parental symptoms (for T2-T3 parameters), they might receive additional points.
Community sample (does not include clinical/ diagnostic groups)	10	Many (older) studies include two subsamples, of which one is usually clinical. Therefore, the final sample includes participants who may have higher levels of internalizing symptoms than our participants. For these participants, the associations may be stronger. Thus, studies with a community sample which is closer to our sample should receive more points.
Meta-analysis	10	Meta-analyses combine information from several studies and thus provide the most comprehensive evidence. Therefore they should receive somewhat more weight.
10 categories (standard 5)	**100 (80)**	**Each study can score between 0 and 100 points (or between 0 and 80 points for T1 → T2 associations).**

Table 1B. Final scoring of all included studies.

Study	T1-T2	lag	cT1	obs	Age	$M_{dep+anx\ (or\ anx)}$	$A_{dep+anx\ (or\ anx)}$	cov_s	cov_i	comm	MA	Score
Points	*10*	*5*	*20*	*15*	*10*	*10*		*5*	*5*	*10*	*10*	*100*
Lovejoy (2000)				x							x	25
*Simons (1993)	x				x							20
McCabe (2014)				x		x					x	35
Pinquart (2017)	x		x				x			x	x	60
Weymouth (2016)							x			x	x	30
Allen (2006)	x	x	x	x	x					x		70
Asbrand (2017)	x			x			x					35
Dadds (1992)				x								15
Dietz (2008)				x		x						25
Griffith neg (2019)	x		x	x				x		x		60
Griffith pos (2019)	x		x	x						x		55
Hofer (2013)	x		x	x	x		x		x	x		80
Jackson (2011)				x								15
Milan (2018, only cs)				x				x	x	x		30
Milan (2018)	x		x	x				x	x	x		60
Nelson (2017)	x			x					x			30
Olino (2016)	x		x	x				x				50
Schwartz (2012)	x		x	x	x		x	x				70
Szwedo (2017)	x		x	x						x		55
Van Doorn (2016)				x				x	x			25

Note. T1-T2 = longitudinal assessment, lag = same time lag used (for longitudinal studies), cT1 = controlling for T1 symptoms (for longitudinal studies), obs = observational assessment of parent-adolescent interaction, age = age range early adolescence, *N* = sample size, M = maternal, A = adolescent, year = publication year, cov_s = controlling for parental symptoms, cov_i = controlling for other interaction behaviors, comm = community sample, MA = meta-analysis, x = indicates that the category is met, grey studies were excluded from the final analyses due to insufficient standardized information.
*study included in aforementioned meta-analysis

Specification of Prior Distributions

To be able to use previous information from studies with various measures, the data of the present study was standardized, and all prior distributions concerned standardized effects. Hence, only information from previous studies that presented or allowed to compute standardized effects *and* the associated standard errors was used[4]. The hyperparameters for the normally distributed prior distributions were a mean and standard deviation.

The longitudinal associations of maternal and adolescent internalizing symptoms with mother-adolescent positive (i.e., model A) and negative interaction behavior (i.e., model B) describe the main parameters in the model (see Figure 1). We did not consider the datapoints from previous studies to be exchangeable with our current dataset, nor to be a previous sample from the same population (Spiegelhalter, 2004). The previous information was thus considered less relevant than the current data, and therefore, needed to be downweighed by power priors. The power prior weights δ were systematically determined through our weighting scheme (Section 2.4.3). Studies with lower relevance obtained lower scores for δ, which means that their variance was inflated. The larger variance (i.e., uncertainty) diminishes its impact on the posterior distribution.

When multiple studies contained information on one parameter, the information needed to be aggregated into one distribution. We evaluated three methods to aggregate previous information: 1) linear pooling, 2) logarithmic pooling, and 3) a normal distribution fitted to the linear pool. Additionally, we conducted sensitivity analyses with default priors from the statistical R package brms as a reference (Bürkner, 2017). The four posterior distributions were compared and evaluated based on estimation issues and interpretability to indicate the role of previous information. The defined informative priors for all longitudinal regression parameters are provided in Table 2. For all other parameters in the model, the following low-informative prior was used: $N(0,10)$.

4 If only the standard error of the unstandardized effect was present, we multiplied that standard error with the standard deviation of the independent variable and divided by the standard deviation of the dependent variable. If a t-statistic was present, the standard error was computed by dividing the standardized effect by t. If a confidence interval for the standardized effect was provided, the difference between upper and lower limit was divided by 2 and by 1.96.

Table 2. Informative priors for the regression parameters in Model A and Model B

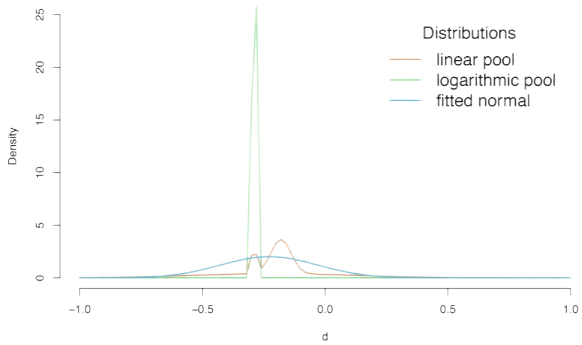
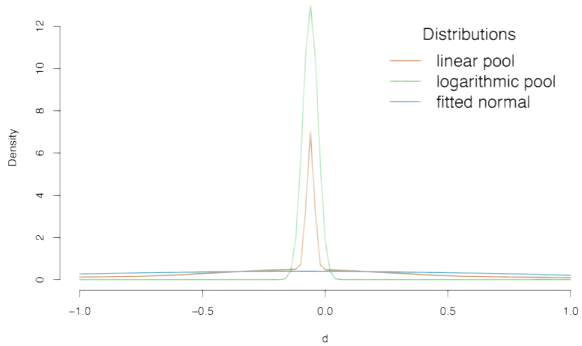
Parameter	Linear pool	Logarithmic pool	Fitted normal	Image
MPonMint	$N(-0.18,0.0179)^{0.4375}+$ $N(-0.21,0.1040)^{0.3125}+$ $N(-0.29,0.0015)^{0.3750}$	$N(-0.29,0.01)$	$N(-0.23,0.20)$	
MPonAint	$N(-0.06,0.0077)^{0.5000}+$ $N(-0.09,0.0950)^{0.3125}+$ $N(-0.12,0.1755)^{0.1875}+$ $N(-0.16,0.6407)^{0.3750}$	$N(-0.06,0.03)$	$N(-0.10,0.98)$	

Table 2. Continued.

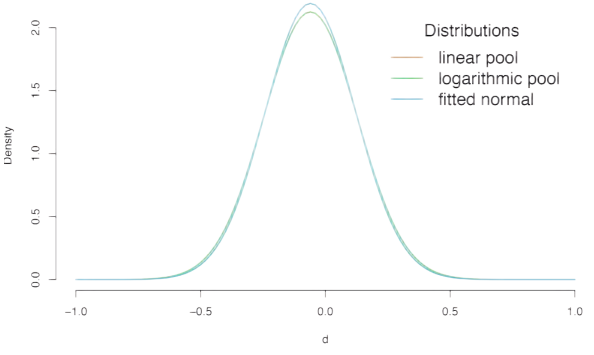
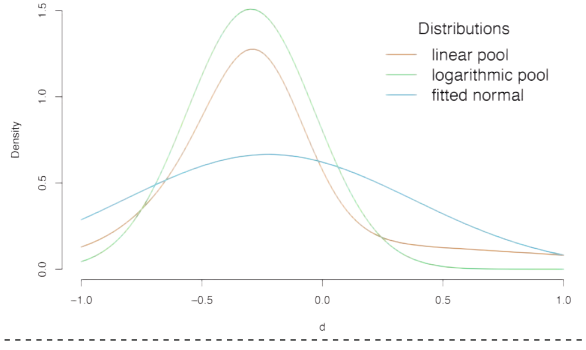
Parameter	Linear pool	Logarithmic pool	Fitted normal	Image
APonMint	$N(-0.06,0.0704)^{0.3750}$	$N(-0.06,0.19)$	$N(-0.06,0.19)$	
APonAint	$N(-0.01,0.1768)^{0.1875}+$ $N(-0.41,0.0871)^{0.3125}+$ $N(-0.26,0.0697)^{0.3750}$	$N(-0.30,0.26)$	$N(-0.22,0.61)$	

Table 2. Continued.

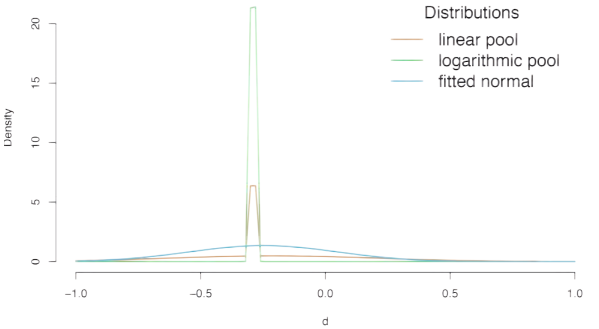
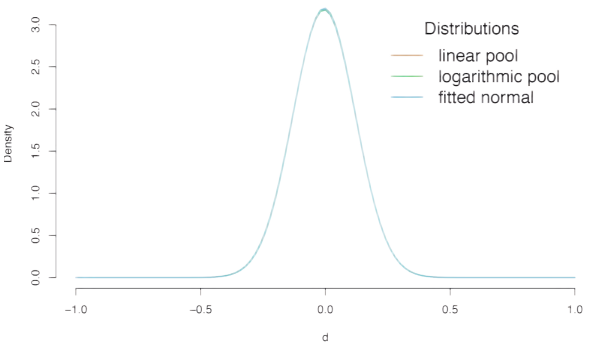
Parameter	Linear pool	Logarithmic pool	Fitted normal	Image
MintonMP	$N(-0.21,0.1040)^{0.2500}+$ $N(-0.29,0.0015)^{0.3000}$	$N(-0.29,0.01)$	$N(-0.25,0.29)$	
MintonAP	$N(-0.01,0.0753)^{0.6000}$	$N(-0.01,0.13)$	$N(-0.01,0.13)$	

Table 2. Continued.

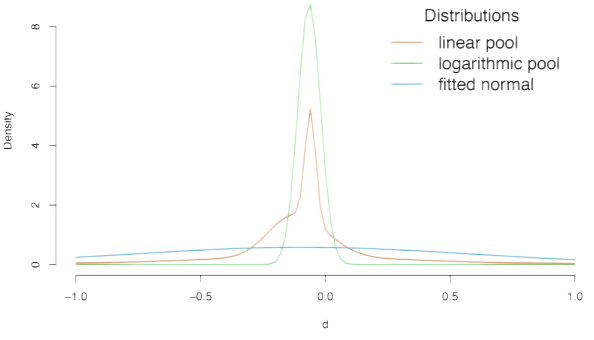
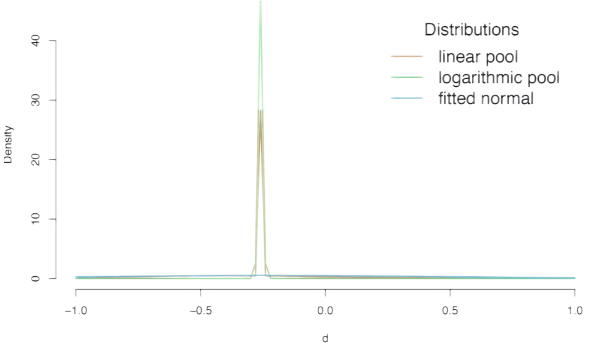
Parameter	Linear pool	Logarithmic pool	Fitted normal	Image
AintonMP	$N(-0.06,0.0128)^{0.6000}+$ $N(-0.16,0.0546)^{0.6000}+$ $N(-0.09,0.0950)^{0.2500}+$ $N(-0.12,0.2219)^{0.1500}+$ $N(-0.05,0.0578)^{0.5500}$	$N(-0.06,0.05)$	$N(-0.10,0.68)$	
AintonAP	$N(-0.01,0.1768)^{0.1500}+$ $N(-0.41,0.0871)^{0.2500}+$ $N(-0.26,0.0014)^{0.3000}$	$N(-0.26,0.01)$	$N(-0.21,0.73)$	

Table 2. Continued.

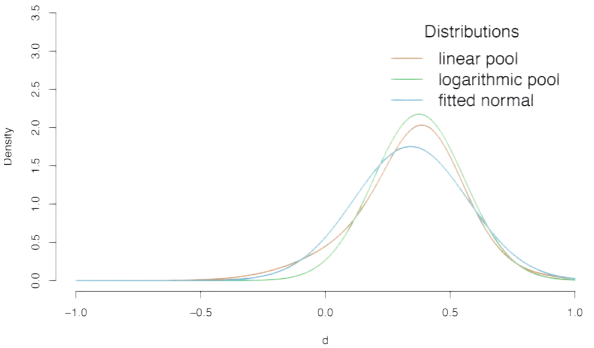
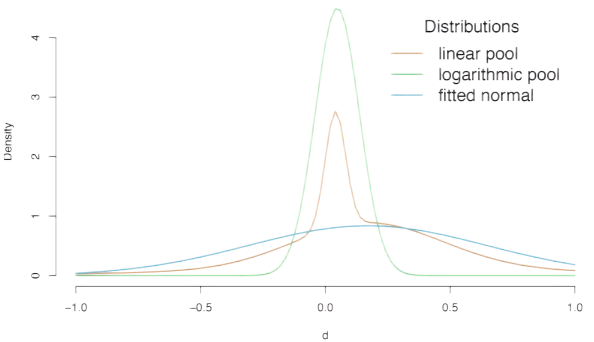
Parameter	Linear pool	Logarithmic pool	Fitted normal	Image
MNonMint	$N(0.40,0.0459)^{0.3125}+$ $N(0.29,0.1030)^{0.3750}$	$N(0.38,0.18)$	$N(0.34,0.22)$	
MNonAint	$N(0.04,0.0204)^{0.5000}+$ $N(0.10,0.0948)^{0.3125}+$ $N(0.27,0.1699)^{0.1875}+$ $N(0.16,0.1020)^{0.3750}+$ $N(0.26,0.1338)^{0.4375}$	$N(0.05,0.09)$	$N(0.17,0.47)$	

Table 2. Continued.

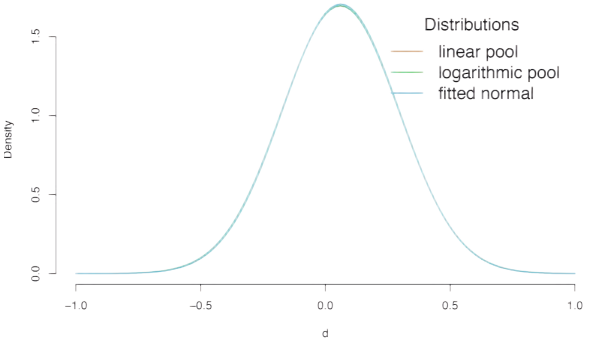
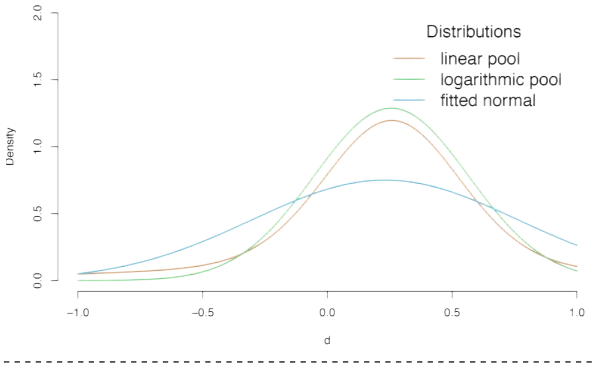
Parameter	Linear pool	Logarithmic pool	Fitted normal	Image
ANonMint	$N(0.06,0.09)^{0.3750}$	$N(0.06,0.24)$	$N(0.06,0.24)$	
ANonAint	$N(0.17,0.1743)^{0.1875}+$ $N(0.28,0.0916)^{0.3125}+$ $N(0.26,0.0875)^{0.3750}+$ $N(0.23,0.1348)^{0.4375}$	$N(0.26,0.31)$	$N(0.23,0.53)$	

Table 2. Continued.

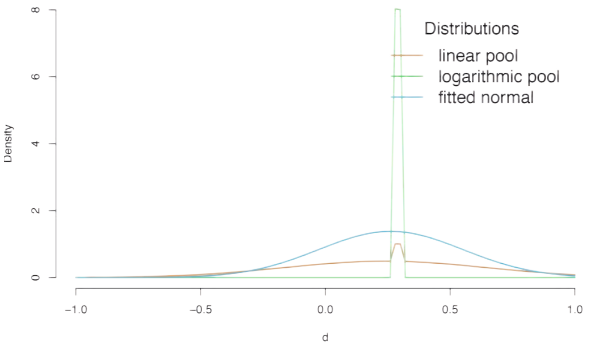
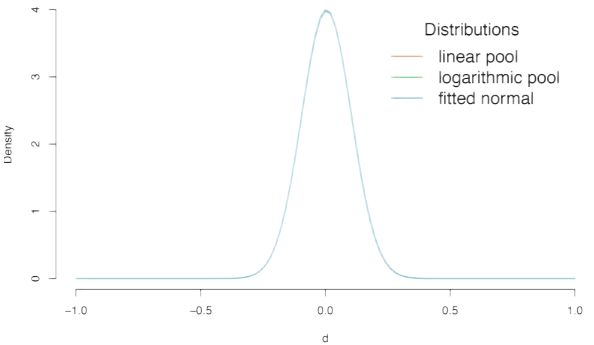
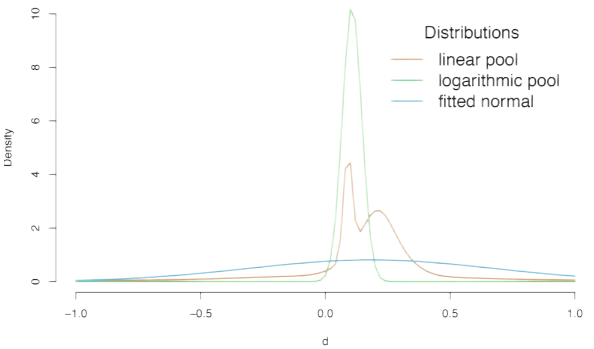
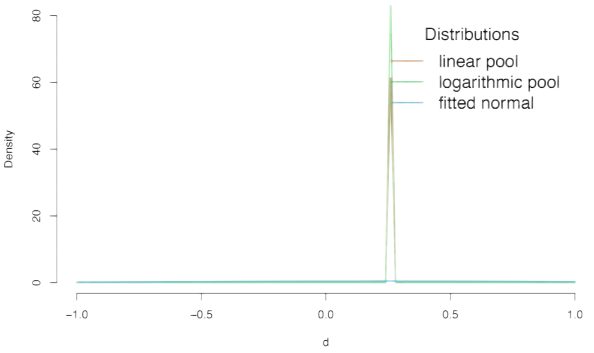
Parameter	Linear pool	Logarithmic pool	Fitted normal	Image
MintonMN	$N(0.24,0.1017)^{0.2500}+$ $N(0.29,0.0010)^{0.3000}$	$N(0.29,0.0046)$	$N(0.27,0.29)$	
MintonAN	$N(0.01,0.0601)^{0.6000}$	$N(0.01,0.10)$	$N(0.01,0.10)$	

Table 2. Continued.

Parameter	Linear pool	Logarithmic pool	Fitted normal	Image
AintonMN	$N(0.09,0.0102)^{0.6000}+$ $N(0.10,0.0948)^{0.2500}+$ $N(0.27,0.1699)^{0.1500}+$ $N(0.21,0.0343)^{0.6000}+$ $N(0.26,0.0260)^{0.3000}+$ $N(0.15,0.0537)^{0.6000}$	$N(0.11,0.04)$	$N(0.19,0.50)$	
AintonAN	$N(0.17,0.1743)^{0.1500}+$ $N(0.26,0.0010)^{0.3000}$	$N(0.25,0.0046)$	$N(0.20,0.82)$	

Note. M = maternal, A = adolescent, int = internalizing, P = positive interaction behavior, N = negative interaction behavior, on = describes the direction of regression (e.g., MPonMint indicates the association from maternal internalizing symptoms at T1 to maternal positive interaction behavior at T2). The hyperparameters of the normal distributions are a mean and a standard deviation.

The linear and logarithmic pool both used the study's normal prior distributions with σ/δ as input for the standard deviation. Subsequently, each of the distributions received an equal weight in the pooling procedure. The normal pool was programmed in Stan (see syntax in the supplemental material). The hyperparameters for the logarithmic pool of normal distributions were calculated according to De Carvalho et al. (2020). To obtain a normal distribution fitting to the linear pool, we first drew 5,000 random samples from each of the weighted normal prior distributions for one parameter. Subsequently, we fitted a normal distribution to these samples (i.e., fitted normal) by means of the fitdist function of the R-package fitdistrplus (Delignette-Muller & Dutang, 2015) using maximum likelihood estimation. The estimated mean and standard deviation associated with the best fit were used as hyperparameters for the priors in Stan.

Statistical Analyses

To evaluate the impact of the different prior distributions, we assessed convergence, conducted prior predictive checks, estimated the posterior distributions and calculated posterior shrinkage.

Convergence was assessed in randomly selected posteriors based on three imputed datasets with the potential scale reduction (PSR; Gelman & Rubin, 1992) and effective sample size (ESS). The PSR (or $\hat{R}$) compares the variance between and within chains. A PSR value near 1.0 indicates convergence. Originally, 1.05 was taken as an upper bound for convergence or even 1.10 with many model parameters, but more recently, smaller values like 1.01 and 1.001 have been recommended (e.g., Vehtari, Gelman, Simpson, Carpenter, & Bürkner, 2019; Zitzman & Hecht, 2019). The ESS quantifies the number of effectively independent draws from the posterior distribution, and is a measure of precision as it indicates how well an estimate is approximated. An ESS larger than 400 is recommended to get a stable estimate (e.g., Vehtari et al., 2019; Zitzman & Hecht, 2019).

In a prior predictive check, samples are taken from the prior distribution to simulate new data based on the sampled parameter estimates. Together, the simulated datasets form the predictive distribution. The predictive distribution encompasses the data that can be expected given the multivariate prior distribution on the parameters. With a predictive distribution, the analyst can evaluate whether the (multivariate) prior relates to sensible data. Furthermore, the current observed data can be compared to the predictive distribution. In the present study, prior predictive distributions were evaluated for each of the four dependent variables and each of the four prior specifications in both models.

Posterior shrinkage (or contraction) *s* describes the degree of reduction in uncertainty from the prior to the posterior distribution of a parameter:

$$s = 1 - \frac{\sigma^2_{\text{posterior}}}{\sigma^2_{\text{prior}}},$$

where $\sigma^2_{\text{posterior}}$ is the variance of the posterior distribution and σ^2_{prior} is the variance of the prior distribution. The inclusion of the likelihood of the data in the posterior tends to decrease the prior uncertainty, resulting in shrinkage. If the data is highly informative compared to the prior, the posterior shrinkage will be close to 1. If the data provides little additional information, the posterior shrinkage will be close to 0.

Missing data was modest and ranged from 2-13% for most variables. Only at T1, 54% of the RADS-2, which is one of the two scales for internalizing problems, was missing because this questionnaire was not assessed with these participants. Based on Little's missing completely at random (MCAR) test that detected no systematic patterns of missingness, normed $\chi^2/df = 1.19$, we inferred that missing data was not likely to bias our analyses. To handle the missing data, multiple imputation was conducted by means of the R-package mice (Van Buuren & Groothuis-Oudshoorn, 2011). All variables that had a correlation >.10 with the variables to be imputed were included as predictors in the imputation model, except for the identification variable. As indicated by the imputation plots and absence of logged events, the 20 imputations were successful. The fraction of missing information (fmi) in all regression paths ranged from .07 to .38.

All Bayesian analyses were conducted in Stan by means of the rstan (Stan Development Team, 2020) and brms (Bürkner, 2017) R-packages in R 4.0 (R Core Team, 2020). We conducted our analyses with 3 chains, each running 8,000 iterations of which the first 3,000 were discarded. The software analyzed each of the 20 imputed datasets separately. Afterwards, the separate posterior distributions were taken together to aggregate the results (Gelman, Carlin, Stern, & Rubin, 2004, *p.* 520; Zhou & Reiter, 2010).[5] We constructed two structural equation models (SEMs) to examine whether maternal and adolescent positive (see Figure 1A) and negative interaction behavior (see Figure 1B) mediated the association between maternal and adolescent internalizing symptoms across time. All models included two-year autoregressive paths for adolescent and maternal internalizing symptoms. We further included correlations between maternal and adolescent interaction behavior. Finally, we calculated eight indirect effects to assess whether maternal and adolescent positive and negative

5 brms includes a function that applies multiple imputation and the aggregation of results in one step, but we did not use it for reasons of comparability between methods.

interaction behavior mediated the associations from mother to adolescent internalizing symptoms as well as from adolescent to maternal internalizing symptoms by multiplying the associations between internalizing symptoms and mother-adolescent interaction behavior from T1 to T2 and from T2 to T3.

RESULTS

Descriptive Statistics

Table 3 displays the means, standard deviations, and correlations among all study variables. Interaction behavior correlated moderately to strongly, both within mother and adolescent interaction behavior as well as between mother and adolescent interaction behavior. Maternal and adolescent interaction behavior correlated moderately with maternal and adolescent internalizing symptoms.

Convergence and Precision

PSR values were <1.01 and ESS >1,000 for all parameters in all viewed analyses for the analyses with logarithmic pooled priors, fitted normal priors, and default priors. However, the analyses with linear pooled priors also showed some insufficient results with respect to convergence and precision. In Model A, a PSR of 1.02 was observed for maternal internalizing symptoms at T1 predicting maternal positive interaction behavior at T2 (MPonMint), and a PSR of 1.04 for maternal positive interaction behavior at T2 predicting maternal internalizing symptoms at T3 (MintonMP). The ESS was <200 for maternal internalizing symptoms at T1 predicting maternal positive interaction behavior at T2 (MPonMint), maternal positive interaction behavior at T2 predicting maternal internalizing symptoms at T3 (MintonMP), and in some analyses also for adolescent positive interaction behavior at T2 predicting maternal internalizing symptoms at T3 (MintonAP). In model B, two PSR values >1.05 were observed: 1.07 for maternal negative interaction behavior at T2 predicting maternal internalizing symptoms at T3 (MintonMN), and 1.12 for adolescent negative interaction behavior at T2 predicting adolescent internalizing symptoms at T3 (AintonAN). The regression of adolescent internalizing problems on adolescent negative interaction behavior (AintonAN) was also repeatedly associated with a particularly low ESS (i.e., <50).

Table 3. Descriptives of All Study Variables

Variable	*M*	*SD*	1	2	3	4	5	6	7
1 Adolescent internalizing T_1	−0.12	0.95							
2 Adolescent internalizing T_3	−0.02	0.88	.426						
3 Maternal internalizing T_1	0.19	0.17	.103	.093					
4 Maternal internalizing T_3	0.19	0.18	−.189	.246	.506				
5 Maternal positive interaction T_2	3.50	0.79	−.363	−.217	−.245	−.175			
6 Maternal negative interaction T_2	1.48	0.72	.169	.189	.204	.186	−.674		
7 Adolescent positive interaction T_2	3.12	0.82	−.213	−.161	−.168	−.161	.627	−.449	
8 Adolescent negative interaction T_2	1.43	0.79	.131	.204	.130	.294	−.400	.425	−.691

Prior Predictive Check

We evaluated the predictive distributions of the four dependent model variables in both studies for each of the four methods (i.e., 32 predictive distributions). Figure 3 displays a selection of four illustrative predictive distributions.

For each of the informative prior specifications, there was a considerable spread in predicted likelihoods and their associated means and standard deviations. The predicted means mostly ranged from -40 to +40, centered around the observed data mean of 0 (all variables were centered). The predictive distribution for the default Stan priors, however, almost had an infinite range including many implausible predicted likelihoods. This behavior was expected, as default priors are not supposed to direct the estimation process, but it also demonstrates that default priors are no sensible priors.

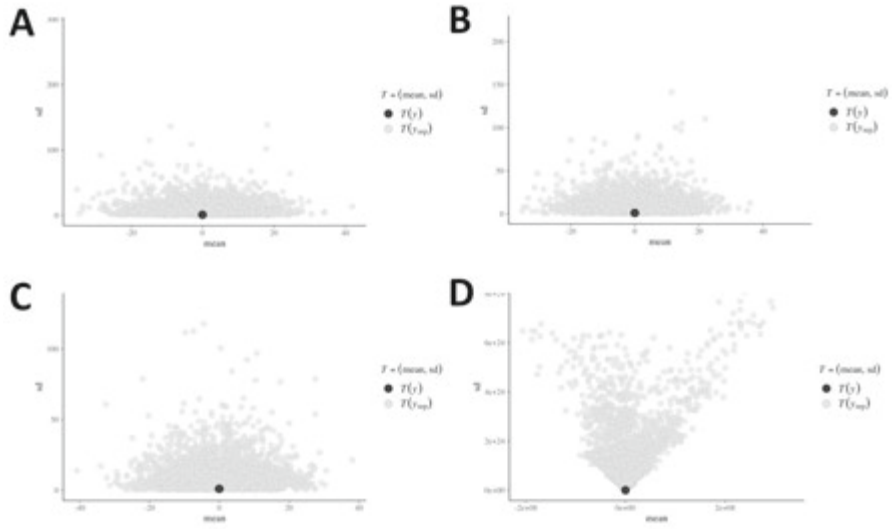


Figure 3. Means (x-axis) and standard deviations (y-axis) in the prior predictive distribution for the four prior specifications: (A) linear pool, (B) logarithmic pool, (C) fitted normal distribution, (D) default. The dark-blue dot represents the mean in the observed data (centered at 0).

Table 4. Shrinkage in model A and B

	Linear pool	Logarithmic pool	Fitted normal	Default
MPonMint	0.67	−0.00	0.54	1.00
MPonAint	0.89	0.04	0.89	1.00
APonMint	0.51	0.50	0.51	1.00
APonAint	0.83	0.60	0.81	1.00
MintonMP	0.54	0.00	0.66	1.00
MintonAP	0.27	0.39	0.32	1.00
AintonMP	0.89	0.10	0.84	1.00
AintonAP	0.82	0.01	0.84	1.00
MNonMint	0.57	0.49	0.57	1.00
MNonAint	0.82	0.20	0.77	1.00
ANonMint	0.58	0.58	0.58	1.00
ANonAint	0.81	0.67	0.80	1.00
MintonMN	0.58	−0.00	0.69	1.00
MintonAN	0.28	0.30	0.30	1.00
AintonMN	0.86	0.07	0.81	1.00
AintonAN	0.87	−0.01	0.88	1.00

Note. M = maternal, A = adolescent, int = internalizing, P = positive interaction behavior, N = negative interaction behavior, on = describes the direction of regression, indirect effects are reported in direction of the association (e.g., MintMPAint describes the indirect effect from maternal to adolescent internalizing symptoms via maternal positive interaction behavior).

Shrinkage

The posterior shrinkage for all parameters of interest and all prior specifications in both models can be found in Table 4. In all cases, the posterior shrinkage for the default Stan priors was 1.00, indicating that the data strongly diminished the posterior variance as compared to the prior variance. This finding was expected as default priors usually have an extremely wide variance to let the likelihood of the data predominate the posterior results. The logarithmic pool generally showed the lowest posterior shrinkage. In 56.3% of the posterior parameter distributions, the posterior shrinkage for the logarithmic pooled prior was <.20, and in 37.5% it was even < .05. In these cases, the logarithmic pooled prior greatly affected the posterior results. The shrinkage of the linear pooled prior and the fitted normal prior were relatively similar and varied between .27 to .90. It should be noted however, that the multimodality of the linear pooled prior and its associated posterior was not captured by our shrinkage measure that summarizes the distributions by their variances. Consequently, even though the shrinkage was larger than that of the fitted normal prior in 50.0% of the cases, we cannot interpret this outcome as if the likelihood had a larger impact on the posterior of the linear pooled prior than the fitted normal.

Indirect Pathways through Maternal and Adolescent Interaction Behavior

Positive interaction behavior. The results for the positive interaction behavior model as analyzed with the three different prior settings are provided in Table 5. Based on the analysis with linear pooled priors, we found that only for the longitudinal associations from maternal and adolescent internalizing symptoms at T1 to maternal positive interaction behavior at T2 ($M_{maternal}$ = -.24, 95% HPD=[-.30, -.13], $M_{adolescent}$ = -.15, 95% HPD=[-.35, -.04]), the 95% highest posterior density (HPD) interval did not include 0 as probable value. The completely negative 95% HPD indicates that higher levels of maternal and adolescent internalizing symptoms predicted lower levels of subsequent maternal positive interaction behavior one year later. Although there was limited evidence that maternal and adolescent internalizing symptoms predicted adolescent positive interaction behavior as the 95% HPD included both negative and positive values, the values were mostly negative. This indicates that there was more probability toward such a negative effect, but still some probability that the effect was positive. For all other associations, negative as well as positive values were part of the 95% HPD. Hence, we are not certain if and how positive interaction behavior at T2 predicted maternal or adolescent internalizing symptoms at T3, one year later. Furthermore, the 95% HPD of the autoregressive paths from maternal and adolescent internalizing symptoms at T1 to their internalizing symptoms at T3 were completely positive ($M_{maternal}$ = .49, 95% HPD=[.33, .66], $M_{adolescent}$ = .44, 95% HPD=[.27, .61]), indicating that maternal and adolescent symptoms showed modest stability across time. All mediational paths included negative as well as positive values in their 95% HPD, indicating that there was no clear evidence on the existence and direction of the

indirect effects from maternal to adolescent or adolescent to maternal internalizing symptoms through maternal or adolescent positive interaction behavior.

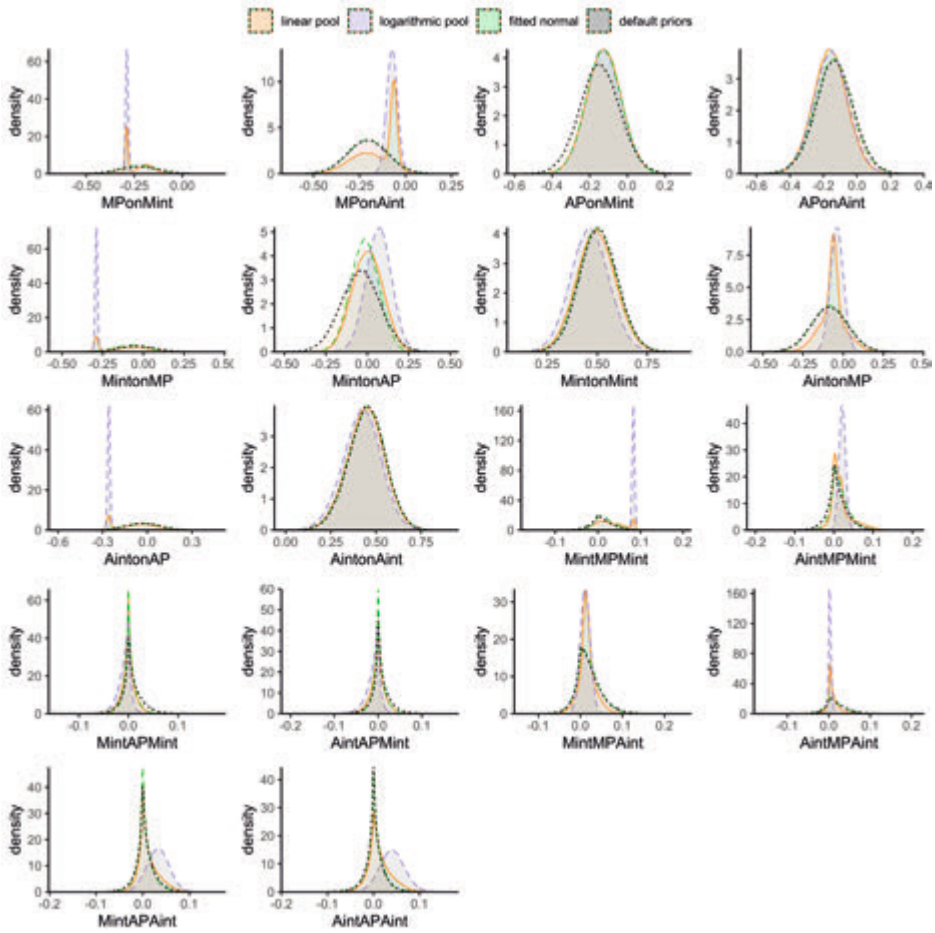


Figure 4. Posterior distributions of the final results involving positive interaction behavior; linear pooled priors are displayed in orange, logarithmic pooled priors in light-purple, fitted normal priors in green, and default priors in grey.

Table 5. Parameter Estimates using Different Prior Settings for Model A

	Linear pool priors			Logarithmic pool priors			Normal fitted to linear pool priors			Default priors		
parameter	mean	95% HPD		mean	95% HPD		mean	95% HPD		mean	95% HPD	
MPonMint	−0.24	−0.3	−0.13	−0.29	−0.3	−0.28	−0.23	−0.38	−0.07	−0.23	−0.4	−0.06
MPonAint	−0.15	−0.35	−0.04	−0.07	−0.12	−0.02	−0.2	−0.38	−0.03	−0.21	−0.39	−0.03
APonMint	−0.12	−0.28	0.03	−0.13	−0.28	0.03	−0.13	−0.28	0.03	−0.15	−0.32	0.03
APonAint	−0.16	−0.33	0.01	−0.16	−0.33	0.01	−0.14	−0.33	0.05	−0.14	−0.32	0.05
MintonMP	−0.12	−0.29	0.1	−0.29	−0.3	−0.28	−0.07	−0.24	0.09	−0.04	−0.24	0.15
MintonAP	0	−0.15	0.15	0.07	−0.06	0.19	−0.02	−0.16	0.12	−0.04	−0.24	0.16
MintonMint	0.49	0.33	0.66	0.46	0.3	0.62	0.5	0.35	0.66	0.51	0.35	0.67
AintonMP	−0.06	−0.2	0.07	−0.04	−0.11	0.03	−0.08	−0.27	0.1	−0.08	−0.27	0.11
AintonAP	−0.09	−0.26	0.12	−0.26	−0.27	−0.24	−0.03	−0.23	0.16	−0.03	−0.22	0.17
AintonAint	0.44	0.27	0.61	0.42	0.24	0.59	0.45	0.27	0.61	0.45	0.28	0.61
AintMPMint	0.02	−0.01	0.08	0.02	0.01	0.04	0.01	−0.02	0.06	0.01	−0.03	0.06
AintAPMint	0	−0.03	0.03	−0.01	−0.04	0.01	0	−0.02	0.03	0.01	−0.03	0.04
MintMPAint	0.02	−0.02	0.05	0.01	−0.01	0.03	0.02	−0.02	0.07	0.02	−0.02	0.08
MintAPAint	0.01	−0.02	0.05	0.03	−0.01	0.07	0	−0.02	0.04	0	−0.03	0.04

Note. M = maternal, A = adolescent, int = internalizing, P = positive interaction behavior, N = negative interaction behavior, on = describes the direction of regression, indirect effects are reported in direction of the association (e.g., MintMPAint describes the indirect effect from maternal to adolescent internalizing symptoms via maternal positive interaction behavior).

The analyses based on logarithmic pooled priors showed generally similar results. As for the analyses with the linear pooled priors, both higher levels of maternal and adolescent internalizing symptoms at T1 predicted lower levels of maternal positive interaction behavior at T2 ($M_{maternal}$ = -.29, 95% HPD=[-.30, -.28], $M_{adolescent}$ = -.07, 95% HPD=[-.12, -.02]). In contrast to the linear pooled priors, lower levels of maternal and adolescent positive interaction behavior at T2 predicted higher levels of their own, but not the other's internalizing symptoms at T3 ($M_{maternal}$ = -.29, 95% HPD=[-.30, -.28]; $M_{adolescent}$ = -.26, 95% HPD=[-.27, -.24]). For all other direct associations, the 95% HPD included both positive and negative values. Similar to the linear pooled priors, maternal and adolescent internalizing symptoms at T1 predicted their own respective symptoms at T2. Furthermore, maternal positive interaction behavior mediated the association between adolescent and maternal internalizing symptoms, as indicated by the 95% HPD of the indirect effect that was completely positive ($M_{indirect}$ = .02, 95% HPD=[.01, .04]). This suggests that higher levels of adolescent internalizing symptoms predicted higher levels of maternal internalizing symptoms two years later through decreased positive maternal interaction behavior. No other indirect effects were found.

Based on the analysis with normal distributions fitted to the linear pooled priors, we detected similar results as for the analysis using linear pooled priors. Comparable to the analyses with both linear and logarithmic pooled priors, maternal and adolescent internalizing symptoms at T1 predicted maternal positive interaction behavior at T2 ($M_{maternal}$ = -.23, 95% HPD=[-.38, -.07]; $M_{adolescent}$ = -.20, 95% HPD=[-.38, -.03]). However, we found no evidence for associations between maternal or adolescent interaction behavior and their subsequent internalizing symptoms, which is in line with the linear pooled priors, but only partially in line with the logarithmic pooled priors. As in the other analyses using linear and logarithmic pooled priors, maternal and adolescent internalizing symptoms at T1 predicted their own respective symptoms at T2. No indirect effects were found. Further sensitivity analyses with default priors, which relied on prespecified non-informative priors, yielded the same conclusions as the analysis with fitted normal priors.

Examining the posterior samples per parameter (see Figure 4) indicated that the linear pooled priors affected posterior samples for some parameters in such a manner that they became bimodal. For example, the posterior distribution of the association between maternal internalizing behavior and subsequent maternal positive interaction behavior (MPonMint) shows that there was some strong evidence from previous studies. This previous evidence supports an effect that is larger than what is found in the current data, as indicated by the shift in modes as compared to the analyses with default priors. On the other hand, for the association between adolescent internalizing symptoms and subsequent maternal positive interaction behavior (MPonAint), the posterior distribution still reflects some strong evidence from previous studies for an effect smaller than found in the data.

Negative interaction behavior. The results for the negative interaction behavior model as analyzed with the three different prior settings are provided in Table 6. Based on the analysis with linear pooled priors, we found that higher levels of maternal, but not adolescent internalizing symptoms predicted higher levels of subsequent maternal, but not adolescent negative interaction behavior one year later (M = .23, 95% HPD=[.06, .39]). In turn, maternal negative interaction behavior at T2 predicted adolescent, but not maternal internalizing symptoms one year later at T3 (M = .11, 95% HPD=[.01, .23]). There was limited evidence that adolescent negative interaction behavior at T2 predicted their own or their mothers' internalizing symptoms at T3, one year later. Although for these associations the 95% HPD included both positive and negative values, the values were mostly positive. This indicates that there was more probability toward a positive effect, but still some probably that the effect was negative. Maternal negative interaction behavior mediated the association between maternal and adolescent internalizing symptoms, as indicated by the 95% HPD of the indirect effect that was completely positive ($M_{indirect}$ = .03, 95% HPD=[0, .06]). This suggests that higher levels of maternal internalizing symptoms predicted higher levels of adolescent internalizing symptoms two years later through increased maternal negative interaction behavior. No other indirect effects were found.

The analyses with logarithmic pooled priors again demonstrated generally similar results. Higher levels of maternal, but not adolescent internalizing symptoms at T1 predicted higher levels of maternal negative interaction behavior at T2 (M = .23, 95% HPD=[.08, .39]). Maternal negative interaction behavior at T2 in turn predicted adolescent internalizing symptoms (M = .10, 95% HPD=[.04, .16]) and, in contrast to the linear pooled priors, also maternal internalizing symptoms at T3 (M = .29, 95% HPD=[.28, .30]). Higher levels of adolescent negative interaction behavior at T2 further predicted higher levels of subsequent adolescent internalizing symptoms at T3 as indicated by the completely positive 95% HPD (M = .26, 95% HPD=[.25, .27]), which contrasts with the analysis using linear pooled priors. For all other direct associations, the 95% HPD included both positive and negative values. Similar to the linear pooled priors, we detected evidence for an indirect effect from maternal to subsequent adolescent internalizing symptoms through increased maternal negative interaction behavior ($M_{indirect}$ = .02, 95% HPD=[0, .05]).

Based on the analysis with fitted normal priors, we found slightly different results. While maternal internalizing symptoms at T1 also predicted maternal negative interaction behavior one year later at T2 (M = .22, 95% HPD=[.06, .38]), there was only limited evidence that maternal negative interaction behavior predicted subsequent adolescent internalizing symptoms at T3 as the posterior distribution was wider and the 95% HPD thus included positive and negative values (M = .11, 95% HPD=[-.04, .27]. However, adolescent negative interaction behavior predicted maternal internalizing

symptoms one year later at T3 (*M* = .11, 95% HPD=[.00, .23]). No indirect effects were found in this analysis. Further sensitivity analyses using default priors again yielded the same conclusions as the normal priors fitted to the linear pool. For the association between adolescent negative interaction behavior at T2 and subsequent maternal internalizing symptoms at T3, the effect size doubled in size compared to the linear pool, logarithmic, and fitted normal priors. The 95% HPD was even further from 0 (*M* = .22, 95% HPD=[.06, .38]), indicating stronger evidence that negative behaviors of adolescents predicted internalizing symptoms in mothers.

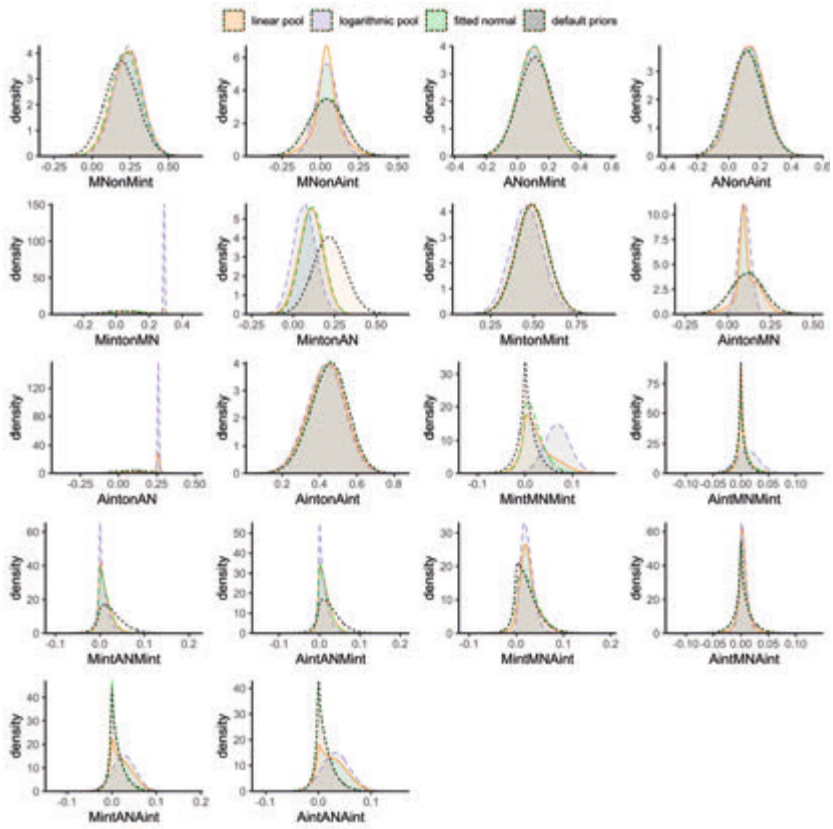


Figure 5. Posterior distributions of the final results involving negative interaction behavior; linear pooled priors are displayed in orange, logarithmic pooled priors in light-purple, fitted normal priors in green, and default priors in grey.

Table 6. Parameter Estimates using Different Prior Settings for Model B

	Linear pool priors			Logarithmic pool priors			Normal fitted to linear pool priors			Default priors		
parameter	mean	95% HPD		mean	95% HPD		mean	95% HPD		mean	95% HPD	
MNonMint	0.23	0.06	0.39	0.23	0.08	0.39	0.22	0.06	0.38	0.19	0.01	0.37
MNonAint	0.04	−0.1	0.19	0.04	−0.08	0.16	0.04	−0.15	0.22	0.04	−0.15	0.23
ANonMint	0.1	−0.06	0.27	0.1	−0.06	0.27	0.1	−0.06	0.27	0.11	−0.07	0.3
ANonAint	0.13	−0.04	0.3	0.13	−0.04	0.3	0.12	−0.05	0.29	0.11	−0.06	0.29
MintonMN	0.11	−0.07	0.29	0.29	0.28	0.3	0.08	−0.07	0.23	0.03	−0.14	0.19
MintonAN	0.11	−0.01	0.23	0.07	−0.05	0.18	0.11	0	0.23	0.22	0.06	0.38
MintonMint	0.49	0.34	0.64	0.46	0.3	0.61	0.49	0.34	0.65	0.49	0.34	0.65
AintonMN	0.11	0.01	0.23	0.1	0.04	0.16	0.11	−0.04	0.27	0.11	−0.05	0.27
AintonAN	0.2	−0.02	0.26	0.26	0.25	0.27	0.1	−0.06	0.26	0.1	−0.06	0.26
AintonAint	0.44	0.27	0.6	0.43	0.26	0.59	0.45	0.28	0.61	0.45	0.28	0.61
AintMNMint	0	−0.01	0.03	0.01	−0.02	0.05	0	−0.02	0.03	0	−0.02	0.02
AintANMint	0.01	−0.01	0.04	0.01	−0.01	0.03	0.01	−0.01	0.04	0.02	−0.01	0.08
MintMNAint	0.03	0	0.06	0.02	0	0.05	0.02	−0.01	0.07	0.02	−0.01	0.07
MintANAint	0.02	−0.01	0.06	0.03	−0.02	0.07	0.01	−0.01	0.04	0.01	−0.01	0.05

Note. M = maternal, A = adolescent, int = internalizing, P = positive interaction behavior, N = negative interaction behavior, on = describes the direction of regression, indirect effects are reported in direction of the association (e.g., MintMNAint describes the indirect effect from maternal to adolescent internalizing symptoms via maternal negative interaction behavior).

Some deviations between the results above stand out. For example, even though the mode of the pooled priors is closer to zero than the data (see Figure 5; AintonMN) maternal negative interaction behavior at T2 predicted adolescent internalizing symptoms at T3 with both pooled priors, but not with the fitted normal and default priors. Apparently, the density in the region slightly above 0 was so high that 0 was excluded from the 95% HPD for the pooled priors. On the other hand, adolescent negative interaction behavior at T2 only predicted maternal internalizing symptoms at T3 with default priors, suggesting that the prior for this parameter had a higher probability in the region around zero than our data. The posterior distribution of the association between adolescent negative interaction behavior and subsequent adolescent internalizing symptoms seemed strongly affected by the prior distribution as well, as there was a small region with extremely high probability (i.e., a peak) in the posterior around .25 in the analyses using linear and logarithmic pooled priors (see Figure 5; AintonAN); the 95% HPD of the linear pooled results, however, still included 0.

DISCUSSION

The present study used Bayesian estimation with systematically obtained results from previous studies and systematically defined prior weights, following three prior aggregation methods. The illustrative empirical research question behind this analysis concerned the mediation of bidirectional associations between maternal and adolescent internalizing symptoms from early to mid-adolescence by mother-adolescent positive and negative interaction behavior. We retrieved 47 effect sizes from 9 studies that provided information on some of the relevant parameters of our model and were thus integrated into our analyses.

Empirical Discussion: The Mediating Role of Mother-Adolescent Interaction Behavior

Consistent with theoretical and empirical evidence that internalizing symptoms can lower maternal positive interaction behavior toward their children (Goodman & Gotlib, 1999; Lovejoy et al., 2000; McCabe, 2014; Simons et al., 1993), the distributions consistently showed that higher levels of maternal internalizing symptoms predicted lower levels of their own, but generally not adolescent positive and negative interaction behavior in the following year. Mothers with increased internalizing symptoms might be emotionally unavailable, easily irritated, and unable to sensitively respond to their children's needs, which can suppress encouraging or nurturing behaviors and exacerbate hostile, rejecting behaviors in subsequent interactions with their children (Lovejoy et al., 2000). As maternal internalizing symptoms can disrupt interactional processes between mothers and adolescents, they are likely to drive relationship erosion in the long term (Coyne, Burchill, & Stiles, 1991; Meeus, 2016). Interestingly, although the analyses using linear and logarithmic pooled priors suggested a clear negative association from adolescent internalizing symptoms to maternal positive

interaction behavior as well, our findings generally provided only little evidence for the theoretical propositions that adolescent internalizing symptoms disrupt interactions in the family (Berg-Nielsen et al., 2002; Sheeber et al., 2001).

Despite theoretical propositions and empirical findings that less positive and more negative mother-adolescent interaction behavior predict adolescent internalizing symptoms (McLeod, Weisz, et al., 2007; McLeod, Wood, et al., 2007; Pinquart, 2017; Yap et al., 2014), we found that maternal or adolescent internalizing symptoms predicted later mother-adolescent interaction behavior more often than that mother-adolescent interaction behavior predicted later internalizing symptoms. This is in line with one of the few mediation studies that found associations between maternal internalizing symptoms and observed maternal interaction behavior, but not between interaction behavior and adolescent internalizing symptoms (van Doorn et al., 2016). One possible reason for this finding may be that mother-adolescent interaction behavior is more likely to influence immediate, short-term emotions in mothers or adolescents. While particularly maternal internalizing symptoms may have long-lasting effects, maladaptive interactions may exert their effects at a shorter time interval than we could detect with annual assessments. Alternatively, highly negative and less positive interactions between mothers and adolescents are quite common in early to mid-adolescence as mother-adolescent conflicts become more intense (Hadiwijaya, Klimstra, Vermunt, Branje, & Meeus, 2017). It is possible that because such behaviors are relatively typical during this time in adolescence, they are experienced as tied to that specific interaction and thus do not directly influence adolescent mood in the long term.

The limited evidence that we found for the associations between mother-adolescent interaction behavior and later internalizing symptoms concerned mainly negative interaction behavior in the analyses using linear and logarithmic pooled priors. This may be expected given that the impact of negative events and emotions is generally stronger than the impact of positive events or emotions (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001). Although the effect sizes were generally comparable in all analyses, using different informative priors yielded somewhat different conclusions based on the distributions and credibility intervals. Together with the detected indirect effect that maternal negative interaction behavior mediated the associations between maternal internalizing and subsequent adolescent symptoms using linear pooled and logarithmic priors, however, they suggest that negative interaction behavior may play a role in the transmission of internalizing symptoms. Hostile behaviors might make interaction partners feel rejected and helpless, undermine their self-esteem, and elicit negative self, which might in turn increase their risk for internalizing symptoms in the long-term (Garber & Flynn, 2001; Gottman et al., 1997). Interestingly, we also found that decreased maternal positive interaction behavior mediated the associations

between adolescent internalizing symptoms and subsequent maternal internalizing symptoms, but this indirect effect was only evident using logarithmic pooled priors.

The different conclusions using different priors also warrant caution. Specifically, they suggest that our data contrasts with previous findings. In the linear pooled prior distribution, we indeed detected two peaks toward a positive distribution for the associations from maternal negative interaction behavior to later adolescent internalizing symptoms, whereas the logarithmic pooled priors suggested one extreme dense, narrow distribution closer to zero and the fitted normal priors indicated a similar, but flat distribution. The detected peaks in the linear and logarithmic pooled priors resulted from information found in previous studies, which drove these conclusions, whereas associations that we only detected with fitted normal and default priors suggested that our data provided stronger evidence than previous findings. Using different approaches to define informative priors allowed us to compare their impact on the results and evaluate the robustness of our conclusions. Once we updated the information collected in previous studies with our new data, the posterior distributions shifted to a varying degree depending on how we specified the priors. Differences between the posteriors were particularly pronounced when our data strongly diverged from previous studies. While the posteriors generated from logarithmic pooled priors were strongly influenced by previous data and thus only shifted little compared to the prior distributions, the linear pooled priors often resulted in bimodal distributions that reflected the discrepancy between previous and new data. These differences in priors and previous compared to new data emphasize that for some associations, we may not yet have enough evidence to draw final conclusions.

The Role of Different Informative Priors

While we were able to include a range of findings relating to our model parameters, these studies reflected our own study's design to a varying degree and might thus introduce potential bias (Hobbs et al., 2011; Viele et al., 2014). Each included study provides a varying amount of relevant information and certainty, which is essential to take into account when specifying informative priors. How much a previous study contributes, depends on the focus and methodological considerations of the specific study. A weighting scheme therefore needs to be tailored to each new study's purpose and design. To avoid bias, such as increased subjectivity, it is important to engage content experts who can judge the relevance of weighting aspects and justify all decisions transparently in an accessible logbook (Zondervan-Zwijnenburg et al., 2017). Therefore, we involved content experts to design a weighting scheme and scoring system that allowed us to consider each study's specific contribution with respect to our data. Our illustrative example represented a longitudinal, multi-method design, which constituted the core of our weighting scheme. As cross-sectional studies cannot be used to disentangle the temporal order of associations, they provided only weak

evidence for our parameters. Similarly, longitudinal studies that did not control for previous levels of psychopathological symptoms at an earlier point in time are not useful to measure change, and therefore received less weight as well. While a weighting scheme is an essential tool to combine findings from more or less comparable studies, it needs to be carefully constructed and reviewed to avoid inaccurate inferences and conclusions. In this study, we followed recommendations, such as including experts for the composition of the weighting scheme or the estimation of the weights (e.g., Zondervan-Zwijnenburg et al., 2017), which can further help to reduce subjectivity. Instead of weights based on the match between previous studies and the design of the study at hand, weights can also be based on optimality criteria (e.g., maximum entropy, minimum Kullback-Leibler divergence) or modelled by means of a prior on the weights (e.g., Veen et al., 2017; Carvalho, 2020). These methods do not take the content of studies into account, which can be regarded their strength because of increased objectivity, but also their weakness because previous studies are not valued based on criteria that are considered important by experts.

Our statistical evaluation showed that analyses based on linear pooled priors may suffer from estimation problems (i.e., insufficient convergence and precision), where other prior specifications do not show the same issues. Furthermore, the prior predictive distributions were comparable across prior specification methods, except for the default prior, which does not produce a meaningful predictive distribution. Generally, we found that the posterior distributions based on the analyses with linear pooled priors displayed bimodal distributions and strong peaks in multiple occasions. The posteriors resulting from the logarithmic pooled priors were peaked and highly driven by the previous information as confirmed by the low associate shrinkage. In the current study, two studies (i.e., Milan & Carlone, 2018; Pinquart, 2017) caused all peaks. These studies reported estimates with extremely small (standardized) standard errors, thus strengthening the evidence for these effects. While Pinquart (2017) conducted a longitudinal meta-analysis on the associations between parental behaviors and adolescent internalizing symptoms with over 1,000 included studies, Milan and Carlone (2018) investigated actor and partner effects in how mother and adolescent internalizing symptoms predicted maternal and adolescent behaviors during an interaction task. Both studies provide important information for our analyses, but do not precisely reflect our study design. Specifically, Pinquart's meta-analysis also included (young) children and reported parental behaviors. Milan and Carlone, on the other hand, only sampled adolescent girls, who have been found to show higher levels of internalizing symptoms (Zahn-Waxler, Shirtcliff, & Marceau, 2008) and to be more sensitive to interpersonal experiences than adolescent boys (Flook, 2011). The design differences were taken into account by using power priors based on a systematic weighting scheme. In the current study, we did not lower study weights based on the specificity on the results. From a perspective of building cumulative knowledge, that would be a questionable practice. From a

more pragmatic perspective, however, it may be sensible to downweigh information that appears unreasonably specific. For example, when expert elicitation is used to form prior distributions, it is suggested that the analyst decides to exclude an expert's distribution if their probability density is too narrow (Carvalho et al., 2020).

Linear pooled priors integrate all available literature to its full avail and consider the influence of potentially differing previous findings. These distributions allow - or even demand - researchers to examine extreme or varying findings and discuss their data more specifically in relation to the literature. In this manner, the linear pooled prior and its associated posterior may also provide directions for future research. However, a multimodal posterior distribution may also render it difficult to interpret the findings directly. Furthermore, extra caution is warranted to establish sufficient convergence and precision.

Logarithmic pooled priors reflect an updating process of previous studies. As such, they are closely tied to the idea of building cumulative knowledge. In the current study, the specificity of some of the previous results overruled other previous findings and the current data in the posterior. Strictly speaking, however, this does not disqualify the logarithmic pooling procedure in general, nor in this case specifically. The posterior still represents our updated previous knowledge.

An alternative to downweighing previous results based on their extreme specificity, is to fit normal distributions to the linear pooled previous information. Similar to logarithmic pooled priors, fitted normal distributions behave well during Bayesian estimation and, similar to linear pooled priors, use previous information to inform the analyses. Particularly if previous research is scarce, contradictory or only few studies are sampled, fitted normal distributions are useful to specify informative priors without overemphasizing the effect of one individual study. Fitted normal priors are best suited when it can be assumed that the previous results are random samples from an underlying normal distribution, or when the analyst considers it a pragmatic midway between the more informative pooled and default priors.

In contrast to informative priors, default priors neglect previous knowledge about how maternal interaction behavior predict later internalizing symptoms. The predictive distribution clearly showed that default priors are highly unspecific with regards to expected future data. The associated shrinkage confirmed that the observed data completely overruled the unspecific previous information. For default priors, this behavior is desired. Previous studies, however, have shown that the use of default, non-informative priors may strongly bias the results and decrease estimation accuracy, particularly in small samples (Smid et al., 2019; Zitzmann et al., 2020).

Strengths, Limitations, and Implications

This study applied Bayesian estimation with informative priors to examine in an illustrative example whether observed mother-adolescent interaction behavior underlies the longitudinal associations between maternal and adolescent internalizing symptoms from early to mid-adolescence. Using a novel, comprehensive approach in which we first systematically quantified previous study findings in a meta-analytic design and then used this previous knowledge as input for the analyses allowed us to draw more precise conclusions about the potential mediating role of mother-adolescent interaction behavior. Such a strategy exceeds a pure meta-analytical approach, because it not only quantifies existing results from previous studies into a prior distribution but also allows to further integrate new data. As such, meta-analyses provide good starting points for new Bayesian analyses. Previous studies generate and raise new research questions, and Bayesian estimation with informative priors allows a cumulative approach that does not ignore existing knowledge, but gradually updates it. This way, existing knowledge will be integrated into the empirical process. Particularly when previous research is scarce or when new studies are needed to address important limitations of previous research, including prior distributions can help to further cumulate knowledge. In our study, information was available on only some parameters, but not on the complete mediation model that we aimed to test. While the limited previous information was not sufficient to perform a meaningful meta-analysis, we were able to use the existing information to conduct new analyses that addressed previous limitations or remaining questions and integrated previous knowledge. By using three different priors, we were further able to show the robustness of our results across different approaches.

Despite these strengths, this study had some limitations with respect to the empirical mediation analysis. First, we only observed mother-adolescent interaction behavior at one time during early to mid-adolescence. While this approach allowed us to reduce the complexity of our model to fit our sample size, summary scores may not accurately reflect the processes that occur during the interactions between mothers and adolescents. It may be important to not only examine which average behaviors mothers and adolescents show during interactions, but also how these behaviors mutually influence each other on a moment-to-moment basis.

Second, a full longitudinal mediation approach would further require the assessment of all variables at each time point to account for the stability of not only internalizing symptoms across time, but also the stability of interaction behavior as well as concurrent associations between interaction behavior and internalizing symptoms. Due to the limited sample size in our data (N = 102 mother-adolescent dyads), we had insufficient information to inform a three-wave fully recursive model, which would have been ideal.

Third, longitudinal studies rarely employ the same time intervals between measurements, which renders comparing the findings from these studies difficult. Parameter estimates often depend on the time interval that was used (e.g., Gollob & Reichardt, 1987)) as the underlying processes that measure change on a micro-scale, such as moment-to-moment or day-to-day, can differ from those on a macro-scale, such as year-to-year (Ebner-Priemer & Trull, 2009; Hollenstein, Lichtwarck-Aschoff, & Potworowski, 2013; Voelkle, Oud, Davidov, & Schmidt, 2012). Consequently, studies with varying time scales might result in different conclusions that are not directly comparable. In our study, we tried to address time dependency by adding additional weight to studies that incorporated the same time interval as we did. However, we were only able to include few longitudinal studies, of which none received this additional weight. Future studies that aim to incorporate more, or exclusively, longitudinal studies might consider continuous rating options, such as continuous-time modeling or continuous-time meta-analytical procedures that allow to account for the effect of time more precisely (e.g., Kuiper & Ryan, 2020; van Montfort, Oud, & Voelkle, 2018). Another option could be to include a selection of varying weighting schemes and subsequently evaluate how different rating decisions affect the results. However, these approaches were beyond the scope of our empirical illustration.

In this study, we made use of differently composed informative priors to compare their effects on the posterior distributions. While our approach allowed us to systematically specify and use informative priors for the analyses of our data, quantifying and weighing each previous study in such a systematic way requires a substantial amount of time and effort. If taken seriously, the task is equivalent to conducting a weighted meta-analysis. By allowing researchers to integrate new data and evaluate novel research questions using existing knowledge, this approach exceeds meta-analytical methods and allows for knowledge to further cumulate over time. As such, Bayesian estimation with informative priors can address important shortcomings of current empirical practices and serve the goal of empirical research to generate scientific growth of knowledge. Nevertheless, in such a systematic approach it is essential effectively use previous information for Bayesian estimation. Knowing the literature and making informed decisions about relevant studies allows researchers to consider the most suitable approach to defining priors for their specific situation. This is important to avoid incorporating information from only one individual sample, while years of research already established well-grounded expectations. Focusing on the 95% HPD for hypothesis testing, our results did not detect many differences between the use of pooled or fitted normal priors.

How results from multiple previous studies on the same parameter should be included in the associated prior depends on theoretical considerations: Should the prior reflect the previous results

as they are (linear pool), be an update of previous results (logarithmic pool), or be considered a set of random samples from an underlying normal distribution (normal fitted to the linear pool)? The differences between the approaches are emphasized when results diverge across previous studies: Are all results plausible and can they coexist in the prior distribution (linear pool), is only the consensual part plausible (logarithmic prior), or is there an underlying truth that is best resembled by a fitted normal distribution (fitted normal)? Additionally, pragmatic considerations can be taken into account. For example, the logarithmic pool is a theoretically sound (Bayesian) approach to aggregate multiple previous results that will emphasize consensual values, but extremely specific results from previous studies lead it to exclude large portions of the sample space. In the same situation, the posterior distributions based on the linear pooled priors do not exclude the observed values. However, the bimodal results that can result from diverging previous findings are difficult to interpret substantively. In these cases, a prior distribution like the fitted normal may be preferred, as it eliminates most of the impact of studies with high density when more studies contribute to the previous information.

CONCLUSION

Testing a comprehensive model that includes mediation effects requires a large sample size to detect small-to-moderate effects that are common in social science. Typically, studies including longitudinal, observational designs include only relatively small samples as they are time-consuming, costly, demand more of the participants, and face recruitment difficulties, such as dropout. Attempting to estimate complex models with traditional analytical techniques can result in estimation problems as well as inaccurate parameter estimates (e.g., van de Schoot et al ., 2017), and thus limit the conclusions that can be drawn from such models. Furthermore, by using informative priors, we gain insight into how our data relate to the results from previous studies.

The findings of our study indicated that posterior distributions were generally stable across different prior distributions with differing levels of existing knowledge on the associations between mother-adolescent interaction behavior and internalizing symptoms. Specifically, we consistently found that even though mother-adolescent interaction behavior might play a relatively limited role in the transmission of internalizing symptoms from early to mid-adolescence, particularly negative interaction behavior might still be relevant. Nevertheless, the choice of prior aggregation did alter the results for some parameters and may well make a difference in other studies. Researchers should carefully consider how to aggregate previous results into one prior distribution, and always conduct sensitivity analyses to demonstrate if the results hold with different prior specifications. As illustrated by our example, using Bayesian estimation with informative priors offers a great opportunity to

use accumulated knowledge to increase the precision of our outcomes. If conducted thoroughly, the approach equals and exceeds a weighted meta-analysis as it not only quantifies previous knowledge, but also integrates new data into a cumulative process. Such precision and accumulation of knowledge is important in moving empirical science forward, but also in informing therapeutic programs that aim to prevent or reduce adolescent internalizing symptoms by targeting often proposed risk factors, such as maladaptive interaction behavior between mothers and adolescents.

SUPPLEMENTAL MATERIAL

(provided on OSF: https://osf.io/c37mv/)

Review of Prior Knowledge on the Role of Mother-Adolescent Interaction Behavior

Several studies have been conducted to support the theoretically proposed associations of maternal and adolescent internalizing symptoms with mother-adolescent interaction behavior. These previous findings provide the core input for informative priors that can be used to inform novel analyses and thus increase the precision of posterior distributions. In constructing such strongly defined informative priors, methodological study differences need to be taken into account and weighed according to how well they represent and match the present study's design and how strongly they support the theoretically proposed hypotheses. Effectively using the quantified information to increase the precision of the results thus requires informative priors that are specified systematically and transparently (van de Schoot, Broere, Perryck, Zondervan-Zwijnenburg, & Van Loey, 2015; Zondervan-Zwijnenburg, Peeters, Depaoli, & Van de Schoot, 2017). In the following, we systematically review and discuss the support from previous meta-analytic and longitudinal studies as input for our informative priors.

Support from meta-analytic studies. In line with theoretical models, meta-analytical studies indeed found that mothers with more internalizing symptoms used less positive and more negative interaction behaviors towards their children than mothers with fewer internalizing symptoms (Lovejoy, Graczyk, O'Hare, & Neuman, 2000; McCabe, 2014). In turn, children whose mothers used less positive interaction behavior and more negative interaction behavior showed higher levels of internalizing symptoms than children whose mothers used less positive and more negative interaction behavior (McLeod, Weisz, & Wood, 2007; McLeod, Wood, & Weisz, 2007; Pinquart, 2017; Yap, Pilkington, Ryan, & Jorm, 2014). Recent longitudinal meta-analyses further found that maternal positive and negative interaction behavior predicted subsequent child internalizing symptoms (Pinquart, 2017; Yap et al., 2014), while prior child internalizing symptoms also predicted lower levels of subsequent mother-child positive, but not negative interaction behaviors (Pinquart, 2017).

As meta-analyses quantify a large body of empirical research, they provide reliable information for the construction of informative priors. However, drawing conclusions from these meta-analyses to our specific developmental period and design is also difficult. First, most meta-analyses included a broad age range from infancy throughout adolescence. Mother-child interactions change in structure and function over time. In adolescence, interactions between mothers and their children increase in conflict as adolescents negotiate their growing independence (Laursen, DeLay, & Adams,

2010) and experience more intense, fluctuating emotions than they did in childhood (Larson et al 1996; Larson et al 2002). Mother-child interactions during early childhood can thus not adequately reflect these changing interactions during adolescence. Second, most meta-analyses included both self-reports and observations to assess mother-adolescent interaction behavior. However, self-reports and observations of interaction behaviors are usually only weakly associated (Herbers, Garcia, & Obradović, 2017; Moens, Weeland, Van der Giessen, Chhangur, & Overbeek, 2018). Although observational assessments of parent-adolescent interactions are more objective and valid to reflect interactions between mothers and adolescents, and are thus clearly preferred in the field (Gardner, 2000; Herbers et al., 2017; Lovejoy et al., 2000; Repetti, Reynolds, & Sears, 2015), to date longitudinal multi-method studies are still scarce. Third, all meta-analyses on the associations between maternal internalizing symptoms and mother-adolescent interaction behavior reflect concurrent associations that do not allow conclusions about temporal associations and direction of effects. As causal or temporal associations between mother-adolescent interaction behaviors and maternal as well as adolescent internalizing symptoms take time to unfold, longitudinal studies can more accurately reflect these mediation processes than cross-sectional studies (Gollob & Reichardt, 1987; Kraemer, Yesavage, Taylor, & Kupfer, 2000; Maxwell & Cole, 2007).

Support from longitudinal studies. Observational, longitudinal assessments in adolescence best reflect our study's design and thus provide the strongest evidence for specifying informative priors. The few findings on longitudinal associations between self-reported maternal internalizing symptoms and observed maternal interaction behavior, or between observed maternal interaction behavior and adolescent internalizing symptoms, remain inconsistent. Maternal internalizing symptoms have been shown to predict subsequent maternal positive (Feng, Shaw, Skuban, & Lane, 2007; Simons, Lorenz, Wu, & Conger, 1993), but not negative interaction behavior (Feng et al., 2007). Furthermore, some studies found that maternal positive and negative interaction behavior predicted adolescent internalizing symptoms (Hofer et al., 2013; Milan & Carlone, 2018), while other studies failed to find such associations (Feinberg, Kan, & Hetherington, 2007; Schwartz et al., 2012). The only study that also tested reversed associations from adolescents to mothers found that observed adolescent positive and negative interaction behaviors did not predict subsequent maternal internalizing symptoms (Milan & Carlone, 2018). Differences in the assessments of internalizing symptoms (e.g., diagnosis vs. symptoms; depressive vs. internalizing symptoms), age ranges, and time intervals (ranging from two weeks to three years) may explain these inconsistent findings.

While the reviewed studies provide some indications that maternal positive and negative interaction behavior mediate the transmission of internalizing symptoms, only few studies tested these associations in a mediation model. These studies found that self-reported maternal negative, but not

positive interaction behavior mediate the prospective association between maternal and adolescent internalizing symptoms (Elgar, Mills, McGrath, Waschbusch, & Brownridge, 2007; Sellers et al., 2014). Another study that also controlled for prior adolescent internalizing symptoms, however, did not find that maternal negative interaction behavior mediated the association between maternal depression history and subsequent adolescent internalizing symptoms (Frye & Garber, 2005). Although these studies support a potential mediation effect of interaction behaviors from maternal to adolescent internalizing symptoms, they did not observe interaction behaviors and thus, do not provide suitable information to construct informative priors in our study. The two studies that tested mediating effects of observed maternal interaction behavior found that maternal depressive symptoms were associated with observed maternal interaction behavior, but only one study found that both maternal positive and negative interaction behavior predicted adolescent depressive symptoms (Olino et al., 2016), whereas the other found that positive interaction behavior did not predict adolescent internalizing symptoms (van Doorn et al., 2016). Both studies failed to detect significant mediation effects.

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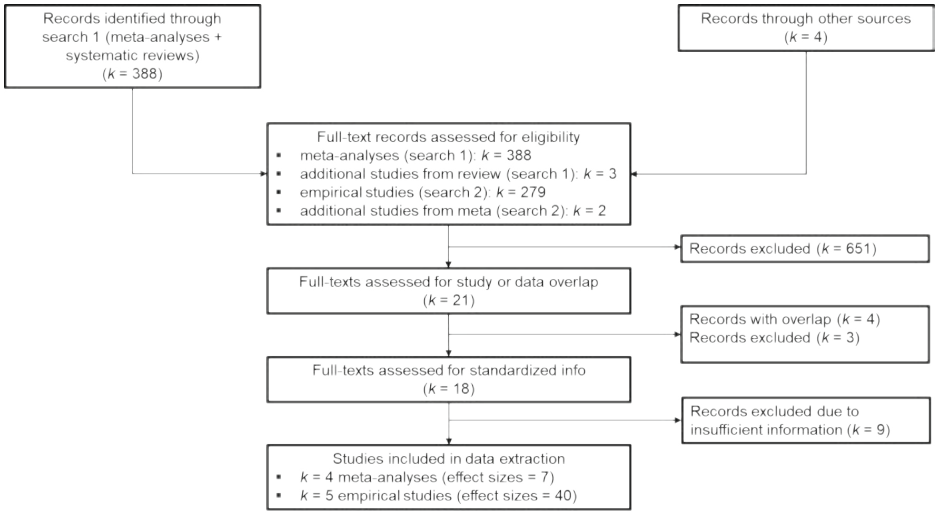
Study selection



Meta-analyses

Inclusion criteria:

- Sample includes adolescence
- Parental and/or adolescent internalizing symptoms
- Parental positive and/or negative interaction behaviors towards adolescent and/or adolescent positive and/or negative interaction behaviors towards parent

Search string: TS=(anxi* OR depress* OR internalizing) AND TS=(parent* OR maternal OR mother) AND TS=(child OR adolescent) AND TS=(positive OR negative OR affect OR warmth OR hostil* OR reject* OR smil* OR interest* OR criticiz* OR interact* OR parenting OR relat*) AND TS=(meta-analy*) → **388 hits** (Web of Science; until 30-3-2020)

Study	Search	Incl ES	Overlap ES	No info	Final nES
Main parameters					
Lovejoy	X	2	1		1
McCabe	X	1			1
McLeod A	X	2	2 (excl)		
McLeod B	X	2	2 (excl)		
Pinquart	X	4			4
Weymouth	X	1			1
Yap	X	4	4 (excl)		
Chapman	X				
*Dietz		11		1	10
*Dadds		8			8
*Jackson		2		2 (excl)	

Note. Search = studies identified through database search, ES = effect size, no info = not all standardized information available, *empirical studies included from Chapman review.

Empirical studies

Inclusion criteria:

- Adolescent sample (range from 7-25 years at first measurement)
- longitudinal associations between maternal and/or adolescent internalizing symptoms and maternal and/or adolescent interaction behavior
- Interaction behaviors observed during mother-adolescent interaction

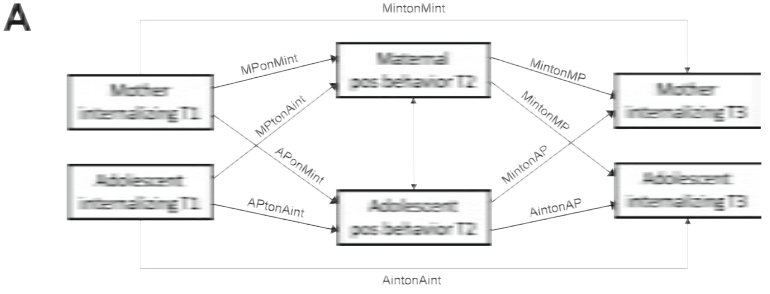
Search string: TS=(anxi* OR depress* OR internalizing) AND TS=(maternal OR mother* OR parent*) AND TS=(adolescen* OR youth OR teen* OR youngst* OR student* OR "emerging adult*" OR "early adult*" OR "young adult*") AND TS=(interacti* OR relation* OR parenting) AND TS=(positive OR negative OR affect OR warmth OR hostil* OR reject* OR smil* OR interest* OR criticiz* OR anger) NOT TS=(autism OR diabet* OR chronic* OR gene* HIV OR ADHD OR preterm OR postnatal OR cancer OR neural OR brain) AND TS=(observation* OR observed OR code* OR rater OR "tape*" OR task* OR record*) → **275 hits** (Web of Science; from 2012 until 30-3-2020)

Study	Search	Meta	Included ES	No info	Final nES
Main parameters					
Allen		X	2	2 (excl)	
Asbrand	X		2		2
Griffith	X		2		2
Hofer	X		4	4 (excl)	
Milan	X		21	3	18
Nelson	X		2	2 (excl)	
Olino	X		2	2 (excl)	
Schwartz	X		3	3 (excl)	
Simons		X	1	1 (excl)	
Szwedo	X		2	2 (excl)	
Van Doorn	X		2	2 (excl)	

Note. Search = studies identified through database search, Meta = studies identified through included meta-analyses or reviews, ES = effect size, no info = not all standardized information available.

Defining priors – List of included effect sizes

Model A) mother-adolescent positive interaction behavior



= effect sizes excluded due to insufficient information

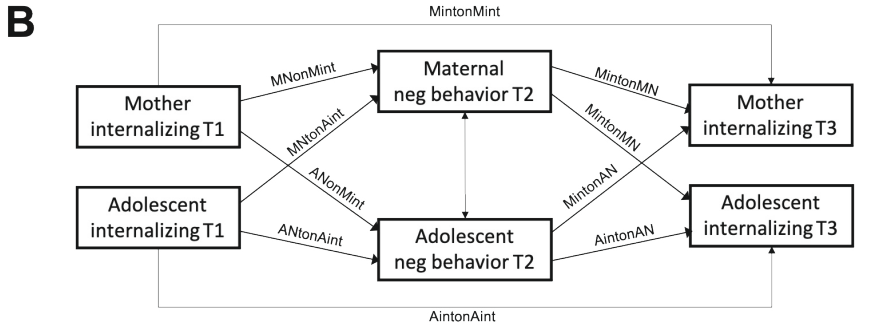
Study	Score	Analysis	stand β	SE/CI	unstand	Comment
M internalizing T1 → M positive interaction behavior T2						
*Simons (1993)	20/80	Correlation/ Path model	r = –.05/ β = -.394			No standardized SE
McCabe (2014)	35/80	Meta-analysis MA path model	ρ = –.18/ β = –.15	CI= –.21, –.14/ SE=.02		
Dietz (2008)	25/80	ANOVA (more vs. less dep)	B = –.21	t(92)=–2.02		
Milan (2018)	30/80	SEM	β = –.29	SE = .01 (unstandard); SD= 0.86 (M pos), SD= 5.56 (M int)	B = –.04	Pos + neg mixed
Van Doorn (2016)	15/80 25/80	Correlation/ Regression	*r* = –.23 β = –.22			No standardized SE

Study	Score	Analysis	stand β	SE/CI	unstand	Comment
			M internalizing T1 → A positive interaction behavior T2			
Milan (2018)	30/80	SEM	–.06	SE = .01 (unstandard); SD= 0.79 (A pos); SD= 5.56 (M int)	B = –.01	Pos + neg mixed
			A internalizing T1 → M positive interaction behavior T2			
Pinquart (2017)	40/80	Meta-analysis	*r* = –.06	CI=–.08; –.05		controlling for interaction T1
Dietz (2008)	25/80	ANOVA	M = 3.2 (dep, n=43)	SD = 0.5		Hi risk = parental diagn of interaction behavior disorder
			M = 3.3 (hi risk, n=28)	SD = 0.5		
			M = 3.3 (lo risk, n=41)	SD = 0.5		
Dadds (1992)	15/80	MANOVA (M= % of pos directed at child)	M = 61.9 (dep, n=18)	SD = 21.4		
			M = 71.6 (no-dep, n=16)	SD = 18.5		
Milan (2018)	30/80	SEM	–.16	SE = .01 (unstandard); SD= 0.86 (M pos), SD= 5.51 (A int)	B = –.01	
			A internalizing T1 → A positive interaction behavior T2			
Dadds (1992)	15/80	MANOVA	M = 28.1 (dep, n=18)	SD = 15.5 (dep)		
			M = 27.8 (no-dep, n=16)	SD = 20.1 (no-dep)		
Dietz (2008)	25/80	ANOVA	M = 2.4 (dep, n=43)	SD = 0.6		Hi risk = parental diagn of interaction behavior disorder
			M = 2.5 (hi risk, n=28)	SD = 0.4		
			M = 2.9 (lo risk, n=41)	SD = 0.4		
Milan (2018)	30/80	SEM	–.26	SE = .01 (unstandard); SD= 0.79 (A pos), SD= 5.51 (A int)	B = –.04	

Study	Score	Analysis	stand β	SE/CI	unstand	Comment
			M positive interaction behavior T2 → M internalizing T3			
Dietz (2008)	25/100	ANOVA	B = –.21	t(92)=–2.02		
Milan (2018)	30/100	SEM	–.29	SE = .01 (unstandard); SD= 0.86 (M pos), SD= 5.56 (M int)	B = –.04	Pos + neg mixed
Van Doorn (2016)	25/100	Regression	–.22 (sig)			No standardized SE
			M positive interaction behavior T2 → A internalizing T3			
Pinquart (2017)	60/100	Meta-analysis	*r* = –.06	CI=–.09; –.04		
Olino (2016)	50/100	Trajectories		*SE* = .79 (unstandard)	*B* =–1.28	No standardized info
Schwartz (2012)	70/100	Path model	Dep: –.09(girls) –.10(boys) Anx: –.02(girls) –.02(boys)	Dep (all unstand): SE=0.77, CI= –2.54;0.49; SD=7.97 (A dep), SD=0.69 (M pos) Anx (all unstand): SE=0.93, CI=-2.02; 1.63; SD=8.72 (A anx)	Dep: B= –1.03 Anx: B= –0.20	Dep = depression, anx=anxiety Girls = 87 (48.9%) Boys = 91
Hofer (2013)	55/100 80/100	Correlation/ Path model	r= –.11 /	SE=.04 (unstandard)	B = –.08	No standardized info
Milan (2018)	60/100	SEM	–.21	SE = .35 (unstandard); SD= 0.86 (M pos), SD= 5.51 (A int)	B = –1.04	Pos + neg mixed
Dietz (2008)	25/100	ANOVA	M = 3.2 (dep, n=43) M = 3.3 (hi risk, n=28) M = 3.3 (lo risk, n=41)	SD = 0.5 SD = 0.5 SD = 0.5		Hi risk = parental diagn of interaction behavior disorder (see comment above)

Study	Score	Analysis	stand β	SE/CI	unstand	Comment
Dadds (1992)	15/100	MANOVA	M = 61.9 (dep, n=18) M = 66.3 (no-dep, n=16)	SD = 21.4 SD = 14.9		
Griffith (2019)	55/100	Logistic regression (support + responsive-ness)	Odds ratio = 1.05 (support) OR = 0.79 (respons)	CI=0.87, 1.26 CI=0.63, 1.00		
A positive interaction behavior T2 → M internalizing T3						
Milan (2018)	60/100	SEM	–.005	SE = .53 (unstandard); SD= 0.79 (A pos); SD= 5.56 (M int)	B = –.04	Pos + neg mixed
A positive interaction behavior T2 → A internalizing T3						
Dadds (1992)	15/100	MANOVA	M = 28.1 (dep, n=18) M = 27.8 (no-dep, n=16)	SD = 15.5 (dep) SD = 20.1 (no-dep)		
Dietz (2008)	25/100	ANOVA	M = 2.4 (dep, n=43) M = 2.5 (hi risk, n=28) M = 2.9 (lo risk, n=41)	SD = 0.6 SD = 0.4 SD = 0.4		Hi risk = parental diagn of interaction behavior disorder (see comment above)
Milan (2018)	30/100	SEM	–.26	SE = .01 (unstandard); SD= 0.79 (A pos); SD= 5.51 (A int)	B = –.04	Pos + neg mixed

Model B) mother-adolescent negative interaction behavior



Study	Score	Analysis	stand β	SE/CI	unstand	Comment
			M internalizing T1 → M negative interaction behavior T2			
Dietz (2008)	25/80	ANOVA		t(92)=2.36	B = .24	No standardized info
Lovejoy (2000)	25/80	Meta-analysis	*d* = .40	CI= .31;.49		
Milan (2018)	30/80	SEM	β = .29	SE = .01 (unstandard); SD= 0.54 (M neg); SD= 5.56 (M int)	B = .04	Pos + neg mixed
			M internalizing T1 → A negative interaction behavior T2			
Milan (2018)	30/80	SEM	.06	SE = .01 (unstandard); SD= 0.63 (A neg); SD= 5.56 (M int)	B = .01	Pos + neg mixed
			A internalizing T1 → M negative interaction behavior T2			
Pinquart (2017)	40/80	Meta-analysis	*r* = .04	CI= –.03; .11		controlling for interaction T1

Study	Score	Analysis	stand β	SE/CI	unstand	Comment
Dietz (2008)	25/80	ANOVA	M = 2.2 (dep, n=43) M = 2.1 (hi risk, n=28) M = 2.0 (lo risk, n=41)	SD = 0.9 SD = 0.8 SD = 0.8		Hi risk = parental diagn of interaction behavior disorder
Dadds (1992)	15/80	MANOVA	M = 10.3 (dep, n=18) M = 3.9 (no-dep, n=16)	SD = 13.4 SD = 8.7		
Szwedo (2017)	35/80	Regression	.15			No standardized SE
Milan (2018)	30/80	SEM	.16	SE = .01 (unstandard); SD= 0.54 (M neg); SD= 5.51 (A int)	B = .01	
Asbrand (2017)	35/80	MANOVA	M = 3.32 (anx, n=27) M = 2.59 (non-anx, n=27)	SD = 1.36 SD = 1.37		
A internalizing T1 → A negative interaction behavior T2						
Dadds (1992)	15/80	MANOVA	M = 17.8 (dep, n=18) M = 12.1 (no-dep, n=16)	SD = 16.9 (dep) SD = 17.8 (no-dep)		
Jackson (2011)	15/80	regression		SE = 0.169 (unstandard)	B = − 2 . 1 2 (dep = more anger)	No standardized info
Dietz (2008)	25/80	ANOVA	M = 2.7 (dep, n=43) M = 2.3 (hi risk, n=28) M = 2.1 (lo risk, n=41)	SD = 1.0 SD = 0.9 SD = 0.8		Hi risk = parental diagn of interaction behavior disorder

Study	Score	Analysis	stand β	SE/CI	unstand	Comment
Milan (2018)	30/80	SEM	.26	SE = .01 (unstandard); SD= 0.63 (A neg); SD= 5.51 (A int)	B = .04	
Asbrand (2017)	35/80	MANOVA	M = 3.88 (anx, n=27) M = 3.20 (non-anx, n=27)	SD = 1.56 SD = 1.31		
M negative interaction behavior T2 → M internalizing T3						
Dietz (2008)	25/100	ANOVA	B = .24	t(92)=2.36		
Milan (2018)	30/100	SEM	.29	SE = .01 (unstandard); SD= 0.54 (M neg); SD= 5.56 (M int)	B = .04	Pos + neg mixed
M negative interaction behavior T2 → A internalizing T3						
Pinquart (2017)	60/100	Meta-analysis	*r* = .09	CI=.07; .11		
Olino (2016)	50/100	Trajectories		*SE* = .81; (unstandard)	*B* = 1.23	No standardized info
Schwartz (2012)	70/100	Path model	Dep: .10 (girls) .12(boys) Anx: .12 (girls) .13(boys)	Dep (all unstand): SE=0.91, CI= –.41; 3.17 Anx (all unstand): SE=1.10, CI= –0.37;3.93	Dep: B=1.38 Anx: B=1.78	Dep = depression, anx=anxiety Girls = 87 (48.9%) Boys = 91
Hofer (2013)	55/100	Correlation/	Neg: r=–.08 Anger:	SE=.06 (unstandard)	B = –.14	No standardized info
	80/100	Path model	r=–.14 /			
Szwedo (2017)	55/100	Regression	.15			No standardized SE
Dietz (2008)	25/100	ANOVA	M = 2.2 (dep, n=43) M = 2.1 (hi risk, n=28) M = 2.0 (lo risk, n=41)	SD = 0.9 SD = 0.8 SD = 0.8		Hi risk = parental diagn of interaction behavior disorder

Study	Score	Analysis	stand β	SE/CI	unstand	Comment
Dadds (1992)	15/100	MANOVA	M = 10.3 (dep, n=18) M = 3.9 (no-dep, n=16)	SD = 13.4 SD = 8.7		
Milan (2018)	60/100	SEM	.21	SE = .35 (unstandard); SD= 0.54 (M neg); SD= 5.51 (A int)	B = 1.04	Pos + neg mixed
Weymouth (2016)	30/100	Meta-analysis	.26	SE = .026		
Griffith (2019)	55/100 60/100	Logistic regression (criticism, conflict)	Odds ratio = 1.04 (criticism) OR = 1.33 (conflict)	CI=0.87, 1.25 CI=1.10, 1.61		Only for conflict also controlling for parental dep (maybe only use conflict then?!)
A negative interaction behavior T2 → M internalizing T3						
Milan (2018)	60/100	SEM	.005	SE = .53 (unstandard); SD= 0.63 (A neg); SD= 5.56 (M int)	B = .04	Pos + neg mixed
A negative interaction behavior T2 → A internalizing T3						
Allen (2006)	50/100 70/100	Correlation/ regression	r = .33 *β = .22*			No standardized SE
Dadds (1992)	15/100	MANOVA	M = 17.8 (dep, n=18) M = 12.1 (no-dep, n=16)	SD = 16.9 (dep) SD = 17.8 (no-dep)		
Jackson (2011)	15/100	regression		SE = 0.169 (unstandard)	B = −2.12 (dep= more anger)	No standardized info
Milan (2018)	30/100	SEM	.26	SE = .01 (unstandard); SD= 0.63 (A neg); SD= 5.51 (A int)	B = .04	Pos + neg mixed

Weight distribution and scoring system for informative priors

Defining informative priors: Scoring individual studies to distribute weight

Study	T1-T2	lag	cT1	obs	Age	$M_{dep+anx\ (or\ anx)}$	$A_{dep+anx\ (or\ anx)}$	cov_s	cov_i	comm	MA	Score
Lagged parameter	*10*	*5*	*20*	*15*	*10*	*10*		*5*	*5*	*10*	*10*	*100*
Lovejoy (2000)				x							x	25
*Simons (1993)	x		n/a		x							20
McCabe (2014)				x		x					x	35
Pinquart (2017)	x		x				x			x	x	60
Weymouth (2016)							x			x	x	30
Allen (2006)	x	x	x	x	x					x		70
Asbrand (2017)	x			x			x					35
Dadds (1992)				x								15
Dietz (2008)				x		x						25
Griffith neg (2019)	x		x	x				x		x		60
Griffith pos (2019)	x		x	x						x		55
Hofer (2013)	x		x	x	x		x		x	x		80
Jackson (2011)				x								15
Milan (2018, only cs)				x				x	x	x		30
Milan (2018)	x		x	x				x	x	x		60
Nelson (2017)	x			x					x			30
Olino (2016)	x		x	x				x				50
Schwartz (2012)	x		x	x	x		x	x				70
Szwedo (2017)	x		x	x						x		55
Van Doorn (2016)				x				x	x			25

Note. T1-T2 = longitudinal assessment, lag = same time lag used (for longitudinal studies), cT1 = controlling for T1 symptoms (for longitudinal studies), obs = observational assessment of parent-adolescent interaction, age = age range early adolescence, *N* = sample size, M = maternal, A = adolescent, year = publication year, cov_s = controlling for parental symptoms, cov_i = controlling for other interaction behaviors, comm = community sample, MA = meta-analysis.
*study include in aforementioned meta-analysis
x = indicates that the category is met
grey-marked studies = excluded, no standardized information available

CHAPTER 5

The Future is Present in the Past: A Meta-Analysis on the Longitudinal Associations of Parent-Adolescent Relationships with Peer and Romantic Relationships

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(preregistered at PROSPERO: bit.ly/3H8O7Ux)

ABSTRACT

Positive peer and romantic relationships are crucial for adolescents' positive adjustment and relationships with parents lay the foundation for these relationships. This longitudinal meta-analysis examined how parent-adolescent relationships continue into later peer and romantic relationships. Included longitudinal studies (k=54 involving peer relationships, k=38 involving romantic relationships) contained demographically diverse samples from predominantly Western cultural contexts. Multilevel meta-regressions indicated that supportive and negative parent-adolescent relationships were associated with supportive and negative future peer and romantic relationships. Meta-analytic structural equation modeling (k=54) indicated that supportive parent-adolescent relationships unidirectionally predicted supportive and negative peer relationships, while negative parent-adolescent relationships were bidirectionally associated with supportive and negative peer relationships. Maintaining mutually supportive relationships with parents may help adolescents to develop positive social relationships.

Keywords: Relationship transmission; Parent-adolescent relationship; Meta-analysis

Author contributions

SuS, HH, TK, and WM conceptualized the study. SuS and HH conducted the systematic review and coded all studies. SuS performed the statistical analyses and drafted the manuscript. All authors provided feedback on the manuscript.

THE FUTURE IS PRESENT IN THE PAST: A META-ANALYSIS ON THE LONGITUDINAL ASSOCIATIONS OF PARENT-ADOLESCENT RELATIONSHIPS WITH PEER AND ROMANTIC RELATIONSHIPS

Adolescence is a crucial developmental period that is marked by rapid physical, cognitive, and social-emotional changes (Dahl, Allen, Wilbrecht, & Suleiman, 2018). Achieving autonomy, gaining more egalitarian relationships with parents, as well as forming and maintaining high quality relationships with peers and romantic partners are among the most salient developmental tasks during this period. As adolescents navigate through changing social demands, their social relationships undergo major modifications and maturation. While parent-adolescent relationships become more egalitarian and reciprocal (Branje, 2018), peer and romantic relationships become increasingly intimate, committed, and mutually responsive (Bagwell & Bukowski, 2018; Furman & Buhrmester, 1992; Lantagne & Furman, 2017). Maintaining positive relationships with parents throughout adolescence is key to establishing positive relationships with other social partners. Social interactional and social cognitive theories (Baldwin, 1992; Bandura, 1977; Furman & Collibee, 2018; Hartup, 1979; Kaufman, Kretschmer, Huitsing, & Veenstra, 2020) commonly emphasize how the family context shapes later social relationships such as peer and romantic relationships. If adolescents fail to form positive relationships with peers and romantic partners, they are at risk for health and adjustment problems, such as increased mortality, loneliness, and depression (Cohen, 2004; Schwartz-Mette, Shankman, Dueweke, Borowski, & Rose, 2020). The role of the parent-adolescent relationship in shaping adolescents' peer and romantic relationships is thus also important for their general functioning.

Research supports theoretical perspectives by demonstrating that adolescents who have more positive parent-adolescent relationships indeed have relatively more positive peer and romantic relationships in adolescence and young adulthood (see Meeus, 2016 for an overview). However, large heterogeneity in effects sizes and assessed relationship dimensions at different ages renders it difficult to draw coherent conclusions about the strength of these associations and how they might differ by age. Furthermore, previous research mainly focused on unidirectional associations from parent-adolescent to future peer or romantic relationships, ignoring potential reversed associations from peer or romantic relationships to parent-adolescent relationships that might gain particular importance during adolescence. By synthesizing available information from longitudinal samples, the current meta-analysis aims to (1) examine how interindividual differences in core dimensions of parent-adolescent relationships predict and precede subsequent interindividual differences in peer and romantic relationships, (2) examine potential bidirectional associations between parent-adolescent and other social relationships, and (3) investigate how these associations differ by age.

Parent-Adolescent Relationships as Foundation for Future Social Relationships

Throughout the lifespan, people engage in relationships with various social partners that fulfill different functions. The very first relationships that children form are those with parents, which are involuntary, closed, and hierarchical by nature (Laursen & Bukowski, 1997). Lifespan and "social mold" theories propose that parent-child relationships influence not only the child's behavioral, emotional, and cognitive development, but also the child's social development (Hartup, 1979), including the formation and development of social relationships that are particularly crucial during adolescence. Relationships with parents at this time continue to affect social developmental processes (Ali, Khatun, Khaleque, & Rohner, 2019; Collins & Laursen, 2004), potentially even more so as relationships with peers and romantic partners become increasingly important and acquire more mature relational functions.

Contrary to parent-adolescent relationships, peer relationships are voluntary, egalitarian, and temporary in nature (Laursen & Bukowski, 1997; Laursen & Veenstra, 2021). These relationships prepare adolescents for interactions among equals and the roles and expectations of adulthood. Early romantic relationships are similar to peer relationships in that both satisfy the adolescent's need for companionship (Shaver & Hazan, 1988). Towards late adolescence, peer and romantic relationships grow more intimate, and romantic relationships begin to take over relationship functions from parent-child relationships (Hazan & Shaver, 1987; Lantagne & Furman, 2017; Selfhout, Branje, & Meeus, 2009; Selman, 1989). As peer and romantic relationships resemble parent-adolescent relationships in their characteristics and functions, parent-adolescent relationships are suggested to provide a direct foundation for these developing social relationships.

Theoretical rationale. In relationships with peers and romantic partners, adolescents apply interaction patterns that reflect relationships with their parents. Such continuity of relationship behaviors is often attributed to internal cognitive models or schemas that adolescents acquire in relationships with parents (Burks & Parke, 1996; Furman & Collibee, 2018). Schemas guide adolescents' social behavior by helping them to understand social cues and form expectations about others' behaviors, and are thus ultimately generalized to other relationships (Baldwin, 1992; Dodge & Pettit, 2003). Additionally, adolescents observe and enact early interactions with parents and model these learned behaviors in future interactions with peers or romantic partners (Bandura, 1977). According to social systems theory (e.g., Hartup, 1979), transfer from one social relationship to another also results from the interdependence between different social systems in which adolescents are embedded. Within the family system, parent-adolescent relationships provide the foundation that allows adolescents to explore their peer environment (Bronfenbrenner & Morris, 2006). As social systems are interrelated, the dynamics that occur within one system, such as structures,

roles, patterns, or power relations, are therefore thought to spill over from one relationship to another (Erel & Burman, 1995). In that way, changes in the parent-adolescent system, which are fundamental during adolescence, can carry over to other relational systems with peers through modified emotions or behaviors (Benson, McWey, & Ross, 2006; Kaufman et al., 2020). Spillover effects from relationships with parents to peer or romantic relationships may be particularly relevant in adolescence as this period is often considered a sensitive period for social learning (Dahl et al., 2018; Laursen & Veenstra, 2021).

Internal schemas are based on aspects of relationship quality that can be categorized into broad dimensions of relationships. Three core dimensions that become particularly important during adolescence when social competence and understanding increase are support, control, and negative interaction (Baumrind, 1991; Furman & Buhrmester, 1992; Furman & Collibee 2018). Supportive parent-adolescent relationships, including aspects such as warmth, responsiveness, and nurturance, help adolescents to acquire positive relationship schemas, to develop prosocial, caring attitudes (Eisenberg, VanSchyndel, & Hofer, 2015), and to build interpersonal competence (Conger et al., 2000). They thus provide a positive foundation from which adolescents can explore other relationships and meet new relationship partners with trust and affection (Collins & Laursen, 2004). As friends become increasingly important sources of support during adolescence (Blos, 1967; Bagwell & Bukowski, 2018), adolescents are likely to generalize positive cognitive models and behaviors to peer and romantic relationships, which facilitates supportive social relationships (Rubin et al., 2015).

Similarly, individuation theory (Fousiani, Van Petegem, Soenens, Vansteenkiste, & Chen, 2014; Youniss & Smollar, 1985) posits that as adolescents strive for autonomy, optimal social development requires maintaining both high levels of support and increasingly lower levels of control in parent-adolescent relationships. Equality and independence become important features of social relationships as they promote adolescent well-being and self-validation (Bukowski, Buhrmester, & Underwood, 2011). Negotiating and balancing control in parent-adolescent relationships are therefore important practice grounds for future egalitarian relationships with peers and romantic partners. Unbalanced controlling relationships, characterized by dominance, power, and lack of autonomy, may predict internalizing and externalizing problem behaviors (Pinquart, 2017a; Pinquart, 2017b).

Negative interaction includes aspects such as conflict and hostility, but also physical and emotional abuse. While conflict interactions allow adolescents to integrate different expectations and can thus promote cognitive development and well-being through negotiation and self-reflection, frequent and intense negative interactions can strain social bonds and result in poor developmental and

health outcomes (Laursen & Hafen, 2010). These processes may increase adolescents' aggressive behavior and decrease their interpersonal competence, which in turn increase negative interaction in later relationships with peers and partners (Conger et al., 2000; Patterson, 1982; Rubin et al., 2015).

Bidirectional effects. Theoretical models on the continuity of social relationships are often based on "social mold" models (Hartup, 1979; Collins & Sroufe, 1999; Furman & Collins, 2009) and assume unidirectional pathways from parents to adolescents (i.e., parent-to-adolescent effects), in which experiences with parents provide the foundation for later experiences with peers and romantic partners. During adolescence, children strive for more independence from parents and spend more time outside the family with friends and romantic partners (Branje, Mastrotheodoros, & Laursen, 2021). As peer and romantic relationships become increasingly important, they can also play a role in transforming parent-adolescent relationships. Transactional models (Bell, 1968; Sameroff, 2009) propose that children actively shape their environment and are thus not only influenced by their parents, but also influence their parents. This may apply even more so in adolescence when major developmental changes such as increasing social understanding, competence, and independence motivate changes in social relationships (Laursen & Bukowski, 1997; Branje et al., 2021; Lantagne & Furman, 2017). In maturing peer and romantic relationships, adolescents practice equality and autonomy interactions, which allow them to acquire new relational schemas and interpersonal skills that are generalized to other relationships (Graziano, 1984). The need for similarity with peers during this period further renders adolescents particularly susceptible to peer influence and the adaptation of their own behaviors and attitudes to those of their peers and romantic partners (Laursen & Veenstra, 2021). In line with the spillover hypothesis (Erel & Burman, 1995), peer or romantic relationships may also affect adolescents' general emotional states through which interaction patterns can carry over from one relational system to another, resulting in bidirectional associations between parent-adolescent and peer or romantic relationships.

Empirical evidence. Research supports the theoretical continuity of parent-child relationship quality into peer and romantic relationships. Evidence from meta-analyses and reviews found small to moderate positive associations between early mother-child attachment and peer relationships in childhood (Pallini, Baiocco, Schneider, Madigan, & Atkinson, 2014; Schneider, Atkinson, & Tardif, 2001), as well as moderate positive associations between parent and peer attachment in adolescence (Gorrese & Ruggieri, 2012). These findings are extended by studies reporting longitudinal associations between parent-adolescent relationships and peer or romantic relationships in adolescence and emerging adulthood (for a narrative review, see Meeus, 2016). For the extreme negative end of the relationship quality distribution, meta-analytic evidence showed links between violent parent-child

relationships and violent romantic relationships in adulthood through witnessing interparental violence and experiencing parental violence (Smith-Marek et al., 2015; Stith et al., 2000).

Most previous meta-analytical evidence focused on perceived attachment or the affective quality of relationships, and not on how the core dimensions of support, control, and negative interaction are transferred from parent-adolescent relationships to peer or romantic relationships in adolescence and emerging adulthood. Furthermore, these meta-analyses focused solely on transfer of similar features of relationship dimensions and thus do not allow for conclusions about whether these aspects also generalize to other relationship dimensions. Perspectives on family socialization (Grusec & Davidov, 2010) indicate that each relationship dimension holds distinct functions and thus, associations might be strongest among similar relationship dimensions with parents and peers or partners. Other perspectives propose that cognitive models and representations are relatively global and that specific relationship aspects thus affect relationship quality more generically (Conger, Cui, Bryant, & Elder, 2000; Overall, Fletcher, & Friesen, 2003; Youniss & Smollar, 1985). Hence, specific relationship dimensions might influence not only the same, but also other relationship dimensions (e.g., highly supportive parent-adolescent relationships might not only predict more supportive, but also less negative peer or romantic relationships). The few studies assessing continuity between parent-adolescent and other social relationships across different relationship dimensions remain inconsistent. While some research suggests associations of more negative parent-adolescent relationships with less supportive future peer (Gayman, Turner, Cislo, & Eliassen, 2011) and romantic relationships (Slominski et al., 2011), others failed to replicate these findings (Andrews et al., 2000). The current study extends previous meta-analyses by examining how the three core relationship dimensions support, negative interaction, and control in parent-adolescent relationships continue into later peer and romantic relationships, both within as well as across relationship dimensions.

Furthermore, while previous studies mainly focused on relationships with parents during (early) childhood, this meta-analysis focuses on relationships with parents during adolescence and emerging adulthood. Adolescence is a particularly important developmental period during which social relationships undergo major changes. As relationships with parents mature and become more similar to relationships with peer and romantic partners, aspects of these changing relationships may be more likely to transfer to peer and romantic relationships than aspects of earlier parent-child relationships (Schneider et al., 2001).

Finally, most studies to date focused on theoretical conceptions emphasizing that parent-adolescent relationships constitute the foundation of later social relationships in adolescence and emerging adulthood and thus mainly focused on unidirectional associations between parent-adolescent and

peer or romantic relationships. However, they often combined cross-sectional and longitudinal evidence, which does not allow for conclusions on directionality and temporal order of these associations. Moreover, changes of social relationships and the increasing importance of peers and romantic partners during adolescence suggest that peer and romantic relationships might influence and thus further transform parent-adolescent relationships. Initial research supports bidirectional links between peer and parent-adolescent relationships in adolescence, with associations between support from parents to peers decreasing from early to late adolescence and associations between negative interaction from parents to peers increasing (De Goede, Branje, Delsing, & Meeus, 2009). Another study, however, failed to detect reversed associations between support from parents and support from peers in adolescence (Luyckx, Missotten, Goossens, & Moons, 2012). Apart from these few inconsistent findings, longitudinal studies to date rarely emphasized the potential lagged effects of peer and romantic relationships on later parent-adolescent relationships. Including such reversed associations is crucial to disentangle whether the associations of parent-adolescent relationships with peer or romantic relationships in adolescence are stronger for parent-to-adolescent or adolescent-to-parent effects. In this meta-analysis, we therefore aimed to examine whether associations between parent-adolescent and later adolescent or emerging adult peer and romantic relationships are unidirectional, and thus provide support for parents as foundation for social relationships, or whether they are bidirectional, and thus provide support for transactional processes.

Moderators of the Associations between Parent-Adolescent and Peer and Romantic Relationships

Time effects. As the time interval between measurement occasions increases, the associations of parent-adolescent relationships with peer and romantic relationships are likely to become smaller. If parent-adolescent relationships are measured in close proximity to peer or romantic relationships, there may be fewer intervening variables and thus parent-adolescent relationships may be more strongly associated with peer or romantic relationships. If they are measured further apart, events or changes in parent-adolescent relationships might occur that alter the quality of peer and romantic relationships, and weaken the associations with parent-adolescent relationships. *Revisionist* perspectives support this idea and propose that current experiences decrease the effects of previous relationship experiences with parents (Fraley & Roisman, 2015).

Alternatively, previous relationships with parents might provide long-lasting working models that continue to shape adolescent development (Sroufe, Egeland, & Kreutzer, 1990). This perspective suggests that the effects of previous parent-adolescent relationships are *enduring*, indicating that the associations with peer or romantic relationships stabilize over time (Fraley & Roisman, 2015).

As both revisionist and enduring perspectives emphasize the importance of time in how parent-adolescent relationships continue into peer and romantic relationships, this meta-analysis accounts for the potentially varying effects of time in the continuity of social relationships.

Adolescent age. As autonomy and voluntary interactions gain more importance, the main focus of adolescent relationships gradually shifts from parents to peers and romantic partners (e.g., Branje et al., 2021). Although parents remain relevant sources for support, adolescents are more likely to turn to peers or romantic partners for support (Bagwell & Bukowski, 2018; Youniss & Smollar, 1985). As adolescents gradually become more autonomous from their parents, parents are less involved in adolescent lives. Their relationships with adolescents might thus become less influential in predicting future peer and romantic relationships over time. Peer and romantic relationships, however, grow more important as adolescents get older and provide optimal grounds to apply interaction patterns based on egalitarian principles (Laursen & Bukowski, 1997; Lantagne & Furman, 2017). As such, they might also become more influential in predicting later relationships with parents over time.

On the other hand, parent-adolescent relationships become more reciprocal and egalitarian throughout adolescence (Eccles, Wigfield, Buchanan, & Flanagan, 1993; Hadiwijaya, Klimstra, Vermunt, Branje, & Meeus, 2017). As they realign towards greater horizontality, they more closely resemble peer and romantic relationships. Similarly, processes and functions that are specific to romantic relationships only emerge later in adolescence and young adulthood (Furman & Collins, 2009). Particularly early romantic relationships often resemble close peer relationships more than intimate sexual relationships. As peer and romantic relationships become more important and more similar to parent-adolescent relationships over time, the links between parent-adolescent and peer or romantic relationships might also become stronger with age.

Longitudinal studies generally support associations of parent-adolescent relationships with later peer or romantic relationships in early adolescence (Rice & Mulkeen, 1995; Kochendorfer & Kerns, 2017), mid-adolescence (Giordano et al., 1998; Kaufman-Parks et al., 2018), and late adolescence or early adulthood (de Goede et al., 2009; Slominski et al., 2011). However, these studies do not permit conclusions about whether and how these associations change over time. While research on how parent-adolescent relationships continue into peer and romantic relationships over time remains scarce, initial findings are in line with separation theories, suggesting that these associations decrease for support, but not for negative interaction or control (e.g., De Goede et al., 2009). Contrasting findings are in line with realignment theories, indicating that the associations between parent-adolescent relationships and romantic relationships increase as adolescents grow older (Kaufman-

Parks, DeMaris, Giordano, Manning, & Longmore, 2018; Meeus, Branje, van der Valk, & de Wied, 2007). Other research, however, did not detect any age effects (De Goede, Branje, van Duin, Vandervalk, & Meeus, 2012). Inconsistent findings, variation in time lags between measurements, and the inclusion of different age groups across studies render it difficult to draw coherent conclusions about the strength of associations between parent-adolescent and peer or romantic relationships across adolescence. This meta-analysis addresses these issues by investigating how time lag between measurements and adolescent age might moderate the effects of parent-adolescent relationships on peer and romantic relationships. Investigating temporal differences will provide insights into short- or long-term effects of parent-adolescent relationship quality.

The Present Study

This longitudinal meta-analysis aims to synthesize and expand previous heterogenous evidence on how core dimensions of parent-adolescent relationships continue into future peer and romantic relationships. This is needed because previous meta-analyses often did not allow for conclusions on the temporal order of associations between parent-adolescent relationships and subsequent peer and romantic relationships, and longitudinal findings largely vary regarding the magnitude of associations between parent-adolescent and other social relationships, both within the same and particularly across different relationship dimensions. In the present meta-analysis, we applied two distinct, but complementary statistical approaches to provide generalizations about the extent of relationship continuity across core dimensions of social relationships: First, we used multilevel meta-analytic regressions (Cheung, 2014) to examine how support, negative interaction, and control in parent-adolescent relationships are related to support, negative interaction, and control in future peer and romantic relationships (see Figure 1A). This design allowed us to include all existing information within and across studies while accounting for multiple effect sizes from the same study. We expected that support, negative interaction, and control in parent-adolescent relationships continue into future peer and romantic relationships, within the same and across relationship dimensions.

Second, we used meta-analytic structural equation modeling (MASEM; Jak, 2015) to investigate how support, negative interaction, and control in parent-adolescent relationship predict relative change in support, control, and negative interaction in future peer relationships, controlling for over-time stability, concurrent correlations, and potential lagged associations of peer on parent-adolescent relationships (see Figure 1B). Unlike the multilevel approach, MASEM allowed us to model structural pathways. We expected that support, negative interaction, and control in parent-adolescent relationships predict peer relationships above and beyond over-time stability, concurrent, and reversed correlations.

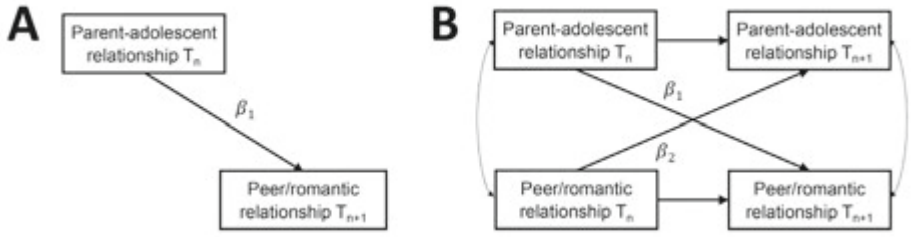

Figure 1. Graphical representation of the conceptual three-level regression model (A) in which β1 denotes the parent-to-peer regression path, and the cross-lagged panel model (B) in which β1 and β2 denote the parent-to-peer and peer-to-parent regression paths, respectively, controlling for autoregressive stability paths and concurrent correlations.

Based on contrasting theories and findings, it remains unclear how and to what extent the links between parent-adolescent relationships and future peer or romantic relationships change as measurements are further apart in time or as adolescents get older. We therefore examined whether the associations of parent-adolescent relationship quality with peer and romantic relationship quality vary depending on time interval between measurement occasions (i.e., short-term vs. long-term effects) and adolescent age. Due to the increasing importance and influence of peer and romantic relationships, potential reversed associations were expected to become stronger as adolescents got older. In addition, we explored other sample and study characteristics that may explain variation within and across studies.

METHODS

The design, research aims, and hypotheses of this meta-analysis were preregistered (https://www.crd.york.ac.uk/PROSPERO/display_record.php?RecordID=103492). All hypotheses were confirmatory in nature, except analyses involving sample and study characteristics, which were explored as potential moderators.

Eligibility Criteria, Data Sources, and Study Selection

Eligibility criteria. Our literature search aimed to identify studies that examined longitudinal associations between parent-adolescent relationship quality and adolescents' peer or romantic relationship quality. We used three main criteria to select studies: First, selected studies included an adolescent or emerging adult sample at the earliest measurement. Studies were excluded if the initial sample at T_n included participants younger than 10 years or older than 25 years. Second, studies assessed relationship aspects for both parent-adolescent and peer or romantic relationships that correspond to core dimensions of relationship quality (i.e., support, negative interaction, or

control). We excluded measures that did not focus specifically on parents, such as measures that focused on family more generally, which could include other family members, aspects that did not constitute relationship quality, such as peer affiliation or peer victimization, or aspects that we could not assign to core dimensions, such as general relationship quality (i.e., composite measures of various core components) or attachment. Studies that measured attachment in the traditional sense (e.g., secure or avoidant attachment) were excluded. Studies that used the term attachment to refer to aspects of relationship quality (e.g., closeness measured with the Inventory of Parent and Peer Attachment; Armsden & Greenberg, 1987) were included as measures of support. Retrospective reports on relationship quality with parents and peers or romantic partners that dated back to > 1 year were excluded as they could introduce potential bias and distortions and thus, do not accurately reflect relationship quality at that time. Third, studies included a longitudinal study design in which parent-adolescent relationship assessments preceded peer and romantic relationship assessments in time. Lastly, we set no language restrictions and if needed, received help from native speakers to decode non-English articles.

Literature search. Our strategy to identify relevant articles was fourfold: First, we searched for peer-reviewed journal articles in the databases ERIC, PsycArticles, PsycInfo, Scopus, and Web of Science for all years until August 2018. We used a combination of search terms that reflected (1) an adolescent or young adult sample, (2) relationship dimensions with parents, peers or romantic partners, and (3) a longitudinal study design. The exact search strings for each of the databases are provided in the supplemental material. Second, we searched the table of contents of the top ten journals deemed most likely to publish studies on adolescent interpersonal relationships. Specifically, we manually searched for articles in the most recent issues and online first or early view sections of the top ten journals identified by Web of Science and SCOPUS as having the most records concerning our search terms. Third, we searched for articles by examining the reference sections of relevant review articles (e.g., Meeus, 2016) and of all articles included in current meta-analysis. Finally, we searched for unpublished materials, such as dissertations, conference proceedings, policy documents, and other reports, in 15 databases (see the supplemental material for more details on the searched databases and screening process).

Our search strategy for published studies yielded 6,753 unique journal articles (see Figure S1). Two subsequent screening procedures of titles and abstracts as well as full texts by the first and third author resulted in an inclusion of 145 articles. Interrater reliabilities on 25% of all studies were good (κ = .91 and κ = .89, respectively). Diverging assessments were discussed among the co-authors until consensus was reached. More than half of the studies (59%) did not report the required effect sizes or reported effect sizes derived from multivariate analyses. In these cases, we contacted the

authors to provide the missing zero-order correlations between continuous study variables, biserial correlations between continuous and dichotomous study variables, and polychoric correlations between dichotomous study variables. If necessary, reminders were sent after two weeks. Of all 97 authors contacted, 47 provided the requested effect sizes, 22 could not provide the requested correlations, and 28 did not reply to our request. This led to an exclusion of 42 studies.

Our search strategy for unpublished materials yielded an additional 1,381 documents (see Figure S1). Two subsequent screening procedures of titles and abstracts as well as full texts by the first author and two graduate students resulted in an inclusion of 67 documents. Interrater reliabilities on 10% of all studies were good (κ = .81-.92 and κ = .88, respectively). Of these 67 documents, 27 were excluded because the relevant correlations had already been published. For studies that did not report the required effect sizes (n = 11), we contacted the authors using the same strategy as reported above for journal articles. One study was excluded because no up-to-date author information could be retrieved. Of all 10 authors contacted, three provided the requested effect sizes, but two of those studies had to be excluded because the relationship constructs combined several relationship dimensions. Seven authors did not reply to our request or could not provide the requested correlations. Hence, nine studies were excluded. All included unpublished studies were dissertations.

As common in longitudinal research, we identified several studies that used the same datasets as other included studies (k = 65, including 42 published and 23 unpublished studies, on 12 different datasets). To ensure that we did not include overlapping data, we excluded all duplicated information based on a three-fold strategy: First, of all studies that used the same dataset, we included the study with the earliest assessment, unless another study on the same dataset used a sample size > 25% of the previous study. Second, if two studies used the same first assessment and sample size, we included the study that contributed the most effect sizes. If the same data was used in published and unpublished materials, we included published studies to increase methodological soundness. Third, if other studies on the same dataset contributed effect sizes that were not assessed in the first study (e.g., additional relationship dimensions or waves), we also included those unique effect sizes. This strategy led to a final inclusion of 87 studies on 80 unique datasets with 100 independent samples, of which 54 studies involved peer outcomes and 38 studies involved romantic outcomes.

Data Extraction

A structured coding manual guided the coding procedures. To obtain estimates of coder reliability, the first and third author coded 25% of all studies. The interrater reliability was good to perfect (κ = .98 to κ = 1.0). Diverging assessments were discussed among co-authors until consensus was

reached. After obtaining agreement, all remaining studies were coded by either one of the two first authors. Unpublished materials were coded by two graduate students (κ = 1.0 on 50% of all studies). More information on the coding procedures and decisions, including interrater reliabilities for each coded category, can be found in the supplemental material. Cases for which we could not clearly retrieve the required information were coded as missing. Table S1 in the supplemental material displays the sample characteristics of all studies for peer (1A) and romantic outcomes (1B).

Publication. We coded publication status, the year of publication, the journal impact factor, and the associated quartile ranking (i.e., higher impact factors and lower quartile rankings, e.g., Quartile 1, indicate higher quality journals) based on the year of publication. In cases in which impact factor and ranking were unknown, we used the impact factor and quartile of the year closest to the year of publication.

Study procedures. We coded information regarding recruitment and waves. Recruitment characteristics included geographical region of the sample (i.e., North America, Western Europe, South America, Eastern Europe, Asia, or Oceania), recruitment location (i.e., local or national), recruitment strategy (i.e., convenience sampling, selective sampling, or random sampling), and percentage of attrition across waves. Wave characteristics included number of waves measuring parent-adolescent relationship and peer or romantic relationship, respectively, and time lag between measures.

Sample. Sample characteristics at the assessment of the parent-adolescent relationship at time point T_n included total sample size, mean age of adolescents, percentage of adolescent boys, percentage of racial-ethnic minorities within the national context, percentage of participants living with both or single parents, and type of population (i.e., community, marginalized, or clinical). If studies reported an age range of the participating adolescents, we estimated the (weighted) average. If studies reported only the grade level(s) of the participating adolescents but no age range (k = 7 for peer, k = 9 for romantic relationships), we estimated age based on the country's general age range per grade level. Marginalized population in this study refers to populations with low socio-economic status or racial-ethnic minorities as specified per study. Sample characteristics at the assessment of the peer or romantic relationship at T_{n+1} included sample size, mean age of adolescents, degree of friendship (i.e., peers, close friend, or best friend), type of romantic sexuality (i.e., straight, sexual minorities only, or all sexual orientations included), and whether the same peers or romantic partners were assessed across waves.

Measures. Measure characteristics regarding the relationship constructs (i.e., with parent, peer, or romantic partner) included type of parent-adolescent relationship (i.e., both parents, mother, or father), number of relationship constructs, relationship dimension (i.e., support, negative interaction, or control), shared informant (i.e., whether the same assessment method was used for parent-adolescent and peer or romantic relationship, such as same reporter), instrument (i.e., type of questionnaire or task), and the interrater reliability of the measure. Relationship aspects such as warmth, responsiveness, nurturance, or prosocial behavior were classified as support; relationship aspects such as conflicts, arguments, hostility, or abuse were classified as negative interaction; and relationship aspects such as authority, dominance, power, and lack of autonomy were classified as control. To avoid additional levels of dependency in our effect sizes, we used weighted means to average multiple effect sizes on the same wave and construct across different types of parent-adolescent relationship (i.e., mother-adolescent and father-adolescent relationship), different reporters (e.g., adolescent reporter, rater), or different instruments.

Effect sizes. We used Pearson's correlations of continuous relationship scores with at least one time lag between assessments to operationalize the longitudinal associations between parent-adolescent relationships and peer or romantic relationships. If studies provided correlations for more than two waves, we recorded all available correlation coefficients. If available, we recorded all stability coefficients between parent-adolescent and peer relationship, all concurrent correlation coefficients between parent-adolescent and peer relationship at T_n and T_{n+1}, and all bidirectional correlation coefficients between peer relationship at T_n and parent-adolescent relationship at T_{n+1}.

Statistical Analyses

Statistical procedures. All statistical analyses were performed using the package metaSEM (Cheung, 2015) for the software program R 3.5.1 (R Core Team, 2019), following the approaches outlined by Cheung (2014) for multilevel meta-analyses and Jak (2015) for MASEM analyses. Random-effect models were used for all analyses to account for heterogeneity among studies. While fixed-effects meta-analysis assumes one single mean population effect size and identical study conditions, which is hardly realistic, random effect models assume a distribution of the mean population effect size and take varying study conditions into account (Hedges & Vevea, 1998). For all analyses, we modeled the raw correlations as recommended for random effects models (Schulze, 2004). Parameter estimates were obtained with maximum likelihood estimation, and missing data was handled with full information maximum likelihood estimation. All standardized estimates can be interpreted in terms of effect size *r*, with values around .10 indicating small effects, values around .30 indicating intermediate effects, and values around .50 indicating strong effects (Bollen, 1989). As current statistical procedures do not yet allow the performance of structural equation modelling

(SEM) on multilevel meta-analytic data, we conducted two complementary analyses to answer our research questions (see Figure 1).

Three-level meta-analyses. Traditional meta-analysis assumes that the effect sizes are statistically independent. However, most included studies reported multiple effect sizes (e.g., the same correlation was measured at different waves), which indicates dependence among these effect sizes. To include all available study information, we used a three-level approach to account for dependent effect sizes. Specifically, the first level reflects the sampling variance around the estimated population effect size, the second level reflects the variance between effect sizes within studies, and the third level reflects the variance between effect sizes between studies (Cheung, 2014). To estimate how supportive, negative, and controlling parent-adolescent relationships are associated with supportive, negative, and controlling future peer relationships (see Figure 1A; Figure 2-I), we performed nine three-level random-effect regression analyses – three analyses for each parent-adolescent relationship dimension (i.e., support, negative interaction, and control), respectively. Similarly, to estimate how supportive, negative, and controlling parent-adolescent relationships are associated with supportive, negative, and controlling future romantic relationships (see Figure 1A; Figure 2-II), we performed nine additional three-level random-effect meta-regression analyses.

Cross-lagged MASEM analyses. To estimate how supportive, negative, and controlling parent-adolescent relationships predict future peer relationships over time (see Figure 1B; Figure 3), we performed nine cross-lagged MASEM analyses – three analyses for each parent-adolescent relationship component, respectively. Specifically, we added five additional paths to the model: (1) the stability of parent-adolescent relationship from T_n to T_{n+1}, (2) the stability of peer relationship from T_n to T_{n+1}, (3) the concurrent correlation of parent-adolescent and peer relationship at T_n (4) the concurrent correlation of parent-adolescent and peer relationship at T_{n+1}, and (5) the reversed correlation from peer relationship at T_n to parent-adolescent relationship at T_{n+1}. MASEM analyses do not allow for multiple effect sizes per study. Therefore, we selected only the first effect size per study for the analyses, as the first assessment point usually contained the largest sample size. To estimate the cross-lagged models, we used the one-stage MASEM approach (OSMASEM; Jak & Cheung, 2020). This approach is an extension of the conventional two-stage approach that first pools correlations coefficients and then fits the structural model to the pooled correlation matrix (TSSEM; Cheung, 2014). In OSMASEM, the structural model is fitted directly on the observed correlation matrix, rather than the pooled correlation matrix. Both approaches result in the same parameter estimates and standard errors in models without moderators (Jak & Cheung, 2020). Unlike previous approaches, however, OSMASEM allows the modelling of continuous moderators on the structural paths while accounting for missing correlations.

Moderator analyses. Heterogeneity across effect sizes was assessed in all models, using the *Q*-statistic and I^2 measure from Stage 1 of the two stage MASEM approach. The *Q*-statistic evaluates whether there is substantial variability in the effect sizes across studies ($\tau^2 \neq 0$). The I^2 quantifies how much variation across studies is attributed to heterogeneity, with values > 25%, > 50%, and > 75% representing small, moderate, and large heterogeneity, respectively (Higgins, Thompson, Deeks, & Altman, 2003). We reported all effect sizes to two decimal places. For small moderation effects, we deviated from this rule if rounding to two decimal places would result in non-informative, imprecise estimates.

If the analyses detected significant heterogeneity between effect sizes, we examined whether hypothesized moderators might explain this heterogeneity. As potential moderators were prespecified, we did not add all moderators successively to the model but conducted all moderator analyses separately to refrain from inflating the type II error rate and maximize statistical power. For both statistical procedures, we tested each moderator's unique contribution by adding them as predictors to the final models and calculating the proportion of explained variance (e.g., Cheung, 2014). R^2_{within} and $R^2_{between}$ indicate how much estimated heterogeneity each predictor explained within (level 2) and between studies (level 3). However, this approach did not allow us to examine the unique effects of one moderator when accounting for other moderators. Hypothesized moderators were adolescent age and time between measurements. Additional moderators included type of population, shared informant, percentage males, percentage racial-ethnic minority, publication status, publication year, journal impact factor, and journal quartile. Continuous moderators were centered on the mean.

Publication bias. Publication bias is a major concern in meta-analyses as it may inflate the associations between two constructs, resulting in false conclusions. To date, most methods assessing publication bias suffer from serious limitations, particularly with regard to the small number of included studies and effect sizes (see van Aert, Wicherts, & van Assen, 2019 for a review). Furthermore, severe heterogeneity between studies prevents a clear detection of publication bias as it often results in an increase of false-positives (Terrin, Schmid, Lau, & Olkin, 2003). To get a more comprehensive picture of the potential presence and implication of publication bias in this study, we used two established methods. First, we examined funnel plots and conducted Egger's tests to estimate potential associations between effect sizes and their precision (i.e., small study effects). Asymmetrical funnel plots indicate the presence of small study effects. The Egger's test outperforms comparable methods and is the recommended method to assess publication bias in meta-analyses (van Aert et al., 2019). However, the Egger's test often fails to produce reliable results in samples with < 10 effect sizes due to insufficient power, resulting in an increase of false positives (Sterne,

Gavaghan, & Egger, 2000; Terrin et al., 2003). Furthermore, it should be noted that asymmetrical funnel plots indicate small study effects, of which publication bias is only one possible cause (Egger, Smith, Schneider, & Minder, 1997). Second, we used the three-parameter selection model (Iyengar & Greenhouse, 1988) to estimate effect sizes corrected for potential publication bias. This approach allows researchers to compare the unadjusted to bias-adjusted models using likelihood ratio tests and outperformed other effect size correction methods in simulation studies (Carter, Schönbrodt, Gervais, & Hilgard, 2019; van Aert et al., 2019).

RESULTS

Table S2 provides a general overview of all main and moderation results.

Three-Level Meta-Regressions on Parent-Adolescent and Future Peer and Romantic Relationships

Sample description. For peer outcomes, our search yielded $N = 431$ effect sizes from $k = 54$ independent studies with a total of 62 samples and 51,891participants. For romantic outcomes, our search yielded $N = 147$ effect sizes from $k = 38$ independent studies with a total of 43 samples and 18,763 participants. Further information regarding the number of effect sizes per sub-analysis is depicted in Table S3A. The majority of studies sampled intact (72.0%), racial-ethnic majority families (63.1%) from Western countries (96.0%) and assessed relationship quality using adolescent informants only (79.0%). Further study characteristics are depicted in Table S1A. Forest plots with all effect sizes, their corresponding confidence intervals, and interpretation are depicted in the supplemental material (Figures S2-S9).

Peer outcomes. Due to the small number of studies assessing control in parent-adolescent or peer relationships ($k = 2$), these analyses were not interpreted (but see Table S4). For all remaining relationship dimensions, parent-adolescent relationship quality was significantly associated with later peer relationship quality (see Figure 2-I; Table S3A). More supportive relationships with parents were associated with more supportive ($\beta = .18$, $p < .001$) and less negative ($\beta = -.12$, $p < .001$) subsequent relationships with peers. Similarly, more negative relationships with parents were associated with more negative ($\beta = .18$, $p < .001$) and less supportive subsequent relationships with peers ($\beta = -.07$, $p = .002$).

Test of moderators. All analyses revealed significant heterogeneity within and across studies (see Table S3A). Levels of heterogeneity were moderate to large between, but negligible to small within studies. Therefore, we conducted several analyses to examine whether theoretical moderators (i.e.,

time lag, adolescent age) and exploratory moderators (i.e., population, shared informant, percentage males, percentage racial-ethnic minority, publication status, publication year, journal impact factor, and journal quartile) significantly explained these differences (see Table S5 for descriptive statistics on all moderators). Only few moderators (i.e., time lag, adolescent age, shared informant) significantly improved the model fit for some relationship dimensions (see Table S6). The results of these moderators are depicted in Table S7 (see also Figure 2-I).

Time lag. Adding time lag as a moderator significantly improved the model fit for two out of four analyses: If the measurements were closer together in time, supportive parent-adolescent relationships were more strongly associated with supportive peer relationships ($\beta = -.002$, $p < .001$, $R^2_{within} = .87$, $R^2_{between} = .07$), and negative parent-adolescent relationships were more strongly associated with negative peer relationships ($\beta = -.002$, $p < .001$, $R^2_{within} < .99$, $R^2_{between} < .01$).

Adolescent age. Adding adolescent age as a moderator significantly improved the model in two out of four analyses: As adolescents got older, supportive parent-adolescent relationships were more strongly associated with supportive peer relationships ($\beta = .01$, $p = .003$, $R^2_{within} = .43$, $R^2_{between} < .01$), and negative parent-adolescent relationships were more strongly associated with negative peer relationships ($\beta = .02$, $p = .037$, $R^2_{within} = .46$, $R^2_{between} < .01$).

Additional moderators. Results of the moderator analyses further indicated significant effects for shared informants for some relationship dimensions. Specifically, studies using shared informants (i.e., the same informant reported on the parent-adolescent and peer or romantic relationship) reported larger associations between supportive parent-adolescent and supportive peer relationships compared to studies using non-shared informants ($\beta = .10$, $p = .046$, $R^2_{within} < .01$, $R^2_{between} = .08$). However, due to little or no variation at one level of the moderator, we could not test the effects of shared informants on the links between negative parent-adolescent relationships and negative or supportive peer relationships ($k = 1$ for non-shared informant).

Adding population, gender, racial-ethnic minority, publication status, publication year, or journal impact factor separately as moderators did not significantly improve the model fit for any relationship dimension. Due to little or no variation at some levels of the moderator, we could not test the effects of population on the links between supportive parent-adolescent relationships and negative peer relationships ($k = 1$ for clinical, $k = 2$ for marginalized population) as well as negative parent-adolescent relationships and negative ($k = 1$ for clinical, $k = 2$ for marginalized population) or supportive peer relationships ($k = 1$ for clinical, $k = 0$ for marginalized population). Similarly, we

could not test the effects of publication status on the links between supportive or negative parent-adolescent relationships and negative peer relationships (k = 1 for unpublished studies).

Romantic outcomes. Similar to peer outcomes, we refrain from interpreting all analyses involving controlling parent-adolescent or romantic relationships due to the small number of studies (k = 1-4) assessing this relationship dimension (but see Table S4). For all remaining relationship dimensions, parent-adolescent relationship quality was significantly associated with later romantic relationship quality (see Figure 2-II; Table S3A). More supportive relationships with parents were associated with more supportive ($\beta = .11$, $p < .001$) and less negative ($\beta = -.09$, $p < .001$) relationships with romantic partners at the next time point. Similarly, more negative relationships with parents were associated with more negative ($\beta = .15$, $p < .001$) and less supportive relationships with romantic partners at the next time point ($\beta = -.10$, $p < .001$).

Test of moderators. The analyses involving negative, but not supportive parent-adolescent relationships revealed significant heterogeneity within and across studies (see Table S3A). Levels of heterogeneity were moderate between, but small within studies for the associations between negative parent-adolescent relationships and supportive as well as negative romantic relationships. Therefore, we conducted several analyses to examine whether theoretical moderators (i.e., time lag, adolescent age) and exploratory moderators (i.e., population, shared informant, percentage males, percentage racial-ethnic minority, publication year, journal impact factor, and journal quartile) significantly explained these differences (see Table S5 for descriptive statistics on all moderators). Contrary to our expectations, none of these moderators significantly improved the model fit for any relationship dimension. Due to little or no variation in the data, we could not test the moderating effects of population and publication status on the associations between negative parent-adolescent relationships and negative (k = 2 for clinical, k = 3 for marginalized population, and k = 2 for unpublished studies) as well as supportive peer relationships (k = 1 for clinical, k = 2 for marginalized population, and k = 1 for unpublished studies).

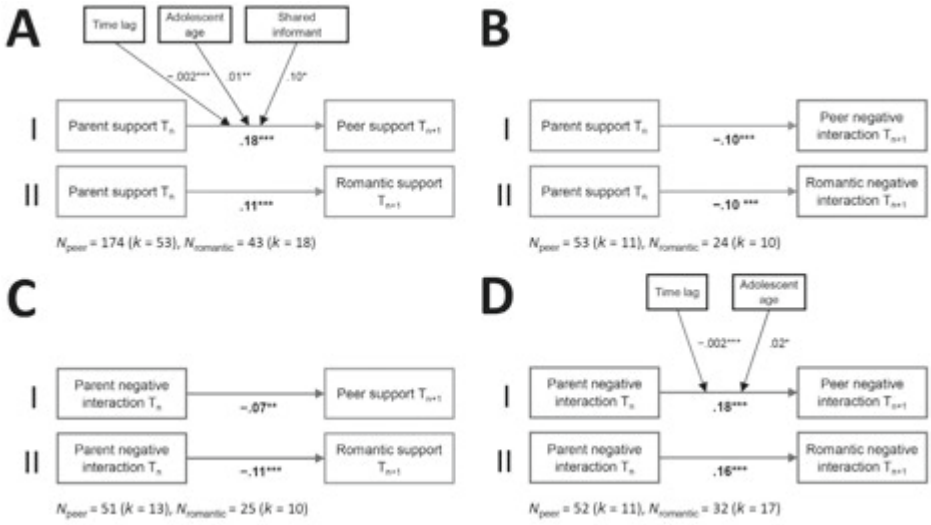

Figure 2. Main and significant moderation outcomes for three-level meta-regression analyses between supportive (A, B) and negative parent-adolescent relationships (C, D) and future peer (I) and romantic relationships (II). All effect sizes refer to standardized βs.
*** $p < .001$ ** $p < .01$ * $p < .05$.

Cross-Lagged Panel Analyses on Parent-Adolescent and Future Peer Relationships

We used MASEM on all independent effect sizes ($N = 54$) to estimate a cross-lagged panel model that accounts for concurrent correlations at each time point, stability of each quality measure as well as the reciprocal effect from peer relationship quality to parent-adolescent relationship quality. Missing data on the separate paths ranged from $n = 1$ to $n = 8$. As too few studies assessed parent-adolescent and romantic relationships simultaneously ($k = 5$), and thus provided stability, concurrent as well as bidirectional correlations, we were unable to estimate a cross-lagged model for the associations between parent-adolescent and romantic relationships.

Main outcomes. The MASEM analyses showed that, controlling for concurrent, stability, and bidirectional correlations, parent-adolescent relationship quality significantly predicted later peer relationship quality for all relationship dimensions (see Figure 3; Table S3B): More supportive parent-adolescent relationships predicted more supportive (β = .07, $p < .001$) and less negative (β = –.06, $p = .016$) relationships with peers at the next time point. More negative relationships with parents predicted more negative (β = .10, $p = .001$) and less supportive (β = –.06, $p = .001$) relationships with peers at the next time point. Out of four possible reversed effects, only the two effects of peer

relationships on negative relationships with parents were significant: More negative relationships with parents were predicted by both more negative (β = .06, p = .026) and less supportive relationships with peers (β = –.06, p = .017) at the previous time point. The two bidirectional findings indicated overlapping confidence intervals for the cross-lagged paths between negative parent-adolescent relationships and negative ($CI_{par→peer}$=[.05, .15], $CI_{peer→par}$ = [.01, .11]) and supportive peer relationships ($CI_{par→peer}$ = [–.10, –.02], $CI_{peer→par}$ = [–0.11, –0.01]), which suggests that these paths did not differ from each other. Both parent-adolescent (βs = .50-.59, ps < .001; see Table S3B) and peer relationship dimensions (βs = .37-.43, ps < .001) remained quite stable across time, even though studies did not always assess the same peers at T_n and T_{n+1}.

Moderator analyses. All analyses revealed significant heterogeneity across studies (see Table S3B). Levels of heterogeneity were substantial for all cross-lagged paths. Therefore, we added potential theoretical (i.e., adolescent age, time lag) and exploratory moderators (i.e., shared informant, percentage males, percentage racial-ethnic minority, publication year, journal impact factor, journal quartile) on the structural cross-lagged parameters to examine whether they explained significant differences between the studies. Most moderators (i.e., adolescent age, time lag, publication year, journal impact factor, journal quartile) significantly improved the model fit for some relationship dimensions (see Table S6). The results of these moderators are depicted in Table S7 (see also Figure 3).

Time lag. Adding time lag as a moderator significantly improved the model fit in one out of four analyses: However, time between measurements did not significantly moderate the cross-lagged associations between supportive parent-adolescent relationships and negative peer relationships ($β_{par→peer}$ = .001, p = .451; $β_{peer→par}$ = .01, p = .067).

Adolescent age. Adding adolescent age as a moderator significantly improved the model fit in three out of four analyses: As adolescents got older, the negative association between negative parent-adolescent relationships and supportive peer relationships became stronger (i.e., became more negative; β = .02, p = .003, $R^2_{par→peer}$ = .05). Reversely, the negative association between supportive peer relationships and negative parent-adolescent relationships also became stronger with adolescent age (β = .03, p = .004, $R^2_{peer→par}$ > .99). For the associations between negative parent-adolescent and negative peer relationships as well as between supportive parent-adolescent and negative peer relationships, on the other hand, only the reversed effects were significant: As adolescents got older, the negative association of negative peer relationships with subsequent supportive parent-adolescent relationships became stronger (β = .03, p = .042, $R^2_{peer→par}$ > .99), while

the positive association with subsequent negative parent-adolescent relationships *became weaker* (β = –.04, p = .002, $R^2_{peer \rightarrow par} > .99$).

Additional moderators. Results of the moderator analyses further indicated significant moderation by publication year, journal impact factor, and journal quartile for some relationship dimensions. Compared to older publications, more recent publications reported slightly smaller bidirectional cross-lagged paths from negative parent-adolescent to supportive peer relationships (β = –.004, p = .013, $R^2_{par \rightarrow peer} = .10$) and reversely from supportive peer relationships to negative parent-adolescent relationships (β = –.01, p = .005, $R^2_{peer \rightarrow par} > .90$) as well as slightly larger reversed paths from supportive peer to supportive parent-adolescent relationships (β = .004, p = .019, $R^2_{peer \rightarrow par} = .07$) as well as from negative peer to negative parent-adolescent relationships (β = .01, $p < .001$, $R^2_{peer \rightarrow par} = .20$).

Adding journal impact factor and journal quartile separately as moderators significantly improved the model fit for some relationship dimensions, but they mainly moderated the reversed paths from peer to parent-adolescent relationships: Publications in journals with higher impact factors reported smaller effects from supportive peer to supportive parent-adolescent relationships (β = –.03, p = .009, $R^2_{peer \rightarrow par} = .05$) and from supportive peer to negative parent-adolescent relationships compared to journals with lower impact factors (β = –.03, p = .009, $R^2_{peer \rightarrow par} = .37$). Similarly, publications in journals with lower quartile rankings reported larger effects from supportive peer to supportive parent-adolescent relationships (β = .04, $p < .001$, $R^2_{peer \rightarrow par} = .24$), but also reversely from supportive parent-adolescent to supportive peer relationships (β = .02, p = .029, $R^2_{par \rightarrow peer} = .13$).

Adding publication status, population, shared informant, gender, or racial-ethnic minority separately as moderators did not significantly improve the model fit for any relationship dimension. As in the three-level meta-regression analyses, due to little or no variation at some levels of the moderator, we could not test the effects of shared informants on the links between negative parent-adolescent relationships and negative or supportive peer relationships and the effects of population on the links between supportive parent-adolescent relationships and negative peer relationships as well as negative parent-adolescent relationships and negative or supportive peer relationships.

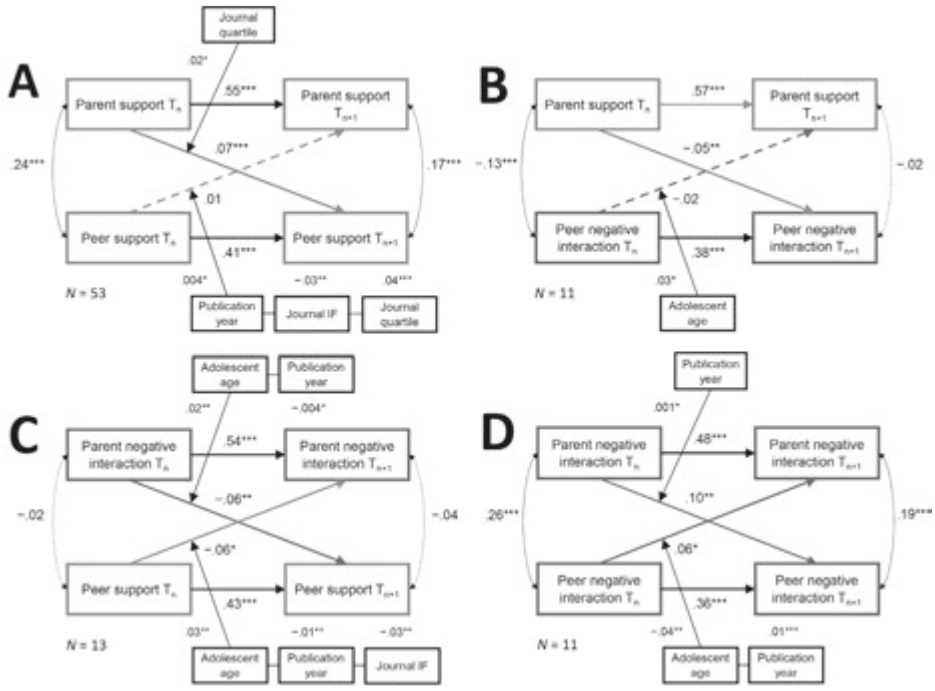

Figure 3. Main and significant moderation outcomes for MASEM analyses between supportive (A, B) and negative parent-adolescent relationships (C, D) and future peer relationships; Journal IF = journal impact factor. All effect sizes refer to standardized βs.
*** $p < .001$ ** $p < .01$ * $p < .05$.

Sensitivity Analyses

To disentangle the effects of relationship continuity within (e.g., from supportive parent-adolescent to supportive peer relationships) and across relationship components (e.g., from supportive parent-adolescent to negative peer relationships), we conducted four additional posthoc analyses on the combined samples with inverted scores for the associations across relationship components. Specifically, we analyzed how peer or romantic relationship dimensions moderated the association between parent-adolescent support and peer or romantic relationships (i.e., supportive and negative peer relationships combined) and the association between parent-adolescent negative interaction and peer or romantic relationships. For peer outcomes, supportive parent-adolescent relationships were more strongly associated with supportive than negative peer relationships in the three-level meta-regression (β = .12, $p < .001$), but not the MASEM analyses (β = –.01, $p = .831$). In the MASEM analyses, the reversed effect was significant: Supportive parent-adolescent relationships were more strongly predicted by supportive than negative peer relationships (β = .33, $p < .001$).

Similarly, negative parent-adolescent relationships were more strongly associated with negative than supportive peer relationships in the three-level meta-regression (β = .13, $p < .001$), but not the MASEM analyses (β = .06, $p = .060$). Again, the reversed effect was significant: Negative parent-adolescent relationships were more strongly predicted by negative than supportive peer relationships (β = .29, $p < .001$). For romantic outcomes, supportive parent-adolescent relationships were more strongly associated with supportive than negative romantic relationships (β = .03, $p = .049$) and negative parent-adolescent relationships were more strongly associated with negative than supportive romantic relationships (β = .10, $p < .001$).

Publication Bias

As many studies in our meta-analysis did not assess the longitudinal associations between parent-adolescent and peer or romantic relationships as (primary) outcomes, we did not expect publication bias to severely impact our results. Nevertheless, we assessed publication bias and its potential impact on our analyses using two methods. First, visual inspection of the funnel plots and results of Egger's tests suggested that the effects were approximately evenly distributed around the mean in all but one subgroup model, all *p*s > .202 (see Table S8). For the association between negative parent-adolescent and supportive romantic relationships, we detected funnel plot asymmetry ($z = 2.47$, $p = .013$). Second, results from the three-parameter selection model (*p* value cut point = .05) indicated that the estimated model adjusted for publication bias did not fit the data significantly better than the unadjusted model in all but one of the main analyses, all *p*s > .116. However, the adjusted model seemed to fit better than the unadjusted model for the association between negative parent-adolescent relationships and supportive peer relationships in the three-level meta-regression analysis ($r_{adjusted} = -.01$, $p = .008$). The weighted average effect sizes from the other adjusted models were similar to those of the unadjusted models. Due to low variation in effect sizes, we were unable to conduct three-parameter selection analyses for all but one association involving romantic relationships. However, recent research suggests that publication bias in relatively homogeneous datasets is weak at best (van Aert et al., 2019). Together, these results suggest that some of the subgroup analyses might be subject to publication bias. However, due to the small number of studies, these analyses of publication bias are likely biased and may be false positives (Carter et al., 2019). Combined with our extensive search for unpublished materials and most correlations pertaining to secondary outcomes, publication bias is not likely to severely impact our results.

DISCUSSION

The present meta-analysis examined associations between dimensions of parent-adolescent relationships and subsequent peer and romantic relationships in adolescence and emerging adulthood, using two complementary analyses on a range of 10 to 53 studies across analyses. First, we used multilevel analyses to examine how parent-adolescent relationships are associated with future peer and romantic relationships. Second, we used meta-analytic structural equation modelling (MASEM) to examine bidirectional associations between parent-adolescent and future peer relationships, controlling for concurrent associations and over-time construct stability. Results indicated that supportive and negative parent-adolescent relationships were associated with future supportive and negative peer relationships, above and beyond concurrent associations and construct stability. Also, supportive and negative parent-adolescent relationships were associated with supportive and negative future romantic relationships. For negative parent-adolescent relationships, the transmission from parent-adolescent to peer relationship quality became stronger as adolescents got older. Despite relatively small effect sizes, these findings indicate that parent-adolescent relationship dimensions are significantly related to subsequent peer and romantic relationship dimensions.

Parent-Adolescent Relationships as Foundation for other Social Relationships

Consistent with our hypotheses based on social interactional and cognitive theories (e.g., Baldwin, 1992; Bandura, 1977; Furman & Collibee, 2018; Kaufman et al., 2020), we found significant associations across time between parent-adolescent and future peer and romantic relationships within and across relationship dimensions. Specifically, more supportive parent-adolescent relationships were associated with more supportive and less negative peer and romantic relationships. Similarly, more negative parent-adolescent relationships were associated with more negative and less supportive peer and romantic relationships. These findings suggest that core parent-adolescent relationship dimensions are related to subsequent social relationship dimensions. The results from the MASEM analyses further extended these findings, indicating that across time more supportive and more negative parent-adolescent relationships predicted more supportive and more negative future peer relationships, above and beyond concurrent associations and construct stability. These findings support relationship continuity from parent-adolescent to future peer relationships and highlight the importance of both support and negative interaction in the transmission of relationship quality.

While the MASEM results indeed indicated unidirectional pathways from supportive parent-adolescent relationships to peer relationships, they also indicated bidirectional associations between negative parent-adolescent and peer relationships. These bidirectional associations are in line with

transactional (Bell, 1968; Sameroff, 2009) and social systems theories (Erel & Burman, 1995), indicating that adolescent experiences with different social partners may spill over to other relationships. The results further suggest that negative interactions might serve a different function than support during adolescence. Adolescence represents a time in which children become more independent and learn to negotiate their own expectations (Collins & Laursen, 2004). Peers become more important and primary relationship partners with whom adolescents practice conflict interactions among equals (Bukowski et al., 2011). These newly practiced and acquired interaction patterns and schemas are thus likely to be generalized to parent-adolescent relationships. Adolescents who are less adept at integrating different objectives and expectations into peer relationships might transfer these negative interaction patterns to the parent-adolescent relationship, which is already susceptible for negative interaction as it realigns towards equality (Branje, 2018). Negative relationship processes increase negative mood, antisocial behaviors and decrease interpersonal competence, through which these negative interactions are likely to spill over to other relationships (Conger et al., 2000; Patterson, 1982), resulting in bidirectional relations between negative interaction patterns in parent-adolescent and peer relationships.

Sensitivity analyses suggested that the transmission of relationship quality from parent-adolescent to peer and romantic relationships was stronger within the same relationship dimensions (e.g., from parent-adolescent support to peer support) than across relationship dimensions (e.g., from parent-adolescent support to peer negative interaction). Similarly, the MASEM findings indicated that the reversed associations from peer relationships to future parent-adolescent relationships were also stronger within than across relationship dimensions. One reason may be that in relationships with parents, adolescents form representations and expectations about social relationships and acquire interaction patterns that they transfer to their relationships with peers and romantic partners (Bandura, 1977). Such representations and interaction patterns are based on specific observed behaviors (e.g., positive or negative relationship behaviors) that form schemata of relationships (Burks & Parke, 1996). In that way, relationship schemata are more easily activated within the same, than across different relationship dimensions (Brewer & Treyens, 1981).

To summarize, the results for both peer and romantic relationships indicate that core dimensions of parent-adolescent relationships continue into other social relationships later in adolescence and emerging adulthood. Even more so, particularly supportive parent-adolescent relationships might provide a foundation for later peer relationships as suggested by dominant theoretical perspectives (e.g., Collins & Sroufe, 1999; Hartup, 1979), while negative parent-adolescent relationships are also predicted by adolescent peer relationships. However, the associations of parent-adolescent relationships with peer and romantic relationships were generally small. These results were in

line with previous meta-analyses on the associations between early parent-child bonds and peer relations (Pallini et al., 2014; Schneider et al., 2001). Relationships with parents during adolescence continue to be associated with later relationships in adolescence and emerging adulthood, and these associations do not seem to become weaker compared to childhood. However, the small effect sizes also suggest that relationship quality with parents does not solely predict later relationship quality with peers and romantic partners. Rather, it is likely that other processes are involved in determining the quality of social relationships (see e.g., Bronfenbrenner & Morris, 2006).

Moderators in the Transmission of Relationship Quality

Effects of time lag. Although we only detected moderating effects of time lag for some relationship dimensions, assessments of parent-adolescent and peer, but not romantic relationships that were further apart in time showed smaller associations. In line with revisionist perspectives (Fraley & Roisman, 2015), increased time between assessments might elicit changes in the parent-adolescent relationship or additional continuous pathways that decrease the association of parent-adolescent relationships with future peer relationships. From a statistical perspective, this finding might reflect serial correlation which specifies that correlations between longitudinal assessments that are closer together in time are stronger than those further apart. Parent-adolescent relationships are thus likely to promptly predict peer relationships.

The associations between parent-adolescent and future romantic relationships did not vary as a function of time intervals, which might suggest stabilizing or enduring effects that are in line with previous empirical findings on the effects of early experiences with parents (Fraley & Roisman, 2015). It may be possible that parent-adolescent relationships provide continuous internal models that are particularly influential for romantic relationships (Sroufe et al., 1990). However, we should note that assessments between parent-adolescent and romantic relationships were generally further apart in time, which resulted in less variation and heterogeneity to detect significant effects between studies. Furthermore, the sample size might have also been too small to detect any potentially significant effects.

While our findings provide preliminary evidence that parent-adolescent relationships transiently affect peer relationships within the same relationship dimension and enduringly affect romantic relationships, we should note that our analyses rendered it difficult to unravel moderating effects of time intervals and adolescent age. It may be possible, for example, that the effects of parent-adolescent relationships in late, compared to early adolescence are more enduring as they realign (Eccles et al., 1993). However, analyses that would assess the effects of time intervals while accounting for adolescent age were too complex to produce reliable, interpretable results. Future studies are

needed to find more suitable ways to disentangle potential time and age effects, and thus allow more substantial conclusions about the long-term effects of parent-adolescent relationships on future peer and romantic relationships. Additionally, parent-adolescent relationships, despite potential changes in their quality, are inherently stable, whereas peer and romantic relationships may dissolve (Laursen & Bukowski, 1997). Most studies, however, did not assess whether relationships with the same peer or romantic partner were assessed across time. Although the perceived quality of peer and romantic relationships remained relatively stable across our analyses, future research might help to unravel potential differences between peer and romantic relationships that remained stable and those that dissolved across time.

Effects of adolescent age. Results indicated that adolescent age significantly moderated the links between parent-adolescent relationships and some future peer, but not romantic relationship dimensions. Specifically, as adolescents got older, the longitudinal associations between parent-adolescent and peer relationships within the same relationship dimensions in the three-level meta-regressions and negative parent-adolescent and supportive peer relationships in the MASEM analyses became stronger. One reason may be that parent-adolescent relationships become more egalitarian and reciprocal towards late adolescence (Eccles et al., 1993; Hadiwijaya et al., 2017) and thus more similar to peer relationships.

We further detected reversed, but inconsistent effects from peer to parent-adolescent relationships. Specifically, as adolescents got older, supportive and negative parent-adolescent relationships were more weakly predicted by negative peer relationships, but negative parent-adolescent relationships were more strongly predicted by supportive peer relationships. It may be possible that the weaker effect from negative peer to supportive and negative parent-adolescent relationship over time may again reflect the change in peer relationships. Not only do adolescents increasingly turn to peers for support (Blos, 1967; Branje et al., 2021), but these relationships with peers also represent a practice ground for egalitarian relationships (Bukowski et al., 2011). These new egalitarian patterns are likely to spill over to relationships with parents, which themselves become more egalitarian and aligned throughout adolescence. Once parent-adolescent relationships are realigned and established by late adolescence, however, they might become less susceptible to the influence of peer relationships. Alternatively, it may be possible that parents worry less about peer relationships as adolescents get older, whereas they may be more concerned about negative peer interactions at younger ages, resulting in more negative and less supportive parent-adolescent interactions during early to mid-adolescence (Hadiwijaya et al., 2017). On the other hand, relationships with peers also mature over time in that peers not only mutually and unconditionally support each other, but also learn to accept needs and interests that differ from their own (Selman, 1989). This development of support

in peer relationships might positively transfer to parent-adolescent relationships and thus, result in less conflictive, negative interactions. While these findings indicate that there may be some moderating effects of adolescent age, for most relationship dimensions the links between parent-adolescent and future peer relationships did not differ with adolescent age but remained equally strong throughout adolescence and adulthood.

No sample or study characteristics explained differences in how parent-adolescent relationships were associated with future romantic relationships, and only few study characteristics explained differences in how they were associated with future peer relationships. This suggests that the associations between parent-adolescent and future peer and romantic relationships were relatively robust across population, informants, adolescent gender, racial-ethnic groups, or publication year. For some relationship dimensions, however, we found that studies published in journals with higher impact factors and associated lower quartile rankings (i.e., indicating higher quality journals) reported smaller associations between parent-adolescent and peer relationships compared to journals with lower impact factors and higher quartiles. One reason could be that these journals require studies to implement more rigorous designs and analyses that often result in smaller effect sizes.

Strengths, Limitations, and Future Directions

This meta-analysis is the first to synthesize the longitudinal associations between parent-adolescent and future peer and romantic relationships, combining novel meta-analytic techniques and establishing temporal order. Multilevel meta-regression allowed us to model the longitudinal associations between parent-adolescent and future peer and romantic relationships, using complete existing evidence from multiple waves of longitudinal studies. Additionally, meta-analytic structural equation modelling extended the multilevel findings and allowed us to additionally model the longitudinal associations between parent-adolescent and future peer relationships in a cross-lagged panel design, to not only control for concurrent associations and over time stability, but also to investigate potential bidirectional pathways. As such, our findings provide a comprehensive and thorough overview of how core dimensions in parent-adolescent relationships predict the same and other dimensions in later peer and romantic relationships, which helps to build and refine theories on adolescent relationships. While theories generally mark relationships with parents in (early) childhood as most influential for a child's later development, our findings indicate that relationships with parents in adolescence might play an important role as well.

Although this is the most extensive and comprehensive work on the transmission of relationship quality in and beyond adolescence, this meta-analysis has some limitations. First, as we decided to analyze subgroups of how parent-adolescent relationship dimensions predict the same and

other peer and romantic relationship dimensions, some cells in our dataset contained only few effect sizes. While we initially intended to assess how control in parent-adolescent relationships continues into peer and romantic relationships, only two studies assessed control, which did not allow for reliable conclusions. As control is a core relationship dimension, future studies should assess whether and how controlling parent-adolescent relationships continue into future peer and romantic relationships.

Second, while our meta-analysis focused on the three core relationship dimensions support, negative interaction, and control, the included studies assessed a variety of different constructs that we combined into the three relationship dimensions. It may be possible that some of these aspects exhibit stronger associations than others. Due to the great number of different constructs and combinations in individual studies, we were not able to examine the effects of more specific individual constructs. However, a meta-analytical review focusing on trust, communication, and alienation as aspects of support, for example, found similarly strong associations across the examined aspects (Gorrese & Ruggieri, 2012).

Third, in some cases there were too few studies to detect or even test moderation effects. For example, most studies relied solely on adolescents to assess relationship quality. Such reports may be biased as different informants might perceive relationship quality differently. To more reliably and objectively assess longitudinal associations between parent-adolescent and peer or romantic relationships, future longitudinal designs should include multi-informant ratings of relationship quality. Related to the sample size problem, we were not always able to retrieve all necessary data. From the articles that did not report the required correlations, 28 authors (29%) did not reply to our requests and 22 authors (23%) replied but were not able to reproduce the data. This is a serious problem that may introduce bias not only in conducting meta-analyses, but in scientific research in general. Open science practices, including data storage and access, help to overcome the problem and should be strongly encouraged, if not required.

Fourth, most studies included samples from White, non-clinical populations, and Western culture, which might not generalize to samples from other populations or cultures. It could be possible, for example, that the associations between parent-adolescent and peer or romantic relationships are stronger in more collectivistic cultures, in which the family context seems to exert greater influence throughout adolescence and young adulthood (Yoshida & Busby, 2012). Cross-cultural longitudinal research is needed to provide crucial insights into how the associations between parent-adolescent and other social relationships generalize to other populations and cultures.

Fifth, our meta-analysis did not allow us to investigate possible differences between mothers and fathers in the association between parent-adolescent and peer or romantic relationships, as such a research question would have required an additional level of dependency in our analyses. Instead, we chose to focus on parent-adolescent relationships more generally. Some studies, however, suggest that relationships with mothers and fathers continue differently into other social relationships (e.g., Möller & Stattin, 2001). Future (meta-analytic) research might further examine how differences in the relationships with mothers and fathers predict the transmission between parent-adolescent relationship dimensions and later relationships with peers or romantic partners. Relatedly, different types of peer groups, such as classmates or peers, might fulfill different socialization functions (Albarello, Crocetti, & Rubini, 2018). Future research might thus investigate how the associations between parent-adolescent and peer or romantic relationships differ among diverse peer groups and romantic partners. Additionally, modes of communication are becoming more diverse with many interactions with peers, romantic partners, and also parents occurring increasingly online. It is possible that online communication, such as social media, offers new opportunities (e.g., meeting platform for peers and romantic partners, sources of support) and challenges (e.g., misperceptions, constant availability, asynchronicity). This may alter the nature and quality of adolescent social relationships (e.g., Nesi, Choukas-Bradley, & Prinstein, 2018). While investigating the effects of different modes of communication on adolescent relationships was beyond the scope of this meta-analysis, future studies might examine the unique role of new modes of communication and social media in the transmission of relationship quality.

Finally, while our findings established both magnitude and relationship continuity from parent-adolescent to other social relationships, we only examined longitudinal associations that do not permit causal conclusions about the influence of parent-adolescent relationships on peer or romantic relationships. Cross-lagged models, however, allowed us to establish temporal order between parent-adolescent and peer relationships. As studies rarely assessed parent-adolescent and romantic relationships simultaneously, we cannot rule out that (particularly later) romantic relationships might also be associated with subsequent parent-adolescent relationships. Future longitudinal studies that simultaneously assess parent-adolescent and romantic relationships over time might more clearly indicate whether romantic relationships are indeed preceded by parent-adolescent relationships or whether they also predict them over time.

Relatedly, studies included in this meta-analysis did not focus on within-family associations, and thus, dynamic processes that occur within families were not examined. Cross-lagged models assess rank-order changes within a group and can thus estimate whether adolescents who have more positive relationships with their parents relative to their peers also develop more positive other

social relationships relative to their peers. To assess within-family dynamics, other designs (e.g., with frequent measurements of discrete parental and adolescent behaviors) or at least other types of models (e.g., dynamic systems approach, Granic, 2005; random-intercept cross-lagged panel models, Hamaker, Kuiper, & Grasman, 2015) would be required. To infer within-family dynamics, future empirical research is needed that utilizes such models and designs. The focus of the present study, however, was on the prediction of relative changes, which combine within- and between-family variance.

CONCLUSION

Adolescence is a critical time in which relationships with peers and romantic partners become increasingly important and relationships with parents become essential prototypes in shaping these new relationships. The results of this meta-analysis emphasize the importance of positive parent-adolescent relationships for the development of positive peer and romantic relationships in and beyond adolescence. Both supportive and negative parent-adolescent relationships seem to equally predict subsequent peer and romantic relationships. Assisting adolescents and parents in maintaining a mutually supportive relationship may help adolescents to develop positive social relationships that are crucial for their overall well-being.

SUPPLEMENTAL MATERIAL

Search for unpublished materials

Based on best practices and recommendations by Cochrane and the Campbell Collaboration (see Bonato, 2018), we conducted a comprehensive search of unpublished materials that spanned 15 databases on dissertations (ProQuest Dissertations, Networked Digital Library of Theses and Dissertations, DART Europe), conference proceedings (OAIster, Scopus, Web of Science), policy documents (Policy Commons, Overton), preregistrations (OSF preregistrations, SocArXiv, PsyArXiv) and other grey literature (Grey literature in the Netherlands, Nederlandse Bibliografie Online, OpenGrey, GreyNet). We used the same search strategy and search strings that we used for our search for published studies (see exact search strings below on pp. 2-7 in the supplemental material). In total, we identified 1,381 unpublished documents, which we screened for title and abstract as well as full text (see Figure S1). Of 184 documents that we screened for full text, 170 were dissertations, four were reports, seven were conference papers, and three were presentations. In cases in which we could not retrieve the full text, we contacted the first author. Out of 30 contacted authors, only three authors replied to our request and one of those authors provided the requested text. Based on the full-text screening, we included 67 studies, of which we excluded a number of documents that had already been published (n = 27) or used data that overlapped with studies included in our meta-analysis (n = 23). For included studies that did not report the required effect sizes (n = 11), we contacted the authors using the same strategy as for published studies. Of all 10 authors contacted, three provided the requested effect sizes, but two of those studies had to be excluded because the relationship constructs combined several relationship dimensions. Seven authors did not reply to our request or could not provide the requested correlations. Hence, nine studies were excluded, resulting in a final inclusion of seven unpublished dissertations.

Search strings for all databases

The exact search strings used for this meta-analytic review are provided below.

EBSCOHOST (ERIC, PsycArticles, PsycInfo)

SU (adolescen* OR teen* OR youth OR youngst* OR student* OR "emerging adult*" OR "early adult*" OR "young adult*") AND SU (Famil* OR parent* OR mother* OR father* OR maternal OR paternal) AND SU (friend* OR peer* OR romantic* OR intimate* OR marital OR marriage OR couple*) AND SU (Relation* OR dating OR warmth OR bond OR affecti* OR attachment OR intimacy OR nurturance OR sensitivity OR support OR aggression OR conflict OR discord OR hostility OR discipline OR abuse OR victimization OR violence) AND SU (Longitudinal* OR prospective OR intergenerational OR transmission OR spillover OR "spill*_over") Journal or Document: Journal Articles (EJ);

SCOPUS

(TITLE-ABS-KEY (adolescen* OR teen* OR youth "emerging adult*" OR "early adult*" OR "young adult*") AND TITLE-ABS-KEY (famil* OR parent* OR mother* OR father* OR maternal OR paternal) AND TITLE-ABS-KEY (friend* OR peer* OR romantic* OR intimate* OR marital OR marriage OR couple*) AND TITLE-ABS-KEY (relation* OR dating OR warmth OR bond OR affecti* OR attachment OR intimacy OR nurturance OR sensitivity OR support OR aggression OR conflict OR discord OR hostility OR discipline OR abuse OR victimization OR violence) AND TITLE-ABS-KEY (longitudinal* OR prospective OR intergenerational OR transmission OR spillover OR "spill*_over")) AND aggression OR conflict OR discord OR hostility OR discipline OR abuse OR victimization OR violence) AND (EXCLUDE (SUBJAREA , "MEDI")) AND (LIMIT-TO (DOCTYPE , "ar") OR LIMIT-TO (DOCTYPE , "ip"))

Web of Science

TOPIC:(adolescen* OR teen* OR youth OR youngst* OR student* OR "emerging adult*" OR "early adult*" OR "young adult*") AND TOPIC: (Famil* OR parent* OR mother* OR father* OR maternal OR paternal) AND TOPIC:(friend* OR peer* OR romantic* OR intimate* OR marital OR marriage OR couple*) AND TOPIC: (Relation* OR dating OR warmth OR bond OR affecti* OR attachment OR intimacy OR nurturance OR sensitivity OR support OR aggression OR conflict OR discord OR hostility OR discipline OR abuse OR victimization OR violence) AND TOPIC: (Longitudinal* OR prospective OR intergenerational OR transmission OR spillover OR "spill*_over") Refined by: DOCUMENT TYPES: (ARTICLE) Timespan: All years. Indexes: SSCI.

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Policy Commons

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victimization OR violence) AND (longitudinal* OR prospective OR intergenerational OR transmission OR spillover OR "spill*_over")

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(adolescen* OR teen* OR youth OR youngst* OR student* OR "emerging adult*" OR "early adult*" OR "young adult*") AND (famil* OR parent* OR mother* OR father* OR maternal OR paternal) AND (friend* OR peer* OR romantic* OR intimate* OR marital OR marriage OR couple*) AND (relation* OR dating OR warmth OR bond OR affecti* OR attachment OR intimacy OR nurturance OR sensitivity OR support OR aggression OR conflict OR discord OR hostility OR discipline OR abuse OR victimization OR violence) AND (longitudinal* OR prospective OR intergenerational OR transmission OR spillover OR "spill*_over")

OSF preregistrations

(adolescen* OR teen* OR youth OR youngst* OR student* OR "emerging adult*" OR "early adult*" OR "young adult*") AND (famil* OR parent* OR mother* OR father* OR maternal OR paternal) AND (friend* OR peer* OR romantic* OR intimate* OR marital OR marriage OR couple*) AND (relation* OR dating OR warmth OR bond OR affecti* OR attachment OR intimacy OR nurturance OR sensitivity OR support OR aggression OR conflict OR discord OR hostility OR discipline OR abuse OR victimization OR violence) AND (longitudinal* OR prospective OR intergenerational OR transmission OR spillover OR "spill*_over")

PsyArXiv / SocArXiv

(adolescen* OR teen* OR youth OR youngst* OR student* OR "emerging adult*" OR "early adult*" OR "young adult*") AND (famil* OR parent* OR mother* OR father* OR maternal OR paternal) AND (friend* OR peer* OR romantic* OR intimate* OR marital OR marriage OR couple*) AND (relation* OR dating OR warmth OR bond OR affecti* OR attachment OR intimacy OR nurturance OR sensitivity OR support OR aggression OR conflict OR discord OR hostility OR discipline OR abuse OR victimization OR violence) AND (longitudinal* OR prospective OR intergenerational OR transmission OR spillover OR "spill*_over")

Information about the coding process

Coding procedure

Coding decisions were based on a coding protocol that was developed prior to the coding process by the first and second author, in consultation with three experts in the field of adolescent family and peer relationships. The protocol included all coding categories and information about how studies should be scored based on these categories. Before the final coding, the protocol was piloted across 7 studies by the first and second author. Based on this piloting phase, we discussed diverging ratings with all experts and adjusted the protocol accordingly. Disagreements in the

piloting and coding phase were presented to the experts and discussed among the experts and raters until consensus was reached among all experts. That mainly concerned the inclusion and categorization of relationship constructs.

To operationalize the relationship dimensions (i.e., support, negative interaction, and control), we developed a list of keywords on often-assessed relationship constructs that corresponded to these broad dimensions (see below). This list was developed in agreement with experts and based on the theoretical framing of the three relationship dimensions. For each study, we consulted the description of each included construct and the used instrument to allocate it to one of the three dimensions. We only included constructs that corresponded to relationship quality, for example if the assessed behaviors or constructs characterized relationships with parents, peers, or partners, or if they were directed from one relationship partner towards the other. Study reports that did not focus on specific relationships, but on social competence or behavior in general (e.g., "makes friends easily", "plays with other children") were excluded.

Similarly, study reports that used a very broad construct (e.g., relationship quality) that did not allow us to disentangle different dimensions or a construct that combined several aspects of different dimensions, such as autonomy (dimension of control) and relatedness (dimension of support), were excluded if these constructs could not be separated. We also detected constructs that we could not allocate to one of the three relationship dimensions, such as 'information management' (for studies focusing on communication aspects, information exchange between relationship partners, including disclosure or secrecy). Due to the specific focus of our meta-analysis and limits regarding manuscript length, these additional dimensions were not included in the present study.

Additionally, most studies (87.5%) did not provide information about whether the assessment of peer relationships referred to the same or different relationships at T1 and T2. Of those that did, most studies indicated including a mixture of both stable and unstable peer relationships (57.1%), and only a small proportion of studies assessed the same peer relationships across time (14.3%). As assessments of relationships mostly focus on perceptions of relationships, however, which are likely to remain similar even across different peers, we still recorded stability coefficients for these different relationships.

List with keywords that correspond to specific relationship dimensions

Support	Control	Negative interaction
Commitment	Dominance	Negative interaction
Satisfaction	Control	Aggression
Trust	Autonomy	Hostility
Identification	Power	Rejection
Connectedness	Dating restriction	Violence (including physical and psychological victimization or perpetration)
Attraction	Overprotection	Antagonism
Warmth		Criticism
Intimacy	Directing of friendships	Threat/abuse
Sensitivity	Psychological control	Punishment/discipline
Relatedness	Jealousy	Hassles
Companionship		Problems
Alienation (reverse-coded)		Derogation
Affection		(Di)stress
Emotional support		Coercion
Closeness		Disagreements
Nurture/care		
Acceptance		
Affiliation		

Interrater reliabilities for all main outcomes

Category	κ	ICC
Publication		
Journal IS	1	
Journal IF		1
Study procedures		
Country	1	
Recruitment location	1	
Recruitment strategy	.993	
Attrition across waves	1	
# Waves family		1
# Waves friends		1
# Waves romantic		1
Time lag between measures		1
Sample		
N adolescents fam T1		.992
N adolescents rom/peer T1		.999
M_{age} adolescent T1		1
M_{age} adolescent T2		.928
Population	.990	
% Adolescent boys		.979
% Racial-ethnic minority		.988
Living with both parents		.995
Living with single parents		.995
Degree of friendship	1	
Type of romantic sexuality	1	
Same peer/romantic partner across waves	.80	
Measures		
Relationship dimension parent-adolescent	1	
Relationship dimension peer/romantic	1	
Construct name parent-adolescent	.992	
Construct name peer/rom	.991	
Family member	.979	
Informant parent-adolescent	1	
Informant peer/romantic	1	
Instrument parent-adolescent	1	
Instrument peer/rom	1	
IRR parent-adolescent	1	
IRR peer/romantic	1	
Effect sizes		
Effect size		.997
Sample size N		1

Table S1A. Sample Descriptives for Peer Relationships

Study (Year)	*N*	# waves family	# waves peer	Time lag (in mo)	Age T_n	% boys	% rac-eth minority	Region	Study cohort	Population	Publication status
Allen et al. (2006)	143	2	2	12.00	13.34	48.25	37.00	North America	study specific	community	published
Appel et al. (2014)	363	2	2	6.00	12.84	45.70		Western Europe	EARSandEYES	community	published
Asendorpf & van Aken (2003)	167	2	2	36.00	12.00	51.74		Western Europe	Munich Longitudinal Study on the Genesis of Individual Competencies	community	published
Bae, 2015	2264	1	3	12.00		52.20		Asia	Korea Youth Panel Survey	community	published
Baril et al. (2009)	29	1	1	84.00	14.63	0.00	0.00	North America	study specific	community	published
Becht et al. (2017)	450	5	5	12.00	13.03	57.00	5.20	Western Europe	Research on Adolescent Development and Relationships (RADAR)	community	published
Benner et al. (2017)	138	2	2	12.00	14.38	50.00	98.00	North America	Schools, Peers, and Adolescent Development Project	racial-ethnic minority	published
Burke et al. (2017)	814	4	4	6.00	13.20	51.00		Western Europe	net-TEEN	community	published
De Goede et al. (2009, sample A)	923	5	5	12.00	12.40	54.10	15.50	Western Europe	Conflict and Management Of Relationships (CONAMORE)	community	published
De Goede et al. (2009, sample B)	390	5	5	12.00	16.70	43.30	15.50	Western Europe	Conflict and Management Of Relationships (CONAMORE)	community	published
Drew (2017)	555	2	2	6.00	12.00	46.49		Western Europe	study specific	community	unpublished
Elmore & Huebner (2010)	398	2	2	12.00		39.60	56.80	North America	study specific	community	published
Fenzel (2000)	118	2	2	3.00	10.80	43.97	10.00	North America	study specific	community	published
Gallagher et al. (2014)	144	2	2	9.00	13.51	28.00	25.00	North America	study specific	clinical	published
Gayman et al. (2011)	1267	1	1	96.00	12.00	74.00	71.00	North America	study specific	community	published
Giordano et al. (1998)	620	2	2	120.00	15.30	44.00	49.00	North America	study specific	marginalized	published
Gore & Aseltine (1995)	1036	2	2	12.00	15.50	43.40	0.00	North America	study specific	community	published
Guan & Fuligni (2016)	478	3	3	24.00	17.81	48.90	80.50	North America	study specific	community	published
Guay et al. (2008)	380	3	3	12.00	17.70	28.30		North America	study specific	community	published
Guay et al. (2017)	639	2	2	12.00		42.00		North America	study specific	marginalized	published
Guinn (2012)	272	1	2	3.00	18.12	35.66	51.70	North America	study specific	community	unpublished
Hazel et al. (2014, sample A)	243	1	5	3.00	11.00	44.45	32.35	North America	study specific	marginalized	published
Hazel et al. (2014, sample B)	223	1	5	3.00	14.60	44.45	32.35	North America	study specific	minority	published
Herd et al. (2018)	167	2	2	24.00	14.13	53.00	20.00	North America	study specific	community	published
Holt et al. (2018, sample A)	153	2	2	48.00	18.60	36.00	27.00	North America	study specific	community	published
Holt et al. (2018, sample B)	138	2	2	48.00	17.67	28.00	32.00	North America	study specific	community	published

Table S1A. Continued.

Study (Year)	*N*	# waves family	# waves peer	Time lag (in mo)	Age T_n	% boys	% rac-eth minority	Region	Study cohort	Population	Publication status
Kretschmer et al. (2016)	1806	1	1	60.00	11.00	49.00	10.00	Western Europe	Tracking Adolescents' Individual Lives Survey (TRAILS)	community	published
Laird et al. (2013)	163	2	2	12.00	12.40	49.50	60.70	North America	study specific	community	published
Laursen et al. (2006)	188	2	2	24.00	15.30	50.00	30.50	North America	study specific	community	published
Letcher et al. (2009)	1141	6	2	24.00	13.00	51.90		Oceania	Australian Temperament Project (ATP)	community	published
Luijpers (2000, sample A)	387	2	2	36.00	13.00	46.86		Western Europe	Wendingen in de Levensloop	community	unpublished
Luijpers (2000, sample B)	444	2	2	36.00	16.00	46.86		Western Europe	Wendingen in de Levensloop	community	unpublished
Luyckx et al. (2012)	429	2	2	9.00	15.75	53.40		Western Europe	Information technology Devices and Education programme for Transitioning Adolescents with Congenital Heart disease (i-DETACH)	community	published
Mak et al. (2018)	567	3	3	18.00	11.27	47.70	10.00	North America	Promoting School-Community-University Partnerships to Enhance Resilience (PROSPER)	community	published
Martin et al. (2017)	278	1	3	12.00	13.00	50.00	73.00	North America	study specific	community	published
Meeus et al. (2007)	350	2	2	72.00	17.59	42.00	1.00	Western Europe	Utrecht Study of Adolescent Development (USAD)	community	published
Mize & Kliewer (2018, sample A)	167	2	2	12.00	10.78	48.40	91.40	North America	Project COPE	marginalized	published
Mize & Kliewer (2018, sample B)	146	2	2	12.00	13.68	43.70	92.00	North America	Project COPE	marginalized	published
Murphy et al. (2012)	107	11	11	6.00	13.00	52.00	95.00	North America	Parents and Children Coping Together (PACT)	minority	published
Musliner & Singer (2014)	15409	2	1	168.00	14.90	52.80	28.30	North America	National Longitudinal Study of Adolescent Health (Add Health)	community	published
Nummer & Seiffge-Krenke (2001)	210	4	4	12.00	13.90	46.80		Western Europe	study specific	community	published
Pinquart & Pfeiffer (2013, sample A)	134	3	3	12.00	15.64	44.00		Western Europe	Marburg Study of Vision Loss	clinical	published
Pinquart & Pfeiffer (2013, sample B)	422	3	3	12.00	14.19	48.00		Western Europe	Marburg Study of Vision Loss	community	published
Puckett (2010)	371	2	2	12.00	13.95	42.10	21.00	North America	study specific	community	unpublished
Rauer et al. (2013)	511	1	1	48.00	12.00	51.00	16.00	North America	Child Development Project	community	published
Rice & Mulkeen (1995)	109	3	3	48.00	13.00	47.71	0.00	North America	study specific	community	published

Table S1A. Continued.

Study (Year)	*N*	# waves family	# waves peer	Time lag (in mo)	Age T_n	% boys	% rac-eth minority	Region	Study cohort	Population	Publication status
Rodríguez et al. (2014)	246	3	3	24.00	12.51	49.00	100.00	North America	study specific	minority	published
Rosario et al. (2008)	613	3	3	12.00	11.80	50.22	97.00	North America	study specific	marginalized	published
Schmidt & Seiffge-Krenke (1996, sample A)	53	3	3	24.00	13.90			Western Europe	Chronisch kranke Jugendliche und ihre Familien	community	published
Schmidt & Seiffge-Krenke (1996, sample B)	36	3	3	24.00	13.90			Western Europe	Chronisch kranke Jugendliche und ihre Familien	clinical	published
Seiffge-Krenke (2003)	103	3	3	24.00	13.00	40.80		Western Europe	study specific	community	published
Seiffge-Krenke & Persike (2017)	145	2	2	48.00	13.60	44.80	7.00	Western Europe	study specific	community	published
Song et al. (2015)	6089	2	2	12.00		51.80		Asia	Korean Educational Longitudinal Study	community	published
Starks et al. (2015)	186	3	3	6.00	18.80	53.40	86.30	North America	study specific	community	published
Tillinger (2013)	93	1	1	96.00	10.00	47.30	10.90	North America	Early Intervention Collaborative Study (EICS)	clinical	unpublished
Umemura & Šerek (2016)	409	1	1	48.00	13.00	50.00		Eastern Europe	European Longitudinal Study of Pregnancy and Childhood	community	published
Van Zalk & Van Zalk (2015)	2194	3	3	12.00	13.58	52.01	7.60	North America	study specific	community	published
Wray-Lake et al. (2016)	4150	3	3	12.00	13.70	44.60	28.20	North America	Social Responsibility and Prevention Project	community	published
Yeung Thompson & Leadbeater (2013, sample A)	361	3	3	24.00	13.50		15.00	North America	Victoria Healthy Youth Survey	community	published
Yeung Thompson & Leadbeater (2013, sample B)	278	3	3	24.00	17.00		15.00	North America	Victoria Healthy Youth Survey	community	published
Zhang et al. (2018)	945	4	4	6.00	13.95	53.00	20.80	Western Europe	Studies on Trajectories of Adolescent Relationships and Sexuality (STARS)	community	published

Note. *N* = sample size, time lag = time between measurements (in months), age T_n = adolescent age at first included measurement (in years), rac-eth = racial-ethnic, community = mostly racial-ethnic majority, non-clinical sample, clinical = clinical sample (including mental or physical disabilities), minority = racial-ethnic minority sample (including refugees), marginalized = economically marginalized, such as low income sample, empty cells = missing data.

Table S1B. Sample Descriptives for Romantic Relationships

Study (Year)	*N*	# waves family	# waves romantic	Time lag (in mo)	Age T_n	% boys	% rac-eth minority	Region	Study cohort	Population	Publication status
Ajayi (2011)	3346	1	4	60.00	15.21	49.10	48.00	North America	National Longitudinal Survey of Youth (NLSY97)	community	unpublished
Allen et al. (2014)	184	2	1	84.00	15.30	46.70	42.00	North America	study specific	community	published
Andrews et al. (2000)	135	1	1	72.00	16.70	42.00	7.00	North America	study specific	community	published
Beach et al. (2017)	380	1	2	126.00	10.50		100.00	North America	Family and Community Health Study (FACHS)	minority	published
Brendgen et al. (2002)	336	1	1	54.00	12.00	100.00		North America	study specific	community	published
Brook et al. (2013)	816	1	1	168.00	19.20	40.00	100.00	North America	study specific	minority	published
Burns & Dunlop (1998)	72	3	1	120.00	14.70	52.60		Oceania	study specific	community	published
Chen et al. (2008)	1560	1	1	156.00	12.80	37.00		North America	study specific	community	published
De Goede et al. (2012, sample A)	218	5	1	48.00	12.40	39.00	21.00	Western Europe	Conflict and Management Of Relationships (CONAMORE)	community	published
De Goede et al. (2012, sample B)	185	5	1	48.00	16.70	30.80	22.00	Western Europe	Conflict and Management Of Relationships (CONAMORE)	community	published
Fosco et al. (2016)	106	1	2	6.00	16.38	39.40	43.20	North America	study specific	community	published
Giordano et al. (1998)	620	1	1	120	15.30	44.00	49.00		study specific	marginalized	published
Goodnight et al. (2017)	240	1	6	24.00	16.00	44.00	19.00	North America	Child Development Project	community	published
Ivanova (2012)	131	3	1	120.00	15.00	56.80		Western Europe	Solna study	community	unpublished
Katz et al. (2013)	182	1	1	60.00	15.00	36.26	4.00	North America	Mater University Study of Pregnancy	clinical	published
Kaufman-Parks et al. (2017)	950	5	5	12.00	15.22	46.60	34.10	North America	Toledo Adolescent Relationships Study (TARS)	community	published
Kaufman-Parks et al. (2018)	950	5	5	12.00	15.22	46.60	34.10	North America	Toledo Adolescent Relationships Study (TARS)	community	published
Kim & Pears (2009)	190	1	1	156.00	15.50	100.00	10.00	North America	Oregon Youth Study (OYS)	community	published
Kochendorfer & Kerns (2017)	192	1	2	24.00	10.00	63.00	28.10	North America	National Institute of Child Health and Human Development Study of Early Child Care and Youth Development (NICHD SECCYD)	community	published
Kogan et al. (2013)	285	2	1	134.40	10.40	40.60	100.00	North America	Family and Community Health Study (FACHS)	minority	published
Kretschmer et al. (2017)	811	1	1	132.00	11.10	49.00	10.00	Western Europe	Tracking Adolescents' Individual Lives Survey (TRAILS)	community	published
Laursen et al. (2006)	167	2	2	24.00	15.30	50.00	30.50	North America	study specific	community	published

Table S1B. Continued.

Study (Year)	*N*	# waves family	# waves romantic	Time lag (in mo)	Age T_n	% boys	% rac-eth minority	Region	Study cohort	Population	Publication status
Lee (2018, sample A)	520	1	1	72.00	18.00	40.20	6.00	North America	Michigan Study of Adolescent Life Transitions	community	published
Lee (2018, sample B)	154	1	1	72.00	18.00	30.50	6.00	North America	Michigan Study of Adolescent Life Transitions	community	published
Linder & Collins (2005)	81	1	2	96.00	13.00	47.93	32.20	North America	Minnesota Longitudinal Study of Parents and Children	marginalized	published
Lohman et al. (2013)	392	1	2	78.00	14.50	48.00	0.00	North America	Family Transitions Project (FTP)	community	published
Makin-Byrd et al. (2013)	401	1	1	60.00		57.00	41.60	North America	Fast Track Multisite Investigation	community	published
Meeus et al. (2004, sample A)	362	3	3	36.00	15.80	42.00	1.00	Western Europe	Utrecht Study of Adolescent Development (USAD)	community	published
Meeus et al. (2004, sample B)	280	3	3	36.00	22.50	42.00	1.00	Western Europe	Utrecht Study of Adolescent Development (USAD)	community	published
Morris et al. (2015)	461	1	1	72.00	11.79	49.00	81.00	Western Europe	Birmingham Youth Violence Study (BYVS)	community	published
Mumford et al. (2016)	1117	1	2	12.00	15.30	52.80	39.60	North America	Survey on Teen Relationships and Intimate Violence (STRiV)	community	published
Puckett (2010)	371	2	2	12.00	13.95	42.10	21.00	North America	study specific	community	unpublished
Scharf & Mayseless (2008)	49	1	2	18.00	17.50	0.00	0.00	Eastern Europe	study specific	community	published
Seiffge-Krenke et al. (2003)	44	3	1	96.00	13.00	40.80	2.00	Western Europe	study specific	community	published
Slominski et al. (2011, sample A)	52	2	1	204.00	13.00	45.00	32.00	North America	study specific	community	published
Slominski et al. (2011, sample B)	41	2	1	204.00	13.00	45.00	32.00	North America	study specific	clinical	published
Starks et al. (2015)	150	3	3	12.00	18.80	53.40	86.30	North America	study specific	community	published
Stocker & Richmond (2007)	110	1	1	36.00	15.06	51.00	20.00	North America	study specific	community	published
Surjadi et al. (2013)	288	1	1	156.00	15.00	43.80	0.00	North America	Family Transitions Project (FTP)	community	published
Sweeten et al. (2016)	617	1	1	48.00	17.60	100.00	80.30	North America	Pathways to Desistance Study	community	published
Tyrell et al. (2014)	142	1	1	24.00	12.29	46.00	100.00	North America	study specific	minority	published
Walper & Wendt (2015)	684	1	1	12.00	18.60	42.40		Western Europe	Panel Analysis of Intimate Relationships and Family Dynamics (PAIRFAM)	community	published

Note. *N* = sample size, time lag = time between measurements (in months), age T_n = adolescent age at first included measurement (in years), rac-eth = racial-ethnic, community = mostly racial-ethnic majority, non-clinical sample, clinical = clinical sample (including mental or physical disabilities), minority = racial-ethnic minority sample (including refugees), marginalized = economically marginalized, such as low income sample, empty cells = missing data.

Table S2. Interpretative Overview of All Significant Main and Moderation Outcomes

		Three-level random effects models – peer outcomes											
		Meta-analytic effect size			*Moderators*								
Parent T_n	Peer T_{n+1}		*r*	*p*	*time*	*age*	*population*	*shared*	*boys*	*rac-eth*	*year*	J_{IF}	J_Q
support	support		+	sig	–	+		+					
	negative		–	sig				N/A					
negative	support		–	sig									
	negative		+	sig	–	+		N/A					
		Three-level random effects models – romantic outcomes											
support	support		+	sig				No heterogeneity in effect sizes					
	negative		–	sig				No heterogeneity in effect sizes					
negative	support		–	sig									
	negative		+	sig									
		Random effects structural equation models – peer outcomes											
support	support	$CL_{par>peer}$	+	sig	–						+	–	+
		$CL_{peer>par}$	+										
	negative	$CL_{par>peer}$	–	sig		+		N/A					
		$CL_{peer>par}$	–										
negative	support	$CL_{par>peer}$	–	sig		+					+	–	
		$CL_{peer>par}$	–	sig		+					+		
	negative	$CL_{par>peer}$	+	sig		–		N/A			–		
		$CL_{peer>par}$	+	sig							–		

Note. Time = time between measurements (in months), age = adolescent age, shared = shared informant (i.e., the same informant reported on parent-adolescent and peer or romantic relationship), year = publication year, boys = percentage of boys, rac-eth = racial-ethnic minority, population = community, marginalized, or clinical sample, J_{IF} = journal impact factor, J_Q = journal quartile, sig = significant, $CL_{par>peer}$ = cross-lagged path from parent T_n to peer T_{n+1}, $CL_{peer>par}$ = cross-lagged path from peer to parent, empty cells = not significant, N/A = too few studies available.

Table S3A. Summary of All Main Three-level Meta-regression Analyses

				Three-level random effects models – peer outcomes						
				Meta-analytic effect size			*Heterogeneity estimates*			
Parent T_n	Peer T_{n+1}	*k*	*n* ES	β	*p*	95% CI	*Q*	p_q	I_w	I_b
support	support	53	174	.18	<.001	[.15, .20]	902.16	<.001	9.6%	68.9%
support	negative	11	53	–.12	<.001	[–.16, –.07]	115.23	<.001	<0.01%	73.4%
negative	support	13	51	–.07	.002	[–.12, –.03]	188.67	<.001	2.7%	74.2%
negative	negative	11	52	.18	<.001	[.12, .24]	163.32	<.001	7.4%	73.7%
				Three-level random effects models – romantic outcomes						
				Meta-analytic effect size			*Heterogeneity estimates*			
Parent T_n	Rom T_{n+1}	*k*	*n* ES	β	*p*	95% CI	*Q*	p_q	I_w	I_b
support	support	18	43	.11	<.001	[.09, .13]	44.57	.364	<0.01%	<0.01%
support	negative	10	24	–.09[6]	<.001	[–.14, –.07]	19.59	.666	<0.01%	<0.01%
negative	support	10	25	–.10	<.001	[–.15, –.05]	49.04	.002	18.8%	50.1%
negative	negative	17	32	.15	<.001	[.11, .19]	206.48	<.001	14.5%	66.0%

Note. *k* = number of independent samples, *n* ES = number of effect sizes, CI = lower and upper limits of confidence interval, *Q* = heterogeneity test, p_q = *p* value of heterogeneity statistic, I_w = percentage of within-study heterogeneity, I_b = percentage of between-study heterogeneity.

6 Due to small variation on the third level of the analyses, the analysis resulted in problems estimating the standard errors. We therefore reran the analysis using the package metafor.

Table S3B. Summary of All Main MASEM Analyses

	Random effects structural equation models – peer outcomes													
	Meta-analytic effect size												*Heterogeneity*	
	CL par>peer	*p*	CL peer>par	*p*	Stab parent	*p*	Stab peer	*p*	CS T_n	*p*	CS T_{n+1}	*p*	*Q*	*p*
support on support (*k*=53)	.07	<.001	.01	.324	.55	<.001	.41	<.001	.23	<.001	.16	<.001	2966.58	<.001
support on negative (*k*=11)	–.06	.016	–.02	.333	.59	<.001	.39	<.001	–.15	<.001	–.02	.403	199.3	<.001
negative on support (*k*=13)	–.06	.001	–.06	.017	.54	<.001	.43	<.001	–.02	.572	–.04	.322	323.32	<.001
negative on negative (*k*=11)	.10	.001	.06	.026	.50	<.001	.37	<.001	.25	<.001	.18	<.001	399.40	<.001

Note. CL par>peer = cross-lagged path from parent T_n to peer T_{n+1}, CL peer>par = cross-lagged path from peer T_n to parent T_{n+1}, Stab parent = stability path from parent T_n to parent T_{n+1}, Stab peer = stability path from peer T_n to peer T_{n+1}, CS T_n = concurrent association between parent T_n and peer T_n, CS T_{n+1} = concurrent association between parent T_{n+1} and peer T_{n+1}.

Table S4. Summary of Main Three-level Meta-Regression Analyses involving Control

				Three-level random effects models – peer outcomes						
				Meta-analytic effect size			*Heterogeneity estimates*			
Parent T_n	Peer T_{n+1}	*k*	*n ES*	β	*p*	95% CI	*Q*	p_q	I_w	I_b
control	control	2	20	.240	<.001	[.209, .270]	43.35	.001	48.6%	5.7%
control	support	1	21	.036	<.001	[.020, .053]	13.17	.870	-	-
control	negative	2	20	.101	<.001	[.076, .127]	32.28	.029	32.7%	3.2%
support	control	2	20	–.055	.002	[–.090, –.019]	17.26	.573	<.01%	23.5%
negative	control	2	20	.105	<.001	[.065, .145]	26.41	.119	3.9%	28.8%

				Three-level random effects models – romantic outcomes						
				Meta-analytic effect size			*Heterogeneity estimates*			
Parent T_n	Rom T_{n+1}	*k*	*n* ES	β	*p*	95% CI	*Q*	p_q	I_w	I_b
control	*control*	*1*	*1*	*–.075*	*.049*	*[–.150, –.000]*	-	-	-	-
control	*support*	*4*	*5*	*–.030*	*.210*	*[–.076, .017]*	*6.99*	*.136*	*<0.01%*	*<0.01%*
control	*negative*	*4*	*5*	*.047*	*NA*	*NA*	*6.74*	*.150*	*<0.01%*	*<0.01%*
support	*control*	*1*	*1*	*.145*	*<.001*	*[.072, .218]*	-	-	-	-
negative	control	2	11	.015	.816	[–.109, –.139]	43.22	<.001	18.3%	69.4%

Note. *k* = number of independent samples, *n* ES = number of independent effect sizes, 95% CI = lower and upper limits of 95% confidence interval, *Q* = heterogeneity test, p_q = *p* value of heterogeneity statistic, I_w = percentage of within-study heterogeneity, I_b = percentage of between-study heterogeneity. Fit attempts for italicized estimates resulted in errors.

Table S5. Descriptive Statistics for Moderator Variables included in Subgroup Analyses

Moderator	*Peer outcomes*				*Romantic outcomes*			
	Frequency (%) or *M* (*SD*)				Frequency (%) or *M* (*SD*)			
	support T_n → support T_{n+1}	support T_n → negative T_{n+1}	Negative T_n → support T_{n+1}	Negative T_n → negative T_{n+1}	support T_n → support T_{n+1}	support T_n → negative T_{n+1}	Negative T_n → support T_{n+1}	Negative T_n → negative T_{n+1}
Time lag (in months)	26.8 (23.0)	20.0 (13.4)	25.4 (18.1)	21.0 (11.6)	59.2 (46.4)	74.6 (40.8)	81.6 (66.1)	59.6 (47.3)
Age T_n (in years)	14.7 (2.0)	14.4 (2.1)	14.5 (2.0)	14.7 (1.8)	16.4 (3.8)	14.7 (2.7)	15.8 (2.5)	15.1 (2.0)
Population	58.6	79.2	94.1	90.4	83.7	79.2	72.0	84.4
Community	36.8	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Marginalized	4.6	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Clinical								
Shared informant	92.0	N/A	70.6	N/A	N/A	N/A	60.0	65.6
Adolescent boys	49.3 (6.9)	50.1 (5.6)	50.5 (9.9)	50.4 (5.5)	43.2 (10.8)	47.0 (6.7)	46.4 (4.9)	52.2 (16.6)
Racial-ethnic minority	55.5 (40.1)	16.4 (9.1)	13.9 (10.6)	17.3 (18.3)	41.9 (39.8)	45.0 (30.2)	41.9 (30.8)	38.2 (25.2)
Publication year	2011 (5.4)	2011 (6.8)	2011 (7.0)	2009 (7.3)	2011 (6.0)	2012 (6.8)	2014 (3.9)	2013 (5.1)
Journal impact factor	2.1 (1.1)	2.4 (1.4)	2.1 (1.2)	1.9 1.3)	2.1 (0.9)	3.2 (1.4)	2.0 (1.0)	2.1 (1.0)
Journal quartile	2.3 (1.0)	2.1 (1.2)	2.2 (1.1)	2.5 (1.2)	1.8 (1.0)	1.3 (0.8)	2.1 (0.8)	2.3 (0.9
Publication status	97.1	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Published	2.9	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Unpublished								

Table S6. Model Fit Evaluation for Moderation Analyses

	Analyses								
	Three-level meta-regression – peer			*Three-level meta-regression – romantic*			*MASEM – peer*		
Moderator	$\Delta\chi^2$	*df*	*p*	ΔX^2	*df*	*p*	$\Delta\chi^2$	*df*	*p*
Time lag (in months)									
support T_n → support T_{n+1}	35.79	1	<.001	-	-	-	–272.68	2	>.999
support T_n → negative T_{n+1}	1.33	1	.249	-	-	-	13.54	2	.001
negative T_n → support T_{n+1}	1.00	1	.317	0.68	1	.410	–20.27	2	>.999
negative T_n → negative T_{n+1}	15.20	1	<.001	1.15	1	.283	1.81	2	.405
Adolescent age (in years)									
support T_n → support T_{n+1}	6.11	2	.047	-	-	-	–35.22	23	>.999
support T_n → negative T_{n+1}	0.08	1	.783	-	-	-	7.125	2	.028
negative T_n → support T_{n+1}	<0.001	1	.992	0.14	1	.704	17.28	2	<.001
negative T_n → negative T_{n+1}	3.94	1	.047	–2.05	2	>.999	9.52	2	.009
Population: community									
support T_n → support T_{n+1}	0.05	1	.816	-	-	-	0.64	2	.726
Population: marginalized									
support T_n → support T_{n+1}	0.10	1	.756	-	-	-	0.27	2	.872
Population: clinical									
support T_n → support T_{n+1}	0.76	1	.384	-	-	-	4.75	2	.093
Shared reporter									
support T_n → support T_{n+1}	3.89	1	.048	-	-	-	5.18	2	.075
negative T_n → support T_{n+1}	0.06	1	.801	0.93	1	.335	5.05	2	.080
negative T_n → negative T_{n+1}	-	-	-	0.29	1	.587	-	-	-

	Analyses								
	Three-level meta-regression – peer			*Three-level meta-regression – romantic*			*MASEM – peer*		
Moderator	$\Delta\chi^2$	*df*	*p*	ΔX^2	*df*	*p*	$\Delta\chi^2$	*df*	*p*
Adolescent boys (%)									
support T_n → support T_{n+1}	–20.52	13	>.999	-	-	-	0.37	2	.831
support T_n → negative T_{n+1}	–9.17	7	>.999	-	-	-	1.57	2	.456
negative T_n → support T_{n+1}	–7.40	7	>.999	–5.49	3	>.999	2.53	2	.282
negative T_n → negative T_{n+1}	–6.18	7	>.999	–4.77	3	>.999	5.65	2	.059
Racial-ethnic minority (%)									
support T_n → support T_{n+1}	–63.19	37	>.999	-	-	-	–181.34	2	>.999
support T_n → negative T_{n+1}	–2.17	8	>.999	-	-	-	6.05	2	.049[7]
negative T_n → support T_{n+1}	–9.56	8	>.999	–5.49	3	>.999	–23.30	2	>.999
negative T_n → negative T_{n+1}	–8.50	13	>.999	–4.77	3	>.999	–29.18	2	>.999
Year of publication									
support T_n → support T_{n+1}	0.05	1	.818	-	-	-	11.83	.2	.003
support T_n → negative T_{n+1}	0.22	1	.642	-	-	-	4.74	2	.093
negative T_n → support T_{n+1}	1.13	1	.288	0.03	1	.853	12.40	2	.002
negative T_n → negative T_{n+1}	0.00	1	.953	0.08	1	.773	11.81	2	.003
Journal impact factor									
support T_n → support T_{n+1}	–77.97	61	>.999	-	-	-	9.74	2	.008
support T_n → negative T_{n+1}	2.01	2	.366	-	-	-	2.44	2	.295
negative T_n → support T_{n+1}	0.16	1	.689	–6.11	4	>.999	8.42	2	.015
negative T_n → negative T_{n+1}	–2.52	2	>.999	–16.45	6	>.999	3.69	2	.158
Journal quartile									
support T_n → support T_{n+1}	7.88	6	.247	-	-	-	21.76	2	<.001

7 Due to small sample size and high model complexity, the analysis resulted in problems estimating reliable standard errors. We therefore do not report and interpret these results. None of the estimates were statistically significant ($\beta_{par \rightarrow peer} = -.004$, $p > .999$; $\beta_{peer \rightarrow par} = -.002$, $p > .999$).

	Analyses								
	Three-level meta-regression – peer			*Three-level meta-regression – romantic*			*MASEM – peer*		
Moderator	$\Delta\chi^2$	*df*	*p*	ΔX^2	*df*	*p*	$\Delta\chi^2$	*df*	*p*
support T_n → negative T_{n+1}	1.58	2	.453	-	-	-	0.94	2	.625
negative T_n → support T_{n+1}	0.08.	1	.781	–5.80	4	>.999	2.62	2	.270
negative T_n → negative T_{n+1}	–1.80	2	>.999	–15.54	6	>.999	2.93	2	.231
Publication status: unpublished									
support T_n → support T_{n+1}	0.86	1	.355	-	-	-	4.51	2	.105

Note. Time lag = time between measurements (in months), adolescent age = age at first included measurement (in years), community = mostly racial-ethnic majority, non-clinical sample, marginalized = economically marginalized (e.g., low income) and/or racial-ethnic minority sample, clinical = clinical sample (including mental or physical disabilities), – = not tested due to insufficient heterogeneity or too few studies.

Table S7. Summary of All Moderation Outcomes

Moderator	*Three-level meta-regression – peer*			*Three-level meta-regression – romantic*			*MASEM – peer*		
	β	*p*	95% CI	β	*p*	95% CI	$CL_{par>peer}$ $CL_{peer>par}$	β	*p*
Time lag (in months)									
support T_n → support T_{n+1}	−.002	<.001	[−.002;−.001]	-	-	-			
support T_n → negative T_{n+1}				-	-	-		.001 .01	.451 .067
negative T_n → support T_{n+1}									
negative T_n → negative T_{n+1}	−.002	<.001	[−.004;−.001]						
Adolescent age (in years)[8]									
support T_n → support T_{n+1}	.01	.001	[.004, .021]	-	-	-			
support T_n → negative T_{n+1}				-	-	-		.01 .03	.482 .042
negative T_n → support T_{n+1}								.02 .03	.003 .004
negative T_n → negative T_{n+1}	.02	.039	[.00, .03]					−.01 −.04	.542 .002
Population: community									
support T_n → support T_{n+1}				-	-	-			
Population: marginalized									
support T_n → support T_{n+1}				-	-	-			
Population: clinical									
support T_n → support T_{n+1}				-	-	-			

8 Rerunning the moderation analyses without the estimated cases based on the country's general age range per grade level yielded similar estimates and conclusions.

Moderator	*Three-level meta-regression – peer*			*Three-level meta-regression – romantic*			*MASEM – peer*		
	β	*p*	95% CI	β	*p*	95% CI	$CL_{par>peer}$ $CL_{peer>par}$	β	*p*
Shared informant									
support T_n → support T_{n+1}	.10	.046	[.00; .20]	-	-	-			
negative T_n → support T_{n+1}									
Boys (%)									
support T_n → support T_{n+1}				-	-	-			
support T_n → negative T_{n+1}				-	-	-			
negative T_n → support T_{n+1}									
negative T_n → negative T_{n+1}									
Racial-ethnic minority (%)									
support T_n → support T_{n+1}				-	-	-			
support T_n → negative T_{n+1}				-	-	-			
negative T_n → support T_{n+1}									
negative T_n → negative T_{n+1}									
Year of publication									
support T_n → support T_{n+1}				-	-	-		.003 .004	.062 .019
support T_n → negative T_{n+1}				-	-	-			
negative T_n → support T_{n+1}								−.004 −.01	.013 .005
negative T_n → negative T_{n+1}								.001 .01	.738 <.001

Moderator	*Three-level meta-regression – peer* β	*p*	95% CI	*Three-level meta-regression – romantic* β	*p*	95% CI	*MASEM – peer* $CL_{par>peer}$ $CL_{peer>par}$	β	*p*
Journal impact factor									
support T_n → support T_{n+1}				-	-	-		–.01 –.03	.263 .009
support T_n → negative T_{n+1}				-	-	-			
negative T_n → support T_{n+1}								–.01 –.03	.264 .009
negative T_n → negative T_{n+1}								-	-
Journal quartile									
support T_n → support T_{n+1}				-	-	-		.02 .04	.029 <.001
support T_n → negative T_{n+1}				-	-	-			
negative T_n → support T_{n+1}									
negative T_n → negative T_{n+1}									
Publication status: unpublished									
support T_n → support T_{n+1}				-	-	-			

Note. CI = lower and upper limits of confidence interval, $CL_{par>peer}$ = cross-lagged path from parent T_n to peer T_{n+1}, $CL_{peer>par}$ = cross-lagged path from peer T_n to parent T_{n+1}, empty cells = model fit did not significantly improve, – = not tested (no heterogeneity).

Table S8. Publication Bias

	Three-level analyses – Peer outcomes				
	Egger's test		Three parameter selection		
	z	*p*	$r_{adjusted}$	χ^2	*p*
support T_n → support T_{n+1}	0.78	.435	.21	0.20	.652
support T_n → negative T_{n+1}	−0.41	.681	-	-	-
negative T_n → support T_{n+1}	−0.86	.388	−.01	7.03	.008[9]
negative T_n → negative T_{n+1}	−1.24	.215	.19	0.03	.863
	Three-level analyses – Romantic outcomes[10]				
support T_n → support T_{n+1}	1.25	.210	-	-	-
support T_n → negative T_{n+1}	−0.90	.368	-	-	-
negative T_n → support T_{n+1}	2.47	.013	-	-	-
negative T_n → negative T_{n+1}	−0.01	.995	.16	0.77	.380
	MASEM analyses – Peer outcomes				
support T_n → support T_{n+1}	−0.94	.349	.19	3.44	.064
support T_n → negative T_{n+1}	−0.62	.538	−.12	0.29	.592
negative T_n → support T_{n+1}	−0.10	.918	−.06	0.65	.419
negative T_n → negative T_{n+1}	−0.78	.435	.20	<.001	.987

9 The analysis resulted in estimation problems.

10 Due to low variation in effect sizes, we were unable to conduct three-parameter selection analyses for all but one association involving romantic relationships. However, recent research suggests that publication bias in relatively homogeneous datasets is weak at best (van Aert et al., 2019).

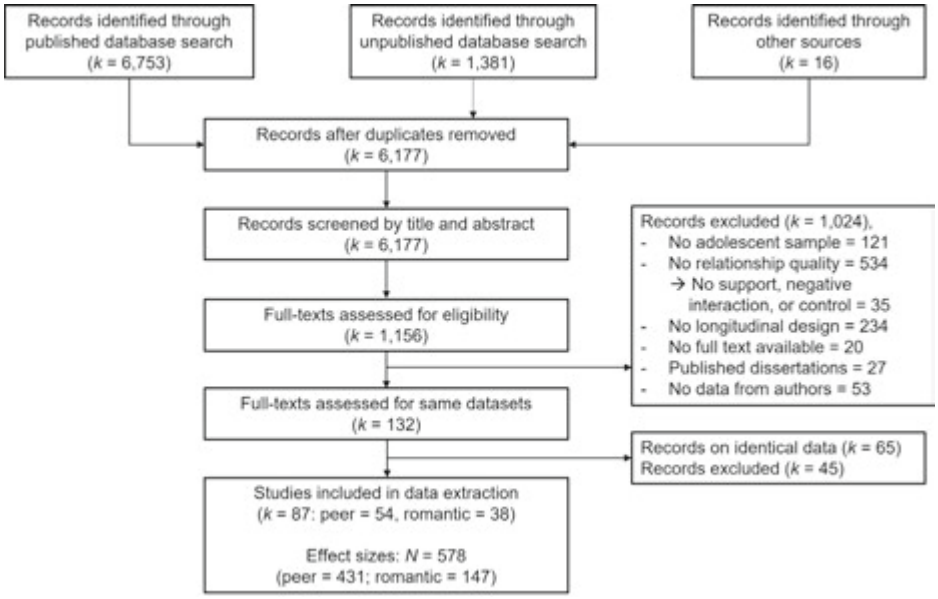
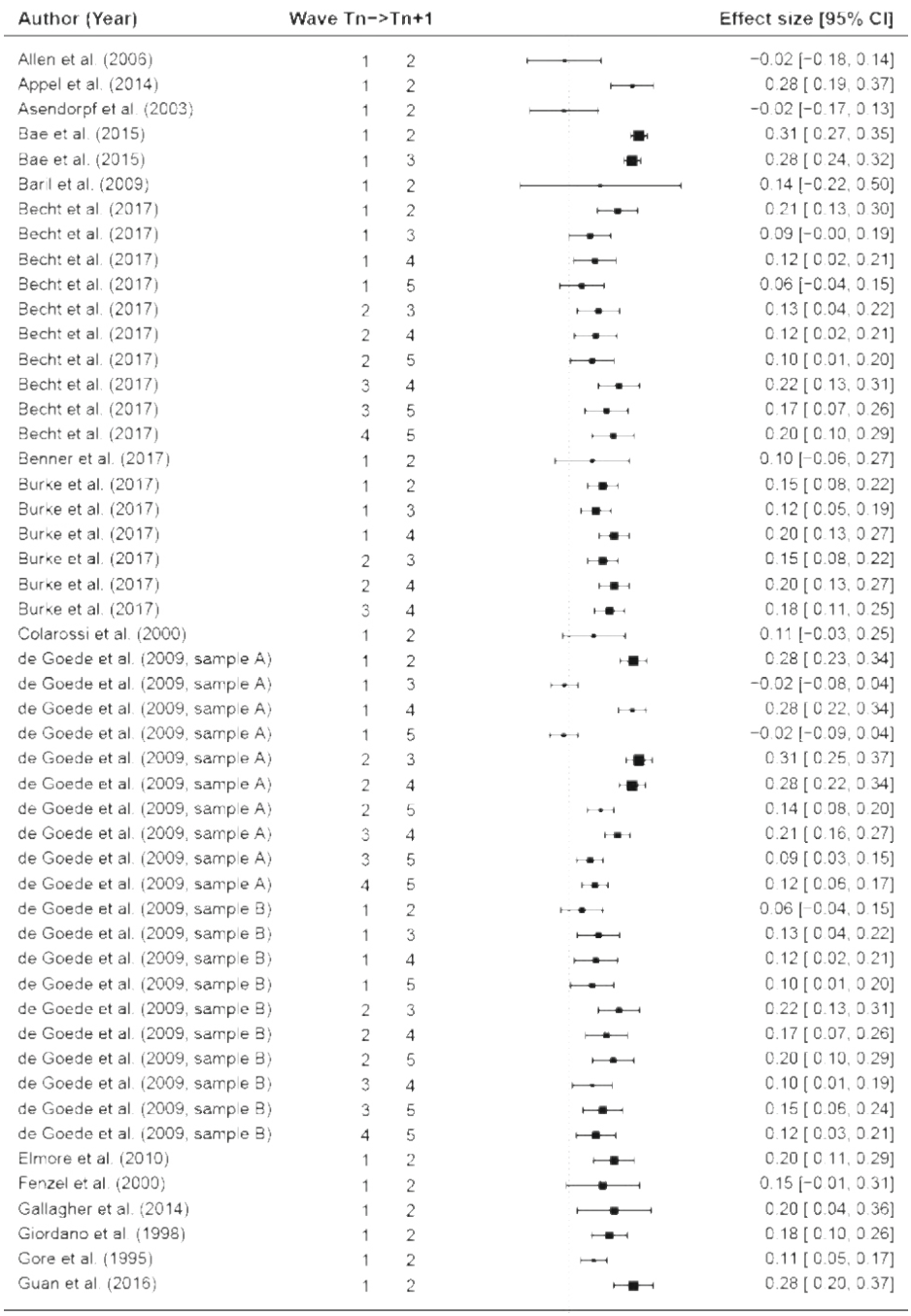


Figure S1. PRISMA flow chart for study inclusion.

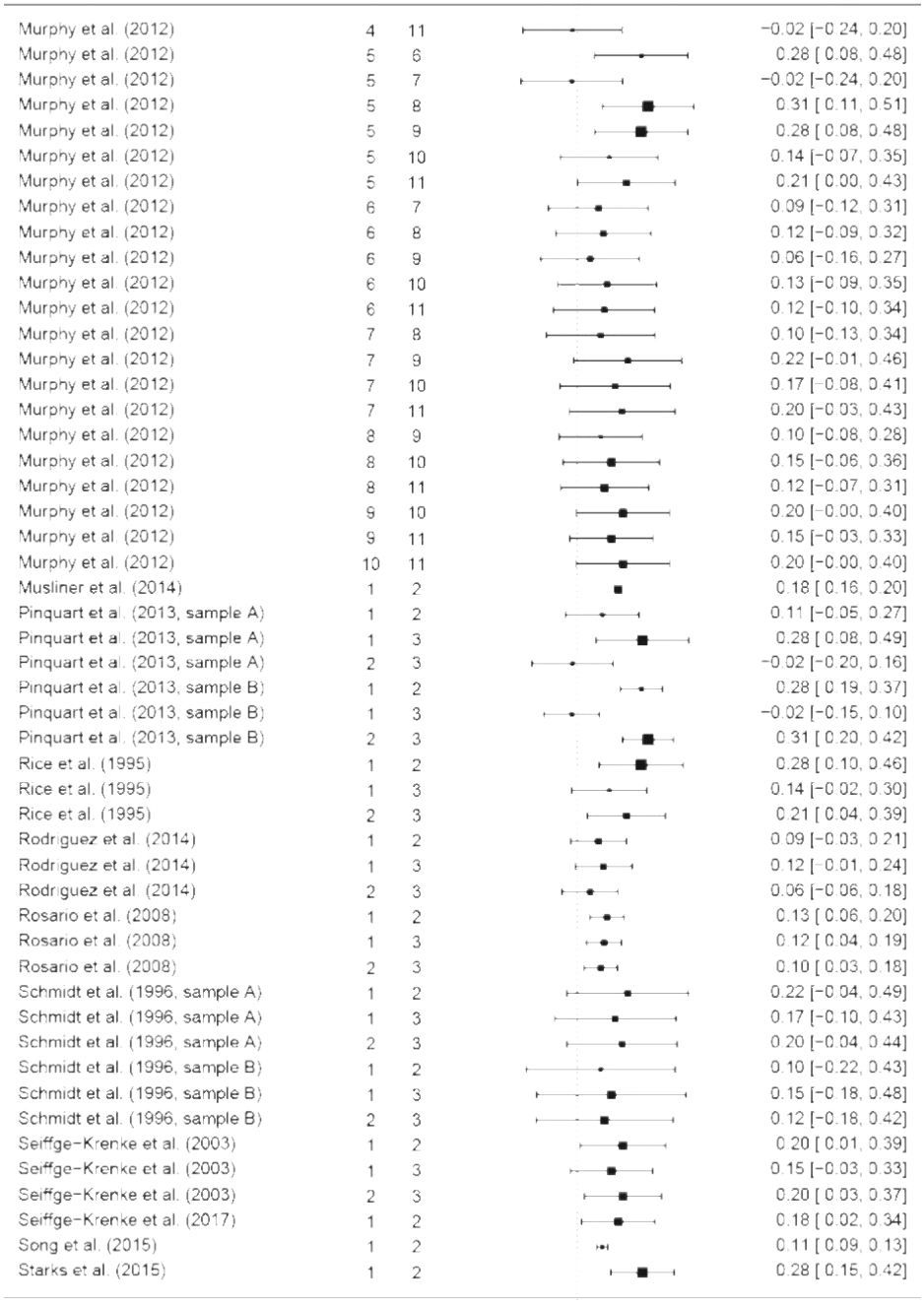
Interpretation of forest plots (Figures S2-S9)

Forest plots regarding peer outcomes (see Figures S2-S5) indicate that particularly for associations across the same dimensions, study findings generally provide conclusive evidence, as indicated by mostly positive small-to-medium effects and many confidence intervals that do not include zero. For associations across different dimensions, study findings seem to exhibit similar conclusions from supportive parent-adolescent relationships to negative peer relationships, but less so from negative parent-adolescent relationships to supportive peer relationships, as indicated by mostly negligible effect sizes close to zero.

Forest plots regarding romantic outcomes (see Figures S6-S9) indicate that study findings generally point in the same direction for all associations with small-to-medium sized effects. While there was one study that also found medium-sized effects in the opposite direction for the association from negative parent-adolescent relationships to supportive romantic relationships, most of the confidence intervals included zero.


Author (Year)
Wave Tn->Tn+1
Effect size [95% CI]
Allen et al. (2006) 1 2 −0.02 [−0.18, 0.14]
Appel et al. (2014) 1 2 0.28 [0.19, 0.37]
Asendorpf et al. (2003) 1 2 −0.02 [−0.17, 0.13]
Bae et al. (2015) 1 2 0.31 [0.27, 0.35]
Bae et al. (2015) 1 3 0.28 [0.24, 0.32]
Barl et al. (2009) 1 2 0.14 [−0.22, 0.50]
Becht et al. (2017) 1 2 0.21 [0.13, 0.30]
Becht et al. (2017) 1 3 0.09 [−0.00, 0.19]
Becht et al. (2017) 1 4 0.12 [0.02, 0.21]
Becht et al. (2017) 1 5 0.06 [−0.04, 0.15]
Becht et al. (2017) 2 3 0.13 [0.04, 0.22]
Becht et al. (2017) 2 4 0.12 [0.02, 0.21]
Becht et al. (2017) 2 5 0.10 [0.01, 0.20]
Becht et al. (2017) 3 4 0.22 [0.13, 0.31]
Becht et al. (2017) 3 5 0.17 [0.07, 0.26]
Becht et al. (2017) 4 5 0.20 [0.10, 0.29]
Benner et al. (2017) 1 2 0.10 [−0.06, 0.27]
Burke et al. (2017) 1 2 0.15 [0.08, 0.22]
Burke et al. (2017) 1 3 0.12 [0.05, 0.19]
Burke et al. (2017) 1 4 0.20 [0.13, 0.27]
Burke et al. (2017) 2 3 0.15 [0.08, 0.22]
Burke et al. (2017) 2 4 0.20 [0.13, 0.27]
Burke et al. (2017) 3 4 0.18 [0.11, 0.25]
Colarossi et al. (2000) 1 2 0.11 [−0.03, 0.25]
de Goede et al. (2009, sample A) 1 2 0.28 [0.23, 0.34]
de Goede et al. (2009, sample A) 1 3 −0.02 [−0.08, 0.04]
de Goede et al. (2009, sample A) 1 4 0.28 [0.22, 0.34]
de Goede et al. (2009, sample A) 1 5 −0.02 [−0.09, 0.04]
de Goede et al. (2009, sample A) 2 3 0.31 [0.25, 0.37]
de Goede et al. (2009, sample A) 2 4 0.28 [0.22, 0.34]
de Goede et al. (2009, sample A) 2 5 0.14 [0.08, 0.20]
de Goede et al. (2009, sample A) 3 4 0.21 [0.16, 0.27]
de Goede et al. (2009, sample A) 3 5 0.09 [0.03, 0.15]
de Goede et al. (2009, sample A) 4 5 0.12 [0.06, 0.17]
de Goede et al. (2009, sample B) 1 2 0.06 [−0.04, 0.15]
de Goede et al. (2009, sample B) 1 3 0.13 [0.04, 0.22]
de Goede et al. (2009, sample B) 1 4 0.12 [0.02, 0.21]
de Goede et al. (2009, sample B) 1 5 0.10 [0.01, 0.20]
de Goede et al. (2009, sample B) 2 3 0.22 [0.13, 0.31]
de Goede et al. (2009, sample B) 2 4 0.17 [0.07, 0.26]
de Goede et al. (2009, sample B) 2 5 0.20 [0.10, 0.29]
de Goede et al. (2009, sample B) 3 4 0.10 [0.01, 0.19]
de Goede et al. (2009, sample B) 3 5 0.15 [0.06, 0.24]
de Goede et al. (2009, sample B) 4 5 0.12 [0.03, 0.21]
Elmore et al. (2010) 1 2 0.20 [0.11, 0.29]
Fenzel et al. (2000) 1 2 0.15 [−0.01, 0.31]
Gallagher et al. (2014) 1 2 0.20 [0.04, 0.36]
Giordano et al. (1998) 1 2 0.18 [0.10, 0.26]
Gore et al. (1995) 1 2 0.11 [0.05, 0.17]
Guan et al. (2016) 1 2 0.28 [0.20, 0.37]

Author (Year)	Wave Tn->Tn+1		Effect size [95% CI]
Guan et al. (2016)	1	3	−0.02 [−0.11, 0.07]
Guan et al. (2016)	2	3	0.28 [0.19, 0.37]
Guay et al. (2008)	1	2	−0.02 [−0.12, 0.07]
Guay et al. (2008)	1	3	0.31 [0.22, 0.40]
Guay et al. (2008)	2	3	0.28 [0.19, 0.37]
Guay et al. (2017)	1	2	0.14 [0.07, 0.21]
Herd et al. (2018)	1	2	0.21 [0.08, 0.35]
Holt et al. (2018, sample A)	1	2	0.09 [−0.06, 0.24]
Holt et al. (2018, sample B)	1	2	0.12 [−0.05, 0.28]
Kretschmer et al. (2016)	1	2	0.06 [0.01, 0.10]
Laird et al. (2013)	1	2	0.13 [−0.02, 0.28]
Laursen et al. (2006)	1	2	0.12 [−0.01, 0.25]
Letcher et al. (2009)	1	2	0.10 [0.05, 0.16]
Luyckx et al. (2012)	1	2	0.22 [0.13, 0.31]
Martin et al. (2017)	1	2	0.17 [0.05, 0.28]
Martin et al. (2017)	1	3	0.20 [0.08, 0.31]
Meeus et al. (2007)	1	2	0.10 [−0.00, 0.21]
Murphy et al. (2012)	1	2	0.15 [−0.03, 0.33]
Murphy et al. (2012)	1	3	0.12 [−0.07, 0.31]
Murphy et al. (2012)	1	4	0.20 [0.02, 0.38]
Murphy et al. (2012)	1	5	0.15 [−0.02, 0.32]
Murphy et al. (2012)	1	6	0.20 [−0.00, 0.40]
Murphy et al. (2012)	1	7	0.18 [−0.02, 0.38]
Murphy et al. (2012)	1	8	0.11 [−0.09, 0.31]
Murphy et al. (2012)	1	9	0.28 [0.08, 0.49]
Murphy et al. (2012)	1	10	−0.02 [−0.24, 0.20]
Murphy et al. (2012)	1	11	0.28 [0.06, 0.50]
Murphy et al. (2012)	2	3	−0.02 [−0.18, 0.14]
Murphy et al. (2012)	2	4	0.31 [0.15, 0.47]
Murphy et al. (2012)	2	5	0.28 [0.12, 0.44]
Murphy et al. (2012)	2	6	0.14 [−0.06, 0.34]
Murphy et al. (2012)	2	7	0.21 [0.02, 0.41]
Murphy et al. (2012)	2	8	0.09 [−0.10, 0.28]
Murphy et al. (2012)	2	9	0.12 [−0.08, 0.31]
Murphy et al. (2012)	2	10	0.06 [−0.15, 0.26]
Murphy et al. (2012)	2	11	0.13 [−0.08, 0.34]
Murphy et al. (2012)	3	4	0.12 [−0.04, 0.28]
Murphy et al. (2012)	3	5	0.10 [−0.07, 0.28]
Murphy et al. (2012)	3	6	0.22 [0.02, 0.43]
Murphy et al. (2012)	3	7	0.17 [−0.04, 0.37]
Murphy et al. (2012)	3	8	0.20 [−0.00, 0.40]
Murphy et al. (2012)	3	9	0.10 [−0.10, 0.30]
Murphy et al. (2012)	3	10	0.15 [−0.06, 0.36]
Murphy et al. (2012)	3	11	0.12 [−0.09, 0.33]
Murphy et al. (2012)	4	5	0.20 [0.04, 0.36]
Murphy et al. (2012)	4	6	0.15 [−0.05, 0.35]
Murphy et al. (2012)	4	7	0.20 [0.01, 0.39]
Murphy et al. (2012)	4	8	0.18 [−0.01, 0.37]
Murphy et al. (2012)	4	9	0.11 [−0.09, 0.31]
Murphy et al. (2012)	4	10	0.28 [0.09, 0.48]


Author (Year)
Wave Tn->Tn+1
Effect size [95% CI]
Murphy et al. (2012) 4 11 −0.02 [−0.24, 0.20]
Murphy et al. (2012) 5 6 0.28 [0.08, 0.48]
Murphy et al. (2012) 5 7 −0.02 [−0.24, 0.20]
Murphy et al. (2012) 5 8 0.31 [0.11, 0.51]
Murphy et al. (2012) 5 9 0.28 [0.08, 0.48]
Murphy et al. (2012) 5 10 0.14 [−0.07, 0.35]
Murphy et al. (2012) 5 11 0.21 [0.00, 0.43]
Murphy et al. (2012) 6 7 0.09 [−0.12, 0.31]
Murphy et al. (2012) 6 8 0.12 [−0.09, 0.32]
Murphy et al. (2012) 6 9 0.06 [−0.16, 0.27]
Murphy et al. (2012) 6 10 0.13 [−0.09, 0.35]
Murphy et al. (2012) 6 11 0.12 [−0.10, 0.34]
Murphy et al. (2012) 7 8 0.10 [−0.13, 0.34]
Murphy et al. (2012) 7 9 0.22 [−0.01, 0.46]
Murphy et al. (2012) 7 10 0.17 [−0.08, 0.41]
Murphy et al. (2012) 7 11 0.20 [−0.03, 0.43]
Murphy et al. (2012) 8 9 0.10 [−0.08, 0.28]
Murphy et al. (2012) 8 10 0.15 [−0.06, 0.36]
Murphy et al. (2012) 8 11 0.12 [−0.07, 0.31]
Murphy et al. (2012) 9 10 0.20 [−0.00, 0.40]
Murphy et al. (2012) 9 11 0.15 [−0.03, 0.33]
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Musliner et al. (2014) 1 2 0.18 [0.16, 0.20]
Pinquart et al. (2013, sample A) 1 2 0.11 [−0.05, 0.27]
Pinquart et al. (2013, sample A) 1 3 0.28 [0.08, 0.49]
Pinquart et al. (2013, sample A) 2 3 −0.02 [−0.20, 0.16]
Pinquart et al. (2013, sample B) 1 2 0.28 [0.19, 0.37]
Pinquart et al. (2013, sample B) 1 3 −0.02 [−0.15, 0.10]
Pinquart et al. (2013, sample B) 2 3 0.31 [0.20, 0.42]
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Rice et al. (1995) 1 3 0.14 [−0.02, 0.30]
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Schmidt et al. (1996, sample B) 1 2 0.10 [−0.22, 0.43]
Schmidt et al. (1996, sample B) 1 3 0.15 [−0.18, 0.48]
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Seiffge−Krenke et al. (2003) 1 2 0.20 [0.01, 0.39]
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Song et al. (2015) 1 2 0.11 [0.09, 0.13]
Starks et al. (2015) 1 2 0.28 [0.15, 0.42]

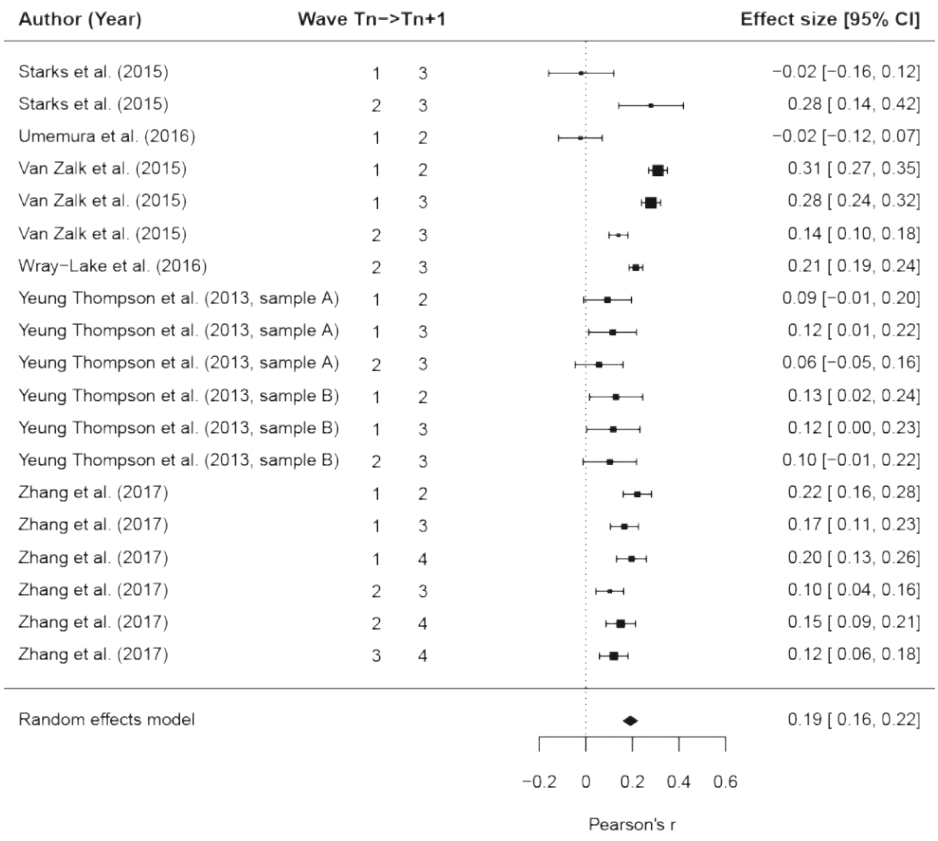

Figure S2. Three-level random effects model for the associations between supportive parent-adolescent relationships and supportive peer relationships. Wave Tn = wave at parent-adolescent relationship; Tn+1 = wave at peer relationship; squared boxes depict weighted effect sizes with larger sample sizes receiving larger weights (indicated by the size of the box); whiskers depict confidence intervals.

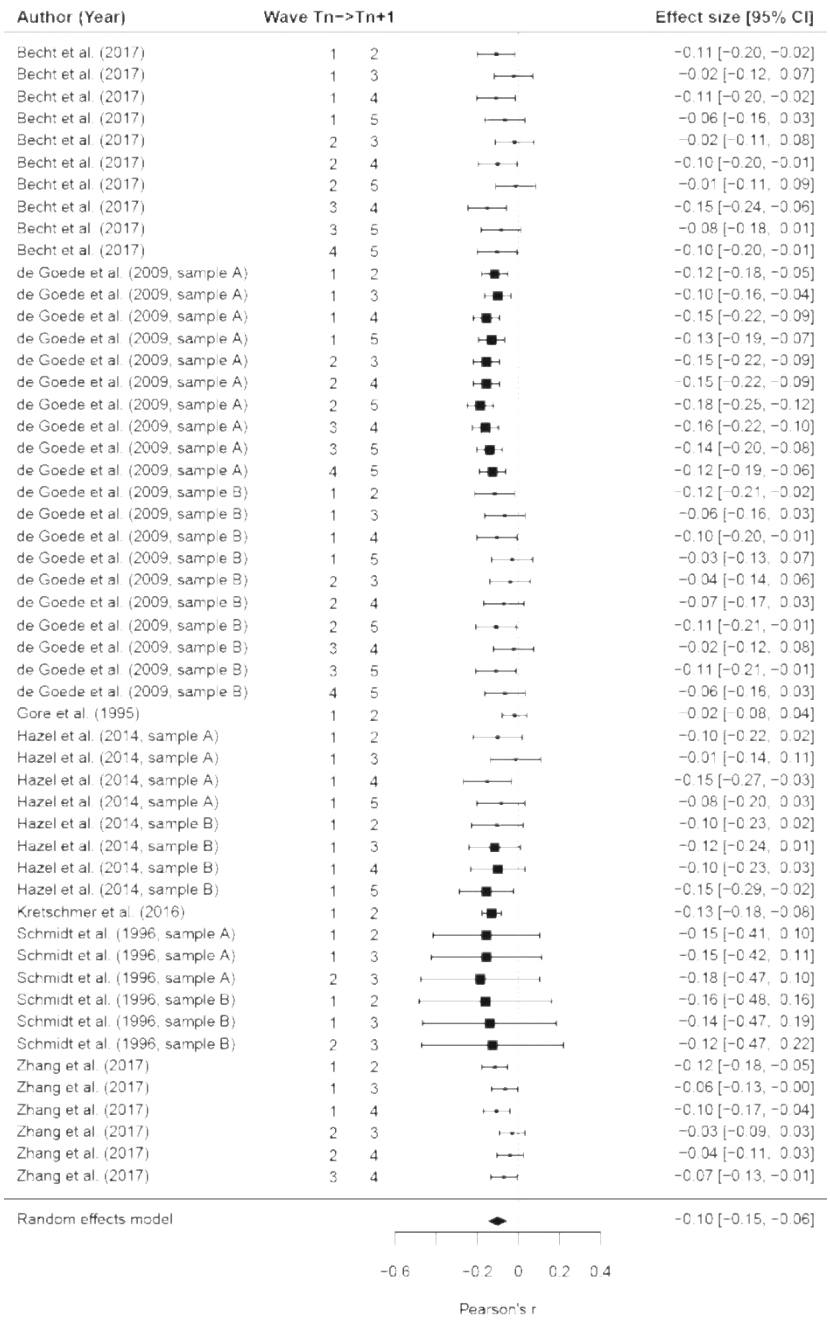

Figure S3. Three-level random effects model for the associations between supportive parent-adolescent relationships and negative peer relationships. Wave Tn = wave at parent-adolescent relationship; Tn+1 = wave at peer relationship; squared boxes depict weighted effect sizes with larger sample sizes receiving larger weights (indicated by the size of the box); whiskers depict confidence intervals.

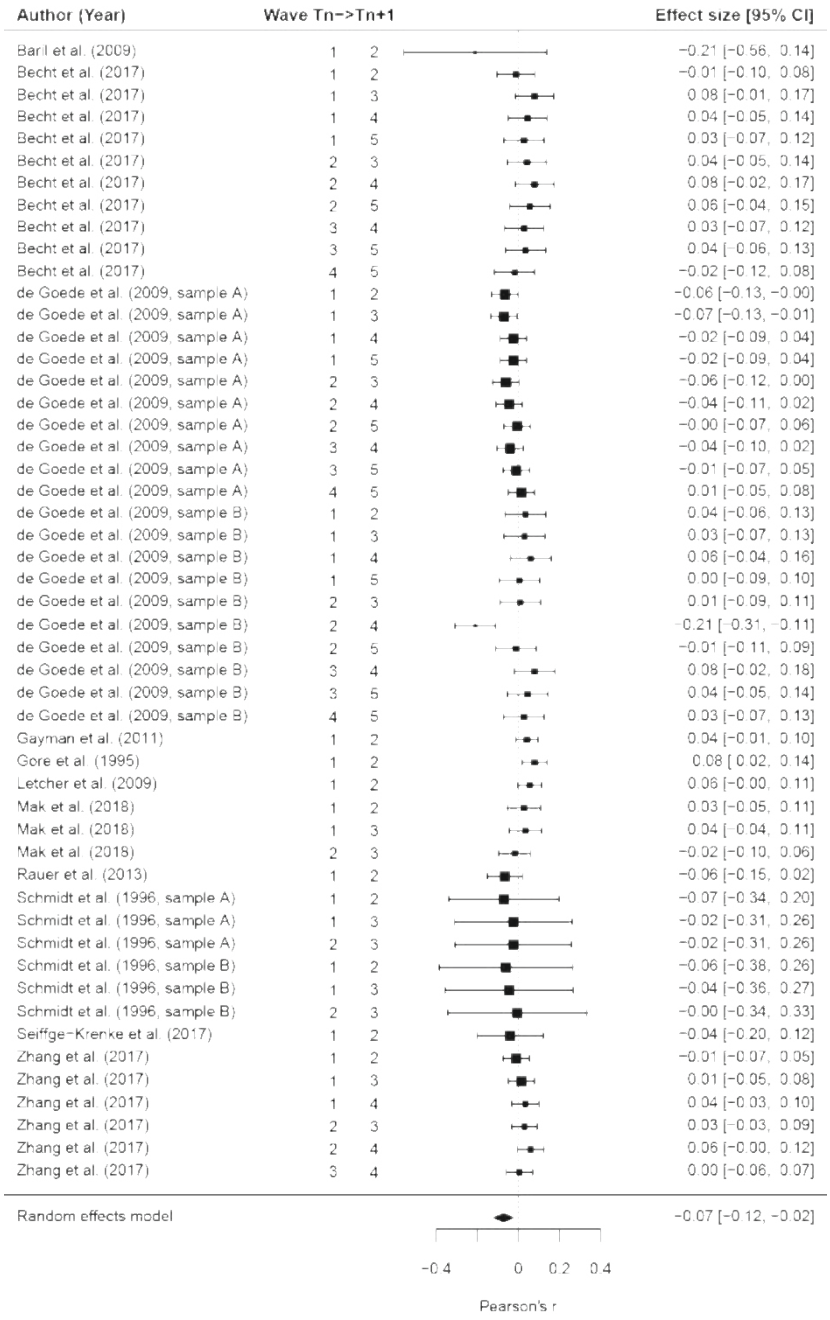

Figure S4. Three-level random effects model for the associations between negative parent-adolescent relationships and supportive peer relationships. Wave Tn = wave at parent-adolescent relationship; Tn+1 = wave at peer relationship; squared boxes depict weighted effect sizes with larger sample sizes receiving larger weights (indicated by the size of the box); whiskers depict confidence intervals.

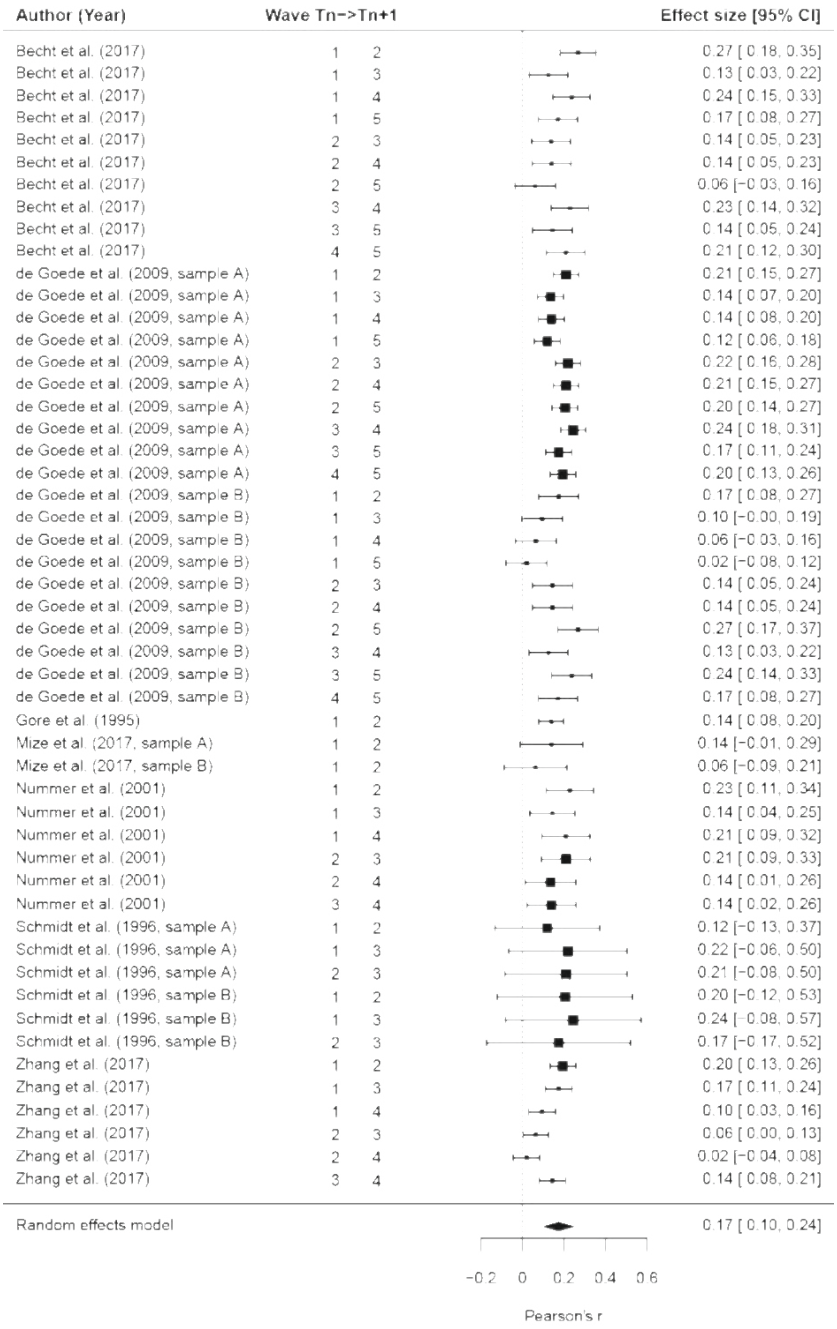

Figure S5. Three-level random effects model for the associations between negative parent-adolescent relationships and negative peer relationships. Wave Tn = wave at parent-adolescent relationship; Tn+1 = wave at peer relationship; squared boxes depict weighted effect sizes with larger sample sizes receiving larger weights (indicated by the size of the box); whiskers depict confidence intervals.

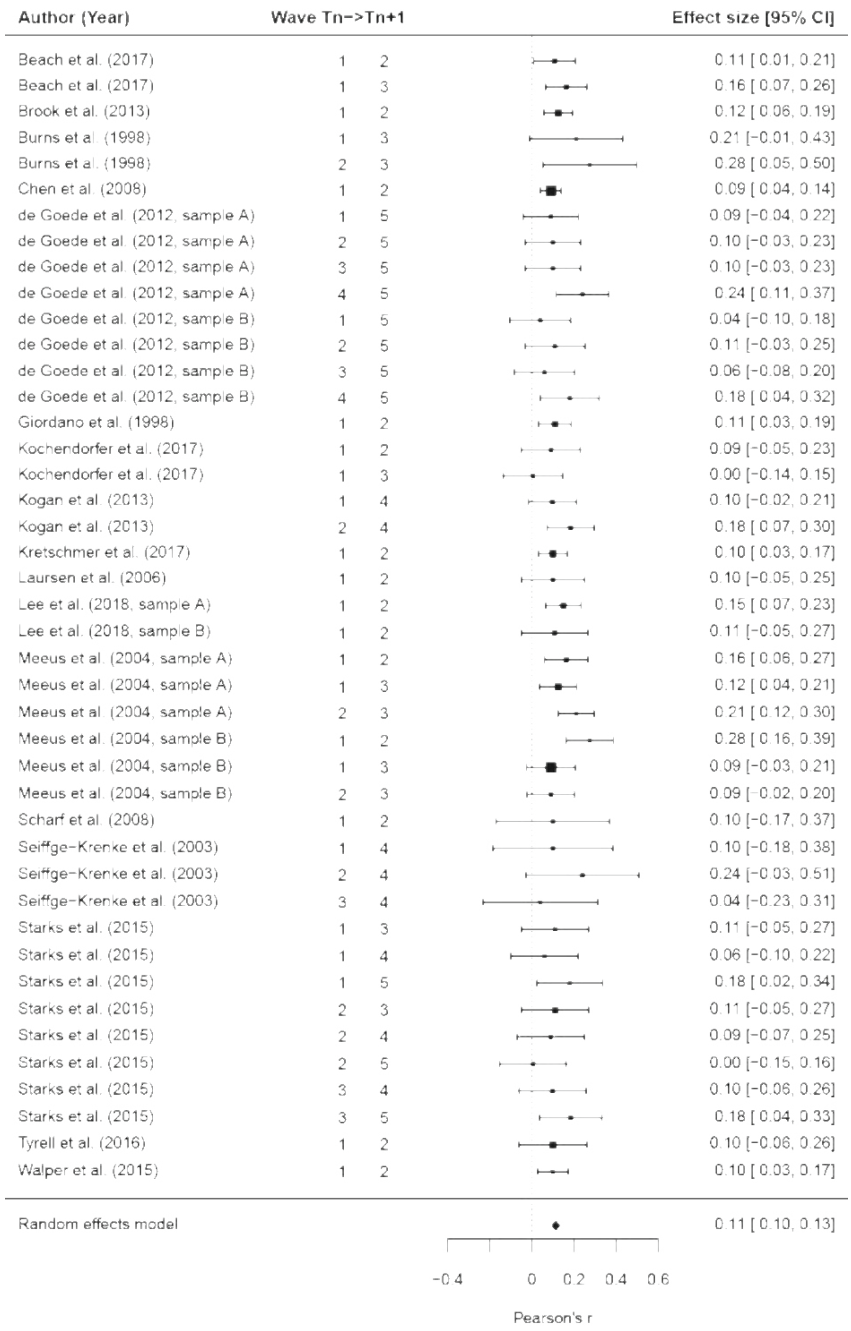

Figure S6. Three-level random effects model for the associations between supportive parent-adolescent relationships and supportive romantic relationships. Wave Tn = wave at parent-adolescent relationship; Tn+1 = wave at romantic relationship; squared boxes depict weighted effect sizes with larger sample sizes receiving larger weights (indicated by the size of the box); whiskers depict confidence intervals.

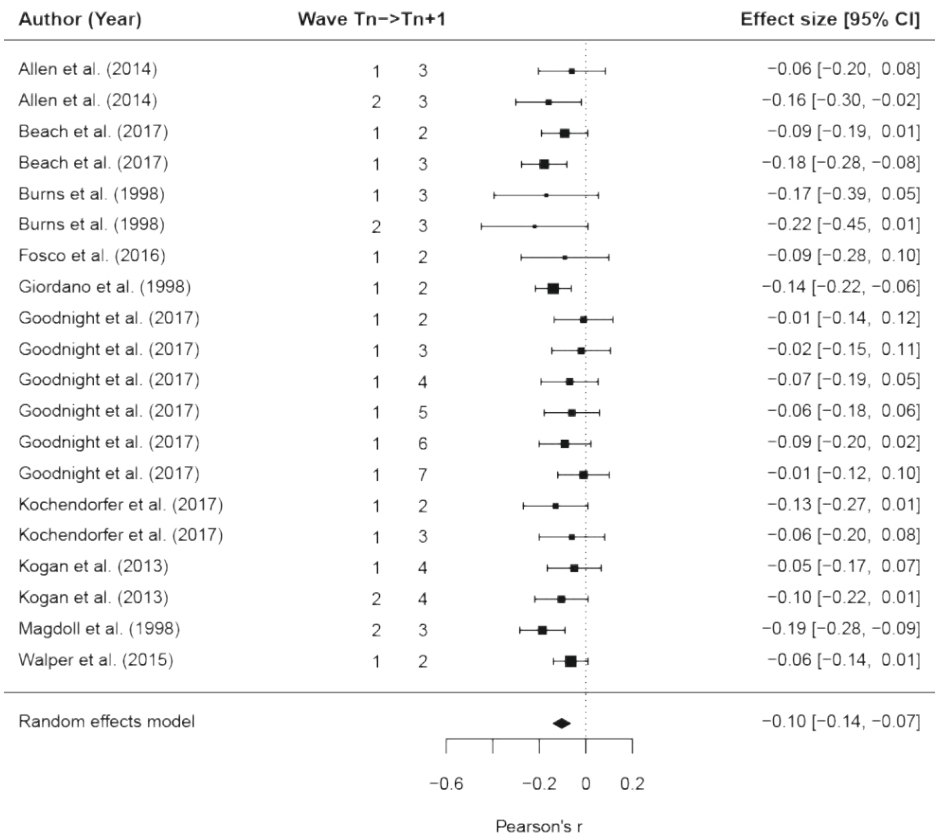

Figure S7. Three-level random effects model for the associations between supportive parent-adolescent relationships and negative romantic relationships. Wave Tn = wave at parent-adolescent relationship; Tn+1 = wave at romantic relationship; squared boxes depict weighted effect sizes with larger sample sizes receiving larger weights (indicated by the size of the box); whiskers depict confidence intervals.

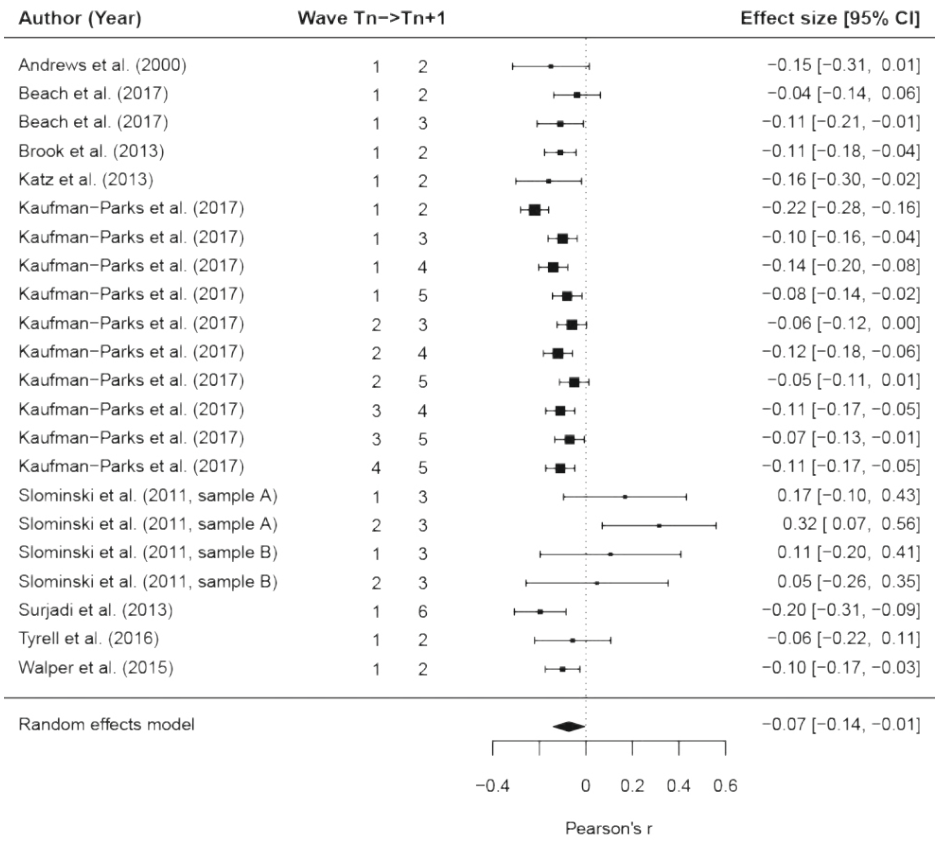

Figure S8. Three-level random effects model for the associations between negative parent-adolescent relationships and supportive romantic relationships. Wave Tn = wave at parent-adolescent relationship; Tn+1 = wave at romantic relationship; squared boxes depict weighted effect sizes with larger sample sizes receiving larger weights (indicated by the size of the box); whiskers depict confidence intervals.

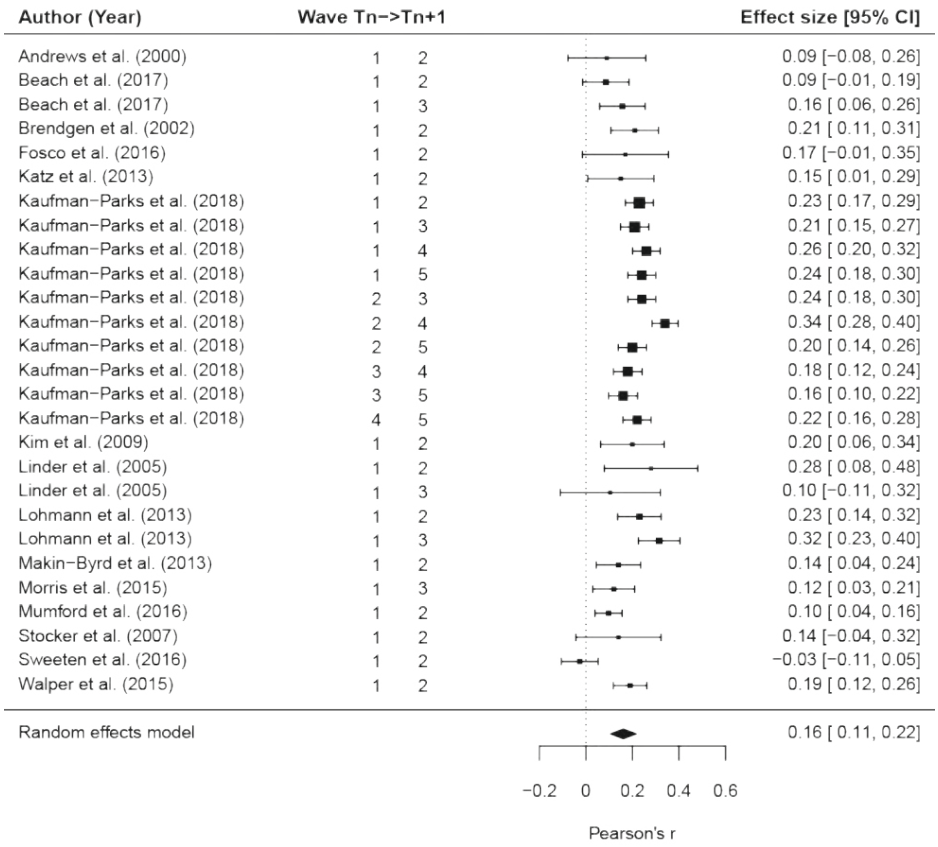

Figure S9. Three-level random effects model for the associations between negative parent-adolescent relationships and negative romantic relationships. Wave Tn = wave at parent-adolescent relationship; Tn+1 = wave at romantic relationship; squared boxes depict weighted effect sizes with larger sample sizes receiving larger weights (indicated by the size of the box); whiskers depict confidence intervals.

CHAPTER 6

General Discussion

Is it true that children turn into their parents? Questions regarding intergenerational transmission are particularly relevant during adolescence, when major transitions occur that set individuals on course to explore and shape who they are and who they will be. How are symptoms and behaviors between parents and adolescents transmitted during these times? And what happens during adolescence that can explain possible intergenerational continuity or discontinuity, and under which circumstances? These are questions that the present dissertation aimed to answer. Specifically, four studies were conducted that addressed three main overarching aims on how intergenerational transmission of psychopathology and relationships occurs across adolescence and young adulthood: 1) to investigate how psychopathological symptoms and relationship behaviors are transmitted across adolescence and whether there are changes in parental influences across time, 2) to shed light on the direction of transmission effects between parents and adolescents, and 3) to provide insights into potential mechanisms involved in intergenerational transmission processes.

The findings of each chapter in this dissertation are summarized in Table 1. Using a multi-method design that included observational, (intensive) longitudinal, and meta-analytical data from early adolescence to young adulthood as well as state-of-the-art statistical analyses, the findings of this dissertation provide novel, comprehensive insights into the study of intergenerational transmission. Specifically, within families parental and child psychopathological symptoms were consistently associated, but only adolescent psychopathological symptoms predicted changes in maternal psychopathological symptoms across adolescence, and not vice versa (Chapter 2). Intensity and temporal fluctuations of maternal daily affect further predicted adolescent and young adult psychopathological symptoms, and these associations were partly moderated by the congruency between maternal and adolescent affect (Chapter 3). Mother-adolescent interaction behavior explained how maternal internalizing symptoms were transmitted to adolescent internalizing symptoms, but not how adolescent symptoms predicted maternal symptoms (Chapter 4). Finally, interaction behaviors between parents and adolescents not only continued into adolescents' later relationships with peers and romantic partners, but were also predicted by peer relationships (Chapter 5).

The findings of this dissertation provide important implications for understanding the processes that are involved in intergenerational transmission. At the same time, they also raise questions and considerations about the nature of intergenerational transmission processes. The next section discusses how the findings of the four chapters relate to the dissertation's main aims and how they might shape and advance the field of intergenerational transmission.

Theoretical Implications

Intergenerational transmission across time: Associations in adolescence are important

The findings of this dissertation indicate that adolescence is an important period during which parents and adolescents not only exhibit somewhat similar symptoms and behaviors (i.e., intergenerational similarity), but also during which parents shape adolescents' development of psychopathology and relationships (i.e., parent effects). Throughout all four chapters, parents' and children's characteristics and behaviors were consistently associated. Specifically, moderately strong concurrent associations were found between parental and adolescent internalizing symptoms (Chapter 2), between maternal and adolescent daily affect (Chapter 3), and between parent-adolescent relationship behaviors and similar peer relationship behaviors (Chapter 5) across adolescence and to some extent even young adulthood.

Consistent with life course theories (Elder, 1998), the lives of parents and adolescents are linked, not only through shared genes and living in the same environments, but also through continuous reciprocal interactions. Because of cumulated negative reciprocal influences in a shared environment, it is possible that some families are characterized by generally high levels of problems. As children's emotional and social repertoire expands and new demands arise during adolescence, similarities with their parents might become more evident as well. Challenges in adolescents' emotional and social development, such as regulating more variable, intense emotions and the emergence of psychopathological symptoms or increased negative interaction and less warmth in relationships with parents (Branje, 2018; Hadiwijaya et al., 2017), can also make adolescents particularly vulnerable and thus susceptible to parental behaviors and changes therein. While the findings suggest that there is substantial intergenerational similarity between parental and adolescent psychopathological symptoms and relationship behaviors, concurrent associations do not provide direct evidence for intergenerational transmission. They indicate that transmission might occur, but other factors could explain these similarities as well. The studies in this dissertation therefore assessed parental and adolescent symptoms and behaviors not only concurrently, but also across time, which allowed for capturing how transmission unfolds from one generation to the next. The results revealed small but consistent longitudinal transmission with regard to psychopathological symptoms (Chapter 3, 4) as well as relationship behaviors from parents to adolescents (Chapter 5). Interestingly, however, longitudinal effects were not detected for the year-to-year transmission of psychopathological symptoms from parents to their children *within* families (Chapter 2). One reason may be that within-family processes in adolescence are driven by evocative processes from adolescents to parents (i.e., adolescent effects), which will be discussed in the next section on the direction of transmission effects. Alternatively, it is possible that within-family effects are less likely to be observed from one

year to the next, but concern individual behaviors within dyads that unfold at a shorter time frame. From a within-family perspective, it is assumed that how symptoms are transmitted is a process that occurs in momentary interactions within the family: Fluctuations in parental symptoms, for example, directly impact parenting behaviors and in that way influence adolescents' symptoms (Goodman et al., 2020).

The findings of this dissertation indicate that transmission processes are evident from early to late adolescence (Chapter 2, 5) and even into young adulthood (Chapter 3). Rather than transient influences, the effects of parental symptoms and behaviors are enduring across adolescence (Fraley & Roisman, 2015), indicating that parents remain important sources of influence. Some further evidence for age effects during adolescence suggests the existence of potential sensitive periods that are characterized by heightened sensitivity to the feelings and behaviors of others (Chapter 3, 5), even though the findings of this dissertation did not reveal a consistent pattern. For example, the effects of parent-child relationship behaviors on peer relationships increased as adolescents got older (Chapter 5), while the effects of maternal affect on children's psychopathological symptoms were generally smaller in young adulthood compared to adolescence or the transition to young adulthood (Chapter 3). Although evidence regarding potential age effects remained inconclusive, it is possible that there are sensitive periods even within adolescence in which not only risk for behavioral continuity but also for discontinuity is particularly increased. At these times, adolescents may not only be most sensitive to negative parental effects, but also to positive effects of interventions. Such potential sensitive periods may differ for specific characteristics under examination. Future research should look more into age effects to determine when parental influence and transmission effects are strongest for specific symptoms and behaviors.

Disentangling directions of effects: Intergenerational transmission processes are transactional

Just as symptoms and behaviors are passed on from parents to their children (i.e., parent effects), changes in adolescence prompt evocative processes. Although reversed effects from adolescents to parents (i.e., adolescent effects) usually do not constitute intergenerational transmission in the traditional sense, many studies that reflect on theoretical concerns in transmission research now emphasize their importance (Branje et al., 2020; Thornberry, 2016). Chapters 2, 4, and 5 provided evidence for adolescent effects, suggesting that adolescents' characteristics and behaviors elicit change in parents' behaviors. Specifically, adolescent effects were found consistently in that less positive and more negative relationships with peers predicted more negative future relationships with parents compared to other families (Chapter 5) as well as adolescent internalizing symptoms predicted increases in maternal symptoms within families (Chapter 2) and less positive maternal interactions behaviors compared to other families (Chapter 4). These findings suggest that reversed

effects from adolescents to parents play a crucial role in the transmission of psychopathology and relationship quality. Adolescents' emotional or behavioral problems can evoke negative feelings, cognitions, and behaviors in parents (e.g., Berg-Nielsen et al., 2002; Patterson, 1982; Serbin et al., 2015), which in turn drive parents' vulnerability to similar problems. While parents have been found to guide children's emotion regulation by driving their negative behaviors towards more positivity (e.g., Van Bommel, Van der Giessen, Van der Graaff, Meeus, & Branje, 2019), children's repeated or accumulated negative feelings or behaviors might make it more difficult for parents to remain positive.

The findings of this dissertation suggest that the intergenerational transmission of psychopathology and relationship quality cannot simply be characterized as unidirectional transfer, but rather describes a complex process that arises through continuous parent-child transactions. Both parent (Chapter 3, 4, 5) and adolescent (Chapter 2, 4, 5) effects were found to contribute to transmission processes, which suggests that these processes are transactional in nature. In line with that, Chapter 2 and 5 also detected bidirectional associations, suggesting that parents and adolescents may reinforce each other's characteristics or problems over time. Such reciprocal associations are also involved in important mechanisms that shape intergenerational transmission in the long run, such as parenting or parent-child interactions.

Explaining intergenerational transmission processes: Parent-adolescent dynamics matter

To further understand why and how parental psychopathological symptoms are related to adolescent symptoms, the third aim of this dissertation focused on examining parent-adolescent dynamics as mechanisms through which transmission processes occur. The findings revealed that maternal affect, as part of the emotional climate in the family, did not only predict children's future adjustment (Chapter 3), but that there was also some evidence that mother-adolescent interactions mediated relative changes in the associations between maternal and adolescent psychopathological symptoms (Chapter 4). Maternal negative characteristics or behaviors, such as psychopathological symptoms, render mothers more emotionally unavailable and less sensitive to their children's needs, which influences how they feel and how they interact with their children (e.g., Goodman et al., 2020; Morris et al., 2007). These feelings and interactions can in turn elicit negative feelings in their children, increasing their risk for psychopathological problems.

Albeit consistent, transmission effects were generally small throughout adolescence and young adulthood. This is in line with previous studies on intergenerational transmission of psychopathological symptoms and relationship behaviors (e.g., Connell & Goodman, 2002; Pallini, Baiocco, Schneider, Madigan, & Atkinson, 2014; Schneider et al., 2001) and suggests that transmission

might not occur in all families. Besides examining mediating factors, this dissertation also aimed to examine parent-adolescent dynamics as moderating factor to explain why problem behaviors are maintained and accumulated in some families, but not others. In Chapter 3, congruency between maternal and adolescent affect signaled resilience against maternal maladaptive affect, indicating that mother-adolescent dynamics did not only explain why certain characteristics were transmitted, but also why they were *not* transmitted in some families. While it may seem counterintuitive that affect congruency explains non-transmission, the functions of short-term congruency and long-term transmission are likely to differ. Similar to interaction patterns, parent-adolescent congruency of positive *and* negative affect might reflect mutual understanding or positive relationship quality between parents and their children, indicating that positive mother-child dynamics could be particularly relevant in disrupting the transmission of negative feelings or behaviors.

The extent to which positive parent-child dynamics were involved in intergenerational transmission differed from negative parent-child dynamics. In Chapter 4, evidence for mediation from maternal to adolescent internalizing symptoms was only found for negative, but not positive, interaction behaviors from mothers towards adolescents. Exposure to an adverse emotional climate in the family seems to play an important role in explaining intergenerational transmission of psychopathology. That is not surprising given that psychopathology strongly revolves around negative affect and the effects of negative experiences or feelings tend to be stronger than positive ones (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001). On the other hand, in Chapter 3, maternal positive, but not negative, affect continued to influence children's adjustment into young adulthood. While negative parent-child dynamics have immediate adverse effects, the effects of a positive family climate may be more long-lasting. Negative behaviors, such as harsh discipline or control assertion, are often considered reactive parenting behavior that occurs as immediate responses to children's behaviors (e.g., Padilla-Walker & Son, 2019). Positive parenting behaviors, such as support, praise, or monitoring, on the other hand are considered proactive in that they focus on long-term change and are motivated by long-term goals. Staying positive and mutually understanding in the face of adversity might reflect over-time resilience in families. The effects for positive and negative mother-child dynamics in Chapters 3 and 4 might also differ because momentary affect and behaviors are bound to situational circumstances. Reciprocating negative affect or behaviors between parents and children might be warranted in some situations, but have negative effects in others. To better understand the role of parent-child dynamics, future research should therefore take the context into account when examining momentary dynamics between parents and children.

The involvement of parent-child dynamics in the intergenerational transmission of psychopathology indicates that short-term processes are related to long-term processes. In Chapter 3, accumulated

daily affect was associated with adolescent long-term adjustment, while in Chapter 4 momentary mother-adolescent interaction behavior explained the transmission of relatively stable symptoms. Mechanisms of intergenerational transmission processes, such as dynamics between parents and their children, unfold in the moment and change quickly. It is therefore best to assess them at the short term in daily (Chapter 3) or momentary (Chapter 4) measurements. Associations between parents' and adolescents' behaviors that occur consistently over a relatively short time can accumulate and shape long-term development (Granic & Patterson, 2006; Lougheed & Keskin, 2021). Specifically, patterns emerge in these dynamic parent-child interactions that become relatively stable internal working models or schemas that drive long-term intergenerational transmission processes. Although the detected indirect and moderation effects remained mostly small and explained continuity or discontinuity of intergenerational associations only to some extent, small effects can thus be still meaningful in shaping later trajectories (Bornstein, 2017). However, there may be processes involved in intergenerational transmission that unfold at different time scales than those investigated in the present studies. For example, parental anger or stress could have direct effects in the moment or exert its effects on the adolescent only after one or several hours, whereas the effects of parental depressive mood or controlling parenting behaviors would only show across several days, weeks, or even months. While theories on parent-child dynamics generally do not specify the time scales at which effects are expected to unfold, future research will need to examine processes between parents and children at different time scales. This will not only contribute to our understanding about which mechanisms at which times are most relevant in explaining intergenerational transmission, but also clarify how processes at different time scales relate to each other – with regard to both how parent-adolescent dynamics shape long-term transmission and how long-term transmission predicts parent-adolescent dynamics.

Methodological Considerations

This dissertation used a variety of different methodological and statistical designs to provide novel and nuanced insights into intergenerational transmission between parents and adolescents. Each of the four studies shed light on different aspects that together draw a more comprehensive picture of the intergenerational transmission of psychopathology and relationships.

The mechanisms that explain intergenerational transmission between parents and their children describe reciprocal processes that occur within families over time. At the micro-level, we are therefore particularly interested in what happens within a family, whereas at the macro-level, we might also be interested in predicting relative change and how families differ. Both approaches have important implications, but resulted in slightly different conclusions. Chapters 3, 4, and 5 found that parental symptoms and behaviors predicted relative change in adolescents' subsequent symptoms and

behaviors, while Chapter 2 indicated that changes in adolescent symptoms predicted changes in their parents' symptoms within families, but not vice versa. Although changes in parental symptoms compared to their own average did not predict changes in adolescents' symptoms, changes in parental symptoms compared to other parents still predicted how adolescents scored compared to their peers across time. Similarly, just because individuals change does not mean they change in comparison to their peers (i.e., differences across families remain). The reasons for between-family differences are likely to differ from the reasons for within-family changes, and might reflect a shared family environment, such as socio-economic status or neighborhood factors. Together the findings provide a comprehensive picture about the role of relative change in intergenerational transmission (Chapter 4, 5) and their mechanisms, which concern the processes and individual behaviors within dyads, such as parent-child dynamics (Chapter 3, 4). How families differ across time might be largely determined by relative changes transmitted from parents to adolescents, while transmission processes that occur within families may be driven by evocative processes.

To disentangle changes within families from changes between families, Chapter 2 employed random-intercept cross-lagged panel models (RI-CLPMs). Besides within-family associations, the findings from these models also showed that parental and adolescent psychopathological symptoms were associated between families, but they did not provide information about how families change over time. In RI-CLPMs, between-family associations are thought to be trait-like, therefore remaining relatively stable and less likely to change over time. Because change across families is meaningful and can differ from associations within families, Chapters 4 and 5 used cross-lagged panel models (CLPMs) to quantify and build on previous findings from between-family rank-order associations. These models provided important information about relative changes in transmission processes and how adolescents change compared to their peers, but could not disentangle within-family associations from between-family associations. Additionally, CLPMs cannot adjust for trait-like stability. Besides the limitations of CLPMs, the assumptions of RI-CLPMs that differences between families are perfectly stable are equally unrealistic, but alternative models, such as Autoregressive Latent Trajectory (ALT) models or Latent Curve Models with Structured Residuals (LCM-SR; Curran, Howard, Bainter, Lane, & McGinley, 2014) are highly complex and often result in model estimation errors and nonconvergence (Orth, Clark, Donnellan, & Robins, 2021). To reliably model how parents and children affect each other within families as well as how families differ from each other across time, better solutions are needed. Before we therefore start to dismiss previous findings from traditional models, we need to carefully reflect on the questions we aim to answer and the information each model can provide.

In Chapter 4 and 5, meta-analytical techniques were used to quantify empirical knowledge about intergenerational transmission processes. Such quantified associations are particularly important as they provide robust, generalizable evidence about whether parental and adolescent symptoms and interaction behaviors are associated. Novel techniques, such as meta-analytical structural equation modelling (MASEM; Chapter 5), allowed for examination of complex bidirectional associations between adolescent relationships with parents and peers. Chapter 4 moved one step further and not only synthesized existing information from previous studies, but also used these accumulated empirical findings to inform new analyses. Specifically, Bayesian estimation with informative priors suggests evidence for associations between parental and adolescent internalizing symptoms through mother-adolescent interaction behaviors. Compared to a pure meta-analytical approach, this cumulative approach did not only identify gaps and limitations in research, but immediately addressed and overcame previous methodological shortcomings, such as predominantly cross-sectional or unidirectional designs, with new data. Combining accumulated knowledge from several decades of intergenerational transmission research with new longitudinal data allowed for updating our knowledge on these transmission processes, and thus generating cumulative knowledge. Given the flexibility to weight and adjust the influence of previous information and update this information with new data, Bayesian analyses are able to manage highly heterogenous studies with differing methods or conclusions and even flawed designs of previous studies that are considered main weaknesses of conventional meta-analyses.

That said, meta-analytical techniques are time-consuming and still risk missing some studies, even when attempting to include unpublished materials. Moreover, Bayesian estimation techniques can be quite complex and the numerous decisions required with regard to study inclusion, weighting, or prior techniques can make them seem too challenging or potentially arbitrary. Despite these challenges, the finding that different decisions, such as the use of different priors, often resulted in similar conclusions emphasized the strengths of the detected associations in this dissertation. Some differing findings also enable the opportunity for dialogue, as they can provide important avenues for future research. Even more so than conventional models, Bayesian techniques require researchers to think critically about previous as well as their own findings, which fits the current call for increased rigor and transparency in empirical science. Moving towards such a cumulative, but laborious approach can be facilitated by collaborative science and investment in one study that can provide more insights than several small ones.

Implications for Clinical Practice

It is not new that parental emotional and behavioral difficulties affect children's adjustment – nor that children's problems influence their parents. Several interventions have been developed that aim

to reduce children's problem behaviors by targeting family systems (e.g., Family Systems Therapy; Bowen, 1978, Multidimensional family therapy; Liddle, 2002) or enhancing parent-child relationship quality (e.g., Tuning into Kids; Havighurst et al., 2013). Despite evidence of the effectiveness of such programs, long-term effects remain unclear and the effect sizes are often small, indicating that not all families benefit (Weisz & Kazdin, 2017). The results of this dissertation provide important implications for current practice.

First, the findings of this dissertation suggest that interventions to reduce psychopathological symptoms or negative behaviors in adolescence should not only focus on children, but also on parents. While parenting interventions are prominent in childhood, they are less common in adolescence. However, parents remain important sources of influence throughout adolescence. They play an important role in adolescents' development not only through directly transmitting symptoms and behaviors (Chapter 3, 4, 5), but also through evocative processes within families (Chapter 2) that might put adolescents at risk for maladjustment. Coercive cycles might arise in which negative characteristics and behaviors are reinforced in families that ultimately affect adolescents' adjustment. Such parent-child dynamics play an important role in maintaining or breaking intergenerational patterns. Through such negative cycles, symptoms or problem behaviors can accumulate and become rigid working models or behavioral patterns that render change in these families particularly difficult. Targeting these patterns successfully and in the long run might therefore be challenging. Adolescence might be an important period to modify relationships with parents and help parents to maintain positive interactions with their children. Changes in this period might make relatively rigid systems susceptible for change and influence. If relationships between parents and their children are in the process of changing, intervention efforts might be particularly successful.

Second, stability in parent-child patterns can be adaptive or maladaptive, depending on the context. While repeated, reinforced negative behaviors and symptoms between parents and their children can signal coercion or accumulated problems in these families, congruency in positive *and* negative affect might increase mutual understanding between parents and adolescents. Enhancing positive relationships and understanding in parent-adolescent dyads might be an important avenue that helps to break potential negative cycles. Importantly, interventions need to target long-term change, not just change in the moment. Only if consistent change in parents' as well as adolescents' affect and behaviors can be achieved, working models can be modified and evoke long-term effects. It may therefore be relevant for programs to provide repeated booster sessions and track improvement in interactions across several years. This should include assessments at different time

scales to understand whether parents and adolescents remain consistent in their change and if not, when they need additional help before they relapse into old patterns.

Finally, individual differences need to be taken into account. What works for some families might not work for others (Janssen, Elzinga, Verkuil, Hillegers, & Keijsers, 2021; Keijsers & van Roekel, 2019). It is therefore relevant to identify heterogeneity in transmission processes and assess a family's individual risk profile to determine the care that they might need. Such tailored interventions can consider family characteristics and context to effect long-term change in parent-child dynamics. Recent developments in parenting intervention research that focus on identifying effective components (e.g., Leijten, Melendez-Torres, & Gardner, 2022) can provide important insights into personalizing family interventions.

Concluding Remarks

Adolescence is an important period in which intergenerational transmission processes play a role. Parental and adolescent psychopathological symptoms and relationship behaviors are generally consistently associated across adolescence and to some extent even into young adulthood, with both parent and adolescent effects involved. Although short-term transmission might reflect different processes than long-term transmission at equivalent age periods, they provide important insights into how parents and adolescents become similar across time. Parent-child dynamics can explain these short-term associations across adolescence. But not all families are the same and parental effects do not affect all equally. While negative parent-child interactions might explain why problem behaviors accumulate in some families, congruency between parents and children can help to break maladaptive patterns. Do children become their parents? The findings of this dissertation do not indicate that they do, but suggest that consistent parent-child transactions increase the chances of sharing similar characteristics and behaviors at least for some families.

Table 1. Summary of Main Findings.

Chapter	Main findings
2	***Bidirectional associations between parental and adolescent psychopathology*** • Between families, maternal, but not paternal, internalizing and externalizing symptoms were associated with adolescent internalizing and externalizing symptoms. • Within families, more maternal and paternal internalizing, but not externalizing, symptoms were associated with more adolescent internalizing, but not externalizing, symptoms in the same year. • From early to late adolescence, more adolescent internalizing, but not externalizing, symptoms in the previous year predicted more maternal internalizing, but not externalizing, symptoms in the following year. • For adolescent boys, these findings were unidirectional from boys' to mothers' symptoms, whereas for adolescent girls these associations were bidirectional. • Paternal psychopathological symptoms and adolescent psychopathological symptoms did not show longitudinal within-family associations.
3	***The moderating role of parent-adolescent dynamics*** • Less maternal positive affect intensity and more inertia predicted higher adolescent and young adult psychopathological symptoms, while more maternal negative affect intensity and inertia predicted higher psychopathological symptoms only in adolescence. • High mother-adolescent congruency of both positive and negative affect buffered the effects of higher maternal affect inertia on higher adolescent symptoms.
4	***The mediating role of parent-adolescent dynamics*** • Higher maternal internalizing symptoms in early adolescence predicted less positive and more negative mother-adolescent interaction behavior one year later, while higher adolescent internalizing symptoms only predicted lower positive mother-adolescent interaction behavior. • More negative mother-adolescent interaction behavior in turn predicted higher maternal and adolescent internalizing symptoms one year later. • Negative mother-adolescent interaction behavior mediated the associations between maternal and adolescent internalizing symptoms.
5	***Bidirectional associations between parent-adolescent and social relationships*** • Less supportive and more negative parent-adolescent relationships predicted less positive and more negative future peer and romantic relationships. • More negative parent-adolescent relationships were predicted by less positive and more negative previous peer relationships. • The associations between parent-adolescent relationships and future peer relationships became stronger as adolescents got older.

APPENDICES

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NEDERLANDSE SAMENVATTING (SUMMARY IN DUTCH)

De adolescentie is een van de belangrijkste ontwikkelingsperioden, gekenmerkt door contextuele, biologische en persoonlijke veranderingen. Deze veranderingen gaan gepaard met nieuwe ontwikkelingstaken voor jongeren, zowel in het affectieve als sociale domein. Zo ervaren adolescenten meer en extremere emoties dan in hun kinderjaren, die ze moeten leren reguleren om flexibel te kunnen reageren op de veranderingen in hun omgeving. Tegelijkertijd worden meer sociale competenties en zelfstandigheid verwacht, die veranderingen in bestaande en nieuwe sociale relaties stimuleren. Adolescenten worden zo uitgedaagd om gezonde relaties te vormen waarbij autonomie en verbondenheid in balans zijn. Enerzijds bieden deze ontwikkelingstaken kansen voor een positieve ontwikkeling. Anderzijds maken ze adolescenten ook kwetsbaar voor internaliserende problemen (bijv. depressie, angst), externaliserende problemen (bijv. agressie, delinquentie) en relatieproblemen (bijv. met ouders of vrienden), die nadelige gevolgen kunnen hebben tot in de volwassenheid.

Ouders spelen een belangrijke rol in de psychosociale ontwikkeling van jongeren. Ze helpen adolescenten hun emoties te reguleren en kunnen positieve sociale interacties stimuleren. Ouders kunnen echter ook een van de belangrijkste risicofactoren voor aanpassingsproblemen van adolescenten zijn. Met name psychische klachten en relatiepatronen worden vaak overgedragen van ouders. Een dergelijke intergenerationele overdracht vindt plaats via genetische en omgevingsfactoren. Ouders beïnvloeden niet alleen het gedrag van jongeren, jongeren hebben ook invloed op het gedrag van hun ouders. Er zijn echter nog weinig empirische studies naar mogelijke bidirectionele verbanden tussen psychische klachten en relaties van ouders en adolescenten over tijd, hoe deze verbanden verklaard kunnen worden en of dit voor alle gezinnen geldt of sterker voor bepaalde gezinnen dan voor andere.

In dit proefschrift is de intergenerationele overdracht van psychische klachten en relatiekwaliteit in de adolescentie en jongvolwassenheid onderzocht. Door middel van vier longitudinale, multi-methodische studies is geprobeerd om meer inzicht te verkrijgen in deze intergenerationele overdracht. De dissertatie had drie overkoepelende doelen: 1) onderzoeken hoe psychische klachten en relatiekwaliteit van ouders en adolescenten aan elkaar zijn gerelateerd tijdens de adolescentie en of de invloed van ouders door de tijd heen verandert, 2) in kaart brengen of overdrachtseffecten voornamelijk van ouders op adolescenten verlopen of ook van adolescenten op ouders en 3) inzicht krijgen in mogelijke mechanismen die de processen van intergenerationele overdracht kunnen verklaren en welke gezinnen meer of minder sterke verbanden laten zien.

Intergenerationele overdracht over tijd: De adolescentiefase is van belang

Uit de resultaten van dit proefschrift bleek dat de adolescentie een belangrijke levensfase is waarin ouders en adolescenten niet alleen gerelateerde psychische klachten en relationeel gedrag vertonen, maar waarin ouders ook de ontwikkeling van hun jongeren vormgeven. In alle hoofdstukken konden verbanden tussen symptomen en gedrag van ouders en adolescenten worden aangetoond – niet alleen op hetzelfde moment in de tijd (cross-sectioneel) maar ook over de tijd (longitudinaal), wat meer inzicht geeft in de overdracht van een generatie op de volgende. Er kwamen duidelijke overdrachtsprocessen naar voren van de vroege tot de late adolescentie en zelfs tot in de jonge volwassenheid voor zowel psychische klachten als relatiekwaliteit, wat suggereert dat ouders gedurende de gehele adolescentiefase van belang blijven voor de ontwikkeling van jongeren.

Richting van intergenerationele overdracht: De effecten zijn bidirectioneel

De resultaten toonden verder aan dat niet alleen ouders hun jongeren beïnvloeden, maar dat adolescenten ook hun ouders beïnvloeden. Zo voorspelden minder positieve en meer negatieve relaties met vrienden een slechtere ouder-kindrelatiekwaliteit op een later moment in de tijd, en waren internaliserende symptomen bij adolescenten gerelateerd aan een toename van vooral internaliserende symptomen van moeders en minder positieve interacties tussen moeders en adolescenten. Deze resultaten benadrukken de cruciale rol van adolescenten zelf in de overdracht van psychische klachten en relatiekwaliteit. De wederzijdse verbanden suggereren bovendien dat ouders en adolescenten elkaars problemen over tijd mogelijk kunnen versterken. De intergenerationele overdracht van psychische klachten en relatiekwaliteit kan dus niet simpelweg worden gekarakteriseerd als een eenzijdig proces van de oudere naar de jongere generatie, maar lijkt eerder een complex bidirectioneel proces dat ontstaat en voortduurt door wederzijdse ouder- en kind-effecten.

Onderliggende mechanismen van intergenerationele overdracht: Ouder-kind dynamiek als verklarende factor

Het emotionele klimaat in het gezin, dat onder meer gekenmerkt wordt door de emoties van ouders en ouder-adolescentinteracties, speelt een cruciale rol in de intergenerationele overdracht van psychopathologie. Ondanks aanzienlijke verbeteringen in de afgelopen decennia, bestaan er nog steeds ongelijkheid tussen mannen en vrouwen en traditionele gezinsrollen. Vooral moeders staan dus nog steeds centraal bij het vormgeven van het emotionele klimaat in het gezin. De resultaten van dit proefschrift lieten zien dat de dagelijkse emoties van moeders het welzijn van adolescenten voorspelden en dat de overdracht van psychische klachten van moeders op adolescenten deels werd verklaard door - voornamelijk negatieve - moeder-kind interacties.

Hoewel er overwegend consistente resultaten werden gevonden, suggereren de relatief kleine effectgroottes dat psychische klachten en relatiekwaliteit niet in alle gezinnen worden overgedragen. De impact van negatieve dagelijkse emoties van moeders, vooral moeilijkheden hun emoties te reguleren, bleek minder sterk wanneer de emoties van moeder en adolescent meer op elkaar leken, wat suggereert dat emotionele afstemming en wederzijds begrip kunnen wijzen op veerkracht tegen een negatief emotioneel klimaat in het gezin. Dit indiceert dat de moeder-adolescent dynamiek enerzijds verklaart hoe intergenerationele overdracht tot stand komt (mediatie), maar laat anderzijds ook zien dat niet in alle gezinnen sprake is van intergenerationele overdracht (moderatie).

Implicaties voor de klinische praktijk

De resultaten van dit proefschrift bieden belangrijke implicaties voor de praktijk. Ten eerste suggereren ze dat ouders belangrijk zijn en blijven in de adolescentie en dat interventies die beogen om psychische klachten en negatief gedrag van jongeren te verminderen zich zowel op de adolescent als op de ouders moeten richten. Het versterken van positieve relaties en wederzijds begrip tussen ouders en jongeren kan een belangrijke stap zijn om negatieve patronen te doorbreken. Het is bovendien van belang dat interventies zich richten op veranderingen op de lange termijn en dat er rekening wordt gehouden met verschillen tussen gezinnen. Wat in sommige gezinnen werkt, werkt niet per se ook in andere gezinnen. Een individueel risicoprofiel van een gezin kan mogelijk helpen om te bepalen welke ondersteunende maatregelen nodig zijn om langdurige veranderingen in de ouder-kindrelatiedynamiek teweeg te brengen.

Conclusie

Samenvattend laten de resultaten van dit proefschrift zien dat intergenerationele overdracht van belang is tijdens de adolescentie. Psychische klachten en relatiekwaliteit van ouders zijn consequent gerelateerd aan symptomen en relaties van adolescenten, waarbij zowel het effect van ouders op adolescenten als van adolescenten op ouders van belang zijn. Dynamische processen tussen ouder en adolescent kunnen deze verbanden verklaren, maar niet alle gezinnen zijn gelijk en de mate van effecten verschilt per gezin. Negatieve ouder-kind interacties kunnen verklaren waarom probleemgedrag in sommige gezinnen opstapelt en relaties waarbij ouder en adolescent op elkaar afgestemd zijn kunnen helpen om negatieve patronen te doorbreken. Worden jongeren net zoals hun ouders? De bevindingen van dit proefschrift wijzen er niet op dat dit altijd het geval is, maar suggereren dat herhaalde ouder-kind interacties de kans op het delen van vergelijkbare psychische problemen en relationele gedragingen vergroten, in ieder geval in een deel van de gezinnen.

DEUTSCHE ZUSAMMENFASSUNG (SUMMARY IN GERMAN)

Die Adoleszenz ist eine der bedeutendsten Entwicklungsabschnitte, gekennzeichnet durch wichtige kontextuelle, biologische und persönliche Veränderungen. Diese Veränderungen gehen mit neuen Entwicklungsaufgaben für Jugendliche einher, sowohl emotional als auch sozial. So erfahren Jugendliche mehr und extremere Emotionen als in ihrer Kindheit. Diese müssen sie lernen zu regulieren, um flexibel auf die Anforderungen ihrer Umgebung reagieren zu können. Zur selben Zeit nehmen ihre soziale Kompetenz und Selbständigkeit zu, was Veränderungen in bestehenden und neuen sozialen Beziehungen stimuliert. Jugendliche werden so mit der Aufgabe konfrontiert, gesunde Beziehungen zu entwickeln, die ein ausgewogenes Verhältnis an Autonomie und Verbundenheit aufweisen. Obwohl eine positive Entwicklung aus diesen neuen Herausforderungen resultieren kann, machen diese Aufgaben Jugendliche auch anfällig für internalisierende Probleme (z.B. Depressionen, Angststörungen), externalisierende Probleme (z.B. Aggression, Kriminalität) und Beziehungsprobleme (z.B. mit Eltern oder FreundInnen), welche sich negativ auf ihre Gesundheit im Erwachsenenalter auswirken können.

Eltern spielen eine zentrale Rolle in der sozialen und emotionalen Entwicklung von Jugendlichen. Sie helfen Kindern, mit ihren Emotionen umzugehen und positive soziale Kontakte aufzubauen. Gleichzeitig sind Eltern jedoch auch ein Risikofaktor für Anpassungsprobleme ihrer Kinder. Vor allem psychische Probleme und Beziehungsmuster werden oft durch genetische und Umweltfaktoren von Generation zu Generation weitergegeben. Eltern beeinflussen dabei nicht nur das Verhalten von Jugendlichen, sondern Jugendliche beeinflussen auch das Verhalten ihrer Eltern. Derzeit ist jedoch nur wenig bekannt über die wechselseitigen langzeitlichen Zusammenhänge zwischen psychische Problemen und Beziehungsqualität von Eltern und Jugendlichen, wie diese Zusammenhänge aufrecht erhalten werden und für welche Familien diese gelten.

In der vorliegenden Dissertation wurden daher die intergenerationelle Weitergabe von psychischen Problemen und Beziehungsqualität im Jugend- und Erwachsenenalter untersucht. In multimethodischen Längsschnittstudien wurde anhand von drei übergreifenden Zielen versucht mehr Einsichten in intergenerationelle Weitergabe zu gewinnen: So wurde untersucht 1) wie psychische Probleme und Beziehungsqualität während des Jugendalters weitergegeben werden und ob sich der Einfluss der Eltern im Laufe der Zeit verändert, 2) ob Transferprozesse hauptsächlich von Eltern zu Kindern oder von Kindern zu Eltern verlaufen, und 3) welche Mechanismen intergenerationelle Weitergabe erklären können und welche Familien mehr oder weniger stark ausgeprägte Zusammenhänge aufzeigen.

Intergenerationelle Weitergabe über die Zeit hinweg: Die Jugendzeit ist bedeutsam

Aus den Ergebnissen dieser Dissertation ist zu schließen, dass die Adoleszenz eine wichtige Lebensphase darstellt, in der Eltern und Jugendliche nicht nur ähnliche psychische Probleme und Beziehungsverhalten zeigen, sondern Eltern auch die Entwicklung ihrer Kinder prägen. In allen vier Kapiteln wurden Zusammenhänge zwischen Symptomen und Verhalten von Eltern und Kindern deutlich – nicht nur zum selben Zeitpunkt (querschnittlich), sondern auch über die Zeit hinweg (längsschnittlich). Sowohl für psychische Probleme als auch für die Qualität der Eltern-Kind-Beziehung zeigten sich klare Übertragungsprozesse von der frühen bis zur späten Adoleszenz und sogar bis ins junge Erwachsenenalter, was darauf hindeutet, dass Eltern für Jugendliche über die gesamte Adoleszenz hinweg von Bedeutung bleiben.

Richtung der intergenerationellen Weitergabe: Effekte sind bidirektional

Die Ergebnisse zeigen zudem, dass nicht nur Eltern ihre Kinder beeinflussen, sondern dass Jugendliche auch Einfluss auf ihre Eltern ausüben. So sagten weniger positive und stärker negative Freundschaftsbeziehungen schlechtere Eltern-Kind-Beziehungen zu einem späteren Zeitpunkt voraus. Auch internalisierende Symptome von Jugendlichen sagten die Zunahme mütterlicher Symptome sowie weniger positive Interaktionen zwischen Müttern und ihren Kindern voraus. Diese Resultate unterstreichen die entscheidende Rolle, die Jugendlichen selbst bei der Übertragung psychischer Probleme und Beziehungsqualität zukommt. Wechselseitige Zusammenhänge deuten zudem darauf hin, dass Eltern und Jugendliche die Probleme des Anderen im Laufe der Zeit verstärken können. Die intergenerationelle Weitergabe von psychischen Problemen und Beziehungsqualität kann demnach nicht schlichtweg charakterisiert werden als einseitiger Prozess von der älteren auf die jüngere Generation, sondern ist ein komplexer Prozess, der durch wechselseitige Eltern-Kind-Effekte entsteht und fortbesteht.

Zugrundeliegende Mechanismen der intergenerationellen Weitergabe: Eltern-Kind-Dynamiken als erklärender Faktor

Das emotionale Klima der Familie, das unter anderem Emotionen der Eltern und Interaktionen zwischen Eltern und Jugendlichen umfasst, spielt eine entscheidende Rolle dabei, wie Verhalten zwischen Eltern und Jugendlichen weitergegeben wird. Trotz erheblicher Fortschritte in den letzten Jahrzehnten bestehen Geschlechterungleichheit und traditionelle Familienrollen weiterhin. Vor allem Mütter prägen daher das emotionale Klima der Familie nach wie vor maßgeblich. Die Ergebnisse dieser Dissertation zeigen, dass die täglichen Emotionen, in Ausmaß und Dauer, von Müttern das zukünftige Wohlbefinden von Jugendlichen voraussagten, und dass die Weitergabe psychischer Probleme von Müttern auf Jugendliche teils durch - hauptsächlich negative - Mutter-Kind-Interaktionen erklärt werden konnte.

Obwohl die Zusammenhänge eindeutig und studienübergreifend zu finden waren, waren die Effekte verhältnismäßig klein. Das deutet darauf hin, dass psychische Probleme und Beziehungsqualität nicht in allen Familien übertragen werden. Der Einfluss negativer täglicher Emotionen von Müttern, vor allem Schwierigkeiten diese zu regulieren, war weniger ausgeprägt, wenn sich die Emotionen der Mutter und des Kindes stärker ähnelten. Emotionale Anpassung und gegenseitiges Verständnis könnten demnach auf Widerstandsfähigkeit gegen ein negatives emotionales Klima in der Familie hinweisen. Dies lässt darauf schließen, dass die Dynamik zwischen Mutter und Kind einerseits erklärt, wie intergenerationelle Weitergabe erfolgt (Mediation), andererseits aber auch zeigt, dass intergenerationelle Weitergabe nicht alle Familien betrifft (Moderation).

Implikationen für die klinische Praxis

Die Ergebnisse dieser Dissertation liefern wichtige Anknüpfungspunkte für die klinische Praxis. Zunächst suggerieren sie, dass Eltern in der Adoleszenz eine wichtige Bedeutung zukommt und dass Interventionen, die darauf ausgerichtet sind, psychische Probleme und negative Verhaltensmuster im Jugendalter zu reduzieren, sich sowohl an das Kind als auch an dessen Eltern richten sollten. Die Stärkung von positiven Beziehungen und gegenseitigem Verständnis zwischen Eltern und Jugendlichen könnte ein wichtiger Schritt sein, um mögliche negative Kreisläufe zu durchbrechen. Wichtig ist dabei, dass Interventionen auf langfristige Veränderungen abzielen und Unterschiede zwischen Familien berücksichtigen. Was in einigen Familien hilft, greift nicht unbedingt auch in anderen. Ein individuelles Risikoprofil per Familie kann hier die Abstimmung von Hilfsmaßnahmen begünstigen, um langfristige Veränderungen in der Eltern-Kind-Dynamik zu bewirken.

Fazit

Zusammenfassend verdeutlichen die Ergebnisse dieser Dissertation, dass die intergenerationelle Weitergabe während des Jugendalters von Bedeutung ist. Psychische Probleme und Beziehungsqualität von Eltern sind eng mit der psychischen Gesundheit und dem Verhalten von Jugendlichen verknüpft, wobei sowohl Übertragung von Eltern auf Jugendliche als auch von Jugendlichen auf Eltern eine Rolle spielt. Dynamische Prozesse zwischen Eltern und Kind können diese Zusammenhänge erklären, aber das Ausmaß des Einflusses der Eltern variiert zwischen Familien. Während negative Eltern-Kind-Interaktionen erklären können, warum sich problematisches Verhalten in manchen Familien häuft, können Beziehungen, in denen Eltern und Kinder aufeinander abgestimmt sind, dazu beitragen, negative Muster zu durchbrechen. Werden Kinder genauso wie ihre Eltern? Die Ergebnisse dieser Arbeit deuten nicht darauf hin, dass dies immer der Fall ist. Sie legen aber für einige Familien nahe, dass es wahrscheinlicher wird, ähnliche psychische Probleme und Beziehungsmuster zu entwickeln, wenn Eltern-Kind-Interaktionen sich permanent wiederholen.

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ABOUT THE AUTHOR

Susanne Schulz was born on May 18th in 1987 in Röbel/Müritz, Germany. She studied *Educational and Pedagogical Sciences, German Language Studies,* and *English Language Studies* at the University of Hamburg, Germany (highest honors). After graduation, she moved to Salzburg, Austria to complete her teacher's training and to work as an elementary school teacher in Bavaria, Germany. During her time in Salzburg, she followed her passion for music and worked voluntarily as a radio host for programmes for young adults, together with Sara Wichelhaus. In 2015, Susanne moved to Amsterdam and completed the *Child Development and Education* Research Master at the University of Amsterdam (cum laude). In her thesis, she conducted an experimental study that examined parents' stress and self-efficacy beliefs in response to disruptive child behavior, supervised by dr. Patty Leijten and prof. dr. Geertjan Overbeek. During her studies at the University of Amsterdam, she worked as a research assistant and as an international student ambassador.

In September 2017, Susanne started her PhD studies at the Youth & Family department at Utrecht University as part of the Consortium on Individual Development (CID) and under supervision of prof. dr. Susan Branje, prof. dr. Tineke Oldehinkel, prof. dr. Wim Meeus, and dr. Stefanie Nelemans. She published several longitudinal studies of this dissertation on the role of parents in adolescent development and contributed to other research projects as co-author. She was also a core team member of the CID *Connecting Data in Child Development* (CD^2) project, in which she represented the RADAR (Research on Adolescent Development And Relationships) cohort.

In addition to her research-related activities, Susanne was involved in organizational and teaching activities. She organized grant-supported international symposia and presented her work at national and international conferences. To disseminate her findings to a broader audience, Susanne also contributed to outreach activities, including a media interview, a family lecture, and a practitioners' report. As representative of the Young Scholars committee of the European Association for Research on Adolescence (EARA), she organized and coordinated conference activities such as preconference workshops, webinars, roundtables, and social activities. In her teaching, Susanne gave small-group lectures, supervised internships and Master's theses, and gave statistical workshops for colleagues, organized by EARA. During her PhD in 2021, she became the mother of Yuna Lou (2021).

In October 2022, Susanne started working as a postdoctoral researcher at the Research Insititute for Child Development and Education at the University of Amsterdam, where she will continue her work on the transmission of psychopathology and on identifying potential targets for breaking negative cycles between parents and children.

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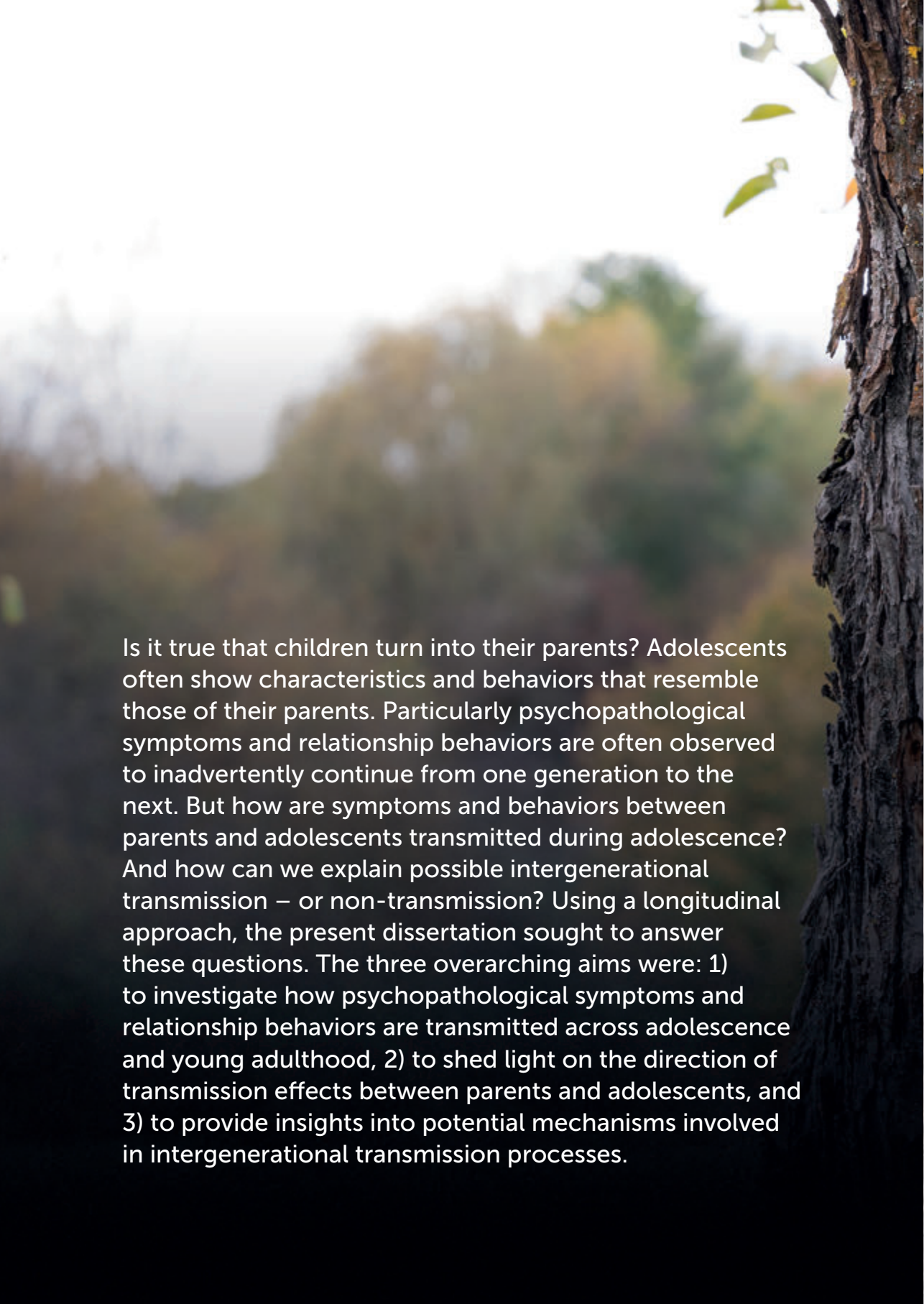

Is it true that children turn into their parents? Adolescents often show characteristics and behaviors that resemble those of their parents. Particularly psychopathological symptoms and relationship behaviors are often observed to inadvertently continue from one generation to the next. But how are symptoms and behaviors between parents and adolescents transmitted during adolescence? And how can we explain possible intergenerational transmission – or non-transmission? Using a longitudinal approach, the present dissertation sought to answer these questions. The three overarching aims were: 1) to investigate how psychopathological symptoms and relationship behaviors are transmitted across adolescence and young adulthood, 2) to shed light on the direction of transmission effects between parents and adolescents, and 3) to provide insights into potential mechanisms involved in intergenerational transmission processes.