AMERICAN PSYCHOLOGICAL ASSOCIATION

Developmental Psychology

ISSN: 0012-1649

2021, Vol. 57, No. 2, 269–283
https://doi.org/10.1037/dev0001144

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

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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

Supplemental materials: https://doi.org/10.1037/dev0001144.supp

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 10 times the health costs of problem behaviors that develop in adulthood (World Health Organization, 2007). Although adolescent internalizing and externalizing symptoms sometimes co-occur, they represent two distinct liabilities within a hierarchical structure of psychopathology (Achenbach, 1966; Lahey et al., 2017), and predict different health trajectories in adulthood (Korhonen et al., 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 et al., 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 ex-

Editor's Note. Jennifer E. Lansford served as the action editor for this article.—EFD

This article was published Online First December 21, 2020.

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Data of the RADAR study were used. RADAR has been financially supported by main grants from the Netherlands Organisation for Scientific Research (GB-MAGW 480-03-005, GB-MAGW 480-08-006), and Stichting Achmea Slachtoffer en Samenleving (SASS), a grant from the Netherlands Organisation for Scientific Research to the Consortium Individual Development (CID; 024.001.003), and various other grants from the Netherlands Organisation for Scientific Research, the VU University Amsterdam, and Utrecht University.

None of the authors have any conflicts of interest to disclose.

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amined 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 et al., 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 et al., 2018; Rutter et al., 2006; Sullivan et al., 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; Granic & Patterson, 2006; Hammen et al., 2004; Lovejoy et al., 2000; 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 et al., 2009; Kim et al., 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 et al., 2011; Serbin et al., 2011). 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 et al., 1995). Specifically, bidirectional associations have been found between parental internalizing symptoms and adolescent internalizing (Hughes & Gullone, 2010; Wilkinson et al., 2013) as well as adolescent externalizing symptoms (Gross et al., 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., 2020). 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 6-year multiinformant 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., 2020), 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 interindividual 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 et al., 2008; Zahn-Waxler et al., 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.

Method

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 com-

pared 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 nonmissing (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 ($\alpha = .91$–$.96$). 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 ($\alpha = .87$–$.91$). 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 ($\alpha = .87$–$.93$). 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 ($\alpha = .74$–$.85$). Higher scores reflected higher mean levels of externalizing symptoms.

Statistical Analyses

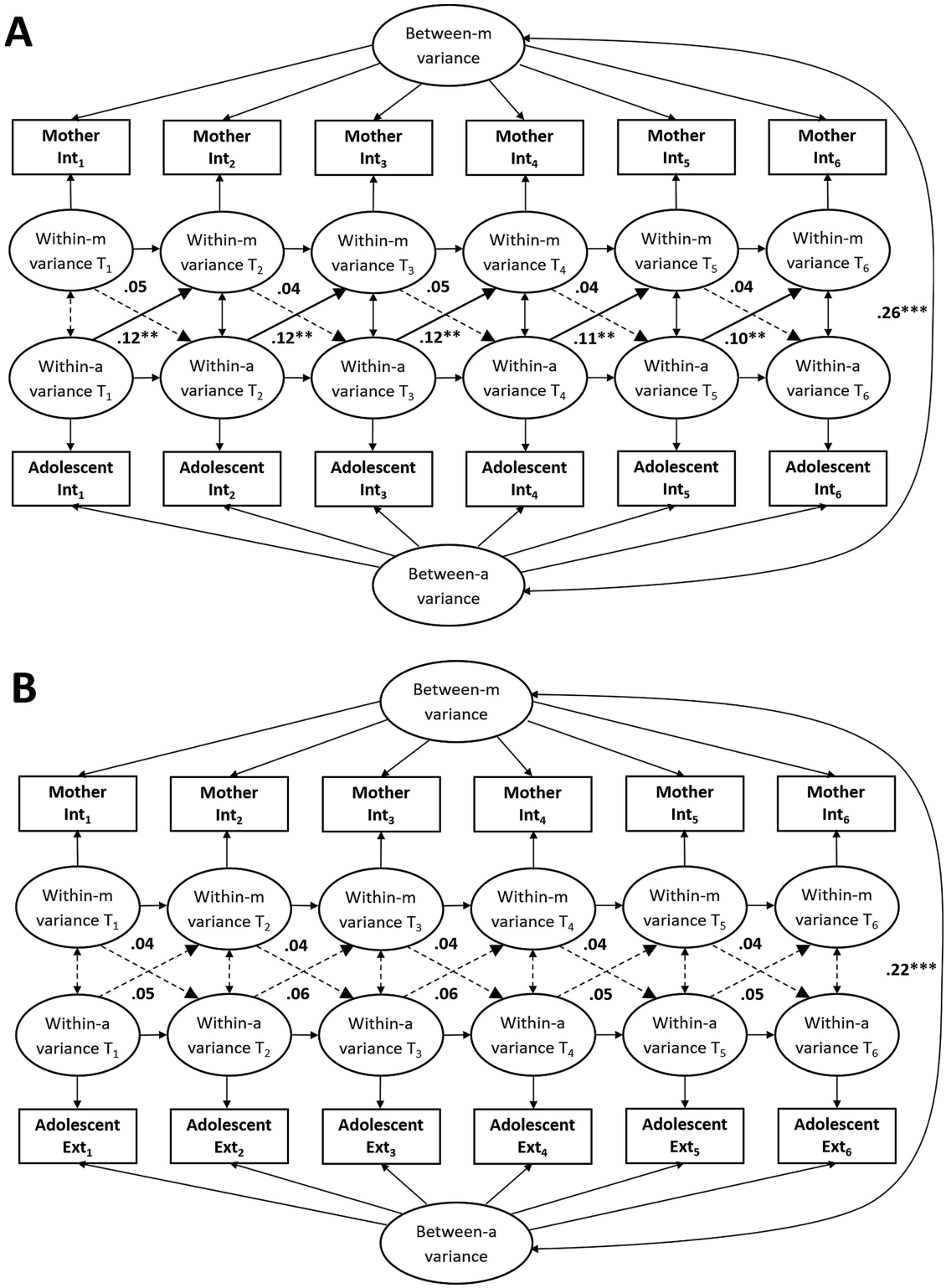
Missing data ranged from 0.4–25.2% for most variables, with overall 14.1% of all values missing. Six adolescents were excluded from the analyses involving internalizing symptoms, because their data was missing on all measurement occasions. Little's missing completely at random (MCAR) test detected no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

We calculated intraclass 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 multilevel 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 1-year autoregressive paths for parental and adolescent symptoms, within-time associations between parental and adolescent symptoms at the same time point, and 1-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

Figure 1

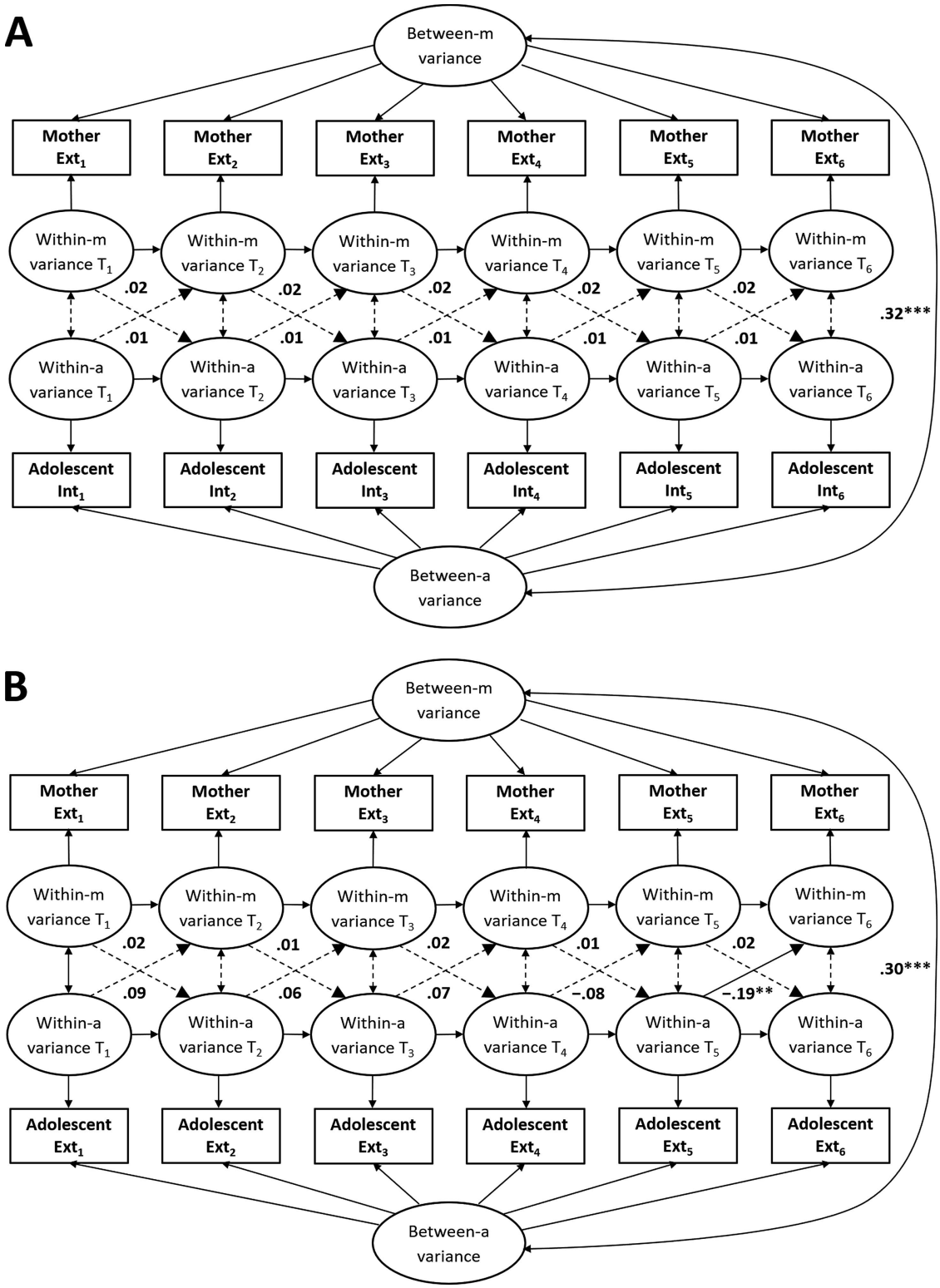
Random-Intercept Cross-Lagged Panel Models on the Associations Between Maternal Internalizing and Adolescent Internalizing (1A) and Externalizing Symptoms (1B), m = Mother, a = Adolescent



Note. ** $p < .01$. *** $p < .001$.

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



Note. ** $p < .01$. *** $p < .001$.

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

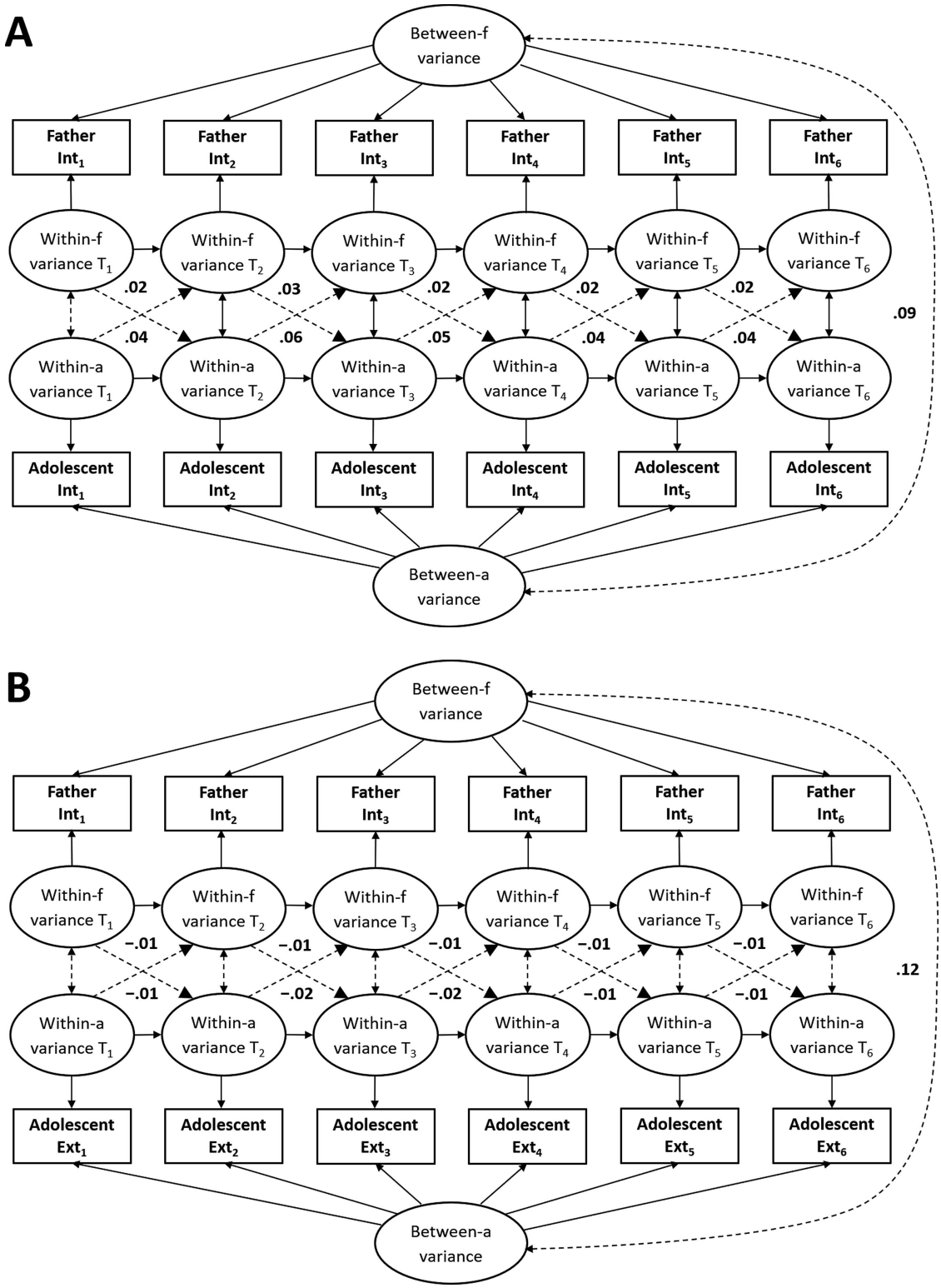
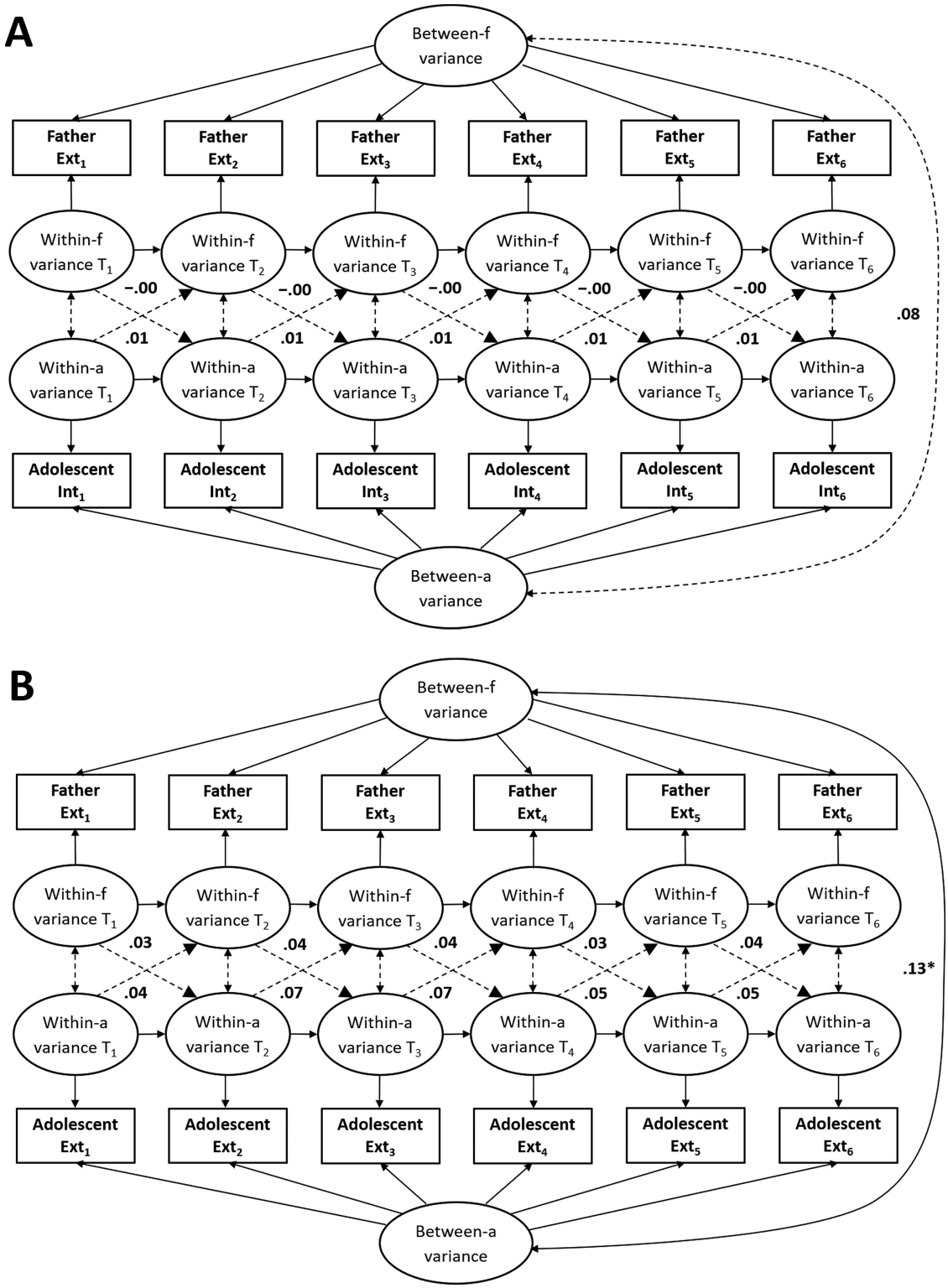


Figure 4

Random-Intercept Cross-Lagged Panel Models on the Associations Between Paternal Externalizing and Adolescent Internalizing (4A) and Externalizing Symptoms (4B), f = Father, a = Adolescent



Note. * $p < .05$.

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 online 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.

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 online supplementary material). Freeing the within-family correlated change did not significantly improve the fit for any model ($ps \geq .118$, 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 ($ps \geq .096$).

Table 1
Range of Descriptives of All Study Variables Across Time

Variable	*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

[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.

Between-Family Associations With Maternal and Paternal Psychopathological Symptoms

The between-family correlations of all RI-CLPMs are presented in Table 2. In families in which mothers reported higher levels of internalizing symptoms than in other families, adolescents reported higher levels of internalizing ($\beta = .26$, $p < .001$; see between-family associations in Figure 1A) and externalizing symptoms ($\beta = .22$, $p < .001$; see Figure 1B) than in other families. Similarly, in families in which mothers reported higher levels of externalizing symptoms than in other families, adolescents reported higher levels of internalizing ($\beta = .32$, $p < .001$; see Figure 2A) and externalizing symptoms ($\beta = .30$, $p < .001$; see Figure 2B) than in other families.

For fathers, we detected only one significant association between paternal and adolescent psychopathological symptoms at the between-family level (see Figures 3–4). Specifically, in families in which fathers reported higher levels of externalizing symptoms than in other families, adolescents reported higher levels of externalizing symptoms ($\beta = .13$, $p = .026$; see Figure 4B) than in other families. This indicates that across families, increased adolescent symptoms are more likely to occur in families with more maternal, but not paternal symptoms compared to other families.

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 ($\beta = .04–.05$, $ps \geq .095$; see cross-lagged paths in Figure 1A) or externalizing symptoms ($\beta = .04$, $ps \geq .182$; 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 ($\beta = .10–.12$, $ps = .001$; see Figure 1A), but not after adolescents reported higher levels of externalizing symptoms than their own average ($\beta = .05–.06$, $ps = .126$; see Figure 1B). No significant longitudinal associations were found from fluctuations in maternal externalizing symptoms to subsequent fluctuations in adolescents' internalizing ($ps \geq .553$; see Figure 2A) or externalizing symptoms ($ps \geq .601$; 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

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

Model		Maternal models			Paternal models		
		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
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 to T6.

reported higher levels of externalizing symptoms than their own average ($\beta_{T1–T5} = -.08–.09$, $ps \geq .194$), 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 ($\beta_{T5–T6} = -.19$, $p = .008$). 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 ($\beta_{T2–T6} = .10–.13$, $ps \leq .002$). 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 ($ps \geq .207$; see Figure 3) or externalizing symptoms than their own average ($ps \geq .153$; 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 ($\beta_{T2–T6} = .05–.08$, $ps \leq .039$; 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 online supplementary material).

Associations for Adolescent Boys and Girls

We conducted multigroup analyses to investigate gender differences between adolescent boys and girls (see online 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, 1976; Hammen et al., 2004; Raposa et al., 2011; Serbin et al., 2011). 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 & Gullone, 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, 1976) than fathers. Furthermore, women tend to be more emotionally expressive with their children (Fivush et al., 2000; Van der Giessen & Bögels, 2018), report to be more empathic toward others (Eisenberg & Lennon, 1983), and have more conflictual interactions with their children than men (Branje et al., 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., corumination; 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 microlevel. 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 interindividual 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 et al., 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.

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Received February 26, 2020
Revision received November 5, 2020
Accepted November 11, 2020 ■