Associations Between Mother–Child Relationship Quality and Adolescent Adjustment: Using a Genetically Controlled Design to Determine the Direction and Magnitude of Effects

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Abstract

This study used a genetically controlled design to examine the direction and the magnitude of effects in the over–time associations between perceived relationship quality with mothers and adolescent maladjustment (i.e., depressive symptoms and delinquency). A total of 163 monozygotic (MZ) twins pairs (85 female pairs, 78 male pairs) completed questionnaires at ages 13 and 14. Non–genetically controlled path analyses models (in which one member of each twin dyad was randomly selected for analyses) were compared with genetically controlled path analyses models (in which MZ–twin difference scores were included in analyses). Results from the non–genetically controlled models revealed a) child–driven effects in the longitudinal associations between adolescent perceived maternal support and depressive symptoms, and b) parent–driven and child–driven effects in the longitudinal association between perceived maternal negativity and adolescent delinquent behaviors. However, results from the genetically controlled models revealed only child–driven effect, suggesting that, purported parent–driven effects were a product of error arising from potential gene–environment correlations (rGE).

Despite changes in the frequency and the form of their interactions, parent–child relationships continue to play an influential role in children's adjustment during the adolescent years. Several studies link the quality of relationships with parents to later adolescent adjustment outcomes such as depression and delinquency (Adams & Laursen, 2007; Branje, Hale, Frijns, & Meeus, 2010; Conger, Ge, Elder, Lorenz, & Simons, 1994). However, previous studies may overstate the importance of parents in shaping adolescent outcomes (i.e., parent–driven effects) because they failed to account for child–driven effects whereby adolescent problem behaviors elicit specific reactions from the parents (Hafen & Laursen, 2009) or uncontrolled third variables such as the child's genotype. Indeed, genes that are linked to child's depression and delinquency or to parent's behaviors may also affect the parent–adolescent relationship through gene–environment correlations (rGE; Plomin, DeFries, & Loehlin, 1977). The present study employs a genetically–controlled design to
assess parent–driven and child–driven effects in the over–time associations between mother–child relationship quality and adolescent maladjustment. To illustrate the pitfalls of conventional analytic procedures, this study will compare non–genetically informed models, in which one twin was randomly selected for analyses, with genetically–controlled models, in which monozygotic (MZ) twin difference scores were included in analyses.

Parent–driven models assume that parenting behaviors, including the quality of the relationship between parents and children, predict adolescent developmental trajectories. Parental negativity, which includes conflict, disagreement and criticism, and lack of parental support, which encompasses instrumental assistance, approval and nurturance, have been linked to adolescent internalizing and externalizing symptoms (DeLay, Hafen, Cunha, Weber & Laursen, 2013; Ehrlich, Dykas, & Cassidy, 2012; Waschbusch, 2002). Despite repeated warnings that the heavy reliance on parent–driven models is unwarranted (e.g. Laursen & Collins, 2009), most studies of parent–child relationship adopt this perspective.

In contrast, child–driven models assume that child behavior will predict parental practices and the quality of parent–child relationship. High levels of adolescent problem behaviors are associated with declines in parental involvement and in effective parenting practices (Simons, Chao, Conger, & Elder, 2001). One study of positive features of parent–child relationship quality found that adolescent externalizing symptoms predicted subsequent changes in perceived parental support (Hafen & Laursen, 2009). Hence, adolescents with the highest levels of behavior problems reported the steepest declines in support from parents. Similar child–driven effects have been found for negative features of parent–child relationship quality, such that parents of children with externalizing behaviors react with more anger and negativity than parents with non–aggressive children (Anderson, Lytton, & Romney, 1986).

Findings from genetically informed research suggest that associations between parent–child relationship quality and adolescent maladjustment may be partly explained by genetic factors (Burt, McGue, Krueger, & Iacono, 2005). Over–time associations between relationship quality with parents and adolescent maladjustment may therefore reflect gene–environment correlations (rGE; Plomin, DeFries, & Loehlin, 1977). Two types of rGE may be implicated: passive and evocative. Passive rGE arises when children receive both genetic and environmental risk factors from their parents. For instance, delinquent parents, who have passed along their genetic make–up to their child, may act more aggressively with their child, which may in turn lead to deterioration in the parent–child relationship quality. The link between parent–adolescent relationship quality and adolescent adjustment could also be explained by an evocative rGE, whereby genetically driven adolescent characteristics evoke specific behavioral responses from the parents. One genetically informed study found that children’s antisocial behaviors evoked increases in parental negativity (Larsson, Viding, Rijstjijk & Plomin, 2008). Therefore, results from genetically informed studies could differ from previous one child per family studies because, as the genetic contribution in the link between parenting and child’s behavior is removed, parent–driven and child,driven effects may be reduced or suppressed (Burt, McGue, Iacono, & Krueger, 2006). To date, genetically informed studies of parent–adolescent relationship quality and adolescent maladjustment have focused on conflicts and externalizing symptoms with mixed results (Hou, Chen,
Natsuaki, Li, Yang, Zhang & Zhang, 2013; Jensen & Whiteman, 2014; Klahr, McGue, Iacono, & Burt, 2011). Outside of these few studies of negative relationship features, little is known about either positive features of parent–child relationships or about adolescent internalizing symptoms.

In the present study, we adopted a genetically controlled design to assess the uni– or bi–directional influence of parent–adolescent relationship quality on adolescent depressive symptoms and delinquency, independent of genetic and family–wise (i.e., shared) environmental effects (Vitaro, Brendgen, & Arseneault, 2009). Monozygotic twins are genetically identical and they share the same family environment, so associations between twin differences on relationship quality with mothers and twin differences in adjustment may be ascribed to the influence of nonshared environmental experiences that are unique to each individual. In this way, the MZ twin difference method eliminates sources of genetic and shared environmental variance, including rGE, which may act as a confound in the association between parent–adolescent relationship quality and adolescent adjustment.

Two goals guided our research. First, we examine parent–driven and child–driven effects to address over–time associations between perceived relationship quality with mothers and adolescent maladjustment. To explore parent–driven effects, analyses examine over–time associations from perceived maternal support and negativity at age 13 to changes in adolescent adjustment from age 13 to age 14, testing the hypothesis that lower maternal support and higher maternal negativity should be associated with increases in adolescent maladjustment. To explore child–driven effects, analyses examine over–time associations from adolescent externalizing and internalizing symptoms at age 13 to changes in mother–child relationship quality from age 13 to age 14, testing the hypothesis that higher initial level of adolescent internalizing and externalizing symptoms are associated with declines in mother–child relationship quality. Second, we sought to determine the degree to which gene–environment correlations inflated previous results describing parent–driven and child–driven effects. To this end, we compare a non–genetically informed model, in which one twin was randomly selected for analyses, with a genetically controlled model, in which MZ twin difference scores were included in the analyses. In the genetically controlled model, we expected that the effects would be reduced compared to the non–genetically controlled model, reflecting the removal of the contribution of rGE and shared family factors.

Method

Participants

The 163 MZ same sex twin pairs (85 female pairs, 78 male pairs) participating in this investigation were part of the Quebec Newborn Twin Study, an ongoing longitudinal study of a population–based sample of twins born between 1995 and 1998 in the greater Montreal area (Boivin et al., 2013). Zygosity was assessed by analysis of 8–10 highly polymorphous genetic markers. Twins were diagnosed as MZ when concordant for every genetic marker. When genetic material was insufficient or unavailable due to parental refusal (43% of cases), zygosity was determined based on physical resemblance questionnaires at 18 months and again at 9 years (Goldsmith, 1991; Spitz et al., 1996). The comparison of zygosity based on genotyping with zygosity based on physical resemblance in a subsample of 237 pairs
revealed a 94% correspondence rate, which is extremely similar to rates obtained in other studies (e.g., Magnusson et al., 2013).

The demographic characteristics of the twin families when the children were 5 months of age resembled those of a sample of single births that was representative of the large urban centers in the province of Quebec (Santé-Québec, Jetté, Desrosiers, & Tremblay, 1998). Eighty–four percent of the families were of European descent, 3% were of African descent, 2% were of Asian descent, and 2% were Native North Americans. The remaining families (9%) did not provide information about ethnicity. Most parents (95%) lived together at the time of birth of their children; 44% of the twins were the eldest siblings; 66% of the mothers and 60% of the fathers were between 25 and 34 years old; 17% of the mothers and 14% of the fathers had not finished high school; a similar proportion of mothers (28%) and fathers (27%) held a university degree; 83% of parents were employed, and 10% of families received social welfare or unemployment insurance.

**Procedure**

Data collection was approved by the Institutional Review Boards of the University of Quebec in Montreal and the Sainte–Justine Hospital Research Center. Instruments were administered either in English (21%) or in French (79%), depending on the language spoken at home by children and parents. Back–translation procedures were employed and bilingual translators verified the semantic similarity between the back–translated items and the original items in the questionnaire. Data for the present study were collected at age 13 (grade 7) and age 14 (grade 8) through personal interviews in the twins’ homes.

**Measures**

**Perceived relationship quality with mothers**—At each wave, participants completed a brief version of the Network of Relationships Inventory (NRI; Furman & Buhrmester, 1985, 1992). **Perceived maternal support** includes six items that describe validation, caring, help and guidance received by the mother. Sample item: “How much does your mother help you figure out or fix things?” **Perceived maternal negativity** includes three items that describe conflict and disagreement with the mother. Sample item: “How much do you and your mother quarrel?” Response options ranged from 1 (not at all) to 5 (most of the time). Item scores were averaged (Cronbach’s alphas = .85–.87).

**Delinquent behaviors**—At each wave, participants completed a brief version of the Self–Report Delinquency Questionnaire (SRDQ; LeBlanc & Frechette, 1989). The SRDQ short form comprises nine items on physical violence, theft and vandalism. Sample item: “During the last 12 months have you stolen something from a store?” Response options ranged from 0 (never) to 3 (very often). Item scores were averaged (Cronbach’s alphas = .66–.67). Approximately 62% of participants engaged in at least one delinquent behavior at least once.

**Depressive symptoms**—At each wave, participants completed the 10–item short form of the Children’s Depression Inventory (CDI; Kovacs 1992). Sample item: “During the last
two weeks, how often have you felt like crying?” Response options ranged from 1 (rarely) to 3 (always). Item scores were averaged (Cronbach’s alpha = .77–78).

**Plan of Analysis**

Twin pairs were included in the present investigation if they participated in the data collection at either age 13 or age 14. Of the 266 MZ twin pairs originally recruited at birth, 140 (53%) completed questionnaires at age 13 and age 14, 13 (0.5%) completed questionnaires at age 13 only, and 10 (0.4%) completed questionnaires at age 14 only. Independent samples t–tests revealed no differences between the original sample and those included in the present study on parent education (d = .03), household income (d = .12) and family structure (d = .08). There was minimal item–level missing data, averaging 2.5% at age 13 and 13% at age 14. Little's MCAR test indicated that data were missing completely at random [$\chi^2 (1599) = 1680.71, p = .08$]. Item–level and wave–level missing data were handled with multiple imputations generated from a Markov chain Monte Carlo simulation in Mplus v7.2 (Muthén & Muthén, 1998–2014). Specifically, 25 independent imputed datasets were generated, which were then used for subsequent model tests using maximum likelihood estimation. Multiple imputation is a robust and accurate estimator of results when less than 50% of data are missing completely at random (Graham, 2009). Parameter estimates were averaged over the imputed data sets.

Before performing the analyses that control for the genetic contributions, it is important to measure the influence of genes on the study variables. By comparing the within–pair correlations of the MZ twin pairs with those of the 119 same–sex DZ twin pairs (61 boys and 58 girls) who also participated in the study, it is possible to estimate the relative strength of genetic and environmental effects on a given variable (Falconer, 1989). The relative strength of genetic effects is approximately twice the MZ and same–sex DZ within–pair correlation difference: $g^2 = 2(r_{MZ} – r_{DZ})$. The relative strength of shared environmental effects can be estimated by subtracting the MZ correlation from twice the DZ correlation: $c^2 = (2r_{DZ}) – (r_{MZ})$. Nonshared environmental effects can be approximated by the extent to which the MZ correlation is less than 1: $e^2 = 1 – r_{MZ}$.

Four sets of path analyses were conducted using Mplus v7.2 (Muthén & Muthén, 1998–2014) to assess parent–driven and child–driven associations between perceived relationship quality with mothers and adolescent adjustment. Parent–driven effects are represented by paths from perceived relationship quality with mothers (i.e., perceived maternal support and negativity) to adolescent maladjustment (i.e., adolescent delinquency and depressive symptoms). Child–driven effects are represented by paths from adolescent maladjustment (i.e., adolescent delinquency and depressive symptoms) to perceived relationship quality with mothers (i.e., perceived maternal support and negativity). The first set of analyses examined associations between perceived maternal negativity and adolescent delinquent behaviors. The second set of analyses examined associations between perceived maternal negativity and adolescent depressive symptoms. The third set of analyses examined associations between perceived maternal support and adolescent delinquent behaviors. The fourth set of analyses examined associations between perceived maternal support and adolescent depressive symptoms.
The second aim of the study was to determine the degree to which rGE inflate results from non–genetically controlled analyses. To this end, each of the four path analyses described in the previous paragraph was conducted twice. First, the non–genetically controlled analyses involved one randomly selected member of each twin dyad, mirroring conventional analytic practices in one child per family studies. Second, the genetically controlled analyses included both members of each dyad. To produce scores free of genetic and shared environmental influence, an MZ twin difference score was calculated for each variable by subtracting one twin's score from the other twin's score on the same variable. These MZ twin difference scores were directional (i.e., not absolute values), with the higher values indicating greater levels of a variable relative to the co–twin. For each variable, each twin pair had one MZ twin difference score, which was included in each analysis only once.

Preliminary multiple group models examined gender as a potential moderator of the bidirectional associations between perceived relationship quality with mothers and adolescent adjustment outcomes from ages 13 and 14. Patterns of association did not differ for boys and girls, so the results were collapse across gender.

Results

Intraclass, Cross–Twin, Cross–Phenotype and Bivariate Correlations

Table 1 presents intraclass and cross–twin, cross–phenotype correlations between study variables separately for MZ twins and same–sex DZ twins. MZ twin intraclass correlations were at least 50% higher than same–sex DZ twin intraclass correlations, indicating that all study variables had a considerable genetic component, whereas shared environmental influences played only a small role. The fact that MZ twin intraclass correlations were considerably less than 1.0 indicates the presence of significant nonshared environmental contributions for most study variables. MZ twin cross–twin, cross–phenotype correlations were also significantly higher than same–sex DZ twin cross–twin, cross–phenotype correlations for most of study variables, indicating that the same set of genetic influences seems to be involved in the associations between adolescent adjustment problems and parent–adolescent relationship quality (Moffitt, 2005).

Table 2 presents bivariate correlations, separately for individual scores and for MZ twin difference scores. Bivariate correlations based on individual scores revealed statistically significant concurrent associations between perceived relationship quality with mothers and adolescent adjustment at ages 13 and 14 with one exception: adolescent delinquent behaviors at age 14 were not associated with maternal support at age 14. There were statistically significant over–time associations between adolescent adjustment and perceived relationship quality with mothers with one exception: maternal support at age 13 was not associated to adolescent adjustment at age 14.

Bivariate correlations based on MZ twin difference scores, which control for genetic and shared environmental influences, revealed statistically significant concurrent correlations between perceived relationship quality with mothers and adolescent adjustment at age 14 with the exceptions of the associations between perceived maternal support and adolescent adjustment. There were statistically significant over–time correlations between adolescent
delinquency at age 13 and perceived maternal negativity at age 14, as well as between adolescent depression at age 13 and perceived maternal support at age 14. Here and in the path analyses, associations involving twin difference scores are interpreted in terms of relative differences between MZ dyad members. For instance, a positive association between MZ twin difference scores of delinquency at age 13 and MZ twin difference scores of perceived maternal negativity at age 14 indicates that, relative to the co–twins, the twins with more delinquent behaviors perceived greater negativity with their mothers.

**Over–Time Associations Between Perceived Maternal Negativity and Adolescent Adjustment**

**Delinquent behaviors**—Figure 1 summarizes the results of the two sets of path analyses examining the associations between perceived maternal negativity and adolescent delinquent behaviors. Results from the non–genetically controlled model revealed that adolescent delinquent behaviors at age 13 were associated with perceived maternal negativity at age 14, indicating a child–driven effect. Higher levels of delinquency predicted greater increases in perceived negativity with mothers. In addition, perceived maternal negativity at age 13 was associated with adolescent delinquent behaviors at age 14, indicating a parent–driven effect. Higher levels of negativity with mothers predicted greater increases in adolescent delinquency. Chi–square difference tests revealed that child–driven paths were significantly larger than parent–driven paths, $\chi^2(1) = 15.64, p > .01$.

Results from the genetically controlled model with the MZ difference scores revealed that adolescent delinquent behaviors at age 13 were associated with perceived mother negativity at age 14, indicating a child–driven effect. Perceived maternal negativity at age 13 was not associated with adolescent delinquent behaviors at age 14. Thus, higher levels of delinquent behaviors predicted greater increases in perceived negativity with mothers but not the reverse. Chi–square difference tests revealed that child–driven paths were significantly larger than parent–driven paths, $\chi^2(1) = 7.67, p > .01$.

**Depressive symptoms**—Figure 2 summarizes the results of two sets of path analyses examining associations between perceived maternal negativity and adolescent depressive symptoms. There were no statistically significant parent–driven or child–driven effects in either the non–genetically controlled or the genetically controlled models.

**Over–Time Associations Between Perceived Maternal Support and Adolescent Adjustment**

**Delinquent behaviors**—Figure 3 summarizes the results of the two sets of analyses examining the associations between perceived maternal support and adolescent delinquent behaviors. There were no statistically significant parent–driven or child–driven effects in either the non–genetically controlled or the genetically controlled models.

**Depressive symptoms**—Figure 4 summarizes the results of the two sets of path analyses examining the associations between perceived maternal support and adolescent depressive symptoms. Results from the non–genetically controlled model revealed that adolescent depressive symptoms at age 13 were associated with perceived maternal support...
at age 14, indicating child–driven effects. Perceived maternal support at age 13 was not associated with adolescent depressive symptoms at age 14. Thus, higher levels of depressive symptoms predicted greater decreases in perceived support with mothers but not the reverse. Child–driven paths were significantly larger than parent–driven paths, $\chi^2(1) = 4.00$, $p > .05$.

Results from the genetically controlled model with the MZ difference scores revealed that adolescent depressive symptoms at age 13 were associated with perceived mother support at age 14, indicating a child–driven effect. Higher levels of depressive symptoms predicted greater decreases in perceived support with mothers. Chi–square difference tests revealed that child–driven paths were significantly larger than parent–driven paths, $\chi^2(1) = 4.44$, $p > .05$.

**Discussion**

The results revealed child–driven associations between adolescent depressive symptoms and perceived maternal support, as well as between adolescent delinquent behaviors and perceived maternal negativity. These findings emerge in both genetically controlled and non–genetically controlled analyses, indicating that these effects are not an artifact of rGE. In contrast, only the non–genetically controlled design revealed parent–driven associations between adolescent delinquent behaviors and perceived maternal negativity. The absence of these findings in the genetically controlled design indicates that purported parent–driven effects were plausibly a product of error arising from potential rGE. After controlling for genetic contributions, adolescent problem behaviors environmentally drive changes in maternal negativity, but maternal negativity does not environmentally drive changes in adolescent problem behaviors.

The child–driven effects between adolescent depressive symptoms and perceived maternal support indicate that higher initial levels of depressive symptoms predict greater subsequent decreases in perceived maternal support. These results are consistent with other reports of evocative child effects, whereby depressed adolescents elicited fewer positive behaviors from their parents when compared to non–depressed adolescents (Joiner & Coyne, 1999). In line with a support erosion process, parents might gradually withdraw from their depressed children, which in turn may affect parent–child relationship quality (Branje et al., 2010). Adolescent depressive symptoms, including daily negative affect and social isolation (Larson, Raffaelli, Richards, Ham, & Jewell, 1990), may cause a deleterious feedback loop, whereby depressed adolescents may further worsen their relational difficulties and, as a consequence, receive less support over–time from their parents.

We also found child–driven effects in the association between adolescent delinquent behaviors and maternal negativity such that greater initial level of behavior problems predicted greater subsequent increases in perceived maternal negativity. Findings are consistent with studies reporting that adolescent behavioral problems elicit increased conflicts and negativity in the parents (Simons, Chao, Conger, & Elder, 2001). For instance, studies have shown that the accumulation of past negative events might foster anger in parents, who become less patient and more negative over–time in their interactions with their children (Anderson et al., 1986). During adolescence, the emergence of delinquent behaviors...
may be particularly frustrating for parents and, as a result, it may exacerbate the negative features of the parent–adolescent relationship quality. Elevated levels of negativity, in turn, predict diminished support for adolescents (Laursen, DeLay, & Adams, 2010), creating a vicious downward spiral in relationship quality.

The child–driven effects for adolescent depressive symptoms and delinquent behaviors remained significant after controlling for genetic and shared environmental contributions. We can therefore rule out the possibility that these associations represent genetically driven effects such as rGE. These results are novel and highlight the importance of early differential life experiences. Indeed, unique environmental experiences, such as prenatal and obstetrical complications, accidents or illnesses, peer influences, and school experiences, differentiate genetically identical children (Pike & Plomin, 1996). In turn, these acquired differences within MZ twin pairs evoke differences in parenting (Guimond et al., 2012), which over-time can snowball into a decrease in the parent–child relationship quality (Mullineaux, Deater–Deckard, Petrill, & Thompson, 2009).

We also found parent–driven effects such that high level of maternal negativity was associated with subsequent increases in adolescent delinquent behaviors but these effects were limited to the non–genetically controlled model. Indeed, parent–driven effects disappeared in the genetically controlled model. Findings are consistent with genetically informed studies reporting that parent–child negativity is not associated with adolescent externalizing symptoms when using MZ difference scores (Burt, McGue, Iacono, & Krueger, 2006; Hou et al., 2013). This result suggests that a substantial portion of the association between maternal negativity and adolescent delinquent behaviors may be an artifact of rGE. Parents with a genetic disposition for hostility and norm–violations are prone to aggression and negativity toward their children and are likely to have children who inherited similar tendencies, indicating a passive rGE. In this scenario, parental negativity and adolescent delinquency may become spuriously related through common genes (Burt, McGue, Iacono, & Krueger, 2006).

Although the effects were in expected direction, we did not find significant associations between maternal negativity and adolescent depressive symptoms nor between maternal support and adolescent delinquency. It is possible that depression symptomatology, such as lower self–worth and optimism, influence adolescents’ perception of the positive features of the parent–child relationship more rapidly than the negative features. Hence, depressed adolescents may perceive less intimacy and support from their parents as immediate responses and it might explain the absence of significant links between maternal negativity and adolescent depressive symptoms. Moreover, it is possible that adolescent delinquency affect to a greater extent the perception of the negative features than the positive ones. Adolescents with delinquent behaviors and aggressive tendencies may perceive higher levels of conflicts and disagreements in the relationship with their parents, and it might explain the absence of significant links between maternal support and adolescent delinquency. Future studies should examine the long term impact of adolescent depression and delinquency on parent–adolescent relationship quality.
Our study has several limitations that need to be considered when interpreting the results. First, shared-reporter variance is a concern. Adolescents were the source of both reports of adjustment problems and reports of relationship quality with mothers. Second, because relationships are dyadic, it is not clear that measures of relationship quality represent child-driven or evocative effects in the traditional sense. Futures studies should examine the perception of both adolescents and their parents. Third, potential moderators that may enhance the associations between parent–adolescent relationship quality and adolescent maladjustment should be examined in future studies. For instance, pubertal maturation is often associated with heightened risk for depression, anxiety and delinquent behaviors, which, in turn, may affect parenting behaviors (Dickson, Laursen, Stattin & Kerr, 2015; Susman & Rogol, 2004). Fourth, some may question the generalizability of findings from MZ twins to singleton populations, although considerable evidence suggests that these concerns tend to be overstated (Barnes & Boutwell, 2013). Fifth, the relatively small sample of the present study limits its statistical power and might be an alternative explanation of the absence of significant findings between maternal negativity and adolescent depressive symptoms and between maternal support and adolescent delinquency. Finally, it is important to note that, although the MZ twins’ difference method affords control for possible genetic influences and shared environmental experiences, it still remains correlational in nature. As such, no definite conclusion can be drawn in regard to the causality of the observed predictions.

Despite these limitations, the present study offers new insights into the complex interplay between parent–child relationship quality and adolescent adjustment. Scholars are quick to pay lip service to the need to test for child-driven effects, but far slower to actually test these effects. Parent-driven models still dominate research on family relationships during adolescence (Laursen & Collins, 2009). The present findings underscore the importance of controlling for child-driven effects and genetic influences to avoid inflating estimates of parent-driven effects that can arise as a result of uncontrolled rGE, especially in the link between parent, adolescent relationship quality and adolescent externalizing behaviors.

References


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Figure 1.
N= 163. Over–time associations between perceived maternal negativity and adolescent delinquent behaviors. Standardized path coefficients are reported. Results for individual scores (non–genetically controlled model) are presented on the left of the slash and results for MZ difference scores (genetically controlled model) are presented on the right of the slash.

*p < .05, **p < .01.
Figure 2.
N= 163. Over-time associations between perceived maternal negativity and adolescent depressive symptoms. Standardized path coefficients are reported. Results for individual scores (non-genetically controlled model) are presented on the left of the slash and results for MZ difference scores (genetically controlled model) are presented on the right of the slash.
*p < .05, **p < .01.
**Figure 3.**

N= 163. Over-time associations between perceived maternal support and adolescent delinquent behaviors. Standardized path coefficients are reported. Results for individual scores (non-genetically controlled model) are presented on the left of the slash and results for MZ difference scores (genetically controlled model) are presented on the right of the slash.

*p < .05, **p < .01.
Figure 4.
N = 163. Associations between perceived maternal support and adolescent depressive symptoms. Standardized path coefficients are reported. Results for individual scores (non-genetically controlled model) are presented on the left of the slash and results for MZ difference scores (genetically controlled model) are presented on the right of the slash.
*p < .05, **p < .01.
Table 1
Intraclass and Cross–Twin, Cross–Phenotype Correlations [and 95% confidence intervals] for Monozygotic (MZ) and Same–Sex Dizygotic (DZ) Twins

<table>
<thead>
<tr>
<th>Variable</th>
<th>MZ twins</th>
<th>DZ twins</th>
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<tbody>
<tr>
<td>Intraclass correlations</td>
<td></td>
<td></td>
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<tr>
<td>Age 13 Delinquent behaviors</td>
<td>.38** [.18, .56]</td>
<td>.24* [-.09, .52]</td>
</tr>
<tr>
<td>Age 14 Delinquent behaviors</td>
<td>.50** [.30, .65]</td>
<td>.29** [.14, .45]</td>
</tr>
<tr>
<td>Age 13 Depressive symptoms</td>
<td>.44** [.31, .57]</td>
<td>-.02 [-.17, .15]</td>
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<tr>
<td>Age 14 Depressive symptoms</td>
<td>.52** [.32, .66]</td>
<td>.23* [.04, .40]</td>
</tr>
<tr>
<td>Age 13 Negativity</td>
<td>.65** [.43, .79]</td>
<td>.00 [-.16, .10]</td>
</tr>
<tr>
<td>Age 14 Negativity</td>
<td>.53** [.39, .69]</td>
<td>.12 [-.11, .40]</td>
</tr>
<tr>
<td>Age 13 Support</td>
<td>.64** [.49, .74]</td>
<td>.25** [.12, .41]</td>
</tr>
<tr>
<td>Age 14 Support</td>
<td>.35** [.16, .56]</td>
<td>.25** [.06, .43]</td>
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<tr>
<td>Cross–Twin, Cross–Phenotype Correlations</td>
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<td>.27** [.11, .45]</td>
<td>.07 [-.07, .21]</td>
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<tr>
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<td>.07 [-.06, .23]</td>
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<td>.08 [-.08, .31]</td>
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<td>-.02 [-.19, .14]</td>
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<td>Age 14 Depression– Support</td>
<td>-.32** [-.47, -.13]</td>
<td>-.06 [-.21, .10]</td>
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Note. MZ twins pairs n = 163. DZ twin pairs n = 119.

* p < .05
** p < .01.
### Table 2

Bivariate Correlations [95% confidence intervals], Means and Standard Deviations

<table>
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<th>Variable</th>
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<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>M (SD)</th>
</tr>
</thead>
<tbody>
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<td>1. Age 13 Delinquency</td>
<td>—</td>
<td>.28 ** [.05,.50]</td>
<td>.38 ** [.19,.55]</td>
<td>.18 * [.03,.34]</td>
<td>.13 [-.10,.34]</td>
<td>.24 ** [.02,.44]</td>
<td>.01 [-.15,.16]</td>
<td>-.08 [-.25,.10]</td>
<td>-.02 (0.17)</td>
</tr>
<tr>
<td>2. Age 14 Delinquency</td>
<td>.64 ** [.53,.75]</td>
<td>—</td>
<td>.25 ** [.08,.41]</td>
<td>.22 * [.02,.41]</td>
<td>.09 [-.10,.27]</td>
<td>.21 * [.04,.37]</td>
<td>-.10 [-.25,.08]</td>
<td>-.02 [-.18,.14]</td>
<td>-.02 (0.16)</td>
</tr>
<tr>
<td>3. Age 13 Depression</td>
<td>.34 ** [.16,.48]</td>
<td>.33 ** [.15,.50]</td>
<td>—</td>
<td>.25 ** [.10,.41]</td>
<td>.08 [-.11,.26]</td>
<td>.11 [-.08,.29]</td>
<td>-.12 [-.29,.07]</td>
<td>-.21 ** [-.37,.03]</td>
<td>0.02 (0.26)</td>
</tr>
<tr>
<td>4. Age 14 Depression</td>
<td>.17 * [.01,.33]</td>
<td>.18 * [.04,.35]</td>
<td>.61 ** [.48,.72]</td>
<td>—</td>
<td>.02 [-.15,.20]</td>
<td>.28 ** [.14,.42]</td>
<td>-.07 [-.23,.09]</td>
<td>-.12 [-.29,.05]</td>
<td>0.02 (0.27)</td>
</tr>
<tr>
<td>5. Age 13 Negativity</td>
<td>.40 ** [.26,.53]</td>
<td>.40 ** [.27,.53]</td>
<td>.23 * [.05,.37]</td>
<td>.19 * [.04,.35]</td>
<td>—</td>
<td>.11 [-.09,.30]</td>
<td>.01 [-.19,.21]</td>
<td>-.02 [-.17,.13]</td>
<td>0.03 (0.79)</td>
</tr>
<tr>
<td>6. Age 14 Negativity</td>
<td>.47 ** [.29,.61]</td>
<td>.51 ** [.36,.64]</td>
<td>.19 * [.00,.37]</td>
<td>.28 ** [.13,.44]</td>
<td>.59 ** [.48,.69]</td>
<td>—</td>
<td>-.12 [-.29,.05]</td>
<td>.01 [-.17,.19]</td>
<td>-.02 (0.71)</td>
</tr>
<tr>
<td>7. Age 13 Support</td>
<td>-.16 * [-.33,.02]</td>
<td>-.04 [-.19,.11]</td>
<td>-.29 ** [-.44,.15]</td>
<td>-.09 [-.27,.08]</td>
<td>-.18 * [-.34,.01]</td>
<td>-.09 [-.26,.08]</td>
<td>—</td>
<td>.23 ** [.06,.38]</td>
<td>0.03 (0.75)</td>
</tr>
<tr>
<td>8. Age 14 Support</td>
<td>-.18 * [-.30,.04]</td>
<td>-.08 [-.22,.06]</td>
<td>-.30 ** [-.42,.17]</td>
<td>-.30 ** [-.44,.15]</td>
<td>-.20 * [-.36,.03]</td>
<td>-.23 * [-.38,.05]</td>
<td>.63 ** [.50,.73]</td>
<td>—</td>
<td>-.04 (0.88)</td>
</tr>
</tbody>
</table>

*M (SD)*

Note. Correlations for individual scores (*n*= 163 randomly selected MZ twins) are presented below the diagonal, and correlations for difference scores (*n*= 163 MZ twin pairs) are presented above the diagonal. Means and standard deviations for individual scores are presented in horizontal rows and means and standard deviation for difference scores are presented in vertical columns and are based on absolute values.

Adolescent delinquency was coded from 0 (*never*) to 3 (*very often*). Adolescent depression was coded from 1 (*rarely*) to 3 (*always*). Maternal negativity and support were coded from 1 (*not at all*) to 5 (*most of the time).*

* *p < .05
** *p < .01