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Parental Knowledge is an Environmental Influence on Adolescent Externalizing

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Abstract

Background—There is evidence both that parental monitoring is an environmental influence serving to diminish adolescent externalizing problems and that this association may be driven by adolescents' characteristics via genetic and/or environmental mechanisms, such that adolescents with fewer problems tell their parents more, and therefore appear to be better monitored. Without information on how parents' *and* children's genes and environments influence correlated parent and child behaviors, it is impossible to clarify the mechanisms underlying this association.

Method—The present study used the Extended Children of Twins model to distinguish types of gene-environment correlation and direct environmental effects underlying associations between parental knowledge and adolescent (age 11-22 years) externalizing behavior with a Swedish sample of 909 twin parents and their adolescent offspring and a US-based sample of 405 White adolescent siblings and their parents.

Results—Results suggest that more parental knowledge is associated with less adolescent externalizing via a direct environmental influence independent of any genetic influences. There

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was no evidence of a child-driven explanation of the association between parental knowledge and adolescent externalizing problems.

Conclusions—In this sample of adolescents, parental knowledge exerted an environmental influence on adolescent externalizing after accounting for genetic influences of parents and adolescents. Because the association between parenting and child development originates in the parent, treatment for adolescent externalizing must not only include parents but should focus on altering their parental style. Thus, findings suggest that teaching parents better knowledge-related monitoring strategies is likely to help reduce externalizing problems in adolescents.

Keywords

Gene-Environment Correlation; Adolescent Externalizing; Parental Monitoring; Parental Knowledge; Extended Children of Twins

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The mechanism of the well-established association between parental knowledge of adolescents' whereabouts, activities, and behaviors and adolescent externalizing problems has been under great debate for decades. Associations between parental knowledge and adolescent externalizing behavior were originally described as a direct environmental association; parents' knowledge of their adolescents' behavior and whereabouts affect their parenting behaviors, perhaps making some parents more (or less) restrictive versus permissive with their adolescents and therefore allowing adolescents less versus more opportunity to engage in externalizing behavior (Dishion & McMahon, 1998; Patterson, DeBaryshe, & Ramsey, 1989). Stattin and Kerr (2000) challenged this notion, suggesting instead that parental knowledge may be a child-driven effect such that children who are generally better behaved are more likely to share their whereabouts, activities, and behavior than children who have more behavior problems. Since that seminal paper, numerous studies using longitudinal and genetically informed designs have been devoted to testing these competing hypotheses. Understanding the direction of the association has important implications for treatment of adolescent externalizing problems. If the association between parenting and child development originates in the parent then treatment must not only include them but should focus on altering parenting style (i.e., family management; Kazdin, 1997; Patterson & Reid, 1973). If the results show evocative effects (genetic or otherwise) the direct treatment of child aggression is suggested (Lochman, 1992). However, no studies have been able to establish causality and disentangle the direction of effects in this association (McAdams et al., 2014). Here we leverage the only model currently capable of establishing causality and disentangling the direction of effects in this association, the extended children of twins model (ECOT, Marceau et al., 2013; Narusyte et al., 2011; Narusyte et al., 2008).

Studies have found support for child effects on parental knowledge (Kerr, Stattin, & Burk, 2010; Racz & McMahon, 2011), including the influence of adolescents' genes (Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007; Neiderhiser et al., 2004), which indicates evocative *r*GE (youth with heritable externalizing problems may choose to disclose less about their activities, and in doing so, elicit less parental knowledge). However, the underlying genetic and environmental origins of the association between parental knowledge and adolescent externalizing have rarely been tested: some evidence suggests adolescent-

based genetic influences explained the association, indicating evocative *r*GE (Reiss, Neiderhiser, Hetherington, & Plomin, 2000). Conversely, longitudinal studies have consistently supported the notion that parental knowledge is a parenting behavior that can exert an influence on children's externalizing-type problems, and reported reciprocal relations between parental knowledge and adolescent delinquency (e.g., Crouter & Head, 2002; Fosco, Frank, Stormshak, & Dishion, 2013; Laird, Pettit, Bates, & Dodge, 2003; Pardini, Fite, & Burke, 2008; Willoughby & Hamza, 2011). Together, there is evidence of both evocative *r*GE and direct environmental influences from parents to adolescents.

There is another possibility that has yet to be tested - that genetic influences common to parents and adolescents could contribute to parental knowledge and adolescent externalizing behavior (passive *r*GE), such that the association is an artifact of shared genetic influences. Cross-lagged longitudinal designs cannot rule out the contribution of genetic influences, or test for evocative or passive *r*GE. Multivariate twin designs and children-of-twins designs can only examine the influence of parents' genes (indicating passive *r*GE, but incapable of ruling out evocative *r*GE) or adolescents' genes (indicating evocative *r*GE, but incapable of ruling out passive *r*GE) and cannot disentangle the direction of effects. Studies that can test all three mechanisms (i.e., direct environmental influence, passive and evocative *r*GE) are needed to advance the field; otherwise, effects that appear environmental may actually be over-estimated, spurious, or actually reflect *r*GE (McAdams et al., 2014). The ECOT model is currently the only method capable of testing all three mechanisms simultaneously and disentangling the direction of effects (Marceau et al., 2013; Narusyte et al., 2011; Narusyte et al., 2008).

Here, we use the ECOT design in order to understand the direction of effects (parents influencing offspring versus offspring influencing parents) and *r*GE underlying the association between parent and youth behavior. The ECOT is a nested model drawing data from two twin studies, one in which twins are parents with adolescent offspring and one in which twins are adolescents with comparable measures of parent and child behavior (see Narusyte et al., 2008 for a more detailed description of the model). By including the multiple sources (parents and adolescents) of genetic and environmental influences, the ECOT design affords examination of three possible mechanisms explaining associations between parent and child characteristics: a) direct environmental effects of parenting behavior on child behavior, free of genetic influences of the parent or child, b) passive *r*GE, suggesting that parents' genes influence both their parenting and their child's behavior, and c) evocative *r*GE, suggesting that children's genes influence both their externalizing behavior and the way they are parented.

Studies using the ECOT design have found that evocative *r*GE plays a prominent role in associations between multiple indices of parenting and adolescent internalizing and externalizing behavior (Marceau et al., 2013; Narusyte et al., 2011; Narusyte et al., 2008), although direct environmental influences were suggested for the association between paternal criticism and adolescent externalizing behavior (Narusyte et al., 2011). The present study builds on these studies to clarify the mechanisms underlying the association between parental knowledge and adolescent externalizing behavior.

Present Study

When firm causal inferences are drawn, it is often important to access very special samples. The present study used data from a sample of adolescents who are twins or siblings, and a sample of parents who are twins with adolescent offspring specifically designed to mirror the adolescent sample in order to investigate the mechanisms underlying the association between parental knowledge and adolescent externalizing behavior. We employed the ECOT approach in order to disentangle the direction of effect of the association as well as the underlying genetic and environmental influences of the association. Drawing from the two predominant theories in the literature, we hypothesized that the association between parental knowledge and adolescent externalizing behavior would be driven in part by direct environmental influences from parents to adolescents, and in part by evocative *r*GE, such that parental knowledge stems in part from adolescent disclosure and youth with heritable externalizing behavior would be less likely to disclose information on their whereabouts and activities.

Method

Participants and Procedures

The present study uses data from the US-based Nonshared Environment in Adolescent Development study (NEAD; Neiderhiser, Reiss, & Hetherington, 2007; Reiss et al., 2000), and the Swedish-based Twin and Offspring Study in Sweden (TOSS; Neiderhiser & Lichtenstein, 2008). All procedures and assessments were approved by the Institutional Review Boards (IRB) of collaborating institutions. Twins are parents in TOSS, and NEAD is a sample of adolescent twins and siblings. TOSS was designed in part to be the mirror image of NEAD, including administration of identical measures of parenting and matched adolescent ages. Previous reports have found the US and Swedish samples to be comparable on a number of key demographic and substantive variables including parental knowledge and externalizing behavior (Neiderhiser, Reiss, Lichtenstein, et al., 2007; Neiderhiser et al., 2004). These two studies have been used together in an ECOT model previously (Marceau et al., 2013).

The Nonshared Environment in Adolescence Study (NEAD)—The NEAD sample was comprised of 721 White (94%) families of twins and siblings who participated in the NEAD project (Neiderhiser, Reiss, & Hetherington, 2007). Families were predominantly recruited through a national market survey of 675,000 families, supplemented with random digit dialing of 10,000 telephone numbers throughout the United States. Zygosity was established using a validated questionnaire (> 90% agreement with genotyping) (> 90% agreement with genotyping; Goldsmith, 1991) on which adolescent twins were rated for physical similarity (Nichols & Bilbro, 1966). Three years after the initial assessment, adolescents who still resided primarily at home were invited to participate in a follow-up assessment. We used data from the 408 twin/sibling pairs who participated in the second assessment (to match the ages of adolescents in TOSS). There were 63 monozygotic twin pairs (MZ), 75 dizygotic twin pairs (DZ), and 58 full sibling pairs (FI) in non-divorced families, and 95 full sibling pairs (FS), 60 half sibling pairs (HS), and 44 genetically

unrelated sibling pairs (US) in stepfamilies who were together for at least 5 years at the time of the first assessment. Adolescents were 11 to 22 years old (M = 15.5 years; SD = 2 years). Siblings were within 4 years of age of each other (M= 1.6 years; SD = 1.3 years), and lived in the same two-parent household at least 50% of the time for at least 5 years prior to the first assessment.

The Twin and Offspring Study in Sweden (TOSS)—The TOSS sample was comprised of 909 White (100%) pairs of twin parents, their spouse/partner, and their adolescent child (Neiderhiser & Lichtenstein, 2008) obtained through the use of the Swedish Twin Registry. This study was desgined subsequent to NEAD and used comparable measures of parenting and child behavior. Zygosity was established using DNA and the same validated questionnaire as used in NEAD (Nichols & Bilbro, 1966). The analysis sample was 854 families for whom zygosity information was available (384 MZ parent pairs, 470 DZ parent pairs). Adolescents ranged in age from 11 to 22 years (M = 15.7 years, SD = 2.5 years). Adolescent cousin pairs were the same sex and within 4 years of age of each other (M = 1.8 years; SD = 1.5 years).

Measures

Parental knowledge—Parental knowledge was measured in each study by mother, father, and adolescent report using identical composite (sum) scores including the Knowledge subscale of the Child Monitoring Scale (Hetherington & Clingempeel, 1992; $\alpha > .88$ across reporters in both samples). Mother, father, and adolescent reports of mothers' and fathers' knowledge were standardized and summed to create the knowledge composites in order to avoid single-measure bias (Bank, Duncan, Patterson, & Reid, 1993; $\alpha = .68$ for TOSS, $\alpha = .70$ for NEAD) and ranked to normalize the distributions, consistent with previous reports (Neiderhiser, Reiss, Lichtenstein, et al., 2007; Neiderhiser et al., 2004).

Adolescent externalizing—Adolescent externalizing behavior was measured using a composite (sum) score of mother, father, and adolescent reported externalizing behavior on the Zill Behavior Problems Inventory (ZIL; Peterson & Zill, 1986) for NEAD and the child behavior checklist (CBCL; Achenbach, 1991) for TOSS. The ZIL was assessed in NEAD as a short-form of the CBCL. The ZIL externalizing behavior subscale is comprised of 20 items (e.g., Breaks things on purpose, deliberately destroys his or her own or other's things) on a 1 (often true) to 3 (never true) scale (reverse coded and summed) over the past three months ($\alpha > .87$ for each reporter). The CBCL externalizing subscale is comprised of 30 items (e.g., I destroy my own things) on a 1 (not true) to 3 (often true) scale (summed) over the past six months ($\alpha > .62$ in NEAD; $\alpha > .70$ in TOSS). The composite scores ranked to normalize the distributions.

Analytic Strategy

Biometric analysis of twin and sibling studies use similarities and differences between twins and siblings with varying degrees of genetic relatedness to decompose the variance in a phenotype into additive genetic (A), shared (C) and nonshared (E) environmental components. Genetic influences are indicated if the correlation of sibling/cousin 1's and sibling/cousin 2's externalizing are more similar among sibling/cousin types sharing more

genes on average than sibling/cousin types that share fewer genes on average (i.e., MZ twin correlation is twice that of DZ/full sibling correlations, and a cascade such that DZ/full siblings > HS > US, and cousin pairs whose parents are MZ twins > whose parents are DZ twins). Shared environmental influences are nongenetic influences that contribute to similarity among family members (indicated by sibling correlations of similar magnitude across the different sibling types and by US correlations > 0), whereas nonshared environmental influences are nongenetic influences that contribute to differences among family members (indicated to the extent that MZ twins' externalizing are not perfectly correlated).

ECOT model—In order to test whether the association between parental knowledge and adolescent externalizing behavior arises because of evocative *r*GE, passive *r*GE, or environmental effects free from genetic confounds, we employed the ECOT model (Figure 1). The lower box on the left of Figure 1 represents the adolescent twin and sibling sample, while the larger box on the right represents the parent twin sample. The ECOT model was run using Mx (Neale, 1999). Missing data was accommodated using full information maximum likelihood data estimation procedures. All analyses were conducted after controlling for age, sex, and age difference (for non-twin siblings in NEAD and cousin pairs in TOSS). Measurement error was estimated, but constrained to be equal across siblings, studies, and phenotypes in order to preserve model identification (ε_1 and ε_2 in Figure 1).

Along the top of Figure 1 parents' genetic and environmental influences are represented as A1 (genetic influences) and E1 (nonshared environmental influences). The influence of the shared environment on parental knowledge for twin parents was not included in the model because the intra-class correlations suggested that effects from the shared environment were negligible (see Table 1) and because the ECOT model performs better when shared environmental influences are estimated on only the child phenotype rather than on both the parent and child phenotype in samples of the current size (Narusyte et al., 2008). Based on the definition of nonshared environmental influences, the correlation for E1 for parent twin 1 and parent twin 2 was fixed at 0. Based on the information provided above regarding average proportions of segregating genes shared by different sibling types, the correlation between A1 for twin parent 1 and twin parent 2 was set to 1 for MZ twin parents and .5 for DZ twin parents. Genetic transmission from parents to adolescents was explicitly modeled using the latent factor A1'. The influence of A1 (influence of parents' genes on their own parenting) on A1' (influence of that same set of genes on adolescent externalizing behavior) was set to .5 (because children inherit half of their genes from each parent), and the influence of those genes that parents and offspring share on externalizing behavior were freely estimated (A1').

Likewise, the variance in adolescent externalizing behavior was parsed into the influence of adolescents' genes (A1', parent and adolescent shared genes, A2: unique genetic influences on externalizing), shared (C2) and nonshared (E2) environments of adolescents on their own externalizing behavior, along the bottom of Figure 1. Based on the information provided above regarding average proportions of segregating genes shared by different sibling types, the correlation between A2 for adolescent sibling 1 and adolescent sibling 2 was set to 1 for MZ twins, .5 for DZ twins and full siblings, .25 for half siblings, and 0 for genetically

unrelated step siblings. The correlation between C2 for adolescent sibling 1 and adolescent sibling 2 was set to 1, and the correlation for E2 for adolescent sibling 1 and adolescent sibling 2 was fixed at 0, based on the definitions of shared and nonshared environmental influences provided above.

Direct paths from parental knowledge to adolescent externalizing behavior (m) and from adolescent externalizing behavior to parental knowledge (n) were freely estimated. Passive rGE is indicated by the presence of genetic influences on parental knowledge in TOSS (A1), associated genetic influences on externalizing behavior in NEAD (path s), and a significant path from parental knowledge to adolescent externalizing behavior (path m). A direct environmental effect of parental knowledge on adolescents' externalizing behavior is indicated by a significant path from parental knowledge to adolescent externalizing behavior (path m) without a significant genetic association (path s). Evocative rGE is indicated by the presence of a significant path from adolescent externalizing behavior to parental knowledge (path n) in combination with genetic influences on externalizing behavior in NEAD (either from A1' or A2 to adolescent externalizing behavior). Detailed information about the specifications and power of the ECOT model can be found in Narusyte et al. (2008).

Model fitting—We fit a full model estimating all paths. 95% confidence intervals were used to determine significance of path estimates. We also used a nested model approach, commonly used to verify the findings from the full models in twin studies (Neale & Cardon, 1992) and the ECOT model (Marceau et al., 2013; Narusyte et al., 2011), where we systematically dropped paths m (parent-to-child) and n (child-to-parent) in order to verify the significance (identified by a significant decrement in model fit when the path estimate was fixed to 0). This approach was also used to verify the other paths deemed non-significant based on confidence intervals separately and as a group. Only parameter estimates from the full model are presented and interpreted.

Assumptions—We assume that environmental influences are equivalent for each sibling type, which has generally been upheld (Loehlin & Nichols, 1976; Neiderhiser et al., 2004; Reiss et al., 2000). We assume that individuals do not systematically choose their mates based on genetically influenced characteristics (assortative mating), although there is moderate evidence of this for antisocial behavior (du Fort, Boothroyd, Bland, Newman, & Kakuma, 2002). Assortative mating may inflate shared environmental influences on adolescent externalizing at the expense of genetic influences, potentially increasing the likelihood of finding passive *r*GE in the ECOT model. The inclusion of genetically-unrelated siblings in the child-based design attenuates this bias (Marceau et al., 2013). We assume there are no systematic differences in genetic and environmental influences on parenting or on adolescent externalizing across the two samples, equivalency of measurement error across phenotypes (i.e., $\varepsilon_1 = \varepsilon_2$), and that the mechanisms underlying the association under examination do not differ in the Swedish and US populations represented in TOSS and NEAD (Marceau et al., 2013; Narusyte et al., 2011; Narusyte et al., 2008).

Results

Intra-Class Correlations

Parental knowledge and adolescent externalizing were moderately negatively correlated in both samples ($r = -.34$ for NEAD, $r = -.25$ for TOSS). Parameter estimates for the full model are presented in Figure 2. There were significant influences of parents' genes and nonshared environment on parental knowledge. Path m (from parental knowledge to adolescent externalizing) was significant, but path s (from A1' to adolescent externalizing) was not, suggesting that parental knowledge exerts a direct environmental effect on adolescent externalizing. Path n (from adolescent externalizing to parental knowledge) was not significant. There were significant genetic influences on adolescent externalizing, but shared and nonshared environmental influences were not significant.

Nested model fitting results confirmed results from the full model. Path n could be dropped without a decrement in model fit but path m could not (see Table 1). Dropping each of the non-significant paths separately or together did not result in a decrement in model fit. The most parsimonious model included effects of A1, E1, m, and A2. Direct environmental influences of parental knowledge on adolescents' externalizing explained the correlation between parental knowledge and adolescent externalizing.

Discussion

We used a rigorous test of three potential mechanisms explaining the association between parental knowledge and adolescent externalizing, and found that parental knowledge reduces adolescents' externalizing via environmental mechanisms. This finding adds to the literature by demonstrating that even after accounting for both parents' and adolescents' genetic influences, knowledge is a successful parenting strategy for reducing adolescents' externalizing problems, supporting the original explanation (Dishion & McMahon, 1998; Patterson et al., 1989), not the role of evocative child effects, contrary to some reports (Crouter & Head, 2002; Fosco et al., 2013; Laird et al., 2003; Pardini et al., 2008; Willoughby & Hamza, 2011).

Combined with other studies using the ECOT design, the present findings highlight that different aspects of parenting are related to adolescent behavior for different reasons. Evocative *r*GE explained the association between maternal and paternal negativity with externalizing behavior in the same samples (Marceau et al., 2013), consistent with many, but not all (e.g., Klahr, McGue, Iacono, & Burt, 2011) genetically informed studies of parental negativity and externalizing problems. Thus, based on evidence from ECOT models, it may be that parents can exacerbate adolescent externalizing behaviors by responding with negativity, but diminish adolescent externalizing behaviors by gaining knowledge regarding their adolescents' whereabouts and activities. The ECOT design has been used previously to test specific aspects of negative parenting (i.e., over-involvement and criticism, Narusyte et al., 2011; Narusyte et al., 2008). This is the first investigation of associations between a positive parenting behavior and adolescent behavior to employ the ECOT model. Continuing to apply the ECOT model to test the mechanisms underlying associations between parenting and adolescent behavior will be important for informing intervention both

in terms of reducing negative behavior and in terms of promoting positive development by identifying optimal targets of intervention.

A recent study using the same sample of adolescent children suggested that parental knowledge was associated with substance initiation in young adulthood via two distinct mechanisms: one, a shared environmental influence common to decreased parental knowledge and deviant peer affiliation during mid-adolescence and substance initiation by early adulthood, and the other a common genetic liability such that adolescents' genetic influences contributed to the same three factors (Neiderhiser, Marceau, & Reiss, 2013). This finding suggests that multiple mechanisms may simultaneously influence how parents gain knowledge of their adolescents' whereabouts and activities, and the effectiveness of parental knowledge. Here, the association between parental knowledge and reduced externalizing was not large, so we may not have had sufficient statistical power (see below) to uncover both a direct environmental influence from parents to adolescents and a smaller evocative effect from adolescents to parents as the literature suggests (e.g., Laird et al., 2003; Pardini et al., 2008; Willoughby & Hamza, 2011).

Limitations and Future Directions

The limitations the ECOT model have been discussed previously (Marceau et al., 2013; Narusyte et al., 2011; Narusyte et al., 2008). It is important to highlight that the ECOT model probes this association at a single time, and does not account for reciprocal associations across development. In the future, the ECOT model should be extended in order to test the mechanisms underlying longitudinal associations (no samples capable of supporting a longitudinal ECOT analysis are currently available). Further, there was a wide age range among our adolescents, and therefore our results represent a mechanism operating across all of adolescence. Given evidence that *r*GE in family relationships may change with child age (Ulbricht & Neiderhiser, 2009), evocative *r*GE (i.e., child self-disclosure) may contribute more at specific stages in development, but have been washed out in the current study because of the wide age range. Relatedly, the ECOT model may not be sensitive enough to uncover bidirectional effects of very small magnitude (Narusyte et al., 2008). Although simulation analysis showed that we had adequate power to detect the effects found in this report with the current sample size (N=2498, power = .95) we were underpowered to detect bidirectional effects of lower magnitude (power = .63). Thus we urge caution in concluding that evocative effects do not play a role. Finally, the present study used samples comprised of adolescents without elevated symptoms of externalizing behavior, making the findings generalizable to normatively developing adolescents. It is possible that the association would have different underlying mechanisms among samples with clinical levels of externalizing behavior. In future studies, the ECOT model could be extended to testing the mechanisms of associations between parenting and adolescent behavior in samples with elevated levels of problems.

Conclusions

The ECOT model continues to be a powerful tool for probing the mechanisms underlying associations between parenting and adolescent behavior. Here, we conducted a very rigorous

test of the mechanisms of the association between parental knowledge and adolescent externalizing, accounting for genetic influences of parents and adolescents using the ECOT model. Our findings provide strong support for the hypothesis that parents' knowledge about their adolescents' activities and whereabouts exerts an environmental influence serving to reduce adolescent externalizing. This finding is particularly important for prevention, because it suggests that increasing parental knowledge is a viable target for interventions aiming to reduce or prevent adolescent externalizing in normative samples, before severely elevated levels of problems emerge.

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Abbreviations

ECOT	Extended Children of Twins Design
***r*GE**	Genotype-Environment Correlation
TOSS	Twin and Offspring Study in Sweden
NEAD	Nonshared Environment in Adolescent Development

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Key Points

- There is evidence both that parental monitoring is an environmental influence serving to diminish adolescent externalizing problems and that this association may be driven by adolescents' characteristics via genetic and/or environmental mechanisms, such that adolescents with fewer problems tell their parents more, and therefore appear to be better monitored.
- We used the novel, powerful ECOT design in order to understand the direction of effects (parents influencing offspring versus offspring influencing parents) and possible gene-environment correlation underlying the association between parental knowledge and adolescent externalizing using a sample of adolescent twins and a sample of parents who are twins with adolescent offspring.
- Results suggest that more parental knowledge is associated with less adolescent externalizing via a direct environmental influence independent of any genetic influences, with no child-driven effects.
- Findings have implications for prevention efforts, as they suggest that increasing parental knowledge is a viable target for interventions aiming to reduce or prevent adolescent externalizing in normative samples, before severely elevated levels of problems emerge.

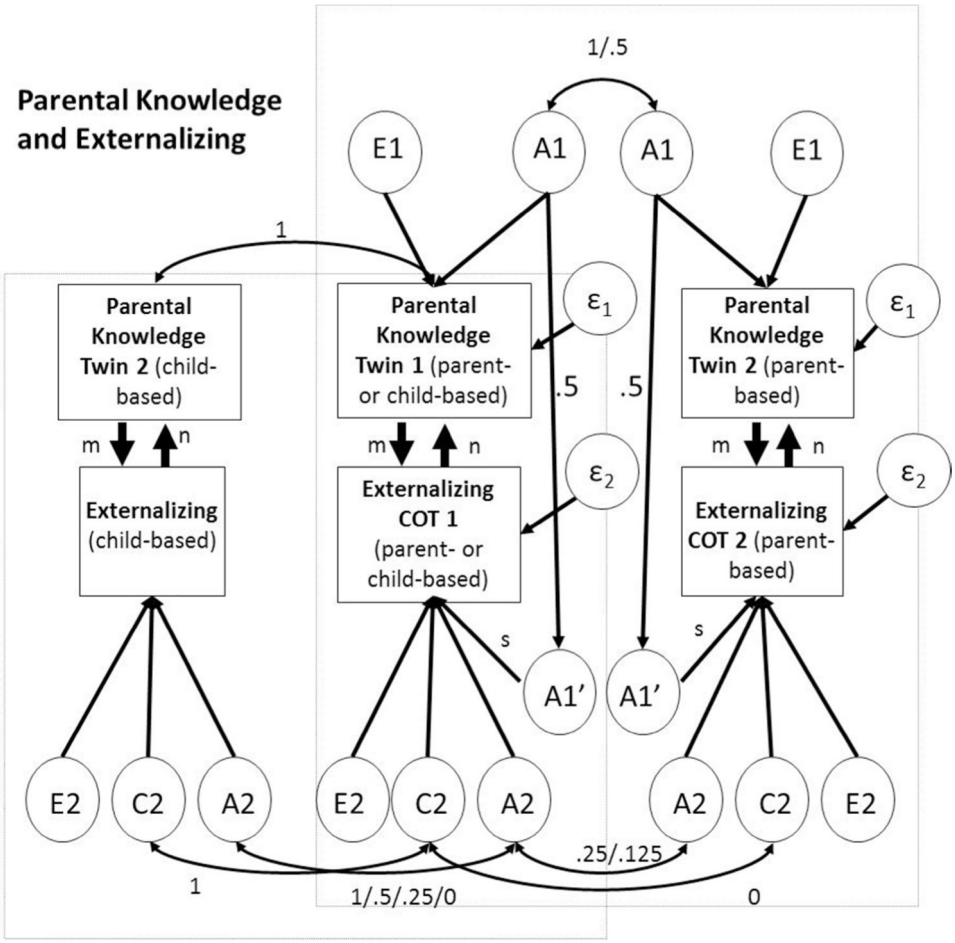

Figure 1. Extended Children of Twins Model

This is a representation of the ECOT model. The lower left-hand box represents the adolescent twin/sibling sample (NEAD). The correlation for parental knowledge for sibling 1 and sibling 2 was fixed to 1 because in NEAD the adolescent siblings share parents. The larger right-hand box represents the parent twin sample (TOSS). A1 = latent genetic influences of parents on their parental knowledge; E1 = latent nonshared environmental influences of parents on their parental knowledge; A2 = latent genetic influences of adolescents on their externalizing behavior; C2 represents latent shared environmental influences of adolescents on their externalizing behavior; E2 = latent nonshared environmental influences of adolescents on their externalizing behavior; A1' = the effect of genes shared by parents and adolescents, that contribute to parental knowledge, on adolescents' externalizing behavior. Path m = direct environmental effects of parenting on adolescents' externalizing behavior; path n = child evocative effects of adolescents' externalizing behavior on parenting; path s = the influence of shared genes of parents and adolescents; significant path s and m = passive *r*GE while significant path n and either A2 or s = evocative *r*GE. Measurement error is estimated as ε_1 and ε_2, and constrained to be equal during model fitting. Based on the information provided above regarding average proportions of segregating genes shared by different sibling types, the correlation between A1 for twin parent 1 and twin parent 2 was set to 1 for MZ twin parents and .5 for DZ twin

parents. The influence of A1 (influence of parents' genes on their own parenting) on A1' (influence of that same set of genes on adolescent externalizing behavior) was set to .5 (because children inherit half of their genes from each parent). Based on the information provided above regarding average proportions of segregating genes shared by different sibling types, the correlation between A2 for adolescent sibling 1 and adolescent sibling 2 was set to 1 for MZ twins because MZ twins share 100% of their segregating genes, .5 for DZ twins and full siblings who share on average 50% of their segregating genes, .25 for half siblings and cousin pairs whose parents are MZ twins, who share on average 25% of their segregating genes, .125 for cousin pairs whose parents are DZ twins and share on average 12.5% of their segregating genes, and 0 for genetically unrelated step siblings who share no genes systematically. The correlation between C2 for adolescent sibling 1 and adolescent sibling 2 was set to 1, and the correlations for E1 for twin parent 1 and twin parent 2 and E2 for adolescent sibling 1 and adolescent sibling 2 were fixed at 0, based on the definitions of shared and nonshared environmental influences.

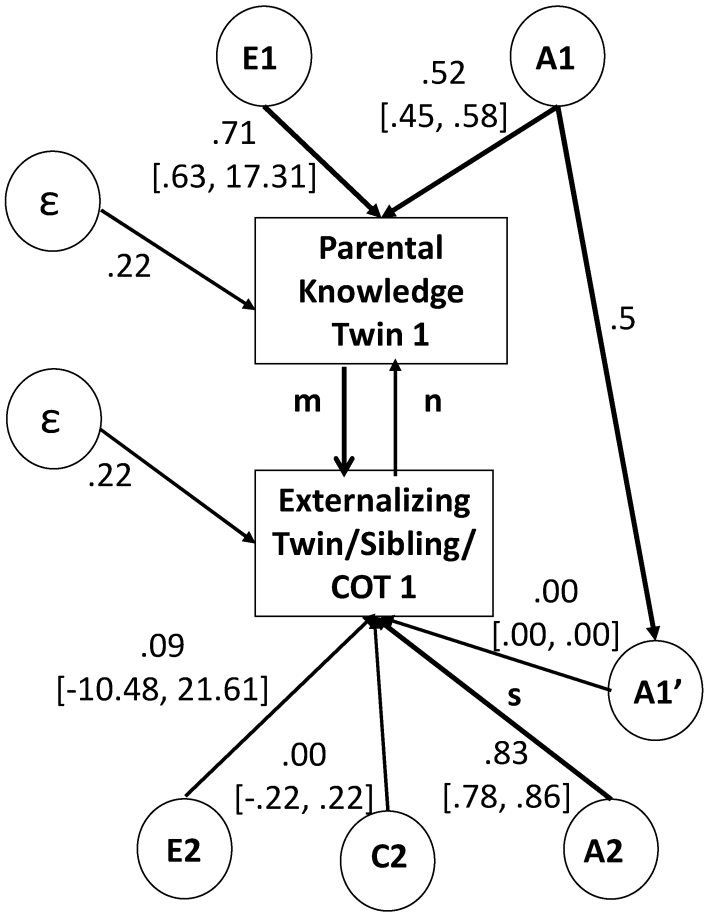

Figure 2. Results for full ECOT model

This figure is reduced from Figure 1 in order to more succinctly present results. Unstandardized path estimates and 95% confidence intervals (in brackets) are provided for each estimated path. A1 represents latent genetic influences of parents on their parenting, E1 represents latent nonshared environmental influences of parents on their parenting. A2 represents latent genetic influences of adolescents on their externalizing problems, C2 represents latent shared environmental influences of adolescents on their externalizing problems, E2 represents latent nonshared environmental influences of adolescents on their externalizing problems. A1' represents the effect of genes shared by parents and adolescents on adolescents' externalizing problems. Path m represents direct environmental effects of parenting on adolescents' externalizing problems while path n represents child evocative effects of adolescents' externalizing problems on parenting. Path s represents the influence of shared genes of parents and adolescents; significant path s and m signifies passive *r*GE while significant path n and either A2 or s signifies evocative *r*GE.

Table 1

Model fitting results

	Full Model	Child→Parent path (n)=0	Parent→Child path (m)=0	Final model
AIC	3509.5	3507.5	3676.0	3501.5
−2Lnl	13343.5	13343.5	13512.0	13343.5
df	4917	4918	4918	4921
$\Delta \chi^2$		0.0	168.5*	0.0
df difference		1	1	4

Note. The final model includes A1, E1, m, and A2. AIC = Akaike information criterion, lower scores = better fit. −2Lnl = loglikelihood function, df = degrees of freedom, $\Delta\chi^2$ = chi square change. * $p < .05$, signifies a significantly worse-fitting model.