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Non-shared Environmental Mediation of the Association between Deviant Peer Affiliation and Adolescent Externalizing Behaviors over Time: Results from a Cross-lagged Monozygotic Twin Differences Design

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Abstract

It has been argued that peers are the most important agent of adolescent socialization, and more specifically, that this socialization process occurs at the child-specific (or non-shared environmental) level (Harris, 1998; Plomin & Asbury, 2005). The present study sought to empirically evaluate this non-shared environmental peer influence hypothesis, examining the association between externalizing behaviors and deviant peer affiliation in a sample of 454 pairs of monozygotic (genetically identical) twins, assessed at ages 14 and 17, within a cross-lagged twin differences design. Results argued against a causal non-shared environmental influence of peer affiliation on the development of externalizing behaviors, and in favor of non-shared environmental "selection". In particular, the twin with more externalizing behaviors at age 14 reported increased deviant peer affiliation relative to his or her co-twin three years later, regardless of one's genetic predispositions towards externalizing behavior. Such findings suggest that adolescents with higher levels of externalizing behaviors select or shape (either intentionally or inadvertently) subsequent environmental experiences to involve increased affiliation with deviant peers. Implications are discussed.

Keywords

Deviant peer groups; Non-shared environmental influences; Twin Differences; Externalizing behaviors

In her 1998 book entitled "The Nurture Assumption", Judith Harris provocatively suggested that peers (and not parents) were the primary environmental agents of adolescent socialization. Her theory was based largely on behavioral genetic findings highlighting the very limited involvement of the shared environment (i.e., family-level environmental influences that increase similarity between siblings), as well as the prominent influence of both genetic and non-shared environmental effects (i.e., child-specific environmental influences that serve to make siblings different from one another). She specifically postulated that, because peers are not typically shared by siblings, peers were the most obvious causal source of these large non-shared environmental influences (Harris, 1998).

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This theory initially spawned a great deal of criticism (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Vandell, 2000), though chiefly in defense of the role of parents in offspring socialization. Indeed, subsequent research has generally not supported the contention that parenting does not matter. For example, behavioral genetic research is now suggesting that, contrary to initial hypotheses, shared environmental influences are often moderate and significant prior to adulthood (Burt, in press-b; Burt, McGue, Krueger, & Iacono, 2007). Moreover, the parent-child relationship appears to have a causal and longitudinally predictive association with child and adolescent outcomes, even when considering/controlling for genetic influences (Burt, McGue, Iacono, & Krueger, 2006; Burt, McGue, Krueger, & Iacono, 2005; Dishion, Andrews, Kavanagh, & Soberman, 1996; Forgatch & DeGarmo, 1999).

The importance of peers to adolescent development, however, has been less contentious, and indeed, theorists have continued to build on this argument. For example, when discussing the modest progress made in identifying non-shared environmental sources of variance, Plomin & Asbury (2005) noted that, “it seems reasonable that experiences outside of the family, with peers and individual life events, for example, might prove to be richer sources of nonshared environment” (pg. 90; Plomin & Asbury, 2005). Given this continued attention, it is quite surprising that the core proposition of the peer socialization theory, that peers are a causal non-shared environmental influence on child and adolescent outcomes, remains essentially unexamined to date.

To be sure, a number of studies have suggested that peer affiliation itself is largely non-shared environmental in origin (though not all; see (Pike, Manke, Reiss, & Plomin, 2000). Iervolino and colleagues (2002) found that self-reported adolescent peer group delinquency was significantly influenced only by the non-shared environment across both a large sample of twins and other sibling pairs (accounting for 77% of the total variance) and a sample of same-sex adoptive and biological siblings (accounting for 73% of the total variance) (Iervolino et al., 2002). Importantly, these findings of very strong non-shared environmental origins to peer deviance were later replicated using teacher informant-reports of peer deviance and observer-ratings of deviant friendship process (non-shared environmental influences accounted for 74–97% of the total variance) (Bullock, Deater-Deckard, & Leve, 2006).

Such findings are at least consistent with the premise of non-shared environmental peer socialization, in that they confirm that peer group affiliations are, as expected, largely non-shared environmental in origin. However, we would further expect the associations of deviant peer affiliation with various outcomes to be non-shared environmental in origin. Leve (2001) conducted a methodologically sophisticated twin study to address this very question, in which observers rated videotapes of dyadic twin-friend interactions (each twin participated with a different friend). She found that friends do account for non-shared environmental variance in twins’ externalizing behaviors (Leve, 2001). Other studies have reported similar findings (Loehlin, 1997).

Critically, however, any conclusions regarding the non-shared environmental role of peers must remain speculative, as direction of causation has yet to be examined. As noted in other studies of the non-shared environment (Asbury, Dunn, Pike, & Plomin, 2003; Burt et al., 2006; Caspi et al., 2004), most studies of non-shared environmental effects typically interpret their findings to be causal, such that differential exposure to the non-shared environmental factor causes differences in child outcomes. Such conclusions are premature, however, particularly as deviant peer affiliation and externalizing behaviors appear to be bidirectional. Numerous studies have suggested that individuals with delinquent tendencies selectively affiliate with others like themselves, while other studies have suggested that exposure to deviant peers may augment these preexisting tendencies (Deater-Deckard, 2001; Dishon, McCord, &

Poulin, 1999). Reciprocity therefore remains a possibility for non-shared environmental effects as well. For example, it may be that adolescents with high levels of externalizing behaviors environmentally select or shape (either inadvertently or intentionally) their subsequent experiences to involve increased affiliation with deviant peers. We thus cannot be sure whether these non-shared environmental effects causally shape the development of adolescent externalizing behaviors.

To date, we know of only one study that has examined the respective influences of both "non-shared environmental causation" and "non-shared environmental selection" on the association between deviant peer affiliation and adolescent externalizing behaviors (though their primary focus was on genetic and shared environmental contributions to their developmental overlap) (Kendler, Jacobson, Myers, & Eaves, 2007). They examined retrospectively-reported Conduct Disorder and deviant peer affiliation at ages 8–11, 12–14, and 15–17 in 373 adult male twin pairs. They found that the non-shared environmental influences on Conduct Disorder and deviant peer affiliation were almost entirely age-specific. However, the small cross-lagged correlations that were present went from Conduct Disorder to deviant peer affiliation (and not the reverse), results which appear to support the "non-shared environmental selection" hypothesis.

Though provocative, this study suffered from some key limitations in regards to clarifying the "non-shared environmental causation" hypothesis put forward by Harris (1998) and Plomin & Asbury (2005). First and foremost, their data were collected from adults (aged 24 to 62 years) using retrospective life history calendars. Though this is the preferred way of collecting retrospective data, such data are not fully free from retrospective recall bias. Second, because their main focus was on understanding genetic and shared environmental contributions to the developmental relationship between Conduct Disorder and deviant peer affiliation, their sample contained both monozygotic (MZ; identical) and dizygotic (DZ; fraternal) twins without regard to peer overlap. This is problematic when studying peers as non-shared environmental influences because, as noted by Kendler et al. (2007), MZ twins are more likely to share peer groups than are DZ twins (and non-shared environmental effects do not vary by zygosity by definition). Moreover, when studying the impact of the non-shared environment in particular it makes more sense to focus on environmental experiences that are explicitly not shared by the twins. Given this, a prospective longitudinal study, with a particular focus on twins who do not share all of their friends, is needed.

The current study sought to fill this gap in the literature, evaluating differential externalizing behaviors and differential affiliation with deviant peers within a prospective, longitudinal, cross-lagged, "MZ twin differences" design (Burt et al., 2006). The MZ twin differences design capitalizes on the unique features of MZ twins, specifically that they share 100% of their genes and 100% of their shared environment. As differences between MZ twins accordingly cannot be confounded by these factors, this design allows researchers to directly evaluate whether experiential differences are *environmentally* linked to child-specific behavior problems. To specifically consider issues regarding the direction of causation, we took advantage of this design within a longitudinal cross-lagged framework (Burt et al., 2006), thereby allowing us to examine the direction of causation between sibling differences in deviant peer affiliation and externalizing behaviors over time, irrespective of their pre-existing relationships and their stabilities over time. Analyses were conducted first on the full sample, and were then repeated for those twins that did not share all or nearly all of their friends, thereby allowing us to specifically examine environmental experiences that differed (at least to some extent) across the twins. In this way, we were able to evaluate how differential deviant peer affiliation and adolescent externalizing behaviors influenced each other over time at a solely non-shared environmental level.

METHOD

SAMPLE

The sample was drawn from participants in the ongoing Minnesota Twin Family Study (MTFS). The MTFS is a population-based, longitudinal study of same-sex adolescent twins born in the state of Minnesota and their parents. More than 90% of twin births between 1971 and 1985 were located using public databases. Among twins who met study eligibility criteria (i.e., neither of the twins had a cognitive or physical handicap that would preclude completing the MTFS day-long, in-person assessment and the family lived less than one day's drive from our Minneapolis laboratory), 83% agreed to participate. The participating families were broadly representative of the Minnesota population at the time the twins were born; approximately 98% are Caucasian. Analysis of more than 80% of the non-participating families via a brief mail or telephone survey revealed that parents in participating families had slightly, albeit significantly more education (0.25 years) than parents in non-participating families. In socioeconomic status and self-reported mental health problems, however, there were no significant differences between participating and non-participating families. Further information regarding the design, recruitment procedures, and participation rates of the MTFS can be obtained elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999). In the current study, the overall sample consisted of 908 (470 male and 438 female) same-gender, reared-together MZ twins (454 pairs) assessed at age 14 (on average; the range was 13 to 16 years of age). Of these, 406 (86%) of the boys and 400 (91%) of the girls also completed an assessment at age 17 (on average; the range was 16 to 18 years of age).

ZYGOSITY DETERMINATION

Zygosity of the twins was determined by the agreement of several separate estimates: 1) Ponderal and cephalic indices and fingerprint ridge counts were measured; 2) MTFS staff evaluated visage, hair color, and face and ear shape for physical similarity; and 3) Parents completed a standard zygosity questionnaire. When these estimates did not agree, a serological analysis was performed to determine zygosity. A previous validation study (n=50 pairs) found that when the three estimates agreed, the indicated zygosity was uniformly confirmed by the serological analysis, suggesting that our method of zygosity determination is accurate.

ASSESSMENT OF EXTERNALIZING BEHAVIORS

We made use of two different indices of adolescent externalizing behaviors. The first was a self-reported questionnaire, the Delinquent Behavior Index (DBI). The DBI (Burt & Donnellan, 2008; Burt et al., 2007; Farrington & West, 1971; Gibson, 1967) is a 36-item inventory of minor (e.g., skipping school) and more serious (e.g., using a weapon in a fight) delinquent behaviors. Participants were asked whether they had engaged in each behavior "during adolescence" (1=no; 2=yes). Items were summed such that higher scores reflect endorsement of more delinquent behaviors. If fewer than four items were missing, items were prorated and added to the scale score. The scale demonstrated very good internal consistency reliabilities, with alphas of .90 and .91, respectively, at ages 14 and 17.

We also made use of an externalizing symptom count variable corresponding to the sum of endorsed criterion A symptoms for Conduct Disorder and Oppositional Defiant Disorder over the last three years. Participants were assessed for DSM-III-R mental disorders (DSM-III-R was current at the study's onset) by trained bachelor and masters-level interviewers. Symptoms were assessed using the Diagnostic Interview for Children and Adolescents-Revised (DICA-R) (Reich & Welner, 1988). The MTFS version of this instrument contained supplementary probes and questions, added after consultation with one of the DICA-R's authors, to ensure complete coverage of each symptom. Twins were interviewed by separate interviewers. Following the interview, a clinical case conference was held in which the evidence for every

symptom was discussed by at least two advanced clinical psychology doctoral students. Only symptoms that were judged to be clinically significant in both severity and frequency were considered present. As actual diagnoses were not used, duration rules were excluded for both disorders. The Conduct Disorder and Oppositional Defiant Disorder symptom counts were then summed to create an overall measure of externalizing symptoms (referred to as EXT hereafter). To adjust for positive skew, EXT was log-transformed prior to analysis.

To create the externalizing difference scores used the final analyses, we subtracted the symptom count/score of the second born twin from that of the first born twin. Accordingly, at any given assessment, we only included data if it was present on both members of the twin pair. Of the 454 pairs included in the current study, DBI data was available on 422 pairs at age 14 and 329 pairs at age 17. EXT data was available on 444 pairs at age 14 and 389 pairs at age 17.

Importantly, prior MTFS work has demonstrated that those who did not complete the follow-up assessment did not have more externalizing psychopathology than those who did complete follow-up (McGue, Elkins, Walden, & Iacono, 2005). Comparisons of MZ difference scores across those with and without follow-up data in the current sample fully supported those findings: the DBI (Cohen's d =.04, p =.69), EXT (Cohen's d = −.03, p =.75), and deviant peer affiliation (Cohen's d =.01, p =.92). We also ran a multivariate logistic regression model of participation. As before, participation at follow-up was not predicted by any of the MZ difference score variables (all p >.55).

ASSESSMENT OF DEVIANT PEER AFFILIATION

Twins reported on their peer group affiliation using the Friends Inventory, a measure of peer characteristics developed by MTFS staff and administered by computer to the present sample (Walden, McGue, Iacono, Burt, & Elkins, 2004). Twins were instructed to provide ratings for their entire peer group, with items scored using a 4-choice response format (1 = *none of my friends are like that*, 2 = *just a few of my friends are like that*, 3 = *most of my friends are like that*, and 4 = *all of my friends are like that*). Item ratings were summed to yield a score indexing deviant peer affiliation (9 items: e.g., *my friends know where to buy drugs; my friends break the rules*). If one item was missing, that item was prorated and added to the scale score. If two or more items were missing, the scale score was coded as missing. The deviant peer affiliation scale demonstrated good internal consistency reliability across both assessments (α's were .88 and .90 at ages 14 and 17, respectively). To create the signed difference scores used the final analyses, we again subtracted the score of twin B from that of twin A. Accordingly, at any given assessment, we only included peer affiliation data if it was present on both members of the twin pair. Peer affiliation data were available on 417 pairs at age 14 and 332 pairs at age 17.

One additional item, also administered as part of the Friends scale, was used to determine the extent to which the siblings' peer groups overlapped. Twins were asked "How many of your friends do you and your twin share?", with items scored using a forced choice format (i.e., 1 = *all or nearly all of our friends*, 2 = *many but not all of our friends*, 3 = *a few of our friends*, 4 = *none of our friends*). This item showed good cross-informant, within-pair reliability: twin sibling agreement was relatively high at both ages (.53–.56, p<.001). Consistent with the observation that MZ twins tend to share friends, 62% of twins reported that they shared "all or nearly all" of their friends at age 14 and/or age 17. Of those twins who consistently reported that did not share all of their friends (i.e., those who denied sharing all of their friends at 14 and then again at 17; 38% of the sample), most (76%) reported that they shared "many but not all" of their friends, 23% reported that they shared "a few" friends, and 2% reported that they did not share any friends.

As one would expect, sharing friends moderated the degree of self-reported similarity between the twins' friends. Those twin pairs who shared all or nearly all of their friends were correlated .87 in their deviant peer affiliation, while those who shared many, a few, or none of their friends were correlated .73, .53, and .39 in their deviant peer affiliation, respectively. Moreover, those 62% of twins that shared all or nearly all of their friends reported more similar levels of deviant peer affiliation than did the 38% of twins who shared fewer friends. Specifically, at age 14, mean sibling differences in deviant peer affiliation were 2.28 for those twins who shared all of the friends versus 2.84 for those twins who did not share all of their friends (p =.021, Cohen's d =.23). At age 17, mean sibling differences in deviant peer affiliation were 2.12 for those twins who shared all of the friends versus 2.98 for those twins who did not share all of their friends (p =.002, Cohen's d =.40). Such results suggest that twins that share all or nearly all of their friends appear to be experiencing more similar levels of deviant peer affiliation than are twins who share fewer friends.

STATISTICAL ANALYSES

Monozygotic (MZ) or identical twins share 100% of their genetic material as well as those family-wide environmental forces that act to further increase their similarity. Accordingly, differences between these reared-together MZ twins are due only to child-specific or unique environmental influences (and measurement error). This rationale can be understood using the following logic (as presented in Rabe-Hesketh, Skrondal, & Gjessing, 2008): the parametric form for phenotype (y) for the jth twin (j=1,2) in the ith twin pair (i=1,…N) can be given by:

$$y_{ij}=\mu+aA_{ij}+dD_{ij}+cC_{ij}+eE_{ij}$$

where μ is the population mean; A, D, C and E correspond, respectively, to the additive genetic, dominant genetic, shared environmental, and non-shared environmental deviations, all normed to unit variance; and a, d, c and e are the corresponding path coefficients. Under the standard assumption that the deviations are uncorrelated, this model corresponds to the standard variance component model used in biometrical analysis:

$$\sigma_y^2=\sigma_a^2+\sigma_d^2+\sigma_c^2+\sigma_e^2$$

where σ_a^2, σ_d^2, σ_c^2, and $\sigma_e^2$are, respectively, the additive genetic, dominant genetic, shared environmental, and non-shared environmental variance components, and the effects due to measurement error are included in the non-shared component of variance.

When applied to phenotypic differences between the two members of a twin pair (j = 1,2; ordering is arbitrary but does not affect results), the model can be represented by:

$$(y_{i1}-y_{i2})=a(A_{i1}-A_{i2})+d(D_{i1}-D_{i2})+c(C_{i1}-C_{i2})+e(E_{i1}-E_{i2})$$

For MZ twin pairs, $A_{i1} = A_{i2}$ and $D_{i1} = D_{i2}$ because MZ twins are genetically identical and $C_{i1} = C_{i2}$ for reared together twins. Consequently, MZ twin phenotypic differences exclusively reflect differences in their non-shared environments (plus measurement error). In other words, the MZ sibling differences approach functions as a "direct estimate" of non-shared environmental influences.

The MZ differences cross-lagged model is presented in Figure 1. The cross-age but within-trait coefficients (i.e., b_{11}, b_{22}) index the stability of differences in externalizing behaviors and deviant peer affiliation, respectively, over time. We were also able to evaluate the relationship

between differential deviant peer affiliation and differences in externalizing behaviors at each age via age-specific correlations (i.e., r_1, r_2). Finally, the cross-lagged coefficients (i.e., b_{12}, b_{21}) allowed us to determine whether differences in deviant peer affiliation and externalizing behaviors at age 14 *independently* impacted the other at age 17, controlling for the stability of differences in each trait over time. Written in the form of two linear equations, where the dependent variable Extern_17 represents differential externalizing at age 17, the dependent variable Peer_17 represents differential deviant peer affiliation at age 17, and the independent variables, Peer_14 and Extern_14, respectively represent differential deviant peer affiliation and differential externalizing at age 14, this model can be summarized as:

$$\text{Extern\_17} = b_{11}{}^{*}\text{Extern\_14} + b_{21}{}^{*}\text{Peer\_14} + e_1$$
$$\text{Peer\_17} = b_{22}{}^{*}\text{Peer\_14} + b_{12}{}^{*}\text{Extern\_14} + e_2$$

As a reminder, because our analyses are based on MZ twin differences, any path or correlation greater than zero is explicitly indicative of non-shared environmental influence. As noted, models were run on signed sibling differences (i.e., twin A – twin B; difference scores could thus be positively or negative signed). As a result, we were able to meaningfully interpret the signs of our effects (e.g., a positive cross-lagged coefficient would suggest that the twin with more of one trait evidenced more of the other, etc.)

For each measure of externalizing, model-fitting analyses were conducted first on the full sample. They were then repeated for those twins who consistently reported having (at least some) different friends during both assessments (i.e., 38% of the full sample). Should the cross-lagged effects be particularly pronounced in the sub-sample of twins who did not share all of their friends, it would imply (albeit indirectly) that our results reflect actual exposure to different peers rather than different perceptions of the same peers. Of note, whether or not twins share their friends is not a random event; indeed, the peer selection process is itself very likely related to the outcomes in question. Importantly, however, any such differences within MZ twin pairs cannot, by definition, be genetic or shared environmental in origin (i.e., regardless of whether or not their share friends, MZ twins in our data are genetically-identical and grew up in the same household). In other words, the peer selection process itself must also be non-shared environmental in origin *in these data*, and thus relevant to our study.

To address the missing difference score data, we made use of Full-Information Maximum-Likelihood (FIML) raw data techniques, which produce less biased and more efficient and consistent estimates than other techniques, such as pairwise or listwise deletion, in the face of missing data (Little & Rubin, 1987). FIML assumes that data are MAR and are thus ignorable. AMOS, a structural-equation modeling program (Arbuckle, 2003), was used to fit cross-lagged models to the observed difference score data.

RESULTS

In order to index the severity of externalizing behaviors over time, mean EXT and DBI scores were computed separately by age (see Table 1). Paired-samples t-tests indicated that mean externalizing behaviors increased significantly with age ($p<.01$), as did deviant peer affiliation ($p<.01$). Collectively, the tabled statistics indicate that there is growth in externalizing behaviors and affiliation with deviant peers from mid- to late-adolescence. Furthermore, average sibling difference scores in the DBI increased from ages 14 to 17. (Please note that the absolute values of the difference scores are presented to highlight the magnitude of sibling differences as signed sibling differences are typically centered at zero. However, to permit examination of the direction of effects, all subsequent analyses were conducted on signed

sibling differences). However, there were no appreciable changes in absolute sibling difference scores with age for either EXT or deviant peer affiliation.

CORRELATIONS

Correlations were computed between differential deviant peer affiliation and differential externalizing behaviors across ages 14 and 17 (see Table 2). As a reminder, non-significant correlations are indicative only of a lack of non-shared environmental influence, rather than a lack of association between those phenotypes in general (indeed, at the phenotypic level, deviant peer affiliation and externalizing behaviors were correlated between .44 and .73 across all ages and measures (all $p<.01$)). There was evidence of non-shared environmental influences on within-trait stability for externalizing behaviors, with correlations ranging from .14 to .39. There was also evidence of moderate non-shared environmental influences on within-trait stability for deviant peer affiliation ($r = .26$ and .32). The within-age, cross-trait associations were strongly suggestive of non-shared environmental mediation at both ages (r's ranged from .22 to .68). The cross-age, cross-trait associations were also generally significant and moderate in magnitude. Specifically, differences in deviant peer affiliation and externalizing behaviors at age 14 each predicted the other at age 17, though the relationship between differences in externalizing behaviors at 14 and differences in deviant peer affiliation at 17 seemed slightly stronger and was more consistently greater than zero.

MULTIVARIATE MODELING

As it is well-known that male adolescents display a higher frequency of externalizing behaviors than do female adolescents, we first examined the moderating effects of gender. The cross-lagged model was fit both allowing for differences in parameter estimates across gender and constraining the parameter estimates to be equal across gender. Associations were equivalent across gender across all measures of externalizing behaviors, in both the full sample and the sub-sample of twins who did not share all or nearly all of their friends ($\Delta\chi^2 \leq 3.1$ on 4 df, $p \geq .54$). The uniformly improved fit of the no-gender-differences model suggests that although males are more symptomatic than females, the non-shared environmental relationship between differential deviant peer affiliation and externalizing behaviors does not vary across gender. Accordingly, final model-fitting analyses were conducted irrespective of gender.

Table 3 presents the standardized parameter estimates for the associations between differential deviant peer affiliation and differential externalizing behaviors separately for the full sample and the sub-sample of pairs who did not share all or nearly all of their friends, respectively. The percentage of variance accounted for by a given path can be obtained by simply squaring the path coefficient. Sibling differences in externalizing at ages 14 and 17 (i.e., b11) evidenced significant stability over time, predicting between 1.7% and 21.1% of the variance three years later. Sibling differences in deviant peer affiliation (i.e., b22) also evidenced some stability, predicting between 4.0% and 8.4% of the variance at age 17. In no case, however, did sibling differences in deviant peer affiliation at 14 predict sibling differences in externalizing behaviors at age 17 (the proportion of variance accounted for was uniformly less than 1%). Instead, differential externalizing behaviors at age 14 consistently predicted differential deviant peer affiliation at age 17, accounting for somewhere between 1.2% and 5.8% of the variance across all analyses. Moreover, this effect appeared to be particularly pronounced in those twins that did not share all of their friends (1.2–2% in the full sample versus 5.3–5.8% in the sub-sample that did not share all of their friends).[1] Such results suggest that, across multiple measures of

[1]As a final check on our results, we re-ran the model for those twins who reportedly shared all or nearly all of their friends (62% of the sample). The cross-age stability of any observed sibling differences (i.e., paths b11 and b22) generally remained significant (estimates ranged from .09 to .21). Critically, however, b12 was no longer significant; it was estimated to be .03 for EXT ($p = .64$) and .06 for the DBI ($p = .43$). Given these results, we conclude that differential externalizing behaviors at age 14 appear to predict differential deviant peer affiliation only in those twins that do not share all or nearly all of their friends.

externalizing, the twin with more externalizing behaviors at age 14 reported having more deviant peers three years later.

DISCUSSION

The aim of the present study was to explicitly evaluate whether and how child-specific environmental processes impacted the association between adolescent externalizing behaviors and deviant peer affiliation from mid- to late-adolescence. To do so, we examined MZ-twin differences in deviant peer affiliation and externalizing behavior from ages 14 to 17 within a cross-lagged model. Analyses revealed moderate-to-strong cross-sectional associations between differential deviant peer affiliation and differential externalizing behaviors. However, the cross-lagged analyses revealed little evidence in support of the non-shared environmental causation hypothesis, as the prediction of differential externalizing behaviors at 17 by differential deviant peer affiliation at 14 was uniformly quite small (i.e., less than 1% of the variance) and non-significant. By contrast, there was ample support for the non-shared environmental selection hypothesis. The twin with more externalizing behaviors at age 14 consistently reported increased deviant peer affiliation relative to his or her co-twin three years later, accounting for as much as 5.8% of the variance in differential deviant peer affiliation at age 17. Moreover, the effect was largely specific to those twins who did not share all or nearly all of their friends, thereby implying (at least indirectly) that the effect may reflect actual exposure to different peers rather than different perceptions of the same peers. Importantly, these results persisted across both measures of externalizing behaviors, suggesting that the associations are robust to method considerations. Such findings are thus thought to strongly suggest that different levels of adolescent misbehavior at age 14 exert an environmentally-mediated influence on deviant peer affiliation at age 17, *even when controlling for the adolescents' genetically-influenced characteristics.*

Critically, our results are fully consistent with those of Kendler and colleagues (2007). Both studies suggested that the non-shared environmental association between externalizing behaviors and deviant peer affiliation was largely age-specific. However, to the extent that these non-shared environmental effects persisted over time, both studies supported the non-shared environmental selection hypothesis rather than the non-shared environmental causation hypothesis. Furthermore, the magnitude of our non-shared environmental effects (1–5%) is notably consistent with those of other studies of the non-shared environment (Asbury et al., 2003; Turkheimer & Waldron, 2000).

Even so, there are limitations to the present study. First, our measure of peer affiliation was not based on sociometric data or teacher informant-reports, but rather on adolescent self-reports of his or her peer group's general characteristics. It thus remains unclear how these results would have changed with the use of other measures of peer affiliation, or the use of multiple informants (so as to avoid the well-known issues of shared method variance that complicate interpretation of all self-report data). Even so, it is noteworthy that teacher informant-reports of deviant peer affiliation and observer ratings of videotaped interactions between each twin and his or her closest friends also appear to be largely non-shared environmental in origin (74–97% of the total variance) (Bullock et al., 2006). Furthermore, it remains uncertain whether our results reflect exposure to differentially deviant peers or differential perceptions of the same peers, as we have no way to confirm the twins' self-reports of peer similarity. It is similarly unclear how the twins interpreted the instruction to rate their "entire peer group". That said, results were essentially specific to those twins who reported that they did not share all or nearly all of their peers, thereby circumstantially implying that the observed association reflects differential exposure. However, future research should seek to confirm this finding using more nuanced measures of peer affiliation.

Second, although the reporting periods for EXT did not overlap across assessments, it is unclear whether this also applies to the DBI. In particular, participants were asked the report on DBI behaviors "during adolescence". It is thus possible that participants were reporting on some of the same actions at both assessments. That said, mean levels of the DBI did increase over time, suggesting that not all behaviors overlap across the two assessments. Moreover, the DBI results are fully consistent with those for EXT. We thus conclude that our core findings are unlikely to be a function of overlapping reporting periods.

Third, the origins of the differences in externalizing behaviors observed between MZ twins at age 14 were not explored here. As noted, these differences must also be non-shared environmental in origin (since MZ twins are genetically identical, differences between them must be environmental in origin). Consistent with this, prior analyses in these data have suggested that they are, at least in part, a function of differential parent-child relationships (Burt et al., 2006). Future research should examine other childhood precursors of discordant MZ twin pairs, with a focus on environmental experiences that are likely to differ between twins (e.g., obstetrical complications). Fourth, it is worth noting that, although it is one of the more useful correlational designs for testing the direction of causation between variables, the cross-lagged model employed here does not entirely overcome the limits of correlational data when trying to determine causality. Experimental data allows for much stronger statements regarding causality.

Next, though our results center on non-shared environmental underpinnings to social selection, they do not exclude additional genetic (or shared environmental) influences on this process (Burt, 2008; Kendler et al., 2007). Indeed, Burt (2008) found evidence of a particular gene underlying experimentally-derived estimates of popularity in late adolescence, an evocative rGE association that appears to be partially mediated via rule-breaking behaviors (Burt, in press-a). Similarly, Kendler et al. (2007) found that genetic influences on Conduct Disorder partially drove the subsequent selection of deviant peer groups.

In addition, the current results apply only to mid- to late-adolescence and not to other developmental periods. Adolescents, by virtue of their increasing independence, likely have more choice in their friends than do younger children. Moreover, adolescence is a developmental period that is characterized by the normalization of antisocial and externalizing behaviors (Moffitt, 1993), a social norm that is less applicable in either childhood or adulthood. It thus remains unclear whether these findings would generalize to earlier or later developmental stages. Finally, genetically-identical siblings share friends more often than do same-sex dizygotic twins or full siblings (Walden et al., 2004). Indeed, in these data, just over half of the available MZ pairs (62%) indicated that they shared all or nearly all of their friends at one or both assessments. Accordingly, although the examination of differential peer group affiliation within MZ pairs offers researchers the opportunity to directly control for genetic confounds when studying non-shared environmental processes, the relatively limited differences between MZ twins' peer groups may be impacting our results. Similarly, although length of time between assessments varied somewhat across participants (the range was 2–5 years, with an average of 3 years), our analyses did not consider this effect. That said, there is relatively little evidence that our conclusions varied with the length of the lag; indeed, repeating our analyses separately for those with longer versus those with shorter lags between assessments (dividing at the 50% mark) did not alter our core conclusions in any way (i.e., differential deviant peer affiliation did not predict differential externalizing behaviors over time, whereas differential externalizing behaviors continued to predict differential deviant peer affiliation). We thus conclude that our core findings are unlikely to be a function of the lag between assessments.

Despite these limitations, the results of the current study have several important implications. First, the current findings suggest that peer "selection" is not solely a genetically-mediated (or active/evocative rGE) process. Instead, our results suggest that externalizing behaviors lead to increased deviant peer affiliation via non-shared environmental mechanisms. In other words, engaging in these deviant behaviors somehow shapes subsequent environmental experiences to involve increased affiliation with deviant peers. This could take the form of a true selection process, such that individuals engaging in these behaviors subsequently select peers engaging in similar behaviors. Alternately, it could take the form of an evocative process, such that those engaging in externalizing behaviors make themselves more appealing to peers engaging in similar behaviors (and/or progressively restrict their social options in regards to more prosocial peers). The latter is generally consistent with Moffitt's concept of "snares" (Moffitt, 2003), in which adolescent misbehavior sometimes results in consequences that make it difficult to desist from these behaviors and transition to a more conventional adult lifestyle (although she was generally referring to more damaging snares, such as conviction of a crime or teenage pregnancy). Future research should seek to understand how adolescent behavior shapes his or her subsequent environmental experiences.

Second, predictions of differential externalizing behaviors at age 17 by differential deviant peer affiliation at age 14 were uniformly small (less than 1% of the variance) and non-significant. By contrast, the twin with more externalizing behaviors at age 14 reported significantly increased deviant peer affiliation relative to his or her co-twin three years later. Such findings clearly run contrary to the hypothesis that peers are the primary (or at least a critically important) causal source of non-shared environmental influences on developmental outcomes. Indeed, rather than being a non-shared environmental cause of adolescent externalizing behaviors, deviant peer affiliation appears to be a non-shared environmental consequence of these behaviors.

This latter point has key implications for the field of behavioral genetics. The non-shared environment is generally viewed as the preeminent environmental influence within the field (Turkheimer, 2000), a view that has held (Plomin & Asbury, 2005) despite the fact that the many existing studies of the non-shared environment have failed to account for much of its variance (see Turkheimer & Waldron, 2000). In response to these (largely null) findings and the provocative hypothesis of Harris (1998), some researchers have postulated that extrafamilial experiences, including (and perhaps especially) peers, are particularly salient to developmental outcomes at a non-shared environmental level (Plomin & Asbury, 2005). Unfortunately, this source of non-shared environmental variance also does not behave as expected.

One interpretation of such findings is that the non-shared environment should be reconceptualized as a function of idiosyncratic and/or transient environmental influences (Turkheimer & Waldron, 2000). Though this sort of reconceptualiztion would diminish the hypothesized importance of the non-shared environment (Plomin & Daniels, 1987), it does not follow that temporally-specific (i.e., within-age, cross-trait) associations are necessarily meaningless. Rather, they may play a role in transiently shaping the adolescent's experience and behaviors at a given age (Turkheimer & Waldron, 2000), experiences that may compound over time to substantively alter individual outcomes (Plomin & Asbury, 2005). This latter possibility is consistent with the strong within-age, cross-trait correlations observed in these data. Future twin research should make use of experimentally-based designs with real-time, microanalytic observer-ratings of dyadic peer interactions (building, for example, on the work of Dishion et al., 1999, and the work of Leve, 2001) to further evaluate this hypothesis.

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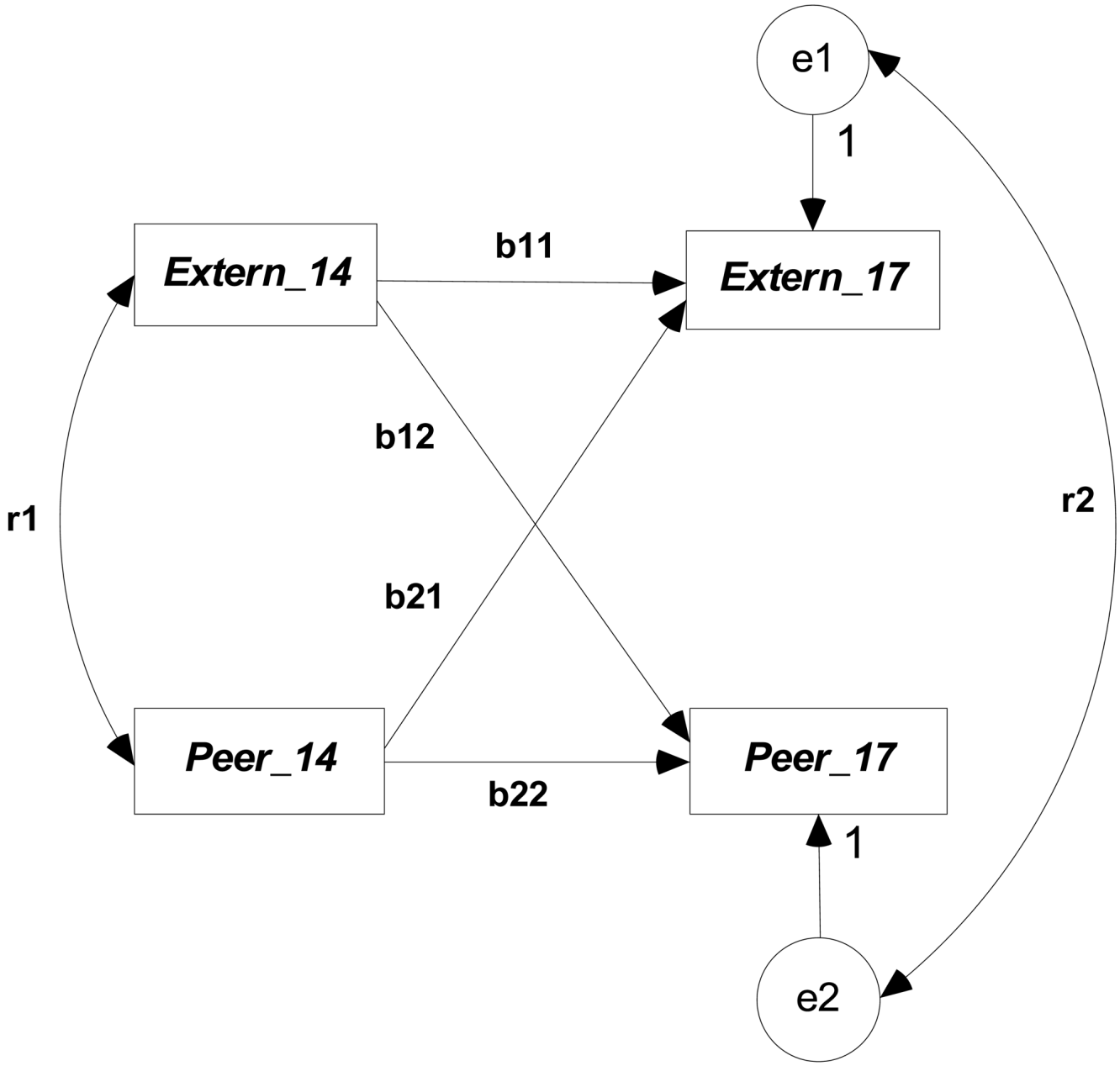

Figure 1.

Cross-lagged model of sibling difference scores in externalizing behaviors (Extern) and deviant peer affiliation (Peer) across mid- to late-adolescence.

Note. Cross-age paths (i.e., partial regression coefficients) are indicated by a “b” followed by 2 numerals. Within-age correlations are indicated by an “r” followed by a single numeral. The residual variance in differential deviant peer affiliation and differential externalizing at age 17 are represented by an “e” followed by a single numeral.

Table 1

Mean level of externalizing and deviant peer affiliation scores at ages 14 and 17, and sibling differences in those scores.

	Individual scores				Absolute sibling difference scores			
	Mean	**SD**	**Min**	**Max**	**Mean**	**SD**	**Min**	**Max**
DBI_14	4.87**	5.29	0	32	2.89**	3.23	0	24
DBI_17	6.06**	5.90	0	33	3.29**	3.46	0	23
EXT_14	1.63*	2.33	0	16	1.33	1.72	0	9
EXT_17	1.75*	1.93	0	13	1.21	1.67	0	8
PEER_14	13.97**	4.12	9	36	2.57	2.46	0	15
PEER_17	16.04**	4.47	9	33	2.62	2.19	0	11

Note. On the left-hand side of the table, mean levels of individual traits are presented. Externalizing symptom counts (EXT) could conceivably range from 0 to 21 (raw data is presented here to facilitate interpretation; however, to adjust for positive skew, these data were log-transformed for analysis). Delinquency questionnaire (DBI) scores could range from 0 to 36 and deviant peer affiliation (PEER) could range from 9 to 36. On the right-hand side of the table, sibling difference scores are presented (in absolute value form, so as to highlight the true magnitude of sibling differences). Of note, however, to examine the direction of any significant effects, final analyses were conducted on signed sibling differences.

** *and* * indicate that scores change across age at $p<.01$ and $p<.05$, respectively.

Table 2

Correlations among Sibling Differences in Deviant Peer Affiliation (PEER) and Externalizing Behaviors at ages 14 and 17.

	1	2	3	4
DBI				
1. DBI_14	---	.39**	*.68***	**.32****
2. DBI_17	.29**	---	**.19~**	*.61***
3. PEER_14	*.57***	**.20****	---	.32**
4. PEER _17	**.23****	*.49***	.26**	---
EXT				
1. EXT_14	---	.19*	*.24***	**.30****
2. EXT_17	.14**	---	**.05**	*.30***
3. PEER _14	*.22***	**.06**	---	.32**
4. PEER _17	**.16****	*.22***	.26**	---

Note. DBI and EXT represent various measures of externalizing behaviors: a delinquency questionnaire and a combined CD/ODD symptom count. Difference score correlations for the full sample are presented below the diagonal, while difference score correlations for the sub-sample of pairs that did not share all their friends are presented above the diagonal. Within-age, across-trait correlations are italicized. Cross-age, cross-trait correlations are bolded.

** *and* * indicate that correlation is significantly greater than zero at p<.01 and p<.05, respectively.

Table 3

Standardized Path Estimates from the MZ Differences Cross-Lagged Model.

	b11	b22	b12	b21	r1	r2
Full sample						
DBI	.27**	.20**	.14*	.08	.57**	.45**
EXT	.13*	.24**	.11*	.03	.22**	.19**
Sub-sample of twins who did not share all or nearly all of their friends						
DBI	.46**	.21~	.24*	−.04	.67**	.57**
EXT	.19*	.29**	.23*	.02	.24**	.24*

Note. DBI and EXT represent various measures of externalizing behaviors: a delinquency questionnaire and a combined CD/ODD symptom count. The path estimate labels (i.e., b11, b22, b12, and b21) correspond to those presented in Figure 1: b11 represents the stability of differential externalizing behaviors over time, b22 represents the stability of differential deviant peer affiliation over time, b12 represents the association between differential externalizing behaviors at 14 and differential deviant peer affiliation at 17, and b21 represents the association between differential deviant peer affiliation at 14 and differential externalizing behaviors at 17. All b paths function as partial regression coefficients. The correlation labels (i.e., r1 and r2) also correspond to those presented in Figure 1: r1 represents the correlation between differential externalizing behaviors and differential deviant peer affiliation at age 14, while r2 represents the residual correlation between across differential externalizing behaviors and differential deviant peer affiliation at age 17, controlling for the age 14 contributions.

** *and* * indicate that estimate is significantly greater than zero at p<.01 and p<.05, respectively.

~ indicates that estimate is approaching significance at p<.10.