Published in final edited form as:
Behav Genet. 2009 September ; 39(5): 532–540. doi:10.1007/s10519-009-9283-y.

Are there Shared Environmental Influences on Adolescent behavior? Evidence from a Study of Adoptive Siblings

Jacob P. Buchanan, **Matt McGue**, **Margaret Keyes**, and **William G. Iacono**
Department of Psychology, University of Minnesota, 75 East River Rd., Minneapolis, MN 55455, USA

Dorret Boomsma and **John Hewitt**

Abstract

The failure to identify specific non-shared environmental influences on behavior coupled with the belief that shared environmental factors contribute minimally to individual differences in behavior has led to the concern that major environmental determinants of behavior may be idiosyncratic, and therefore undetectable. We used data on adoptive (N = 246) and biologically related (N = 130) same-sex sibling pairs (mean ages = 16.1 years older sibling; 13.8 years younger sibling) from the Sibling Interaction and Behavior Study (SIBS) to determine whether non-idiosyncratic environmental factors shared by siblings contributed to individual differences in a diverse set of behavioral outcomes. Evidence for shared environmental influence was sought for eight composite measures covering a wide array of adolescent functioning: Academic Achievement, Total IQ, Substance Use Disorders, Externalizing Disorders, Internalizing Disorders, Peer Groups, Disinhibited Personality, and Negative Emotionality. For six of eight composites, significant shared environmental effects, accounting for 14–22% of the variance, were observed for these same-sex sibling pairs. These findings support the use of adoptive sibling designs to directly estimate shared environmental effects and implicate the existence of systematic environmental influences on behavior that are potentially detectable.

Keywords

Shared environment; Adoption study; Adolescent development; Age moderation; Range restriction

Characterization of shared environmental influence on adolescent behavior: evidence from the Sibling Interaction and Behavior Study

One of the most provocative findings to emerge from behavioral genetic research concerns the nature of environmental influence. In their landmark review, Plomin and Daniels (1987) concluded that while behavioral genetic research unequivocally implicated the existence of substantial environmental influences on virtually every behavioral outcome investigated, these influences had an unexpected form. Namely, the major source of environmental influence on individual differences in behavior appeared to be factors that create differences (what behavioral geneticists term non-shared environmental influences) rather than similarities (shared environmental influences) among reared-together relatives. Plomin and Daniels' review was the impetus for large-scale research programs, such as the NEAD

bucha057@umn.edu .

(Nonshared Environment in Adolescent Development) study by Reiss et al. (2000), which sought to characterize the non-shared environmental influences on development. Despite considerable effort, however, the specific factors that underlie non-shared environmental effects remain largely unknown. Thus, Turkheimer and Waldron (2000) concluded that environmental influences on human behavior may be largely idiosyncratic.

There is potential, however for the identification of specific shared environmental influences to inform behavioral development. Studies of twins reared together (Plomin et al. 2000) or designs that compare twins reared apart to twins reared together (Bouchard et al. 1990) find little evidence of shared environmental effects. However, these studies measure shared environmental effects indirectly. Turkheimer et al. (2005) note that when variables are measured at the family level, necessitating equating them for twins reared together, it is not possible to separate genetic from environmental influences, and the typical methods of twin data analysis, namely structural equation modeling and DeFries–Fulker analysis, are not sufficient to overcome this confound. Additionally, the magnitude of any shared environmental effects may vary across behavioral domains. For example, there is some evidence that shared effects are greater in the ability (Rowe et al. 1998; Chipuer et al. 1990) than in the personality domain (Loehlin et al. 1998; Loehlin and Martin 2001). Finally, the Plomin and Daniels review focused primarily on studies with adults, but the magnitude of shared environmental influences may vary across development. Specifically, it seems likely that shared environmental influences, to the extent they exist, will be maximal during childhood and adolescence, when children in a family are still living together, and decline in adulthood, when they are likely to be living apart. Consistent with this expectation, Bergen et al. (2007), in a recent meta-analysis of twin studies, concluded that shared environmental influences decreased and genetic influences increased for a broad range of psychological outcomes during the transition from late adolescence to early adulthood.

The present study sought evidence of shared environmental effects for a range of behavioral outcomes in a large sample of adoptive and non-adoptive adolescent siblings. In the absence of selective placement effects, the similarity of reared-together non-genetically related (i.e., adoptive) siblings provides a direct assessment of the contribution of shared environmental effects. Our adolescent sample allows us to focus on the developmental stage when shared environmental effects are likely to be maximal, while our investigation of functioning in various behavioral domains, including adolescent problem behavior, personality, mental health, and academics, and intelligence, allows us to demarcate the range of shared environmental effects. While the adoptive sibling design has several advantages over other designs for identifying shared environmental effects on behavior, it is not without its own limitations. Specifically, shared environmental influences may be underestimated because adopted adolescents experience a restricted range of environments (Stoolmiller 1999) or because age differences within sibling pairs reduce the likelihood that siblings share key developmental experiences (McGue et al. 1996). We address these problems by both applying the Pearson–Lawley equation (Pearson 1903; Lawley 1943) to correct for the impact of range restriction and testing whether sibling age difference moderates sibling similarity. It should also be noted that using an adoptive sibling design, as opposed to a twin design, reduces the power to detect significant genetic influences on behavior.

While there have been several earlier studies of adoptive sibling similarity, these studies have several limitations relative to the goals of the current investigation. First, most previous adoptive sibling studies have had a rather specific focus on either cognitive ability (e.g., Segal et al. 2007; Petrill and Deater-Deckard 2004) or adolescent substance abuse (e.g., Slomkowski et al. 2005). Our goal here is to investigate evidence for shared environmental effects across a broad array of behavioral outcomes. Second, in several studies the sample of adopted siblings includes a relatively large number of unlike-sex pairs and shared

environmental effects may be moderated by gender. The sibling sample used here is restricted to like-sex pairs in order to maximize the opportunity for observing shared environmental effects. Finally, several of the previous studies have been based on modest sized samples or unrelated sibling pairs created through divorce rather than adoption. The current study is based on a relatively large number of non-genetically related sibling pairs (N = 246) all created through adoption in infancy, rather than family dissolution.

Method

Participants

The Sibling Interaction and Behavior Study (SIBS) sample is comprised of 409 adoptive and 208 biologically related families, each consisting of two rearing parents and a pair of adolescent siblings no more than 5 years apart in age. Adoptive families were systematically ascertained through three large adoption agencies and consist of either two non-biologically related adoptive siblings (adopt-adopt, N = 285), or one adopted and one biological child of the original rearing parents (bio-adopt, N = 124). Adopted siblings must have been permanently placed in their current family before 2 years of age (mean age of placement = 4.7 months, SD = 3.4 months). Biologically related families were ascertained through state birth records and consist of two biologically related full-siblings, selected to be comparable to the adoptive sample in terms of age and gender distributions. The biologically related and adoptive families undergo a 5-h in-person assessment and complete self-report questionnaires at home that cover a wide range of behavioral domains. Recruited families received reimbursement for travel costs and a small honorarium for their participation. McGue et al. (2007) provide a complete description of the recruitment procedures used in SIBS including an analysis of non-participants showing that biologically related and adoptive families are reasonably representative of the populations from which they were sampled.

For this study, only same-sex sibling pairs were utilized because of the possibility that unlike-sex sibling pairs were less likely to share environmental factors then like-sex sibling pairs (Sharma et al. 1998). Consistent with this expectation, the like-sex sibling correlation was greater than the unlike-sex correlation for all of the measures, and significantly so for four of the eight outcome composites (described below) in both the adoptive (r difference mean = 0.19, range = 0.01–0.33) and biologically related (r difference mean = 0.25, range = 0.07–0.44) sibling samples. The same-sex sibling pairs consisted of 246 adoptive (61% female) and 130 biologically related (52% female) families (Table 1). Of note is the ethnic breakdown of the sample. Reflecting the adoption practices of the Minnesota agencies, a majority of the adopted adolescents are East Asian. Reflecting the demographics of the state of Minnesota for the birth years considered, the vast majority of the non-adopted adolescents were Caucasian (Table 1). East Asian participants did not score significantly differently than Caucasian participants and participants of other ethnic groups on any of the outcome measures of interest, with the exception of the internalizing cluster (see below), on which they scored somewhat higher. Moreover, adopted sibling pairs in which both siblings were of East Asian descent did not differ significantly in similarity from pairs with other ethnic compositions. Sibling correlations also did not differ significantly between male and female pairs or between adopt-adopt and adopt-bio sibling pairs, justifying pooling in both cases in the results presented here.

Procedure and measures

Participating families were assessed at our labs at the University of Minnesota. The assessments consist of interviews, self-report measures, and cognitive tests. The interviewers were extensively trained and had attained at least a B.A. in Psychology or a closely related

field. For some self-report measures, families were mailed the forms in advance, and were asked to complete the measures and return them upon their arrival for the on-site assessments. The assessment battery typically took 5 h to complete. In the event that the assessment took longer than anticipated, participants were asked to complete some self-report measures at home and return these via mail.

Our selection of individual indicators to include in the current analyses was guided by two factors. First, we sought representative indicators across a wide variety of adolescent functioning. Second, we included indicators that were administered to all adolescents regardless of age (e.g., some personality scales were administered only to adolescents age 16 and older). Unless otherwise noted, scales were developed in the context of research on adolescents and their families undertaken at the Minnesota Center for Twin and Family Research (Iacono et al. 2006).

Where needed, behavioral indicators were log-transformed to reduce skewness, and, with the exception of total IQ, all indicators were corrected for age and sex prior to analysis (McGue and Bouchard 1984). The variables of interest were assigned to one of eight a priori defined clusters as given in Table 2. These clusters (number of measures, average cluster measure intercorrelation) are: Academic Achievement (6, 0.50), Total IQ (1, not applicable), Substance Use Problems (4, 0.41), Externalizing Disorders (6, 0.36), Internalizing Disorders (2, 0.24), Peer Groups (2, 0.37), Disinhibitory Behavior (6, 0.83), and Negative Emotionality (3, 0.46). In all cases except Total IQ, cluster scores were created by summing the standardized component scores, after reflecting behavioral indicators with opposite directionality where appropriate, to ensure that scores on the component scales summed to create the same directional effect for each component included in the composite measure.

The Academic Achievement cluster consisted of both parent and sibling reports of the following: grade point average (GPA) in English, Mathematics, Science, and Social Studies ($\alpha = 0.77$), a 9-item academic motivation scale (e.g., “Has a good attitude about school,” $\alpha = 0.85$), and a 3-item academic problems scale (e.g., “Talks excessively in class,” $\alpha = 0.84$).

Total IQ was assessed via the WISC-R (for siblings age 15 years and younger) or WAIS-R (for siblings age 16 years and older), using two verbal subtests (Vocabulary and Information) and two performance subtests (Block Design and Object Assembly). The use of these four subtests together is known to correlate 0.90 with overall IQ (Kaufman 1990).

The Substance Use Disorders cluster consisted of DSM-IV substance disorder symptom counts obtained through in-person interviews with the revised Diagnostic Interview for Children and Adolescents (Welner et al. 1987; Reich 2000; DICA-R) for adolescents aged 15 years and younger or the Substance Abuse Module (SAM, updated to cover DSM-IV substance use criteria; Robins et al. 1987) for adolescents aged 16 years and older, covering Nicotine Dependence, Alcohol Dependence, and Dependence on Any Other Drug, as well as the number ($\alpha = 0.75$) of substances ever used (from a list of 12). Symptoms were coded as either present or absent. Assignment of symptoms was reached by consensus of a team consisting of at least two diagnosticians with advanced clinical training.

The Externalizing Disorders cluster included the Delinquent Behavior Inventory (DBI), a 36-item self-report of minor (i.e., truancy) and major (i.e., weapon use in a fight) indicators of delinquent behavior ($\alpha = 0.89$; Gibson 1967), as well as DSM-IV symptom counts for Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), and Attention-Deficit/Hyperactivity Disorder (ADHD). The Internalizing Disorders cluster included DSM-IV symptom counts for Major Depressive Disorder (MDD) and Separation Anxiety Disorder (SAD). For both the Externalizing Disorders and Internalizing Disorders clusters, DSM-IV symptoms were coded as present at a full, sub-threshold, or absent level, incrementing the

symptom count by 1, 0.5, or 0, respectively, based on diagnostic team consensus. Each listed disorder was assessed through administration of the Diagnostic Interview for Children and Adolescents (DICA-R; Welner et al. 1987; Reich 2000) for Children and Parents (DICA-P). Additionally, adolescents aged 16 years and older (and their parents) are also interviewed with the Structured Clinical Interview for DSM-III-R and DSM-IV (SCID, Spitzer et al. 1992) to cover adult mood and anxiety criteria. A best estimate procedure was employed (Leckman et al. 1982) such that a symptom was considered present if endorsed by either the parent or the child.

The Peer Groups cluster was assessed using a 10-item self-report of positive peer models (five items, e.g., peers are “Liked by teachers” and “Get good grades,” $\alpha = 0.79$), and negative peer models (five items, e.g., peers “Break the rules” and “Get into trouble with police,” $\alpha = 0.89$).

Relevant scales from the Multidimensional Personality Questionnaire (MPQ; Tellegen and Waller 2008) were utilized to form the composites for the Disinhibited Personality cluster and the Negative Emotionality cluster. The following MPQ scales, being the only 6 of the 11 scales of the MPQ included in the younger adolescent assessment, were included in this study: well being ($\alpha = 0.88$), stress reaction ($\alpha = 0.90$), alienation ($\alpha = 0.86$), aggression ($\alpha = 0.81$), and control ($\alpha = 0.83$). In addition, adolescents completed a 40-item attitudes and opinion self-report from which the following scales were computed: antisociality ($\alpha = 0.87$), prosociality ($\alpha = 0.77$), aggressiveness ($\alpha = 0.87$), and family attitudes ($\alpha = 0.85$). The Disinhibited Personality cluster consisted of a composite of scores for aggression, control, antisociality, prosociality, aggressiveness, and family attitudes scales. The Negative Emotionality cluster consisted of a composite of scores for well-being, stress reaction, and alienation.

To assess the effect of restriction of range on adoptive sibling correlations, the Socioeconomic Status (SES) and parental disinhibitory behavior composites derived by McGue et al. (2007) were used. SES was based on a composite of each parent's level of education, coded on a 1 (less than high school) to 5 (professional degree) scale; and occupational status, assessed using Hollingshead's six-point classification scheme that ranges from 1 (professional/managerial) to 6 (manual labor). Following earlier research by Krueger et al. (2002) implicating the existence of a general latent dimension of disinhibitory psychopathology, a disinhibitory behavior composite was formed for each parent by summing the number of symptoms for the following DSM-IV disorders: Antisocial Personality Disorder (ASPD), assessed with the Structured Clinical Interview for DSM-III-R and DSM-IV (SCID; Spitzer et al. 1992), and substance use disorders including Alcohol Dependence and Drug Dependence (the latter including symptoms of cannabis, amphetamine, sedative, cocaine, hallucinogen, inhalant, opioid, and PCP dependence), assessed with the Substance Abuse Module (SAM, updated to cover DSM-IV substance use criteria; Robins et al. 1987). Scores for mothers and fathers were summed and the result standardized to a mean of 0 and SD of 1.0 in the non-adoptive family sample to form the SES and parent disinhibitory behavior family-level composites.

Data analysis

A univariate biometric model based on the comparison of the resemblance of adopted sibling pairs to biologically related sibling pairs was fit to each of the eight factor scores. Additive genetic (a^2), shared environmental (c^2), and non-shared environmental (e^2) standardized components of variance were estimated using the MX software package (Neale 1999) under the assumption that the expected adopted sibling correlation equals c^2 and the expected biologically related sibling correlation is $1/2a^2 + c^2$. To determine whether shared environmental effects are greater among near-in-age siblings, we used Mx to investigate the

moderating effect of sibling age difference on estimates of c^2. The shared environmental component was modeled as containing a portion of c^2 not moderated by the difference in sibling ages and a second portion that is moderated by the difference in sibling ages as a proportion of the maximum age difference between siblings allowed in the study (5 years). Finally, we applied the Pearson–Lawley (Pearson 1903; Lawley 1943) formula to correct the adopted sibling correlations for range restriction on parental SES and disinhibitory behavior, using the biologically related families as the unselected referent. McGue et al. (2007) provides a complete description of the adjustment procedure.

Results

Adoptive and biologically related sibling correlations for each of the behavioral factors are given in Table 3. Except for Academic Achievement and Negative Emotionality in the adoptive siblings, all correlations were significant at $p < 0.05$. The significant adoptive sibling correlations for IQ, Substance Use Disorders, Externalizing Disorders, Internalizing Disorders, Peer Groups, and Disinhibitory Behavior, suggest the presence of shared environmental influences for these factors. Also given in Table 3 are the adopted sibling correlations corrected for range restriction on the parent SES and disinhibitory behavior composites. Because parent SES and disinhibitory behavior are minimally associated with the various behavioral outcomes, the corrected adoptive sibling correlation differed in no case by as much as 0.01 from the uncorrected adoptive correlation. Consequently, we report only the uncorrected correlations in Table 3. These findings suggest that range restriction, at least on these parent factors, does not have a great effect in attenuating shared environmental effects in the adoptive sibling sample.

Table 4 gives the results from the biometric analysis. Consistent with the non-significant adoptive sibling correlations for these variables, estimates of shared environmental effects were small and non-significant for Academic Achievement ($c^2 = 0.01$) and Negative Emotionality ($c^2 = 0.11$). The estimate of shared environmental effect was, however, moderate and statistically significant for Total IQ ($c^2 = 0.19$), Substance Use Disorders ($c^2 = 0.21$), Externalizing Disorders ($c^2 = 0.22$), Internalizing Disorders ($c^2 = 0.20$), and Disinhibited Personality ($c^2 = 0.20$). Additionally, weaker but still significant evidence for shared environmental influences in the Peer Groups factor ($c^2 = 0.14$) was found. The estimate of genetic influence was significant only for Academic Achievement ($a^2 = 0.43$) and Total IQ ($a^2 = 0.65$); estimates of genetic influence for the other factors were all non-significant and generally modest in magnitude ($a^2 = 0.10$ to $a^2 = 0.39$).

Test statistics for fitting the sibling age difference moderation model to each of the phenotypes are summarized in Table 4. Significant age moderation of the shared environmental influence was observed only for the Substance Use Disorders cluster. The age difference moderation effect is plotted in Fig. 1 for the range of age differences in our sample. As can be seen, estimates of c^2 ranged from nearly 50% for siblings very near in age to 0% for those who were 5 years apart.

Discussion

Biometric analysis of a large sample of biologically related and adoptive sibling pairs revealed consistent evidence of shared environmental influences on a diverse set of behavioral outcomes. Measures of substance use, internalizing and externalizing psychopathology, IQ, disinhibitory behavior, and peer group characteristics all showed significant shared environmental effects, accounting for approximately 20% of the variance in most cases. Only for measures of academic achievement and negative emotionality did we not find evidence for shared environmental effects.

Because resemblance among the members of intact nuclear families may reflect their shared genes as well as their shared environments, behavioral geneticists have sought alternative research designs to identify the existence of shared environmental influences on behavior (McGue and Bouchard 1998). By far the most common behavioral genetic research design is the study of reared-together twins. In this design, inference on the existence and magnitude of shared environmental effects is indirect, being based on a comparison of MZ and DZ correlations, and may lack statistical power (Martin et al. 1978; Turkheimer et al. 2005). In principal, the adoptive sibling correlation provides a more direct assessment of shared environmental influences, although adoption studies are not without their own limitations. Specifically, Stoolmiller (1999) has argued that restriction in environmental exposure within adoptive families results in shared environmental effects being substantially underestimated by the adoptive sibling correlation. McGue et al. (2007) have shown, however, that bias in the adoptive sibling correlation occurs only when there is range restriction on factors that are associated with offspring outcome. There was no evidence that either parental SES or parental disinhibitory behavior, the domains upon which restriction is greatest, was related to any of the diverse set of outcomes investigated here. Consequently correction for range restriction had little impact on the adoptive sibling correlation. Although our results suggest minimal bias in the adoptive sibling correlation, we note that we used our sample of biologically related families as the referent for the range restriction correction. The biologically related families were selected to be representative of Minnesota families that have two biologically related full siblings. Thus, for example, families with parents whose marriages dissolved after the birth of their first child would not be included in our study. Due to this requirement, this sample, in all likelihood, under represents extreme levels of poverty and family disorganization. Our estimates of shared environmental effects, although non-trivial, may still underestimate the true contribution of shared experience to sibling similarity.

A second concern with the adoptive sibling design is that siblings who differ markedly in age may experience less similar family environments than siblings who are near in age, attenuating estimates of shared environmental influence. Nonetheless, for only one of the factors we investigated, substance use, did we find that the estimate of c^2 was moderated by sibling age difference. Of interest, in a separate sample McGue and Sharma (1995) also found sibling age difference moderated adoptive sibling similarity for adolescent alcohol use. They speculated that the age moderation of the sibling correlation reflected sibling effects, whereby near-in-age siblings are more likely to influence each other's substance use behavior than distant-in-age siblings (McGue et al. 1996). Although our study does not directly test for the source of the shared environmental effects, it does suggest that in some cases they could be substantial as estimates of c^2 approached 40% for siblings who were 1 year apart.

The most significant finding from the present study is the consistent demonstration of the existence of systematic environmental influences on adolescent behavior. The general failure to find the specific factors underlying non-shared environmental effects coupled with the belief that shared environmental influences are relatively unimportant has led to concern that researchers will not be able to identify the environmental contributors to individual differences in behavior because these influences are largely idiosyncratic (Turkheimer and Waldron 2000). Shared environmental influences must be systematic since they contribute to sibling similarity. Consequently, our finding that 20% of the variance in a diverse set of behavioral outcomes owes to environmental factors shared by siblings implies the existence of systematic environmental influences that are, in principle, detectable. These estimates are consistent with studies of shared environmental effects utilizing twin methodology to examine behavioral phenotypes such as anti-social behavior (Rhee and Waldman 2002) and substance use (Dick et al. 2007).

Our failure to find significant heritable effects in all domains except two (IQ and academic achievement) is somewhat surprising given the extensive evidence of genetic influences on most domains of psychological functioning (McGue and Bouchard 1998). Several factors should be considered in interpreting these results. First, the major aim of SIBS is to identify and characterize shared environmental effects on adolescent development, and the study was designed to maximize our power to detect shared environmental effects. Our sibling design provides power >0.80 to detect shared environmental effects accounting for as little as 10% of the variance and >75% to detect heritable effects accounting for at least 50% of variance. Consequently, our failure to identify significant heritable effects is nonetheless consistent with heritabilities that are moderate in magnitude. Secondly, the variance on many behavioral measures is markedly different when comparing the adoptive sibling pairs to the non-adoptive biological sibling pairs. This accounts for most of the apparent discrepancy between the sibling correlations and the estimated proportions of variance generated by Mx.

Two additional research findings provide additional context for interpreting our results. First, there is growing evidence for the existence of genotype-environment interaction (G × E) effects on behavior (Caspi et al. 2002, 2003, 2005). Our findings complement rather than stand in opposition to evidence for G × E. That is, the evidence for shared environmental influence reported here cannot be accounted for by G × E since the adopted siblings in our sample are not genetically related to one another. Thus, our findings imply the existence of environmental main effects. Although in theory G × E can exist in the absence of main effects, in practice crossover interactions are uncommon (Moffitt et al. 2005; Eaves 2006). Our findings thus suggest that the shared family environment may be one fruitful source of environmental components in G × E models. Second, our adolescent sample allowed us to focus on a developmental stage when shared environmental effects are likely to be maximal. There is extensive evidence that shared environmental effects may wane with age, especially with the attainment of early adulthood when siblings typically move apart (Hood et al. 1996; Eaves et al. 1997; Koenig et al. 2005). Alternatively, developmental decreases in shared environmental influences are typically accompanied by increases in heritability, as was shown in the recent meta-analysis by Bergen et al. (2007) that reported statistically significant increases in heritability in five of eight behavioral domains during the transition from adolescence to early adulthood. Consequently, our failure to observe significant heritable influences on several of the behavioral composites may change as the current sample ages into early adulthood.

The findings of our study support the use of adoptive sibling designs as another useful tool in the direct estimation of the effects on behavioral development due to shared-environmental influences. Our results suggest the existence of non-idiosyncratic environmental determinants of behavior, which cannot be idiosyncratic to the individual, since shared-environmental influences instead cause, by definition, a given pair of individuals to become more similar. Such non-idiosyncratic factors should be detectable, measurable, and systematic phenomena, further advancing our understanding of the etiology of behavior.

Acknowledgments

This research was made possible in part by support from USPHS Grants Numbers AA11886 and MH066140. We also thank the participating families of the Sibling Interaction and Behavior Study for their time and cooperation.

References

Bergen SE, Gardner CO, Kendler KS. Age-related changes in the heritability of behavioral phenotypes over adolescence and young adulthood: a meta-analysis. Twin Res Hum Genet 2007;10:423–433. [PubMed: 17564500]

Bouchard TJJ, Lykken DT, McGue M, Segal N, Tellegen A. Sources of human psychological differences: the Minnesota study of twins reared apart. Science 1990;250:223–228. [PubMed: 2218526]

Caspi A, McClay J, Moffitt T, Mill J, Martin J, Craig IW, et al. Role of genotype in the cycle of violence in maltreated children. Science 2002;297:851–854. [PubMed: 12161658]

Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, Harrington H, et al. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. Science 2003;301:386–389. [PubMed: 12869766]

Caspi A, Moffitt TE, Cannon M, McClay J, Murray R, Harrington H, et al. Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-*O*-methyltransferase gene: longitudinal evidence of a gene × environment interaction. Biol Psychiatry 2005;57:1117–1127. [PubMed: 15866551]

Chipuer HM, Rovine MJ, Plomin R. LISREL modeling: genetic and environmental influences on IQ revisited. Intelligence 1990;14:11–29.

Dick DM, Pagan JL, Viken R, Purcell S, Kaprio J, Pulkkinen L, Rose RJ. Changing environmental influence on substance use across development. Twin Res Hum Genet 2007;10:315–326. [PubMed: 17564520]

Eaves LJ. Genotype × environment interaction in psychopathology: fact or artifact? Twin Res Hum Gen 2006;9:1–8.

Eaves L, Martin N, Heath A, Schieken R, Meyer J, Silberg J, et al. Age changes in the causes of individual differences in conservatisim. Behav Genet 1997;27:121–124. [PubMed: 9145550]

Gibson HB. Self-report delinquency among school boys and their attitudes to police. Br J Soc Clin Psychol 1967;20:303–315.

Hood, RW., Jr; Spilka, B.; Hunsburger, B.; Gorsuch, R. The psychology of religion: an empirical approach. 2nd edn.. Guilford Press; New York: 1996.

Iacono WG, McGue M, Krueger RF. Minnesota Center for Twin and Family Research. Twin Res Hum Genet 2006;9:978–984. [PubMed: 17254440]

Kaufman, AS. Assessing adolescent and adult intelligence. Allyn & Bacon; Needham Heights, MA: 1990.

Koenig LB, McGue M, Krueger RF, Bouchard TJ Jr. Genetic and environmental influences on religiousness: findings for retrospective and current religiousness ratings. J Pers 2005;73:471–488. [PubMed: 15745438]

Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiological connections among substance dependence, antisocial behavior and personality: modeling the externalizing spectrum. J Abnorm Psychol 2002;111:411–424. [PubMed: 12150417]

Lawley DN. A note on Karl Pearson's selection formulae. Proc R Soc of Edinb 1943;62:28–30.

Leckman JF, Scholomskas D, Thompson WD, Belanger A, Weisman MM. Best estimate of lifetime psychiatric diagnosis: a methodlogical study. Arch Gen Psychiatry 1982;39:879–883. [PubMed: 7103676]

Loehlin JC, Martin NG. Age changes in personality traits and their heritabilities during the adult years: evidence from Australian twin registry samples. Pers Indiv Differ 2001;30:1147–1160.

Loehlin JC, McCrae RR, Costa PT Jr, John OP. Heritabilities of common and measure-specific components of the Big Five personality factors. J Res Pers 1998;32:431–453.

Martin NG, Eaves LJ, Kearsey MJ, Davies P. The power of the classical twin study. Heredity 1978;40:97–116. [PubMed: 272366]

McGue M, Bouchard TJ Jr. Adjustment of twin data for the effects of age and sex. Behav Genet 1984;14:325–343. [PubMed: 6542356]

McGue M, Bouchard TJ Jr. Genetic and environmental influences on human behavioral differences. Annu Rev Neurosci 1998;21:1–24. [PubMed: 9530489]

McGue M, Sharma A. Parent and sibling influences on adolescent alcohol use and misuse: evidence from a U.S. adoption cohort. J Stud Alcohol 1995;57:8–18. [PubMed: 8747496]

McGue MK, Sharma A, Benson P. The effect of common rearing on adolescent adjustment: evidence from a U.S. adoption cohort. Dev Psychol 1996;32(60):4–613.

McGue M, Keyes M, Sharma A, Elkins I, Legrand L, Johnson W, et al. The environments of adopted and non-adopted youth: evidence on range restriction from the Sibling Interaction and Behavior Study (SIBS). Behav Genet 2007;37:449–462. [PubMed: 17279339]

Moffitt TE, Caspi A, Rutter M. Strategy for investigating interactions between measured genes and measured environments. Arch Gen Psychiatry 2005;62:473–481. [PubMed: 15867100]

Neale MC. Possible confounds and their resolution in multivariate genetic analyses: comment on Bussell et al. (1999). Dev Psychol 1999;35:1260–1264. [PubMed: 10493651]

Pearson K. Mathematical contributions to the theory of evolution: XI. On the influence of natural selection on the variability and correlation of organs. Philos Trans R Soc Lond A 1903;200:1–66.

Petrill SA, Deater-Deckard K. The heritability of general cognitive ability: a within-family adoption design. Intelligence 2004;32:403–409.

Plomin R, Daniels D. Why are children in the same family so different from one another? Behav Brain Sci 1987;10:1–60.

Plomin, R.; DeFries, JC.; McClearn, GE.; McGuffin, P. Behavioral genetics. 4th edn.. Worth Publishers; New York: 2000.

Reich W. Diagnostic interview for children and adolescents (DICA). J Am Acad Child Adolesc Psychiatry 2000;39:59–66. [PubMed: 10638068]

Reiss, D.; Neiderhiser, JM.; Hetherington, M.; Plomin, R. The relationship code: deciphering genetic and social patterns in adolescent development. Harvard University Press; Cambridge: 2000.

Rhee SH, Waldman ID. Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. Psychol Bull 2002;128:490–529. [PubMed: 12002699]

Robins, LM.; Baber, T.; Cottler, LB. Composite International diagnostic interview: expanded substance abuse module. Authors; St. Louis: 1987.

Rowe DC, Vesterdal WJ, Rodgers JL. Herrnstein's syllogism: genetic and shared environmental influences on IQ, education, and income. Intelligence 1998;26:405–423.

Segal NL, McGuire SA, Havlena J, Gill P, Hershberger SL. Intellectual similarity of virtual twin pairs: developmental trends. Pers Indiv Differ 2007;42:1209–1219.

Sharma AR, et al. The psychological adjustment of United States adopted adolescents and their non-adopted siblings. Child Dev 1998;69:791–802. [PubMed: 9680685]

Slomkowski C, Rende R, Novak S, Lloyd-Richardson E, Niaura R. Sibling effects on smoking in adolescence: evidence for social influence from a genetically informative design. Addiction 2005;100:430–438. [PubMed: 15784052]

Spitzer RL, et al. The structured clinical interview for DSM-III-R (SCID). I: History, rationale, and description. Arch Gen Psychiatry 1992;49:624–629. [PubMed: 1637252]

Stoolmiller M. Implications of restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. Psychol Bull 1999;125:392–409. [PubMed: 10414224]

Tellegen, A.; Waller, NG. Exploring personality through test construction: Development of the Multidimensional Personality Questionnaire.. In: Boyle, GJ.; Matthews, G.; Saklofske, DH., editors. Handbook of personality theory and testing, vol II. Personality measurement and assessment. Sage; Thousand Oaks, CA: 2008. p. 261-292.

Turkheimer E, Waldron M. Nonshared environment: a theoretical, methodlogical, and quantitative review. Psychol Bull 2000;126:78–108. [PubMed: 10668351]

Turkheimer E, D'Onofrio BM, Maes HH, Eaves LJ. Analysis and interpretation of twin studies including measures of the shared environment. Child Dev 2005;76:1217–1233. [PubMed: 16274436]

Welner Z, Reich W, Herjanic B, Jung K, Amado H. Reliability, validity, and parent-child agreement studies of the Diagnostic Interview for Children and Adolescents (DICA). Journal of the Academy of Child and Adolescent Psychiatry 1987;26:649–653.

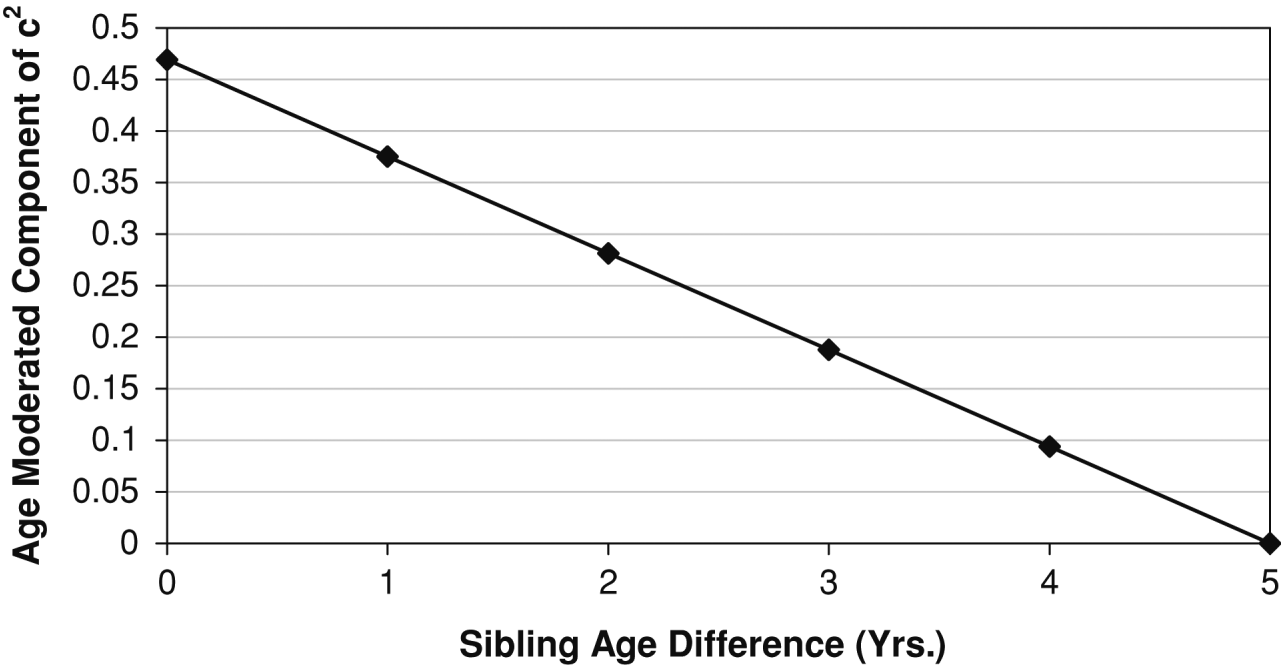

Fig. 1.
How sibling age difference moderates the estimate of the shared environmental effect for the Substance Use Disorders composite

Table 1

Demographic characteristics of the Sibling Interaction and Behavior Study sample

	Adoptive families	Biologically related families
Number of families	246	130
Older sibling		
% Female	61	52
Age (years)		
M	16.1	16.3
SD	1.6	1.4
Ethnicity		
% Asian	62.2	0
% Caucasian	29.7	93.8
% Other	8.1	6.2
IQ		
M	106.6	107.1
SD	14.1	13.0
Younger sibling		
% Female	61	52
Age (years)		
M	13.8	14.1
SD	1.7	1.5
Ethnicity		
% Asian	46.3	0
% Caucasian	43.9	93.8
% Other	9.8	6.2
IQ		
M	107.0	107.7
SD	14.4	12.2
Mother		
% College degree	58.5	46.9
# Externalizing symptoms		
M	1.7	2.0
SD	4.2	3.1
Father		
% College degree	61.4	44.6
# Externalizing symptoms		
M	3.1	5.1
SD	3.3	7.2

Only same sex pairs were included in this study

Table 2

Behavioral composites defined by rational Clustering of the Component Variables

Behavioral composite	Reporter	Component variables
Academic achievement	Parent	GPA
		Academic motivation
		Academic problems
	Adolescent	GPA
		Academic motivation
		Academic problems
Total IQ	N/A	Total IQ
Substance use disorders	Adolescent	Nicotine dependence symptoms
		Alcohol dependence symptoms
		Other drug dependence symptoms
		Number of drugs used
Externalizing disorders	Parent	Behavior problems
	Adolescent	Delinquent behavior inventory score
		Behavior problems
	Best estimate	Oppositional defiant disorder symptoms
		Conduct disorder symptoms
		Attention-deficit/hyperactivity disorder symptoms
Internalizing disorders	Best estimate	Major depressive disorder symptoms
		Separation anxiety disorder symptoms
Peer groups	Adolescent	Positive peer models
		Negative peer models
Disinhibited personality	Adolescent	Aggression
		Control (reverse scored)
		Antisociality
		Prosociality (reverse scored)
		Aggressiveness
		Family attitudes (reverse scored)
Negative emotionality	Adolescent	Well being (reverse scored
		Stress reaction
		Alienation

"Best estimate" indicates a composite report in which one or both of the adolescent and the parent reporter has endorsed a symptom as present in the adolescent

Table 3

Biologically related and adoptive sibling correlations (95% confidence intervals)

Behavioral factor	Biologically related (*N* = 130)	Adoptive (*N* = 246)
Academic achievement	0.25* (0.08, 0.41)	0.02 (–0.11, 0.15)
Total IQ	0.46* (0.31, 0.58)	0.21* (0.09, 0.33)
Substance use disorders	0.30* (0.13, 0.45)	0.22* (0.10, 0.34)
Externalizing disorders	0.33* (0.16, 0.47)	0.25* (0.13, 0.37)
Internalizing disorders	0.29* (0.12, 0.45)	0.22* (0.10, 0.34)
Peer groups	0.34* (0.18, 0.48)	0.13* (0.01, 0.25)
Disinhibitory behavior	0.33* (0.17, 0.48)	0.22* (0.10, 0.34)
Negative emotionality	0.24* (0.07, 0.40)	0.09 (–0.04, 0.21)

All sibling pairs are like-sex and all behavioral variables, with the exception of Total IQ, were age-sex corrected prior to analysis

* 95% Confidence intervals that do not include 0. Adopted Sibling correlations were unchanged when corrected for range restriction due to parental education level and disinhibitory behavior problems

Table 4

Estimates of additive genetic (a^2), shared environmental (c^2), and non-shared environmental (e^2) influences for the eight behavioral factors

Behavioral Composites	Variance component estimate (95% confidence interval)			Age moderation model fit statistics			
	a^2	c^2	e^2	χ^2_{diff}	*df*	*p*	AIC
Academic Achievement	0.43* (0.01, 0.75)	0.01 (0.00, 0.15)	0.55* (0.25, 0.90)	0.00	1	0.99	–2.00
Total IQ	0.65* (0.29, 0.92)	0.19* (0.08, 0.30)	0.16 (0.00, 0.48)	2.27	1	0.10	0.72
Substance use disorders	0.10 (0.00, 0.49)	0.21* (0.08, 0.32)	0.66* (0.35, 0.87)	7.49	1	<0.01	5.49
Externalizing disorders	0.13 (0.00, 0.51)	0.22* (0.10, 0.33)	0.64* (0.33, 0.85)	0.21	1	0.65	–1.79
Internalizing disorders	0.39 (0.00, 0.76)	0.20* (0.08, 0.31)	0.41* (0.11, 0.78)	2.07	1	0.15	0.07
Peer groups	0.29 (0.00, 0.67)	0.14* (0.02, 0.27)	0.56 (0.26, 0.86)	1.42	1	0.23	–0.58
Disinhibitory behavior	0.23 (0.00, 0.61)	0.20* (0.07, 0.32)	0.57* (0.27, 0.84)	0.00	1	1.00	–2.00
Negative emotionality	0.18 (0.00, 0.58)	0.11 (0.00, 0.23)	0.71* (0.38, 0.95)	0.00	1	0.99	–2.00

95% confidence intervals were computed via Mx. The final four columns summarize the fit statistics for the ACE model in which the c^2 estimate is allowed to vary as a function of the difference in age between the siblings in each pair. Significant results indicate a better fit when the age-moderated component of c^2 is allowed to vary freely

*Significant parameter estimates (the 95% CI's do no include zero)