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Additional Evidence against Shared Environmental Contributions to Attention-Deficit/Hyperactivity Problems

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Abstract

A recent meta-analysis (Burt, 2009) indicated that shared environmental influences (C) do not contribute to Attention-Deficit/Hyperactivity Disorder (ADHD). Unfortunately, the meta-analysis relied almost exclusively on classical twin studies. Although useful in many ways, some of the assumptions of the classical twin model (e.g., dominant genetic and shared environmental influences do not simultaneously influence the phenotype) can artifactually decrease estimates of C. There is thus a need to confirm that dominant genetic influences are not suppressing estimates of C on ADHD. The current study sought to do just this via the use of a nuclear twin family model, which allows researchers to simultaneously model and estimate dominant genetic and shared environmental influences. We examined two independent samples of child twins: 312 pairs from the Michigan State University Twin Registry (MSUTR) and 854 pairs from the PrEschool Twin Study in Sweden (PETSS). Shared environmental influences were found to be statistically indistinguishable from zero and accounted for less than 5% of the variance. We conclude that the presence of dominant genetic influences does not account for the absence of C on ADHD.

Keywords

ADHD; nuclear twin family model; shared environment; genetic influences

One often-cited conclusion from behavioral genetics research has been that the more crucial environmental influences result in differences between siblings (i.e., referred to as “non-shared” effects), whereas environmental influences that create similarities between siblings (i.e., “shared” effects; C) are indistinguishable from zero (Plomin & Daniels, 1987; Turkheimer, 2000). Although this finding does appear to hold in adulthood, a recent meta-analysis of twin and adoption studies (n=490) indicated that C makes important contributions to most forms of psychopathology during childhood and adolescence (Burt, 2009). Indeed, analyses revealed that C generally accounted for 10–30% of the variance within conduct disorder, oppositional defiant disorder, anxiety, depression, and broad internalizing and externalizing spectrum disorders. The only exception to this robust pattern of results was observed for Attention-Deficit/Hyperactivity Disorder (ADHD), which instead appeared to be largely genetic in origin, with no observable influence of the shared environment (a finding that was also reported in Nikolas & Burt, 2010).

In response to the results of Burt (2009), others argued that, rather than reflecting a true absence of shared environmental influences on ADHD, the results of Burt (2009) were instead a function of the limitations of the classical twin design (Wood, Buitelaar, Rijdsdijk,

Asherson, & Kuntsi, 2010). Wood and colleagues (2010) based this argument on both a quantitative summary of prior twin studies and a discussion of several methodological limitations that may account for the findings of Burt (2009). Although their summary of ADHD twin studies was hampered by several key limitations (i.e., their estimate of C was not weighted by sample size, non-independent data were treated as though they were independent, and those studies in which C was fixed to zero were not accommodated statistically), their discussion of twin study limitations was incisive and thoughtful. One particularly strong point¹ (made also in Burt, 2009) was that shared environmental influences are confounded with dominant genetic influences (i.e., allele-to-allele interactive effects at a single genetic locus; D) in the classical twin design (Keller & Coventry, 2005; Keller & Medland, 2008; Keller, Medland, & Duncan, 2010). Should both influences be important for a given phenotype (a biologically plausible scenario), it would lead to the under-estimation of both C and D. As an example, Keller and Medland (2008) simulated data in which additive genetic (A), non-additive genetic, and shared environmental variances were equal to .40, .15, and .15, respectively. Using the classical twin design, the ACE model (and not the ADE model) was chosen as the better fitting model, even though C and D effects were in fact equivalent in magnitude. Moreover, additive genetic influences were estimated at .60, whereas shared environmental influences were estimated at .02 (Keller & Medland, 2008). It is therefore possible that C does in fact influence ADHD but that these effects are obscured by the simultaneous presence of non-additive genetic effects.

To address this concern, Burt (2010) meta-analytically compared shared environmental influences on ADHD across twin and adoption studies. Because adoptive siblings do not share any segregating genetic material, dominant genetic influences are no longer a meaningful confound for their similarity. Analyses revealed that shared environmental influences were near-zero and non-significant in both twin and adoption studies (0% and 4% of the variance, respectively). Moreover, C could be constrained to be equal across twin and adoption studies without a significant decrement in fit (Burt, 2010). In short, there was virtually no evidence of C on ADHD in either twin or adoption studies, arguing against the notion that confounding with D accounts for the absence of C observed in Burt (2009).

Although the findings of Burt (2010) are helpful in resolving the concerns of Wood and colleagues (2010), they are limited by the fact that only one adoption study of ADHD (of two non-independent adoption studies) was actually available for analysis. Additional studies, and particularly studies using independent samples and alternate analytic designs, are thus clearly needed to confirm or refute this result. Fortunately, there is another analytic design, referred to as the “nuclear twin family design”, that can directly address this need. The nuclear twin family model is a straightforward extension of the classical twin model, in which data on the twin parents is incorporated along with the standard twin data. The nuclear twin family model thus provides four pieces of information on which to base parameter estimates: the covariance between MZ twins, the covariance between DZ twins, the covariance between parents, and the covariance between parents and children (see Figure 1). The addition of the latter two pieces of information to the information typically contained in the classical twin design allows for the simultaneous estimation of C and D, thereby expanding the otherwise unnested ACE and ADE models of the classical twin design into a single ADCE model. Applying the nuclear twin family model to ADHD data thus affords us the opportunity to more definitively ascertain whether the absence of shared environmental influences observed for ADHD in Burt (2009) and in Nikolas & Burt (2010) stems from the confounding of C and D within the classical twin design.

¹Wood and colleagues (2010) also noted that classical twin studies are limited by issues of low statistical power to detect shared environmental influences. While an important point, analyses in Burt (2010) indicated that low power was highly unlikely to explain the meta-analytic results of Burt (2009). Indeed, Burt (2009) had more than 80% power to detect C as small as 5%.

The current study sought to do just this in two independent samples of child twins and their parents. To make our statistical tests as conservative as possible, we chose to focus our analyses on parental informant reports of the twins using a questionnaire format, as shared environmental effects are larger when using these two particular methodological approaches (see Burt, 2009). Should *C* still be estimated to be at or near zero even when using this otherwise “shared environment friendly” design, it would provide additional compelling evidence against the notion that dominant genetic influences are masking the presence of *C* on ADHD.

METHODS

PARTICIPANTS

Sample 1—The Michigan State University Twin Registry (MSUTR) includes several independent twin projects (Klump & Burt, 2006). The 312 families included in the current study were assessed as part of the on-going Twin Study of Behavioral and Emotional Development in Children within the MSUTR. Children gave informed assent, while parents gave informed consent for themselves and their children. Rearing mothers ($n=312$, including 1 rearing grandmother) ranged in age from 26–59 years (mean (SD) = 39.8 (5.3) years), and rearing fathers ($n=272$, including 1 rearing grandfather) ranged in age from 29–69 years (mean (SD) = 41.7 (5.9) years). The twins ranged from in age 6 to 10 years (although some twins had turned 11 by the time they participated) and were 47% female. To be eligible for participation, neither twin could have a cognitive or physical condition (as assessed via parental screen) that would preclude completion of the assessment.

Families were recruited via State of Michigan birth records in collaboration with the Michigan Department of Community Health (MDCH). The MDCH manages birth records and can identify all twins born in Michigan. Birth records are confidential in Michigan; thus, the following recruitment procedures were designed to ensure anonymity of families until they indicated an interest in participating. MDCH identified twins in our age-range and made use of the Michigan Bureau of Integration, Information, and Planning Services database to locate current addresses through parent drivers’ license information. MDCH then mailed pre-made recruitment packets to families who lived within 120 miles of our laboratory. A reply postcard was included for parents to indicate their interest in participating. Interested families were contacted directly by project staff. Parents who did not respond to the first mailing were sent additional mailings approximately one month apart until either a reply was received or up to four letters had been mailed. Although data collection is not yet complete, our current response and participation rates are 62% and 84%, respectively. These rates are similar to or better than those of other twin registries that use similar types of anonymous recruitment mailings (Baker, Barton, & Raine, 2002; Hay, McStephen, Levy, & Pearsall-Jones, 2002). Moreover, participating families endorsed ethnic group memberships at rates comparable to other area inhabitants (e.g., Caucasian: 84.2% and 85.5%, African-American: 6.8% and 6.3% for the participating families and the local census, respectively). Parental education was also generally comparable to that of the area population (i.e., high school graduates: 93.8% and 91.2% for the participating families and the local census, respectively). Similarly, 14.4% of families in our sample lived below federal poverty guidelines, the same proportion seen for the state of Michigan more generally. Our recruitment strategy thus appears to yield a sample that is broadly representative of the area population.

Zygosity was established using physical similarity questionnaires administered to the twins’ primary caregiver (Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). On average, the physical similarity questionnaires used by the MSUTR have accuracy rates of 95% or better.

Sample 2—Parents of all twins born in Sweden between January 2004 and May 2005 were identified through the population-based medical birth register and contacted as part of the PrEschool Twin Study in Sweden (PETSS) one month prior to the twins' 5th birthday. Questionnaires were sent to parents and pre-school teachers of 1261 twin pairs. Non-responding families received up to three reminders. Parents were approached separately, resulting in 828 (65%) maternal responses and 698 (55%) paternal responses, for a total of 854 participating pairs in the current study. Data collection was approved by the Ethics Committee at Karolinska Institutet. Parents gave informed consent for themselves and their children.

In line with prior work (Heath et al., 2003), zygosity was established by fitting a 2-class (i.e., MZ or DZ) latent class model in Mplus Version 4.1 (Muthén & Muthén, 2006) to standard physical similarity questions (Lichtenstein, Tuvblad, Larsson, & Carlstrom, 2007). Latent class models were fitted separately for mother and father reports. Zygosity was scored as unknown for 25 twin pairs due to contradictions between the mother reports and father reports (20 twin pairs) or due to low predicted probabilities of class membership (5 twin pairs were assigned as MZ or DZ with a probability lower than .95). The majority (97%) of the 530 like-sexed twin pairs were assigned as MZ or DZ with a probability greater than or equal to 0.9999, and all twin pairs could be assigned as MZ or DZ with a probability greater than or equal to 0.95.

MEASURES

Sample 1—Mothers and fathers² in the MSUTR completed the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001) separately for each twin. We utilized the Attention Deficit Hyperactivity Problems (ADHP) scale, which comprises 7 items (i.e., fails to finish things, can't concentrate, can't sit still, impulsive, inattentive or easily distracted, talks too much, and unusually loud) that were viewed as very consistent with the DSM-IV diagnostic category of ADHD. Further validation work (Achenbach & Rescorla, 2001) indicated that the ADHP scale accurately captures inattentive/hyperactive symptoms and diagnoses (*r*s were .80 and .60, respectively). Parents rated how often particular behaviors occurred during the past six months using a 3-point Likert scale (0=never true; 1=sometimes/somewhat true; 2=often true). Internal consistency estimates were adequate for both mother and father informant-reports ($\alpha=.80$).

Consistent with manual recommendations (Achenbach & Rescorla, 2001), analyses were conducted on the raw scale scores. To adjust for positive skew, the ADHP scale was log-transformed prior to analysis to better approximate normality. Because prior twin and adoption studies (see Appendix B in Burt, 2009) have indicated that ADHD heritability estimates do not vary significantly across sex, sex was regressed out of ADHP prior to analysis (McGue & Bouchard, 1984). Maternal-reported CBCL data was available for 100% of the twins; paternal-reported CBCL data was available on 87% of the twins. When only one informant-report was available, that report was used. When both informant reports were available, data were averaged across maternal- and paternal-informant reports, creating composites of twin ADHP. The use of this combined informant approach allowed for a more

²Teacher reports of child ADHD were also collected in both samples. These informant-reports were correlated with mother and father reports of child ADHD (*r*'s ranged from .24 to .35 across samples and informants), magnitudes that are fully consistent with typical cross-informant correlations (Achenbach, McConaughy, & Howell, 1987). Unfortunately, teacher reports of child ADHD were not associated with maternal or paternal reports of their own ADHD in either sample (*r*'s were .00 and .10, respectively, in sample 1, both ns, and .02 and -.01, respectively, in sample 2, both ns). They are thus inappropriate for analysis using the nuclear twin family model, as additive genetic effects would be estimated to be zero under this scenario (an implausible result given the findings presented here, the results of prior twin and adoption studies (Burt, 2009), and the meta-analytically confirmed association of specific candidate genes with ADHD (e.g., DAT1; Faraone & Khan, 2006)). Future studies should explore the role of informant effects on results obtained from the nuclear twin family model.

complete assessment of twin symptomatology than would the use of either informant alone (Achenbach et al., 1987).

Parents also completed the Adult Self-Report (ASR; Achenbach & Rescorla, 2003), which includes a 13-item ADHP scale. Items included most of those listed above for the CBCL ADHP scale, as well as additional items specific to adult ADHD (e.g., is disorganized, loses things, poor work performance). As with the CBCL, items on the ASR scale were viewed as “very consistent” with the DSM-IV diagnostic category of ADHD by at least 13 of 21 highly experienced adult psychologists and psychiatrists (mean years of experience was 17.8). Internal consistency reliabilities were adequate for mothers and fathers ($\alpha = .79$ and $.78$, respectively). Data were again log-transformed prior to analysis to better approximate normality. In keeping with the parameterization of the model (described below), ADHP data were omitted for those parent figures who did not share 50% of their genes with the twins (i.e., rearing grandparents and rearing step-parents). Data were thus available on 99% of mothers, and 86% of fathers.

Sample 2—Mothers and fathers in the PETSS completed the DuPaul ADHD Rating Scale IV (DuPaul et al., 1998) separately for each twin. The DuPaul ADHD scale contains 18 items assessing the diagnostic criteria of the DSM-IV (e.g., is easily distracted). Each item was rated on a 4-point scale: 0 (never or rarely), 1 (sometimes), 2 (often), 3 (very often). Internal consistency estimates were adequate for both mother and father informant-reports ($\alpha = .92$). Maternal-reported DuPaul data was available for 93% of the twins; paternal-reported data was available on 79% of the twins. As before, when both informant reports were available, data were averaged across maternal- and paternal-informant reports, creating composites of twin ADHD. When only one informant-report was available, that report was used. This approach yielded data for 96.3% of the sample. The resulting scale was more or less normally-distributed (skew = $.70$), and thus the raw data were analyzed here. Sex was again regressed out of the twin data prior to analysis (McGue & Bouchard, 1984).

Parents also completed the 18-item Adult ADHD Self-Report Scale (ASRS; Kessler et al., 2005), which references the DSM-IV ADHD criteria, albeit with items specific to adult manifestations of ADHD (e.g., How often are you distracted by activity or noise around you?; How often do you have problems remembering appointments or obligations?). Each item was rated on a 5-point scale: 0 (never), 1 (rarely), 2 (sometimes), 3 (often), 4 (very often). Internal consistency reliabilities were adequate for mothers and fathers ($\alpha = .87$ for both). Data were available for 93% of mothers, and 78% of fathers.

ANALYSES

Twin family studies make use of the proportion of genes shared between twin family members to estimate genetic and environmental influences. Monozygotic twins (MZ) share 100% of their genetic material with each other and 50% with each biological parent, whereas dizygotic twins (DZ) share an average of 50% of their segregating genetic material with each other and with each biological parent. Utilizing these genetic proportions, the variance within observed behaviors or characteristics (i.e., phenotypes) can be partitioned into four components: additive genetic, dominant genetic, shared environment, and non-shared environment plus measurement error. The additive genetic component (A) is the effect of individual genes summed over loci, and acts to increase familial correlations (either between twin siblings or between parents and their biological children) relative to the proportion of genes shared. Dominant genetic effects index non-additive or allele-to-allele interactive effects at a single genetic locus (e.g., the interaction between dominant and recessive alleles). Because they involve interactions between two alleles (one inherited from each parent), dominant genetic influences do not contribute to similarity between parents

and their biological children, and moreover, yield MZ correlations that are more than twice as large as those seen in DZ twins. The shared environment (C) is that part of the environment common to family members that acts to make them similar to each other regardless of the proportion of genes shared. The non-shared environment (E) encompasses environmental factors (and measurement error) that differentiate family members. More information on genetically-informative studies is provided elsewhere (Plomin, DeFries, McClearn, & McGuffin, 2008).

In the nuclear twin family model, parent self-report data is added to the classical twin design (see Figure 1), thereby expanding the unnested ACE and ADE models into a fully identified ADCE model. The nuclear twin family model also allows researchers to clarify the role of shared environmental influences (should there be any) on child attention problems. In particular, we are able to disambiguate shared environmental influences into those shared by siblings (S; sibling environmental variance; e.g., peers, school, parenting style) and those passed via vertical “cultural transmission” between parents and their offspring (F; familial environmental variance; e.g., socioeconomic status, social mores). Passive gene-environment correlations are captured in the covariance between A and F (i.e., w in Figure 1). Unfortunately, because there is not enough information in the data to simultaneously estimate D, S, and F effects, we are required to fix one of these estimates to zero. All permutations of these models were fitted here.

Mx, a structural-equation modeling program (Neale, Boker, Xie, & Maes, 2003), was used to perform the model-fitting analyses. Because of the small amount of missing data, we made use of Full-Information Maximum-Likelihood raw data techniques, which produce less biased and more efficient and consistent estimates than pairwise or listwise deletion in the face of missing data. When fitting models to raw data, variances, covariances, and means are first freely estimated to get a baseline index of fit (minus twice the log-likelihood; $-2\ln L$). Model fit for the more restrictive biometric models was then evaluated using four information theoretic indexes that balance overall fit with model parsimony: the Akaike’s Information Criterion (AIC; Akaike, 1987), the Bayesian Information Criteria (BIC; Raftery, 1995), the sample-size adjusted Bayesian Information Criterion (SABIC; Sclove, 1987), and the Deviance Information Criterion (DIC; Spiegelhalter, Best, Carlin, & Van Der Linde, 2002). The lowest or most negative AIC, BIC, SABIC, and DIC among a series of nested models is considered best. Because fit indices do not always agree (they place different values on parsimony, among other things), we reasoned that the best fitting model should yield lower or more negative values for at least 3 of the 4 fit indices (as done in Hicks, South, DiRago, Iacono, & McGue, 2009).

RESULTS

Descriptives

Descriptive statistics of central tendency and range are presented in Table I, separately by sex and sample. As seen there, women and men reported equivalent levels of ADHP/ADHD in both samples. However, mean levels of twin ADHP/ADHD varied significantly across sex, such that boys evidenced higher rates of these behaviors than did girls ($p < .01$).

Correlations

Phenotypic and intraclass correlations for both samples are presented in Table II. As expected (Humbad, Donnellan, Iacono, & Burt, 2010), there was evidence of modest assortative mating across both samples, such that spouses were more similar in their inattentive and hyperactive behaviors than would be expected by chance (spousal $r = .14$ and $.18$ in samples 1 and 2, respectively, both $p < .05$). Similarly, parental ADHP/ADHD was

moderately correlated with twin ADHP/ADHD across both samples (r s range from .22–.37, all $p < .01$). Such results are consistent with the presence of additive genetic and/or familial environmental influences on the etiology of ADHP/ADHD.

Intraclass correlations were calculated using the double-entry method, which removes the variance associated with the ordering of siblings within a pair. These correlations offer a preliminary indication of genetic and environmental influences on twin ADHP/ADHD: higher MZ than DZ correlations implicate genetic influences whereas equivalent MZ and DZ correlations implicate shared environmental influences. The MZ correlations were at least twice as large as their corresponding DZ correlations, suggesting that additive and perhaps dominant genetic effects are important for ADHP/ADHD. There was little evidence of shared environmental influences in either sample.

Model fitting results

Test statistics for a series of nested nuclear twin family models are reported in Table III, separately by sample. As seen there, the AE and ADE models provided the best fit to the data in samples 1 and 2, respectively. Such findings indicate that additive genetic, non-shared environmental, and (at least in sample 2) dominant genetic influences all contribute to the etiology of ADHP/ADHD. Shared environmental influences, both S and F, could be omitted across both samples without a significant decrement in model fit.

Parameter estimates from the three “full” models and the best-fitting sub-models (across both samples, for comparison purposes) are presented in Table IV. As can be seen there, the S and F parameters were estimated to be at or near zero and were generally non-significant across both samples, a finding that held regardless of whether D was also estimated. Such findings are clearly in keeping with those reported in Burt (2009; 2010) and in Nikolas & Burt (2010), and suggest that the assumptions of the classical twin model did not unduly influence prior meta-analytic conclusions as they relate to ADHP/ADHD. Dominant genetic influences were significant in only one of the two samples (a finding that likely reflects the smaller sample size in sample 1), but were generally estimated to account for 10–19% of the variance. Additive genetic effects were moderate-to-large in magnitude across both samples. Non-shared environmental influences were consistently moderate in magnitude.

DISCUSSION

The goal of the current study was to constructively replicate and extend prior meta-analytic findings (Burt, 2010) indicating that dominant genetic influences do not account for the absence of shared environmental influences on ADHD, as reported in two prior meta-analyses (Burt, 2009; Nikolas & Burt, 2010). To do so, we made use of the nuclear twin family model, which is far better suited to the simultaneous detection of dominant genetic and shared environmental influences than is the classical twin model. Results across both samples were fully consistent with prior work. Shared environmental influences on ADHD could uniformly be constrained to zero without a decrement in model fit. Moreover, even when estimated, they uniformly accounted for less than 5% of the variance, regardless of whether D was also estimated. In short, the limitations of the classical twin model do not appear to account for the absence of shared environmental influences previously observed for ADHD.

There are several limitations to the current study. The twin samples examined here, for example, ranged in age from 5- to 11-years old. Our results should thus be considered specific to the developmental period of middle childhood. That said, because shared environmental influences are thought to generally decrease from childhood to adulthood, it seems likely that the absence of C observed here would persist. Similarly, it remains unclear

how these results may generalize to more deviant populations. Consistent with the epidemiological nature of these samples, rates of ADHD were relatively low (roughly 5–10%). Future research should seek to extend current findings to higher risk samples. Indeed, it may well be the case that there is detectable C in such samples, as available research on conduct problems has indicated that shared environmental influences are larger in high risk environments and lower in average and low risk environments (Tuvblad, Grann, & Lichtenstein, 2006).

Next, the use of small sample sizes (such as that in sample 1) is an important limitation when studying shared environmental influences alongside genetic influences, particularly when the former are modest in magnitude (Martin, Eaves, Kearsley, & Davies, 1978). In the current MSUTR study, however, our ability to detect shared environmental influences was hampered more by the absence of estimable C than by our smallish sample size (i.e., C accounted for less than 1% of the variance in those data). Moreover, the fact that these findings persisted to sample 2 (N=854 twin pairs) further indicates that the absence of C detected here is robust to sample size considerations. Similarly, the current study made use of dimensional questionnaires rather than diagnoses or diagnostic symptom counts. As with the use of the parental report of twin, prior meta-analytic work has strongly suggested that estimates of C are larger (and estimates of D are smaller) when using dimensional questionnaires as opposed to diagnostic assessments, findings that are thought to stem largely from differences in skewness across the two methodologies (Burt, 2009). Indeed, the relatively low estimates of D obtained here may be a function of our use of more normally distributed questionnaires. Even so, we would argue that our inability to detect shared sibling environmental influences when using dimensional questionnaires offers particularly compelling evidence against the notion that D is masking the presence of C. Regardless, because diagnoses are considered the gold standard in ADHD research, future studies should seek to extend these results to diagnostic data.

Next, a critical issue facing all nuclear twin family studies is that of etiological development in the phenotype with age. Specifically, there may be developmental changes in etiology over time, such that different genes “turn on” over the course of development. These gene-by-age interactions would act to decrease genetic covariation between parents and their children but not between twin siblings, since the latter are necessarily the same age. And because both A and D are shared by twin siblings, whereas only A is shared by parents and their children (see Figure I), decreasing the genetic covariation across, but not within, generations should result in overestimates of D/underestimates of A. It is thus theoretically possible that the dominant genetic influences discovered for ADHD are in fact a by-product of gene-by-age interactions, particularly since longitudinal twin studies have suggested age specific effects on ADHD (Chang, Lichtenstein, Asherson, & Larsson, in press; Larsson, Lichtenstein, & Larsson, 2006). That said, classical twin studies of ADHD, which are not influenced by gene-by-age interactions, have also found evidence of significant dominant influences on ADHD (Burt, 2009; Nikolas & Burt, 2010), arguing against the notion that our findings of D stem solely from gene-by-age interactions. Nevertheless, future studies with other designs are necessary to completely rule out the possibility that the D identified here stems from gene-by-age interactions.

Finally, parents reported on symptom presence in themselves and their twins in both samples. Although the use of parental informant reports was generally advantageous in that it constituted a particularly strong test of our hypothesis (i.e., estimates of C appear to be larger for parental informant reports as compared to any other informant; Burt, 2009), there are three complications that arise from this decision. First, the use of both parent report of self and parent report of twin raises the specter of shared “method” (i.e., informant) effects. Because we would generally expect shared informant effects to increase estimates of C

(since this sort of shared parent-child method variance should be invariant across zygosity), however, the absence of shared environmental influences in our results serves to undercut this possibility.

The second issue raised by our use of parental informant reports relates to rater contrast effects (i.e., when parents overemphasize differences in hyperactivity in their DZ twins relative to their MZ twins). As noted by Wood et al. (2010), rater contrast can mimic non-additive genetic effects by accentuating the difference in similarity between MZ and DZ twins (and in this way, inhibit our ability to detect C). Importantly, however, rater contrast effects cannot explain the absence of C when using separate informant-reports for each twin (e.g., when each twin rates only themselves, and those reports are correlated, rater contrast cannot be influencing the results). When child self-reports of ADHD were meta-analyzed in Burt (2010), shared environmental influences were estimated to be zero or near-zero (1% of the variance) and were not significant. To the extent that child self-reports are a valid way to measure ADHD (still an open question; Hart, Lahey, Loeber, & Hanson, 1994; Jensen et al., 1999), such findings argue against rater contrast as an explanation for the lack of C in Burt (2009). To confirm that our results were not a function of rater contrast, we conducted post-hoc analyses using different informants for each twin (i.e., mother report of twin 1 and father report of twin 2), thereby circumventing the possibility of rater contrast. Given our objectives, we specifically focused on the results of the ASFE, ASE, and AFE models. S was uniformly estimated to be zero across all relevant models in both samples. F was also observed to be quite small (1% of the variance in sample 2, and 3.3% in sample 1) and was uniformly non-significant. In short, there is little evidence that rater contrast effects explain the absence of shared environmental influences observed in these data.

Third, recent work has suggested that adults with ADHD may underreport the severity of their symptoms, serving to increase measurement error and thus decrease estimates of genetic influences (Saviouk et al., 2011). Should this interpretation of the low heritabilities of adult ADHD be correct, it could suggest that results from the nuclear twin family model are themselves biased in some way (e.g., D could be over- or under-estimated). Unfortunately, the extent to which the current results are influenced by decreased genetic influences on adult ADHD are not clear, and moreover, cannot be examined here (as the parents are not twins). Future research should explore this possibility in more depth.

Conclusions

Wood and colleagues (2010) argued that, because shared environmental effects are confounded with dominant genetic influences in the classical twin design, prior twin studies (and by extension, meta-analyses of those twin studies) have not been able to detect these effects. The current study sought to address this question via the use of a nuclear twin family model, which allows researchers to simultaneously estimate dominant genetic and shared environmental influences. Results revealed that, regardless of the specific nuclear twin family model fitted to the data, shared environmental influences accounted for less than 5% of the variance in ADHD. Such findings argue against the notion that dominant genetic influences are masking the presence of shared environmental influences on ADHD, thereby constructively replicating the adoption study results of Burt (2010).

For those who hope to identify specific environmental factors contributing to ADHD, such findings could be considered a mixed bag. On the one hand, the likely absence of shared environmental influences on ADHD, as implied by these results, means that specific environmental factors may be more difficult to identify. Indeed, one key advantage of shared environmental influences is that they have (thus far) proven to be “identifiable”. For example, several independent studies have now suggested that the association between parental divorce and adolescent behavior problems is largely shared environmental in origin

(see, for example; Burt, Barnes, McGue, & Iacono, 2008), as is the association between the parent-child relationship and adolescent externalizing, at least in part (see, for example; Klahr, McGue, Iacono, & Burt, 2011; Narusyte et al., 2011).

Extant research has also suggested that shared environmental influences may persist over time (at least during childhood and adolescence). Tuvblad and colleagues (2011) examined the etiology of antisocial behavior across ages 8, 13, 16, and 19 years, and found that shared environmental influences accounted for 51% of their stability over time (Tuvblad, Narusyte, Grann, Sarnecki, & Lichtenstein, 2011). A longitudinal adoption study of adolescent antisocial behavior similarly suggested that shared environmental influences were particularly critical for stability over time (Burt, McGue, & Iacono, 2010). Such findings are collectively consistent with the notion that, although they appear to dissipate by adulthood, the shared environment is a persistent and identifiable source of individual differences in most forms of psychopathology prior to adulthood.

Non-shared environmental influences, by contrast, are less stable over time prior to adulthood and instead appear to be largely assessment-specific (Bartels et al., 2004; Burt et al., 2010). Thus, rather than being a function of important and identifiable environmental influences that serve to differentiate siblings prior to adulthood, the non-shared environment may be largely comprised of idiosyncratic experiences with negligible long-term implications (Burt, 2009; Turkheimer & Waldron, 2000). Moreover, similar to individual genetic variants (but in sharp contrast to specific shared environmental influences), specific non-shared environmental risk factors typically account for no more than 2% of the variance in the outcome (Turkheimer & Waldron, 2000). The finding that environmental influences on ADHD may be primarily non-shared environmental in origin thus does not bode particularly well for efforts to identify specific environmental influences.

Even so, the small literature examining non-shared environmental influences on ADHD (which, as noted previously, are moderate in magnitude) has strongly suggested that these effects reflect more than measurement error. For example, work examining identical twins discordant for ADHD found that the affected twins had lower birth weights and delayed physical and motor maturation as compared to their unaffected co-twins (Hultman et al., 2007; Lehn et al., 2007). Studies of neural anatomy have further indicated that the caudate is significantly smaller in affected as compared to unaffected twins (Castellanos et al., 2003). These differences across identical twins indicate the presence of meaningful contributions from the child-specific environment in the development of ADHD. Thus, despite the generally lackluster performance of the non-shared environment in studies of specific environmental influences, ADHD may well represent an exception. Future work should specifically explore the role of non-shared environmental influences on ADHD in particular.

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Figure I. Path diagram of a Univariate Nuclear Twin Family Model

Note. The variance in the phenotype (P) is parsed into that which is due to additive genetic effects (A), dominant genetic effects (D), sibling environmental influences (S), familial environmental influences (F; more specifically, m is estimated and f is fixed to 1), and non-shared environmental effects (E). μ indexes primary phenotypic assortment (i.e., assortative mating) between the twin parents, and w indexes the covariance between A and F. The staggered paths from parent A's to twin A's (i.e., $-\rightarrow$) are fixed to 0.5. Paths are squared to estimate the proportion of variance accounted for. D, S, and F effects cannot be estimated simultaneously (only two of the three can be estimated).

Table 1

Descriptives.

	Females					Males					Cohen's <i>d</i> effect size
	Mean (SD)	n	% above cut-off	Observed range	Possible range	Mean (SD)	n	% above cut-off	Observed range	Possible range	
ADHP parent, sample 1	4.21 (3.55)	308	4.7	0–17	0–26	4.34 (3.44)	269	5.5	0–17	0–26	--
ADHD parent, sample 2	19.62 (8.87)	796	5.3	0–60	0–72	20.16 (8.52)	669	5.5	0–48	0–72	--
ADHP child, sample 1	2.65 (2.51)	296	9.2	0–14	0–14	3.37 (2.66)	328	10.7	0–12	0–14	-.29**
ADHD child, sample 2	9.45 (6.24)	822	4.6	0–31	0–54	11.63 (6.83)	823	6.7	0–39.5	0–54	-.33**

Note. ADHP refers to the DSM-oriented Attention Deficit Hyperactivity Problems scale (n=13 items for parent report of self and 7 items for parent report of twin) assessed as part of the Michigan State University Twin Registry (i.e., sample 1). ADHD refers to the 18-item Attention Deficit Hyperactivity Disorder scales assessed as part of the PreSchool Twin Study in Sweden (i.e., sample 2). The % above cut-off refers to the proportion of participants who scored in the marginal- or clinically-significant range on the relevant instruments (based on their internal norms). For parent reports of the twins, the % refers to those in the marginal or clinical range by either mother or father report. Maternal and paternal reports of their own ADHP/ADHD were statistically compared using paired sample t-tests. Fathers and mothers reported equivalent levels of ADHP/ADHD across both samples. Composite reports of twin ADHP/ADHD were compared across boys and girls using independent samples t-tests.

p<.01

Table II

Correlations.

	1. Mother report of own ADHP/ADHD	2. Father report of own ADHP/ADHD	3. Combined informant-reports of twin 1	4. Combined informant-reports of twin 2
1.	---	.18 **	.34 **	.37 **
2.	.14 *	---	.27 **	.31 **
3.	.22 **	.30 **	---	.72 ** / .37 **
4.	.26 **	.31 **	.54 ** / .20 **	---

Note. ADHP refers to the DSM-oriented Attention Deficit Hyperactivity Problems scale assessed as part of the Michigan State University Twin Registry (i.e., sample 1). ADHD refers to the Attention Deficit Hyperactivity Disorder scales assessed as part of the PrEschool Twin Study in Sweden (i.e., sample 2). Intraclass correlations are presented separately across zygosity (i.e., rMZ/rDZ) and are highlighted with bold font. Correlations in samples 1 and 2 are presented below and above the diagonal, respectively. Correlations that are significantly greater than zero are indicated as follows:

**
p<.01,

*
p<.05

Nuclear twin family design model fitting results.

Table III

Sample	Model	-2lnL	df	AIC	BIC	SABIC	DIC
MSUTR sample 1	ADSE	3262.86	1189	884.86	-1782.78	102.77	-690.17
	ASFE	3262.53	1189	884.53	-1782.95	102.60	-690.33
	ADFE	3262.53	1189	884.53	-1782.95	102.60	-690.33
	ADE	3262.86	1190	882.86	-1785.66	101.49	-692.12
	AFE	3262.53	1190	882.53	-1785.82	101.32	-692.29
	ASE	3264.62	1190	884.62	-1784.78	102.36	-691.24
	AE	3264.66	1191	882.66	-1787.63	101.10	-693.17
	ADSE	8300.79	3104	2092.79	-6268.12	-1339.54	-3415.73
PETSS sample 2	ASFE	8300.79	3104	2092.79	-6268.12	-1339.54	-3415.73
	ADFE	8300.85	3104	2092.85	-6268.08	-1339.51	-3415.70
	ADE	8300.93	3105	2090.93	-6271.40	-1341.24	-3418.10
	AFE	8302.63	3105	2092.63	-6270.55	-1340.39	-3417.25
	ASE	8309.31	3105	2099.31	-6267.21	-1337.05	-3413.91
	AE	8328.61	3106	2116.61	-6260.92	-1329.17	-3406.70

Note. MSUTR and PETSS represent the Michigan State University Twin Registry and the PREschool Twin Study in Sweden, respectively. Additive genetic, dominant genetic, environmental influences shared by siblings, environmental influences passed via cultural transmission from parents to offspring, and non-shared environmental influences are represented with A, D, S, F, and E, respectively. The best fitting model for each sample is highlighted in bold font, and is indicated by the lowest AIC (Akaike's Information Criterion), BIC (Bayesian Information Criterion), SABIC (sample size adjusted Bayesian Information Criterion), and DIC (Deviance Information Criterion) values for at least 3 of the 4 fit indices.

Table IV

Nuclear twin family design heritability estimates.

Sample	Model	% A	% D	% S	% F	% E	A-F covariance
MSUTR sample 1	ADSE	48.1 * (36.5, 58.8)	9.5 ^{ns} (0, 23.6)	0.0 ^{ns} (0, 8.0)	---	42.4 * (33.3, 54.2)	---
	ASFE	67.3 * (42.9, 86.8)	---	0.0 ^{ns} (0, 7.6)	1.2 ^{ns} (0, 5.6)	41.6 * (32.6, 53.6)	-10.1 ^{ns} (-26.0, 5.6)
	ADFE	67.3 * (42.9, 86.8)	0.0 ^{ns} (0, 56.3)	---	1.2 ^{ns} (0, 5.6)	41.6 * (32.6, 53.6)	-10.1 ^{ns} (-26.0, 5.6)
	ADE	48.1 * (36.5, 58.8)	9.5 ^{ns} (0, 23.6)	---	---	42.4 * (33.3, 54.2)	---
	AE**	52.5 * (43.3, 60.9)	---	---	---	47.5 * (39.1, 56.7)	---
PETSS sample 2	ADSE	55.6 * (49.1, 61.6)	17.1 * (5.6, 25.9)	1.7 ^{ns} (0, 10.0)	---	25.7 * (21.7, 30.7)	---
	ASFE	86.2 * (67.4, 1.06)	---	4.6 ^{ns} (0, 11.2)	2.6 * (0.3, 7.2)	25.7 * (21.7, 30.7)	-19.1 * (-36.2, -5.5)
	ADFE	37.5 ^{ns} (0, 1.06)	28.5 ^{ns} (0, 60.3)	---	1.3 ^{ns} (0, 21.7)	25.6 * (21.7, 30.4)	7.1 ^{ns} (-36.4, 11.3)
	ADE**	55.5 * (49.1, 61.5)	18.9 * (11.9, 26.0)	---	---	25.6 * (21.7, 30.4)	---
	AE	64.3 * (59.2, 69.0)	---	---	---	35.7 * (31.0, 40.8)	---

Note. MSUTR and PETSS represent the Michigan State University Twin Registry and the PREschool Twin Study in Sweden, respectively. Additive genetic, dominant genetic, environmental influences shared by siblings, environmental influences passed via cultural transmission from parents to offspring, and non-shared environmental influences are represented with A, D, S, F, and E, respectively. The covariances between A and F are also presented when both A and F are estimated in the model (they are necessarily zero otherwise). Each row thus sums to 100%. The best-fitting model is indicated with a double asterisk (**). 95% confidence intervals are presented below each estimate.

* p<.05