

Offspring ADHD as a Risk Factor for Parental Marital Problems: Controls for Genetic and Environmental Confounds

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Background: Previous studies have found that child attention-deficit/hyperactivity disorder (ADHD) is associated with more parental marital problems. However, the reasons for this association are unclear. The association might be due to genetic or environmental confounds that contribute to both marital problems and ADHD. **Method:** Data were drawn from the Australian Twin Registry, including 1,296 individual twins, their spouses, and offspring. We studied adult twins who were discordant for offspring ADHD. Using a discordant twin pairs design, we examined the extent to which genetic and environmental confounds, as well as measured parental and offspring characteristics, explain the ADHD–marital problems association. **Results:** Offspring ADHD predicted parental divorce and marital conflict. The associations were also robust when comparing differentially exposed identical twins to control for unmeasured genetic and environmental factors, when controlling for measured maternal and paternal psychopathology, when restricting the sample based on timing of parental divorce and ADHD onset, and when controlling for other forms of offspring psychopathology. Each of these controls rules out alternative explanations for the association. **Conclusion:** The results of the current study converge with those of prior research in suggesting that factors directly associated with offspring ADHD increase parental marital problems.

■ **Keywords:** behavioral genetics, attention-deficit/hyperactivity disorder, marital conflict, divorce

Numerous studies have found associations between offspring attention-deficit/hyperactivity disorder (ADHD) and parents’ marital functioning (Johnston & Mash, 2001). Parents of children with even mild ADHD symptoms report less marital satisfaction than parents of non-clinical children (Murphy & Barkley, 1996). Parents of children with ADHD have more negative child-rearing discussions than other parents (Johnston & Behrenz, 1993). Similarly, families of children with ADHD have higher rates of marital separation and divorce than families without ADHD (Brown & Pacini, 1989). Although these studies indicate that parents’ marital problems and offspring ADHD are correlated, they do not clarify the explanatory mechanisms.

Direction of Effects in the ADHD–Marital Problems Association

One possibility is that children’s ADHD causes marital problems. Children’s ADHD symptoms may create stress for parents, which may impair their functioning in a variety of domains, including marital functioning. Marital partners may also disagree over optimal parenting strategies

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for children with ADHD, which may impair marital functioning. Alternatively, interparental problems might cause children's ADHD. Few studies have utilized methodologies that allow examination of the direction of effects, however. Longitudinal and experimental or quasi-experimental designs can be helpful in this regard, but very few studies have used such approaches. We focus on studies that used such approaches. Results of one longitudinal study were consistent with a child effects model. Compared with parents of children without ADHD, parents of children with ADHD were more likely to divorce, and their latency to divorce was shorter (Wymbs et al., 2008). A study utilizing an experimental design also revealed results consistent with child effects. Spouses were randomly assigned to interact with a child confederate who was trained to engage in either typical or disruptive behavior (Wymbs & Pelham, 2010). Marital partners who interacted with a disruptive confederate had poorer interactions with one another than partners who interacted with a non-disruptive confederate.

Wymbs and Pelham's (2010) study provides perhaps the strongest evidence of child ADHD effects on marital conflict. Although experiments are very powerful, however, they are also subject to bias (McGue et al., 2010). The very experimental control that allowed the marital-to-child model to be ruled out in Wymbs and Pelham's (2010) study compromised ecological validity. Participants were placed in an artificial situation, interacting with a misbehaving child they did not know. Experiments have many advantages, but given their limitations, additional investigation is needed, using different methodological approaches (with different strengths and weaknesses) to test alternative explanations (Rutter et al., 2001).

Genetic and Environmental Confounds

One alternative explanation involves genetic or environmental confounds, that is, third variables that might account for the ADHD–marital problems association. Environmental confounds, such as financial difficulties, could exacerbate both marital problems and offspring ADHD, but potential environmental confounds have been under-examined. It is also possible that genetic factors related to ADHD in the parent generation could influence parents' marital problems and be passed on to offspring, a passive gene–environment correlation (Scarr & McCartney, 1983). ADHD is highly heritable (Nikolas & Burt, 2010), and there is a genetic link between ADHD and antisocial disorders (Faraone et al., 1997). Researchers have also found associations between ADHD, antisocial disorders, and marital problems (Lahey et al., 1988). Thus, a spurious statistical association between marital problems and offspring ADHD could appear when the ADHD–marital problems association is tested without controlling for genetic confounds.

Providing an initial test of this possibility, Wymbs et al. (2008) also tested whether fathers' antisocial behavior and other parental characteristics predict divorce. Paternal an-

tisocial behavior predicted divorce, and with this variable included, child ADHD no longer predicted divorce. This result suggests that the ADHD–marital problems association might reflect genetic and environmental factors that increase both parents' risk for marital problems and children's risk for ADHD.

The Current Study

The purpose of this study is to examine mechanisms underlying the association between children's ADHD and parental marital problems. We examined the degree to which this association is confounded by unmeasured genetic or environmental factors and by measured parental psychopathology. We hypothesized that such confounds were largely responsible for the association between ADHD and marital problems. We hypothesized that the ADHD–marital problems association was also partially due to effects of marital problems on offspring ADHD. We, therefore, also examined whether this association would remain when using information about the timing of marital problems and ADHD. We expected that after controlling for genetic factors, shared environment, and measured parental psychopathology, as well as examining only the sub-sample in which marital separation and divorce did not occur prior to offspring ADHD onset, ADHD would no longer predict marital problems.

Materials and Methods

Participants

Participants were drawn from the Australian National Twin Register. Three major surveys were conducted: (1) a mailed survey in 1981 ($n = 8,183$), (2) a mailed follow-up survey from 1988 to 1989, and (3) a telephone interview from 1992 to 1993. Assessment of 3,844 spouses was completed via telephone interview in 1994. Data for the current study were drawn from the 1992–1993 twin interviews and the 1994 spouse interviews. Additional information about the sample, its similarity to the initial sample, and data collection is available in Heath et al. (1997) and Slutske et al. (1997).

Twins were selected for the current study if they had a biological child born between 1964 and 1983, and if they or their co-twin had a history of alcohol dependence, conduct disorder (CD), major depressive disorder, or divorce. A control group of twins with no history of alcohol dependence, CD, major depression, or divorce was also randomly selected.

Twins in the current study consisted of 1,296 individuals (MZ females = 445, MZ males = 217; DZ females = 415, DZ males = 219; overall 66% female) nested within 889 twin pairs. Zygosity was determined via questionnaire reports of twins' physical similarity and how frequently they were mistaken for one another, which has been shown to be valid (Slutske et al., 1997). The sample included 407 complete twin pairs (reflecting non-participation of some individual twins within co-twin pairs).

Spouses and offspring of all selected twins were targeted for participation. Spouses of 1,045 twins participated. Offspring ($n = 2,554$; female = 50.6%) participated via telephone interview in 1998. To establish reliability, 176 offspring were re-interviewed approximately 1 year later. The institutional review boards at the authors' institutions approved the study, and informed consent was obtained from all participants.

At the time of the study, mothers' mean age was 45.34 ($SD = 7.17$), fathers' mean age was 48.32 ($SD = 8.00$), and offspring mean age was 25.06 years ($SD = 5.65$; range = 25).

Measures

Twins and their spouses completed the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; see Bucholz et al., 1994, for additional description) and offspring completed the offspring SSAGA.

ADHD. The offspring SSAGA included *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) ADHD items. Offspring provided retrospective self-reports for ages of 6–12. Each item was answered yes/no. The number of items endorsed was summed; sums ranged from 0 to 18. The measure was designed to assess the *DSM-IV* symptoms of ADHD. Test–retest reliability for ADHD symptoms in the re-interviewed sub-sample was high ($r = .75$, $p < .001$). Summing ADHD scores across siblings, the mean number of symptoms per nuclear family was 3.76 ($SD = 4.56$). Nuclear family symptom sums were used in model testing, with statistical controls for the number of children per family.

Offspring also indicated whether they had been diagnosed with ADHD by a mental health professional; 132 offspring (5.20%) reported an ADHD diagnosis, consistent with worldwide ADHD prevalence rates of 5.29% (Polanczyk et al., 2007). There were 132 offspring within 125 nuclear families (9.65%) with at least one offspring diagnosed with ADHD. Of the 132 offspring reporting an ADHD diagnosis, 60% ($n = 79$) met criteria for the Predominantly Inattentive Type (6+ inattention symptoms), 23% ($n = 30$) met criteria for the Predominantly Hyperactive-Impulsive Type (6+ hyperactivity/impulsivity symptoms), and 17% ($n = 23$) met criteria for the Combined Type (6+ inattention symptoms and 6+ hyperactivity/impulsivity symptoms). Regarding comorbidities, 33% of offspring reporting an ADHD diagnosis ($n = 44$) also reported four or more symptoms of oppositional defiant disorder (ODD) (the basis for an ODD diagnosis); by comparison, of the 2,422 offspring not reporting an ADHD diagnosis, only 5% ($n = 122$) reported four or more symptoms of ODD. Similarly, 40% ($n = 53$) of offspring diagnosed with ADHD reported three or more symptoms of CD (the basis for a diagnosis of CD), compared with only 11% ($n = 268$) of those without an ADHD diagnosis.

Offspring also reported their age at onset of ADHD symptoms, although this information was missing for 21 offspring. The mean age at onset was 7.33 years ($SD = 2.27$).

Interparental conflict. Offspring answered two questions about interparental conflict occurring when the offspring was 6–13 years old. One item assessed frequency of conflict in the offspring's presence, and was completed using a 4-point scale ranging from 1 (*often*) to 4 (*never*). The other item assessed amount of conflict, and was completed using a 4-point scale ranging from 1 (*a lot*) to 4 (*none*). Cronbach's alpha for the two items was 0.85. Approximately, 30% of offspring reported their parents had conflict 'Sometimes' or 'Often', and 20% reported 'Some' or 'A lot' of conflict between their parents, consistent with disharmony rates in other community samples (Beach et al., 2005). Responses were reverse-scaled and summed (see Harden et al., 2007, for score distribution information and comparison with other samples). Scores were averaged across siblings within nuclear families; nuclear family scores had a mean of 4.03 ($SD = 1.52$). The averages were standardized to facilitate interpretation of the results. Among offspring who were re-interviewed, test–retest reliability was high ($r = .82$, $p < .001$). Additionally, agreement between siblings was high; Cronbach's alpha was 0.73 for two-sibling families, and higher for larger families. Further, siblings' reports had correlations of $r = .58$, $p < .001$ for reports of firstborns with thirdborns, and larger for other sibling pairs.

Marital separation/divorce. Offspring reported parental marital separations and divorces, and their own age at the time of separation/divorce. Offspring in 338 twin nuclear families reported separation/divorce occurring in their lifetime (a rate of 26%). An Australian survey revealed that 25% of individuals born between 1972 and 1989 (similar to the era when offspring in the current study were born) reported their parents divorced or permanently separated during their childhood (Australian Bureau of Statistics, 2010), suggesting the prevalence of serious marital problems in our sample was similar to that of the overall population. Missing data precluded identifying offspring age at the time of separation/divorce for 50 offspring. For the 551 offspring (within the 338 families) who did provide this information, mean age at the time of separation/divorce was 10.99 years ($SD = 6.82$).

Parental covariates. Twins and spouses reported on lifetime symptoms of CD, alcohol problems, and major depression, and lifetime histories of ever smoking cigarettes or ever using illegal drugs. History of suicidality was assessed using a 5-point Likert scale ranging from 1 (*no thoughts or plans of suicide*) to 5 (*serious suicide attempt*) (Statham et al., 1998). Parents also reported their highest level of education on a 7-point Likert scale ranging from 1 (*less than 7 years'*

schooling) to 7 (university postgraduate training), and their age at the birth of their first child.

Other offspring disorders. Offspring completed items assessing *DSM-IV* symptoms of CD, ODD, and alcohol problems (including alcohol dependence and abuse), and items assessing lifetime diagnosis of *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; American Psychiatric Association, 1987) major depression symptoms. Offspring not endorsing either of the two core symptoms of major depression (e.g., depressed mood) were not administered the remaining depression items.

Data Analyses

We tested offspring ADHD symptoms as a predictor of parents' marital problems using Mplus (Muthén & Muthén, 1998–2007). We accounted for the nesting of the data (i.e., individual twins nested within twin pairs) in all models using a sandwich estimator. Full information maximum likelihood was used to account for missing data, and we controlled for the number of children in the nuclear family.

We ran separate models for marital conflict and separation/divorce, using linear regression for the former and logistic regression for the latter. We first computed the regressions in the entire sample (Model 1). This model tests for an ADHD–marital problems association at the phenotypic level, which compares unrelated families. Model 2 tested the same association, but added statistical controls for the measured maternal and paternal psychopathology. This model tests whether the ADHD–marital problems association remains when controlling for parental traits that could confound the association.

Next, we used discordant twin pairs analyses to test whether genetic confounds explain the ADHD–marital problems association (Model 3). The discordant twin pairs design is useful for dealing with potential genetic or shared environmental confounds (Johnson et al., 2009; McGue et al., 2010). This design facilitates comparing outcomes of co-twins who differ in their exposure to a risk factor. When comparing MZ co-twins, observed differences in outcomes cannot be due to genetic factors, because the twins are identical genetically, and the design rules out environmental factors that make twins similar (Rutter et al., 2001). This analysis is ideal for our purposes because it facilitates examination of whether offspring ADHD is associated with parents' marital problems even when controlling for potential genetic and environmental confounds.

In the discordant twin pairs analyses, we simultaneously regressed marital outcomes on the average level of ADHD in the extended family (i.e., the average ADHD level of all offspring of both twins in a pair) and on each twin's deviation from their extended family's average. This approach provides accurate within-family estimates (Carlin et al., 2005). The analyses tested differences in cousins' levels of ADHD as the predictor of the twins' marital outcomes. The regression

of marital outcomes on the average level of ADHD in the extended family (between-families regression) reflects genetic, environmental, and exposure effects; it tests whether families with higher levels of ADHD were generally more at risk for marital problems. The regression on the deviation score (within-families regression) tests whether the twin who was exposed to more offspring ADHD than the co-twin had more marital problems. Thus, differences in levels of offspring ADHD between nuclear families (comparing cousins) are tested as predictors of co-twins' marital outcomes. This test reflects effects of exposure to offspring ADHD, controlling for shared genes and shared environment. A causal association would be implicated if, within a twin pair, the twin who was exposed to more offspring ADHD had more marital problems. Shared environmental and/or genetic factors would be implicated if the twin who was exposed to more ADHD did not have more marital problems.

Next, we repeated this test, adding controls for the parental covariates (Model 4). Then, we reran the discordant twin pairs analyses using only the monozygotic twins' (MZ) data (Models 5 and 6). These tests allowed us greater control for genetic factors, but reduced the sample size considerably.

Finally, we repeated Models 1–6 using only families in which separation/divorce did not precede or co-occur with ADHD onset [$n = 1,214$ nuclear families ($n_{MZ} = 614$, $n_{DZ} = 600$)]. When offspring reports suggested different occasions of separation/divorce, we used the first occasion reported, to be conservative. These tests increase confidence regarding the direction of effects, because they excluded families in which the timing of ADHD onset and separation/divorce might be more consistent with a marital problems-to-child direction of effects than a child-to-marital problems direction. Using this sub-sample decreased the likelihood that the ADHD–marital problems association was due to an effect of marital problems on ADHD. Similar approaches have been used in other studies (e.g., Jaffee et al., 2004).

We also conducted sensitivity tests to examine the robustness of the findings from our primary models. We examined whether our findings were independent of such factors as family size, greater prevalence of ADHD in males, and other forms of offspring psychopathology. To do this, we conducted separate models testing diagnosis of ADHD as the predictor of marital problems, removing the control for the number of children in the family, adding a control for the number of male children in the family, comparing only same-sex DZ twins, and adding controls for offspring ODD, CD, alcohol problems, and major depression.

Results

Table 1 descriptively presents rates of marital problems as a function of ADHD diagnosis. Nuclear families in which

TABLE 1
Rates of Marital Problems as a Function of Offspring ADHD Diagnosis

Offspring ADHD	Marital conflict		Separation/divorce	
	Mean	N	%	N
<i>Entire sample</i>	0.00	1,139	26.1	1,296
No diagnosis	-0.01	1,032	25.4	1,171
Diagnosis	0.13	107	32.8	125
<i>All discordant twins</i>	0.03	135	28.8	146
No diagnosis	0.03	69	27.4	73
Diagnosis	0.02	66	30.1	73
<i>Discordant MZ twins</i>	0.03	75	28.0	82
No diagnosis	0.01	38	24.4	41
Diagnosis	0.06	37	31.7	41
<i>Discordant same-sex DZ twins</i>	-0.06	40	36.4	44
No diagnosis	-0.12	21	36.4	22
Diagnosis	0.01	19	36.4	22

Note: ADHD = attention-deficit/hyperactivity disorder; MZ = monozygotic; DZ = dizygotic.

at least one offspring was diagnosed with ADHD tended to have more marital problems than families without ADHD.

Primary Analyses

Regression analyses comparing unrelated families revealed that ADHD predicted more marital conflict (Table 2, Model 1). Results indicated that one additional ADHD symptom is associated with a 0.03 SD-unit increase in conflict. When controls for parental covariates were added, the association

remained (Model 2). Comparing co-twins differentially exposed to ADHD, the within-families regression was significant (Model 3). When controlling for parental covariates, the association remained in the same direction, although it was no longer significant (Model 4). The magnitude of the within-twin pair effect remained consistent when using only the MZ sample (Model 5) and with parental covariates added to the MZ model (Model 6). Results of these tests, therefore, are consistent with the inference that offspring ADHD increases parents' marital conflict, because when we controlled for genetic and shared environmental factors and measured parental characteristics, the magnitude of the within-families coefficient remained substantial.

Next, we tested models predicting separation/divorce. The comparison of unrelated families revealed that ADHD predicted separation/divorce (Table 3, Model 1), indicating that one additional symptom of ADHD is associated with a 5% increase in odds of separation/divorce. Adding parental covariates, the association was in the same direction but somewhat reduced (Model 2). Comparing co-twins differentially exposed to ADHD, results were consistent with a causal effect of ADHD on separation/divorce (Model 3). Repeating this test adding parental covariates (Model 4), using only the MZ sample (Model 5), and adding parental covariates to the MZ model (Model 6) produced similar, albeit attenuated, results. Because the regression coefficients for separation/divorce were similar in magnitude to those for marital conflict, and because relatively few couples

TABLE 2
Regression Predicting Marital Conflict

Parameter	Model 1 b (SE)	Model 2 b (SE)	Model 3 b (SE)	Model 4 b (SE)	Model 5 b (SE)	Model 6 b (SE)
Offspring ADHD symptoms						
Unrel	0.03 (0.01)***	0.02 (0.01)**				
Btwn			0.04 (0.01)**	0.02 (0.01)†	0.06 (0.02)**	0.04 (0.02)*
W/in			0.03 (0.01)*	0.02 (0.01)	0.05 (0.02)***	0.05 (0.02)**
No. children	-0.07 (0.04)†	-0.04 (0.04)	-0.07 (0.04)†	-0.04 (0.04)	-0.07 (0.05)	-0.04 (0.05)
Wife covariates						
Alcohol		0.05 (0.03)†		0.06 (0.03)†		0.04 (0.05)
Conduct		0.00 (0.05)		0.00 (0.05)		-0.03 (0.07)
Depression		0.04 (0.01)***		0.04 (0.01)***		0.04 (0.01)*
Suicidality		0.06 (0.03)†		0.06 (0.03)†		0.14 (0.05)**
Drug		-0.07 (0.10)		-0.05 (0.10)		-0.29 (0.16)†
Cigarette		-0.03 (0.07)		-0.03 (0.07)		-0.07 (0.09)
Education		0.03 (0.03)		0.03 (0.03)		-0.03 (0.04)
Age at first birth		-0.01 (0.01)		-0.01 (0.01)		-0.01 (0.02)
Husband covariates						
Alcohol		0.06 (0.02)**		0.05 (0.02)**		0.05 (0.03)†
Conduct		-0.02 (0.03)		-0.02 (0.03)		-0.04 (0.04)
Depression		-0.01 (0.01)		-0.01 (0.01)		-0.03 (0.02)
Suicidality		0.08 (0.04)*		0.08 (0.04)†		0.18 (0.06)***
Drug		0.03 (0.09)		0.03 (0.09)		0.06 (0.13)
Cigarette		0.12 (0.08)		0.13 (0.08)†		0.16 (0.10)
Education		0.00 (0.02)		0.00 (0.02)		0.03 (0.03)
Age at first birth		0.01 (0.01)		0.01 (0.01)		0.01 (0.01)

Note: N = 1,296 for Models 1-4; N = 662 for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.
†p < 0.10, *p < 0.05, **p < 0.01, ***p < 0.001.

TABLE 3
Regression Predicting Marital Separation/Divorce

Parameter	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)	Model 5 OR (95% CI)	Model 6 OR (95% CI)
Offspring ADHD symptoms						
Unrel	1.05 (1.02–1.08)***	1.02 (0.99–1.06)				
Btwn			1.04 (1.01–1.08)*	0.99 (0.99–0.99)**	0.99 (0.90–1.08)	0.97 (0.93–1.01) [†]
W/in			1.08 (1.02–1.14)**	1.05 (0.98–1.12)	1.12 (1.03–1.22)**	1.10 (0.99–1.21) [†]
No. children	0.66 (0.55–0.78)***	0.73 (0.60–0.88)**	0.66 (0.56–0.79)***	0.74 (0.61–0.88)***	0.74 (0.57–0.98)*	0.76 (0.58–1.00) [†]
Wife covariates						
Alcohol		1.04 (0.91–1.19)		1.05 (0.91–1.20)		0.99 (0.83–1.18)
Conduct		0.97 (0.81–1.17)		0.98 (0.82–1.18)		0.74 (0.56–0.98)*
Depression		1.09 (1.04–1.14)***		1.09 (1.04–1.14)***		1.08 (1.01–1.16)*
Suicidality		1.17 (1.04–1.33)*		1.17 (1.03–1.32)*		1.27 (1.06–1.51)**
Drug		1.60 (1.06–2.43)*		1.62 (1.06–2.45)*		2.21 (1.14–4.30)*
Cigarette		0.93 (0.68–1.26)		0.92 (0.68–1.25)		1.06 (0.67–1.69)
Education		0.97 (0.87–1.09)		0.97 (0.86–1.08)		0.89 (0.75–1.06)
Age at first birth		0.92 (0.87–0.98)**		0.92 (0.87–0.98)**		0.92 (0.84–1.00) [†]
Husband covariates						
Alcohol		0.99 (0.90–1.08)		0.99 (0.91–1.08)		1.05 (0.92–1.19)
Conduct		0.93 (0.81–1.07)		0.93 (0.81–1.07)		0.86 (0.70–1.06)
Depression		1.16 (1.10–1.23)***		1.16 (1.10–1.23)***		1.16 (1.06–1.28)**
Suicidality		1.04 (0.89–1.21)		1.04 (0.89–1.21)		1.13 (0.88–1.45)
Drug		1.72 (1.18–2.52)**		1.77 (1.21–2.61)**		1.33 (0.68–2.59)
Cigarette		1.14 (0.77–1.70)		1.13 (0.76–1.67)		1.01 (0.57–1.77)
Education		0.98 (0.88–1.08)		0.98 (0.88–1.08)		0.98 (0.83–1.16)
Age at first birth		1.02 (0.96–1.07)		1.02 (0.96–1.07)		1.02 (0.95–1.11)

Note: $N = 1,296$ for Models 1–4; $N = 662$ for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.

[†] $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

separated/divorced, the lack of statistical significance is likely due, at least in part, to limited statistical power. These results suggest that offspring ADHD elevates parents' risk of separation/divorce.

Next, we reran the models using only the sub-sample for whom separation/divorce did not precede or co-occur with ADHD onset. Thus, we re-examined the possible role of genetic factors after excluding families in which the timing of ADHD onset and separation/divorce suggests a marital problems-to-child direction of effects. Results for marital conflict (Table 4) were essentially the same as for the full sample. Thus, using the smaller sample of families in which separation/divorce did not precede ADHD, results were consistent with the notion that offspring ADHD increases marital conflict. We also reran the models predicting separation/divorce using this sub-sample (Table 5). The results were similar to those for the full sample, although the associations were slightly smaller in magnitude.

Sensitivity Tests

Results of analyses using ADHD diagnoses (Tables 6 and 7) were similar to those using ADHD symptoms, although the standard errors around the estimates were large, likely due to power limitations (because relatively few offspring were diagnosed with ADHD). Further, additional analyses that (1) did not control for number of children in the family (Tables 8 and 9), (2) controlled for number of male children in the family (Tables 10 and 11), and (3) compared only

same-sex DZ twins (Table 12) all provided commensurate results. When we added controls for offspring ODD and CD (Table 13), the association remained basically the same; similarly, when we added controls for offspring alcohol problems and major depression (Table 13), the association was attenuated slightly. The results, therefore, are independent of the number of children in the family, the larger number of female than male twins in our sample, and the greater prevalence of ADHD among males than among females. They are also independent of offspring ODD, CD, depression, and alcohol problems.

Discussion and Conclusions

Our results indicate that environmental factors related specifically to offspring ADHD increase parents' risks of marital conflict and separation/divorce, contrary to our hypotheses. First, comparing unrelated families, we found that offspring ADHD robustly predicted marital conflict when controlling for measured characteristics of both parents (e.g., CD, alcohol problems). The association was robust to controls for genetic and shared environmental selection factors (when comparing MZ twins differentially exposed to offspring ADHD). The association also remained after removing families in which separation/divorce preceded or co-occurred with ADHD onset. The results for separation/divorce were similar, although we had limited statistical power to precisely estimate the associations'

TABLE 4
Regression Predicting Marital Conflict: Timing Sub-sample

Parameter	Model 1 b (SE)	Model 2 b (SE)	Model 3 b (SE)	Model 4 b (SE)	Model 5 b (SE)	Model 6 b (SE)
Offspring ADHD symptoms						
Unrel	0.03 (0.01)***	0.02 (0.01)**				
Btwn			0.04 (0.01)**	0.02 (0.01) [†]	0.06 (0.02)**	0.05 (0.02)*
W/in			0.03 (0.01)*	0.02 (0.01)	0.06 (0.02)***	0.05 (0.02)***
No. children	-0.07 (0.04) [†]	-0.04 (0.04)	-0.07 (0.04) [†]	-0.04 (0.04)	-0.08 (0.06)	-0.05 (0.05)
Wife covariates						
Alcohol		0.05 (0.03) [†]		0.06 (0.03) [†]		0.03 (0.05)
Conduct		0.00 (0.05)		0.00 (0.05)		-0.03 (0.07)
Depression		0.04 (0.01)***		0.04 (0.01)***		0.04 (0.02)*
Suicidality		0.06 (0.04) [†]		0.06 (0.04) [†]		0.14 (0.05)**
Drug		-0.07 (0.10)		-0.05 (0.10)		-0.28 (0.16) [†]
Cigarette		-0.02 (0.07)		-0.02 (0.07)		-0.05 (0.09)
Education		0.03 (0.03)		0.03 (0.03)		-0.04 (0.04)
Age at first birth		-0.01 (0.01)		-0.01 (0.01)		-0.01 (0.02)
Husband covariates						
Alcohol		0.05 (0.02)*		0.05 (0.02)*		0.05 (0.03) [†]
Conduct		-0.01 (0.03)		-0.01 (0.03)		-0.04 (0.04)
Depression		-0.01 (0.01)		-0.01 (0.01)		-0.03 (0.02) [†]
Suicidality		0.07 (0.04) [†]		0.07 (0.04) [†]		0.17 (0.06)**
Drug		0.01 (0.09)		0.01 (0.09)		0.01 (0.13)
Cigarette		0.13 (0.08) [†]		0.14 (0.08) [†]		0.18 (0.10) [†]
Education		0.00 (0.02)		0.00 (0.02)		0.04 (0.03)
Age at first birth		0.01 (0.01)		0.01 (0.01)		0.02 (0.01)

Note: N = 1,214 for Models 1–4; N = 614 for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.
[†]p < 0.10, *p < 0.05, **p < 0.01, ***p < 0.001.

TABLE 5
Regression Predicting Marital Separation/Divorce: Timing Sub-sample

Parameter	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)	Model 5 OR (95% CI)	Model 6 OR (95% CI)
Offspring ADHD symptoms						
Unrel	1.03 (0.995–1.07) [†]	1.00 (0.96–1.04)				
Btwn			1.02 (0.94–1.12)	0.98 (0.94–1.02)	0.97 (0.89–1.06)	0.94 (0.79–1.13)
W/in			1.05 (0.99–1.11)	1.02 (0.94–1.11)	1.07 (0.98–1.17)	1.15 (0.96–1.37)
No. children	0.71 (0.59–0.86)***	0.79 (0.64–0.98)*	0.71 (0.58–0.87)***	0.80 (0.63–0.999)*	0.85 (0.64–1.14)	0.81 (0.49–1.35)
Wife covariates						
Alcohol		1.09 (0.94–1.26)		1.11 (0.95–1.29)		1.11 (0.73–1.68)
Conduct		0.98 (0.80–1.21)		1.02 (0.80–1.29)		0.77 (0.48–1.24)
Depression		1.09 (1.04–1.15)***		1.10 (1.04–1.17)**		1.13 (0.98–1.29) [†]
Suicidality		1.18 (1.04–1.35)*		1.22 (1.05–1.42)*		1.40 (0.95–2.06) [†]
Drug		1.34 (0.84–2.12)		1.40 (0.82–2.37)		2.32 (0.56–9.70)
Cigarette		0.78 (0.56–1.10)		0.75 (0.51–1.10)		3.98 (1.48–10.69)**
Education		1.00 (0.88–1.12)		0.99 (0.86–1.13)		0.87 (0.59–1.27)
Age at first birth		0.93 (0.87–0.99)*		0.92 (0.86–0.99)*		0.93 (0.72–1.18)
Husband covariates						
Alcohol		1.00 (0.91–1.10)		1.00 (0.90–1.12)		1.10 (0.86–1.41)
Conduct		0.94 (0.81–1.10)		0.95 (0.80–1.13)		0.79 (0.54–1.14)
Depression		1.15 (1.08–1.22)***		1.18 (1.10–1.27)***		1.22 (1.03–1.44)*
Suicidality		1.03 (0.88–1.21)		1.02 (0.84–1.24)		1.04 (0.68–1.58)
Drug		1.87 (1.24–2.84)**		2.10 (1.29–3.41)**		0.89 (0.21–3.80)
Cigarette		0.99 (0.65–1.53)		0.98 (0.60–1.61)		0.88 (0.34–2.32)
Education		0.96 (0.85–1.07)		0.94 (0.82–1.07)		0.92 (0.69–1.24)
Age at first birth		1.00 (0.94–1.06)		1.00 (0.93–1.07)		0.95 (0.79–1.15)

Note: N = 1,214 for Models 1–4; N = 614 for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.
[†]p < 0.10, *p < 0.05, **p < 0.01, ***p < 0.001.

TABLE 6
Regression Predicting Marital Conflict Using ADHD Diagnoses

Parameter	Model 1 b (SE)	Model 2 b (SE)	Model 3 b (SE)	Model 4 b (SE)	Model 5 b (SE)	Model 6 b (SE)
Offspring ADHD symptoms						
Unrel	0.17 (0.10) [†]	0.10 (0.09)				
Btwn			0.13 (0.17)	0.07 (0.17)	0.20 (0.28)	0.16 (0.26)
W/in			0.00 (0.17)	-0.05 (0.16)	0.03 (0.20)	-0.03 (0.19)
No. children	-0.02 (0.04)	-0.01 (0.03)	-0.02 (0.04)	0.00 (0.03)	0.02 (0.05)	0.03 (0.05)
Wife covariates						
Alcohol		0.06 (0.03) [†]		0.06 (0.03) [†]		0.03 (0.05)
Conduct		0.00 (0.05)		0.00 (0.05)		-0.03 (0.08)
Depression		0.04 (0.01) ^{***}		0.04 (0.01) ^{***}		0.04 (0.01) ^{**}
Suicidality		0.06 (0.03) [†]		0.06 (0.03) [†]		0.14 (0.05) ^{**}
Drug		-0.06 (0.10)		-0.06 (0.10)		-0.29 (0.16) [†]
Cigarette		-0.02 (0.07)		-0.01 (0.07)		-0.04 (0.09)
Education		0.03 (0.03)		0.03 (0.03)		-0.05 (0.04)
Age at first birth		-0.01 (0.01)		-0.01 (0.01)		-0.01 (0.02)
Husband covariates						
Alcohol		0.06 (0.02) ^{**}		0.06 (0.02) ^{**}		0.05 (0.03) [*]
Conduct		-0.01 (0.03)		-0.01 (0.03)		-0.04 (0.04)
Depression		-0.01 (0.01)		-0.01 (0.01)		-0.02 (0.02)
Suicidality		0.07 (0.04) [†]		0.07 (0.04) [†]		0.17 (0.06) ^{**}
Drug		0.03 (0.09)		0.03 (0.09)		0.07 (0.13)
Cigarette		0.13 (0.08) [†]		0.13 (0.08) [†]		0.16 (0.10)
Education		0.00 (0.02)		0.00 (0.02)		0.05 (0.03)
Age at first birth		0.01 (0.01)		0.01 (0.01)		0.01 (0.01)

Note: $N = 1,296$ for Models 1–4; $N = 662$ for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.

[†] $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

TABLE 7
Regression Predicting Marital Separation/Divorce Using ADHD Diagnoses

Parameter	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)	Model 5 OR (95% CI)	Model 6 OR (95% CI)
Offspring ADHD symptoms						
Unrel	1.71 (1.18–2.46) ^{**}	1.51 (1.02–2.22) [*]				
Btwn			1.47 (1.24–1.74) ^{***}	1.74 (1.26–2.41) ^{***}	1.49 (0.45–4.93)	6.22 (0.00–2985401259.35)
W/in			1.35 (0.67–2.71)	0.98 (0.42–2.29)	1.67 (0.63–4.44)	5.65 (0.00–110661.48)
No. children	0.70 (0.60–0.82) ^{***}	0.73 (0.61–0.88) ^{***}	0.72 (0.62–0.84) ^{***}	0.74 (0.62–0.90) ^{**}	0.79 (0.62–1.01) [†]	1.02 (0.10–10.37)
Wife covariates						
Alcohol		1.04 (0.91–1.19)		1.04 (0.91–1.19)		1.17 (0.42–3.30)
Conduct		0.97 (0.81–1.17)		0.98 (0.81–1.20)		0.73 (0.30–1.78)
Depression		1.09 (1.04–1.14) ^{***}		1.10 (1.05–1.16) ^{***}		1.30 (0.37–4.62)
Suicidality		1.17 (1.04–1.33) [*]		1.19 (1.04–1.36) ^{**}		1.33 (0.38–4.65)
Drug		1.59 (1.05–2.41) [*]		1.69 (1.08–2.64) [*]		3.74 (0.00–14110.46)
Cigarette		0.93 (0.68–1.26)		0.94 (0.68–1.30)		12.15 (0.08–1881.43)
Education		0.97 (0.86–1.08)		0.96 (0.85–1.08)		0.95 (0.56–1.63)
Age at first birth		0.92 (0.87–0.98) [*]		0.92 (0.87–0.98) ^{**}		0.83 (0.53–1.31)
Husband covariates						
Alcohol		0.99 (0.91–1.08)		0.98 (0.89–1.08)		1.10 (0.37–3.24)
Conduct		0.93 (0.81–1.07)		0.93 (0.80–1.08)		0.67 (0.06–7.95)
Depression		1.17 (1.10–1.23) ^{***}		1.18 (1.11–1.25) ^{***}		1.44 (0.33–6.32)
Suicidality		1.04 (0.90–1.21)		1.04 (0.88–1.23)		1.03 (0.56–1.92)
Drug		1.73 (1.18–2.53) ^{**}		1.79 (1.18–2.71) ^{**}		0.73 (0.01–37.13)
Cigarette		1.14 (0.77–1.69)		1.14 (0.75–1.74)		2.24 (0.01–780.99)
Education		0.98 (0.88–1.08)		0.97 (0.87–1.09)		0.95 (0.59–1.52)
Age at first birth		1.01 (0.96–1.07)		1.01 (0.96–1.07)		1.04 (0.70–1.56)

Note: $N = 1,296$ for Models 1–4; $N = 662$ for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.

[†] $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

TABLE 8
Regression Predicting Marital Conflict without Control for Number of Offspring

Parameter	Model 1 b (SE)	Model 2 b (SE)	Model 3 b (SE)	Model 4 b (SE)	Model 5 b (SE)	Model 6 b (SE)
Offspring ADHD symptoms						
Unrel	0.03 (0.01)***	0.02 (0.01)**				
Btwn			0.03 (0.01)*	0.01 (0.01)	0.05 (0.02)**	0.04 (0.02)*
W/in			0.02 (0.01)	0.01 (0.01)	0.05 (0.02)**	0.05 (0.02)**
Wife covariates						
Alcohol		0.05 (0.03)†		0.06 (0.03)†		0.03 (0.05)
Conduct		0.00 (0.05)		0.00 (0.05)		-0.03 (0.07)
Depression		0.04 (0.01)***		0.04 (0.01)***		0.04 (0.01)**
Suicidality		0.06 (0.03)†		0.06 (0.03)†		0.14 (0.05)**
Drug		-0.06 (0.10)		-0.05 (0.10)		-0.27 (0.16)†
Cigarette		-0.02 (0.07)		-0.02 (0.07)		-0.06 (0.09)
Education		0.03 (0.03)		0.03 (0.03)		-0.03 (0.04)
Age at first birth		-0.01 (0.01)		-0.01 (0.01)		-0.01 (0.02)
Husband covariates						
Alcohol		0.06 (0.02)**		0.06 (0.02)**		0.05 (0.03)
Conduct		-0.02 (0.03)		-0.01 (0.03)		-0.04 (0.04)
Depression		-0.01 (0.01)		-0.01 (0.01)		-0.03 (0.02)
Suicidality		0.08 (0.04)†		0.08 (0.04)†		0.18 (0.05)***
Drug		0.03 (0.09)		0.03 (0.09)		0.06 (0.13)
Cigarette		0.12 (0.08)		0.13 (0.08)†		0.17 (0.10)
Education		0.00 (0.02)		0.00 (0.02)		0.03 (0.03)
Age at first birth		0.01 (0.01)		0.01 (0.01)		0.01 (0.01)

Note: *N* = 1,296 for Models 1–4; *N* = 662 for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.
†*p* < 0.10, **p* < 0.05, ***p* < 0.01, ****p* < 0.001.

TABLE 9
Regression Predicting Marital Separation/Divorce Without Control for Number of Offspring

Parameter	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)	Model 5 OR (95% CI)	Model 6 OR (95% CI)
Offspring ADHD symptoms						
Unrel	1.02 (0.99–1.05)	1.00 (0.97–1.03)				
Btwn			1.00 (0.94–1.07)	0.98 (0.91–1.06)	0.97 (0.90–1.04)	0.95 (0.92–0.98)**
W/in			1.05 (1.00–1.11)†	1.01 (0.92–1.11)	1.10 (1.02–1.19)*	1.08 (0.99–1.18)†
Wife covariates						
Alcohol		1.04 (0.91–1.18)		1.24 (1.00–1.55)†		0.99 (0.83–1.18)
Conduct		0.98 (0.82–1.18)		0.93 (0.69–1.25)		0.76 (0.57–1.01)†
Depression		1.10 (1.05–1.15)***		1.13 (1.04–1.23)**		1.09 (1.01–1.17)*
Suicidality		1.18 (1.04–1.33)*		1.23 (0.99–1.54)†		1.28 (1.07–1.53)**
Drug		1.70 (1.12–2.57)*		1.54 (0.71–3.33)		2.33 (1.19–4.57)*
Cigarette		0.94 (0.69–1.27)		1.63 (0.93–2.85)†		1.06 (0.66–1.70)
Education		0.97 (0.87–1.09)		1.07 (0.87–1.32)		0.89 (0.74–1.06)
Age at first birth		0.93 (0.87–0.99)*		0.85 (0.75–0.96)*		0.92 (0.84–1.01)†
Husband covariates						
Alcohol		0.98 (0.90–1.07)		1.07 (0.92–1.24)		1.04 (0.91–1.18)
Conduct		0.94 (0.82–1.08)		0.88 (0.69–1.11)		0.86 (0.70–1.06)
Depression		1.17 (1.10–1.23)***		1.18 (1.08–1.30)***		1.17 (1.07–1.28)***
Suicidality		1.03 (0.89–1.21)		1.05 (0.82–1.34)		1.12 (0.87–1.44)
Drug		1.78 (1.21–2.60)**		1.23 (0.61–2.51)		1.34 (0.69–2.60)
Cigarette		1.14 (0.77–1.69)		1.59 (0.80–3.16)		1.01 (0.57–1.79)
Education		0.97 (0.87–1.08)		0.99 (0.84–1.18)		0.99 (0.83–1.16)
Age at first birth		1.02 (0.96–1.08)		1.02 (0.94–1.12)		1.03 (0.95–1.11)

Note: *N* = 1,296 for Models 1–4; *N* = 662 for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.
†*p* < 0.10, **p* < 0.05, ***p* < 0.01, ****p* < 0.001.

magnitudes. Sensitivity tests indicated that our findings were also independent of family size, sex differences in ADHD prevalence, and other offspring psychopathology.

These findings add converging evidence to the literature, and extend previous studies in several ways. First, we know of no other studies to control for potential genetic or shared

TABLE 10
Regression Predicting Marital Conflict with Control for Number of Male Offspring

Parameter	Model 1 b (SE)	Model 2 b (SE)	Model 3 b (SE)	Model 4 b (SE)	Model 5 b (SE)	Model 6 b (SE)
Offspring ADHD symptoms						
Unrel	0.03 (0.01)***	0.02 (0.01)**				
Btwn			0.04 (0.01)**	0.02 (0.01) [†]	0.06 (0.02)**	0.04 (0.02)*
W/in			0.03 (0.01)*	0.02 (0.01)	0.05 (0.02)***	0.05 (0.02)**
No. children	-0.07 (0.04) [†]	0.00 (0.04)	-0.07 (0.04) [†]	0.00 (0.04)	-0.08 (0.05)	0.00 (0.06)
No. male children	0.00 (0.01)	-0.08 (0.04)*	0.00 (0.01)	-0.08 (0.04) [†]	-0.01 (0.01)	-0.09 (0.06)
Wife covariates						
Alcohol		0.05 (0.03) [†]		0.06 (0.03) [†]		0.04 (0.05)
Conduct		-0.01 (0.05)		0.00 (0.05)		-0.03 (0.07)
Depression		0.04 (0.01)***		0.04 (0.01)***		0.04 (0.01)*
Suicidality		0.06 (0.03) [†]		0.06 (0.03) [†]		0.14 (0.05)**
Drug		-0.06 (0.10)		-0.04 (0.10)		-0.27 (0.16) [†]
Cigarette		-0.03 (0.07)		-0.03 (0.07)		-0.08 (0.09)
Education		0.03 (0.03)		0.03 (0.03)		-0.03 (0.04)
Age at first birth		-0.01 (0.01)		-0.01 (0.01)		-0.01 (0.02)
Husband covariates						
Alcohol		0.06 (0.02)**		0.05 (0.02)**		0.05 (0.03) [†]
Conduct		-0.02 (0.03)		-0.01 (0.03)		-0.04 (0.04)
Depression		-0.01 (0.01)		-0.01 (0.01)		-0.03 (0.02)
Suicidality		0.08 (0.04)*		0.08 (0.04) [†]		0.18 (0.06)**
Drug		0.03 (0.09)		0.03 (0.09)		0.06 (0.13)
Cigarette		0.12 (0.08)		0.12 (0.08)		0.16 (0.10)
Education		0.00 (0.02)		0.00 (0.02)		0.03 (0.03)
Age at first birth		0.01 (0.01)		0.01 (0.01)		0.01 (0.01)

Note: $N = 1,296$ for Models 1–4; $N = 662$ for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.

[†] $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

TABLE 11
Regression Predicting Marital Separation/Divorce with Control for Number of Male Offspring

Parameter	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)	Model 5 OR (95% CI)	Model 6 OR (95% CI)
Offspring ADHD symptoms						
Unrel	1.05 (1.02–1.08)***	1.02 (0.99–1.06)				
Btwn			1.04 (1.00–1.07) [†]	1.00 (1.00–1.00)	0.99 (0.90–1.08)	0.97 (0.93–1.01)
W/in			1.08 (1.02–1.14)**	1.05 (0.96–1.15)	1.12 (1.03–1.22)**	1.10 (1.00–1.21)*
No. children	0.65 (0.55–0.77)***	0.74 (0.59–0.92)**	0.66 (0.55–0.78)***	0.75 (0.59–0.94)*	0.74 (0.56–0.97)*	0.78 (0.58–1.06)
No. male children	0.97 (0.94–1.00) [†]	0.98 (0.78–1.22)	0.97 (0.93–1.00)*	0.97 (0.77–1.23)	0.99 (0.94–1.04)	0.95 (0.68–1.32)
Wife covariates						
Alcohol		1.04 (0.91–1.19)		1.05 (0.90–1.22)		0.99 (0.83–1.18)
Conduct		0.97 (0.81–1.17)		0.98 (0.76–1.28)		0.74 (0.56–0.99)*
Depression		1.09 (1.04–1.14)***		1.09 (1.03–1.15)**		1.08 (1.01–1.16)*
Suicidality		1.17 (1.04–1.33)*		1.17 (1.02–1.34)*		1.26 (1.06–1.51)**
Drug		1.61 (1.06–2.43)*		1.62 (0.92–2.84) [†]		2.25 (1.17–4.33)*
Cigarette		0.93 (0.68–1.26)		0.92 (0.62–1.35)		1.05 (0.66–1.67)
Education		0.97 (0.86–1.09)		0.96 (0.84–1.10)		0.89 (0.75–1.06)
Age at first birth		0.92 (0.87–0.98)*		0.92 (0.88–0.97)**		0.92 (0.84–1.00) [†]
Husband covariates						
Alcohol		0.99 (0.90–1.08)		0.99 (0.89–1.10)		1.05 (0.92–1.19)
Conduct		0.93 (0.81–1.07)		0.93 (0.79–1.11)		0.86 (0.70–1.05)
Depression		1.16 (1.10–1.23)***		1.16 (1.09–1.24)***		1.16 (1.06–1.27)**
Suicidality		1.04 (0.90–1.21)		1.04 (0.82–1.33)		1.13 (0.89–1.45)
Drug		1.72 (1.18–2.52)**		1.77 (1.12–2.81)*		1.32 (0.69–2.53)
Cigarette		1.14 (0.77–1.68)		1.12 (0.70–1.78)		1.00 (0.57–1.75)
Education		0.98 (0.88–1.08)		0.98 (0.86–1.10)		0.98 (0.83–1.15)
Age at first birth		1.02 (0.96–1.07)		1.02 (0.97–1.06)		1.02 (0.95–1.11)

Note: $N = 1,296$ for Models 1–4; $N = 662$ for Models 5 and 6. ADHD = attention-deficit/hyperactivity disorder; Unrel = unrelated families; Btwn = between-families; W/in = within-families. Model 1: phenotypic association; Model 2: phenotypic association plus parental covariates; Model 3: co-twin comparison; Model 4: co-twin comparison plus parental covariates; Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.

[†] $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

TABLE 12
Regression Comparing Same-Sex DZ Twins

Parameter	Marital conflict		Marital separation/divorce	
	Model 5 b (SE)	Model 6 b (SE)	Model 5 OR (95% CI)	Model 6 OR (95% CI)
Offspring ADHD symptoms				
Btwn	0.02 (0.02)	0.00 (0.03)	1.10 (0.93–1.31)	1.02 (0.80–1.29)
W/in	0.02 (0.03)	–0.01 (0.03)	1.05 (0.94–1.17)	0.69 (0.55–0.87)**
No. children	–0.02 (0.07)	0.04 (0.06)	0.58 (0.43–0.78)***	0.91 (0.40–2.08)
Wife covariates				
Alcohol		0.08 (0.04) [†]		1.78 (0.92–3.46) [†]
Conduct		0.04 (0.08)		0.89 (0.41–1.90)
Depression		0.04 (0.02)**		1.08 (0.85–1.35)
Suicidality		0.03 (0.05)		1.77 (0.94–3.32) [†]
Drug		0.24 (0.15) [†]		2.02 (0.37–10.92)
Cigarette		0.04 (0.11)		2.21 (0.54–9.10)
Education		0.11 (0.04)**		1.53 (0.87–2.70)
Age at first birth		–0.03 (0.02) [†]		0.78 (0.56–1.07)
Husband covariates				
Alcohol		0.06 (0.03)		0.82 (0.53–1.26)
Conduct		–0.03 (0.05)		0.82 (0.39–1.74)
Depression		–0.00 (0.02)		1.53 (1.20–1.97)**
Suicidality		0.04 (0.06)		0.72 (0.37–1.39)
Drug		–0.02 (0.13)		0.96 (0.19–4.93)
Cigarette		0.14 (0.13)		13.82 (1.65–115.44)*
Education		–0.04 (0.03)		1.16 (0.78–1.72)
Age at first birth		0.02 (0.01)		1.03 (0.84–1.27)

Note: *N* = 496. ADHD = attention-deficit/hyperactivity disorder; Btwn = between-families; W/in = within-families. Model 5: co-twin comparison, MZ sub-sample; Model 6: co-twin comparison plus parental covariates, MZ sub-sample.
[†]*p* < 0.10, **p* < 0.05, ***p* < 0.01, ****p* < 0.001.

TABLE 13
Control for Offspring ODD and CD/Control for Offspring Alcohol Problems and Depression

Parameter	Marital conflict	Separation/divorce	Marital conflict	Separation/divorce
	Model 6 b (SE)	Model 6 OR (95% CI)	Model 6 b (SE)	Model 6 OR (95% CI)
Offspring ADHD symptoms				
Btwn	0.03 (0.02)	0.95 (0.92–0.99)**	0.03 (0.02)	0.96 (0.92–1.00)*
W/in	0.04 (0.02)*	1.10 (0.99–1.21) [†]	0.03 (0.02) [†]	1.07 (0.97–1.19)
No. children	–0.06 (0.05)	0.73 (0.55–0.98)*	–0.10 (0.06) [†]	0.72 (0.53–0.97)*
Offspring ODD	0.02 (0.02)	1.18 (1.04–1.33)**		
Offspring CD	0.03 (0.02)	0.94 (0.84–1.05)		
Offspring alcohol			0.03 (0.01)*	1.01 (0.94–1.08)
Offspring depression			0.16 (0.07)*	1.62 (1.12–2.32)*
Wife covariates				
Alcohol	0.04 (0.05)	0.97 (0.82–1.16)	0.03 (0.05)	0.98 (0.81–1.17)
Conduct	–0.04 (0.07)	0.74 (0.55–0.99)*	–0.02 (0.07)	0.74 (0.55–0.98)*
Depression	0.04 (0.02)**	1.08 (1.00–1.16)*	0.04 (0.01)*	1.07 (1.00–1.15) [†]
Suicidality	0.13 (0.05)**	1.27 (1.06–1.52)*	0.13 (0.04)**	1.27 (1.06–1.51)*
Drug	–0.30 (0.15) [†]	2.24 (1.13–4.43)*	–0.30 (0.15) [†]	2.31 (1.18–4.51)*
Cigarette	–0.06 (0.09)	1.05 (0.65–1.69)	–0.07 (0.09)	1.12 (0.69–1.81)
Education	–0.03 (0.04)	0.89 (0.74–1.06)	–0.03 (0.04)	0.88 (0.74–1.05)
Age at first birth	–0.01 (0.02)	0.92 (0.84–1.00)*	0.00 (0.02)	0.92 (0.84–1.00) [†]
Husband covariates				
Alcohol	0.04 (0.03)	1.06 (0.93–1.21)	0.04 (0.03)	1.05 (0.92–1.20)
Conduct	–0.05 (0.04)	0.86 (0.69–1.07)	–0.05 (0.04)	0.85 (0.69–1.05)
Depression	–0.03 (0.02)	1.17 (1.07–1.28)***	–0.03 (0.02)	1.17 (1.07–1.28)***
Suicidality	0.18 (0.05)***	1.15 (0.90–1.47)	0.18 (0.05)***	1.13 (0.88–1.45)
Drug	0.05 (0.14)	1.34 (0.69–2.61)	0.08 (0.13)	1.32 (0.69–2.54)
Cigarette	0.16 (0.10)	1.01 (0.57–1.79)	0.14 (0.10)	1.00 (0.56–1.78)
Education	0.03 (0.03)	0.98 (0.83–1.15)	0.03 (0.03)	0.98 (0.83–1.15)
Age at first birth	0.01 (0.01)	1.03 (0.95–1.11)	0.01 (0.01)	1.03 (0.95–1.11)

Note: *N* = 662. ADHD = attention-deficit/hyperactivity disorder; Btwn = between-families; W/in = within-families. The models are co-twin comparisons plus parental covariates, MZ sub-sample.
[†]*p* < 0.10, **p* < 0.05, ***p* < 0.01, ****p* < 0.001.

environmental confounds in studies of offspring ADHD as a predictor of marital problems. Our finding that ADHD predicts marital problems even controlling for genetic and shared environmental factors is novel. Second, few studies have controlled for parental psychopathology, and this is one of the first to do so in both mothers and fathers. Our finding that ADHD predicts marital problems even controlling for parental psychopathology indicates that the ADHD–marital association cannot be explained by the influence of parental psychopathology on marital problems and offspring ADHD. Third, this is one of the first studies of the ADHD–marital association to utilize information about the timing of separation/divorce and ADHD onset, strengthening inferences regarding the direction of effect. Fourth, our offspring sample was evenly divided between males and females (50.6% female), whereas previous work has often included more males than females. Fifth, we examined the association between offspring ADHD and parental marital problems independent of the influence of other offspring psychopathology. Thus, this study's results add considerable novel evidence consistent with earlier evidence, suggesting that offspring ADHD causes interparental problems.

Although we know of no previous work using a behavior genetic approach to examine ADHD as a predictor of marital problems, previous work has addressed related questions with this sample. D'Onofrio et al. (2005) examined genetic and environmental contributions to the association between divorce and offspring externalizing problems, and Harden et al. (2007) examined genetic and environmental contributions to the association between marital conflict and offspring CD. The current study builds on these studies by focusing on ADHD rather than CD or overall externalizing, testing offspring ADHD as predictor of interparental problems (rather than the reverse), and using information about separation/divorce and ADHD timing to test the child-to-marital direction of effects. Our findings are consistent with those of D'Onofrio et al., in indicating a direct connection between problems in the parent and offspring generations. Harden and colleagues, however, found that genetic factors accounted for associations between parents and offspring. Further work is needed, therefore, to further examine these differences.

This study has several limitations. Our findings do not control for unmeasured genetic and environmental characteristics of the twins' spouses, which may be passed on to offspring (Eaves et al., 2005). This issue is particularly important because ADHD is more prevalent among males than females, and 2/3 of our twin sample was female. This could bias the results toward showing greater influence of ADHD on marital problems, if ADHD is inherited from twins' male spouses, a genetic process our analyses do not control for. Addressing this limitation, we reran our models controlling for number of male offspring. Results suggested our findings were not biased by over-representation of

female twins. Nonetheless, because we did not have measures of parental ADHD, we were unable to control for parental ADHD statistically. Future studies should address this limitation.

Further, some plausible environmental confounds might influence only one co-twin's family (D'Onofrio et al., 2005), which would influence the within-family estimates. We examined the possibility that the observed associations are caused by characteristics like parental psychopathology, but it is possible that other factors, such as external stressors, are the true cause. Future work should investigate this possibility. Additional limitations are the measurement of ADHD and marital conflict through retrospective report, and our measurement of marital conflict using only two items. However, test–retest reliabilities for both measures were high. Further, Henry et al. (1994) found that 18-year-olds' retrospective reports of family conflict correlated significantly (albeit modestly) with their mothers' prospective reports of family conflict during the same era. Although retrospective reports do not allow the direction of effects to be determined, we also used timing information to strengthen our efforts to test the child-to-marital direction. We did this by repeating our analyses using only the sub-sample in which separation/divorce did not precede or co-occur with ADHD onset. To further investigate this direction of effects while still controlling for genetic factors and other potential confounds, future work should use longitudinal data from a twin sample.

Another consideration is the use of child-, rather than parent-, reported marital functioning. Although parents in the current study did report separation/divorce, their reports of the timing of separation/divorce were much more limited than children's reports, and parents did not report on marital conflict. Although using child reports of both ADHD and marital functioning results in shared method variance, child and parent reports of marital conflict have been found to intercorrelate significantly (Grych et al., 1992). Further, in this study siblings' reports of marital conflict were highly consistent with one another. Subsequent work should include parent reports for comparison with the current results. Investigation using parents' reports of offspring ADHD would also be informative. Additionally, although assortative mating, the tendency to select a spouse similar to oneself, does not typically represent a confound, it could have biased the results. These limitations are necessary drawbacks, however, because they allow us to rule out some alternative explanations of the ADHD–marital problems association. Additional work is needed, using other methods that are robust to these limitations. Together, such work will produce more firm evidence than any one study.

Although a lack of ecological validity is a weakness of experimental methods, it may be argued that self-report questionnaires also lack ecological validity. However, one strength of questionnaires is that they inquire about behavior occurring naturally, as opposed to observing

behavior under artificial conditions. Questionnaires do have weaknesses, though, such as being subject to self-presentation and recall biases. Thus, the convergence of the current findings using questionnaires with findings from previous experimental work is particularly compelling.

In summary, the current study builds on previous research on the ADHD–marital problems association. It provides converging evidence that offspring ADHD elevates parents’ risk of marital problems, accounting for possible genetic and environmental confounds. In addition, by controlling statistically for measured parental and offspring characteristics, we were able to rule out such factors as parents’ and children’s CD as potential confounds of the ADHD–marital problems association.

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List of Abbreviations

ADHD: attention-deficit/hyperactivity disorder
 CD: conduct disorder
 DZ: dizygotic twins
 MZ: monozygotic twins
 ODD: oppositional defiant disorder

References

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Australian Bureau of Statistics. (2010). *Parental divorce or death during childhood* (Cat. no. 4102.0). Canberra, ACT: Australian Bureau of Statistics. Retrieved from www.abs.gov.au

Beach, S. R. H., Fincham, F. D., Amir, N., & Leonard, K. E. (2005). The taxometrics of marriage: Is marital discord categorical? *Journal of Family Psychology, 19*, 276–285.

Brown, R. T., & Pacini, J. N. (1989). Perceived family functioning, marital status, and depression in parents of boys with attention deficit disorder. *Journal of Learning Disabilities, 22*, 581–587.

Bucholz, K. K., Cadoret, R., Cloninger, C. R., Dinwiddie, S. H., Hesselbrock, V. M., Nurnberger, J. I., Reich, T., Schmidt, I., & Schuckit, M. A. (1994). A new, semi-structured psychiatric interview for use in genetic linkage studies: A report on the reliability of the SSAGA. *Journal of Studies on Alcohol, 55*, 149–158.

Carlin, J. B., Gurrin, L. C., Sterne, J. A. C., Morley, R., & Dwyer, T. (2005). Regression models for twin studies: A

critical review. *International Journal of Epidemiology, 34*, 1089–1099.

D’Onofrio, B. M., Turkheimer, E., Emery, R. E., Slutske, W. S., Heath, A. C., Madden, P. A., & Martin, N. G. (2005). A genetically informed study of marital instability and its association with offspring psychopathology. *Journal of Abnormal Psychology. Special Issue: Toward a Dimensionally Based Taxonomy of Psychopathology, 114*, 570–586.

Eaves, L. J., Silberg, J. L., & Maes, H. H. (2005). Revisiting the children of twins: Can they be used to resolve the environmental effects of dyadic parental treatment on child behavior? *Twin Research and Human Genetics, 8*, 283–290.

Faraone, S. V., Biederman, J., Jetton, J. G., & Tsuang, M. T. (1997). Attention deficit disorder and conduct disorder: Longitudinal evidence for a familial subtype. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences, 27*, 291–300.

Grych, J. H., Seid, M., & Fincham, F. D. (1992). Assessing marital conflict from the child’s perspective: The Children’s Perception of Interparental Conflict Scale. *Child Development, 63*, 558–572.

Harden, K. P., Turkheimer, E., Emery, R. E., D’Onofrio, B. M., Slutske, W. S., Heath, A. C., & Martin, N. G. (2007). Marital conflict and conduct problems in children of twins. *Child Development, 78*, 1–18.

Heath, A. C., Bucholz, K. K., Madden, P. A., Dinwiddie, S. H., Slutske, W. S., Bierut, L. J., Statham, D. J., Dunne, M. P., Whitfield, J. B., & Martin, N. G. (1997). Genetic and environmental contributions to alcohol dependence risk in a national twin sample: Consistency of findings in women and men. *Psychological Medicine, 27*, 1381–1396.

Henry, B., Moffitt, T. E., Caspi, A., Langley, J., & Silva, P. A. (1994). On the ‘Remembrance of Things Past’: A longitudinal evaluation of the retrospective method. *Psychological Assessment, 6*, 92–101.

Jaffee, S. R., Caspi, A., Moffitt, T. E., Polo-Tomas, M., Price, T. S., & Taylor, A. (2004). The limits of child effects: Evidence for genetically mediated child effects on corporal punishment but not on physical maltreatment. *Developmental Psychology, 40*, 1047–1058.

Johnson, W., Turkheimer, E., Gottesman, I. I., Bouchard, J., & Thomas, J. (2009). Beyond heritability: Twin studies in behavioral research. *Current Directions in Psychological Science, 18*, 217–220.

Johnston, C., & Behrenz, K. (1993). Childrearing discussions in families of nonproblem children and ADHD children with higher and lower levels of aggressive-defiant behavior. *Canadian Journal of School Psychology, 9*, 53–65.

Johnston, C., & Mash, E. J. (2001). Families of children with attention-deficit/hyperactivity disorder: Review and recommendations for future research. *Clinical Child and Family Psychology Review, 4*, 183–207.

Lahey, B. B., Hartdagen, S. E., Frick, P. J., McBurnett, K., Connor, R., & Hynd, G. W. (1988). Conduct disorder: Parsing the confounded relation to parental divorce and antisocial personality. *Journal of Abnormal Psychology, 97*, 334–337.

- McGue, M., Osler, M., & Christensen, K. (2010). Causal inference and observational research: The utility of twins. *Perspectives on Psychological Science*, 5, 546–556.
- Murphy, K. R., & Barkley, R. A. (1996). Parents of children with attention-deficit/hyperactivity disorder: Psychological and attentional impairment. *American Journal of Orthopsychiatry*, 66, 93–102.
- Muthén, L. K., & Muthén, B. O. (1998–2007). *Mplus user's guide* (5th ed.). Los Angeles, CA: Muthén & Muthén.
- Nikolas, M., & Burt, S. A. (2010). Genetic and environmental influences on ADHD symptom dimensions of inattention and hyperactivity: A meta-analysis. *Journal of Abnormal Psychology*, 119, 1–17.
- Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. *American Journal of Psychiatry*, 164, 942–948.
- Rutter, M., Pickles, A., Murray, R., & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behavior. *Psychological Bulletin*, 127, 291–324.
- Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype – environment effects. *Child Development*, 54, 21.
- Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P. A. F., Bucholz, K. K., Dunne, M. P., Statham, D. J., & Martin, N. G. (1997). Modeling genetic and environmental influences in the etiology of conduct disorder: A study of 2,682 adult twin pairs. *Journal of Abnormal Psychology*, 106, 266–279.
- Statham, D. J., Heath, A. C., Madden, P. A., Bucholz, K. K., Bierut, L., Dinwiddie, S. H., Slutske, W. S., Dunne, M. P., & Martin, N. G. (1998). Suicidal behaviour: An epidemiological and genetic study. *Psychological Medicine*, 28, 839–855.
- Wymbs, B. T., & Pelham, W. E. (2010). Child effects on communication between parents of youth with and without ADHD. *Journal of Abnormal Psychology*, 119, 366–375.
- Wymbs, B. T., Pelham, W. E., Molina, B. S. G., Gnagy, E. M., Wilson, T. K., & Greenhouse, J. B. (2008). Rate and predictors of divorce among parents of youths with ADHD. *Journal of Consulting and Clinical Psychology*, 76, 734–744.

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