

# Parental Alcoholism and Offspring Behavior Problems: Findings in Australian Children of Twins

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We examine the impact of rearing by an alcoholic parent on risk for child behavior problems using data on 2492 offspring drawn from two ongoing studies of children of female and male same- and opposite-sex twin pairs. Results of regression models predicting child behavior problems from parent and co-twin lifetime history of alcohol use disorder (AUD) provide support for genetic but not environmental transmission of externalizing and a measure of total problem behaviors. Results for internalizing behavior were inconclusive with respect to transmission of risk.

**Keywords:** behavior problems, parental alcoholism, children of twins

Children of alcoholics (COAs) are at increased risk for a range of externalizing difficulties, notably conduct-disordered and other 'acting out' behaviors (Kuperman et al., 1999; Sher et al., 1991; Reich et al., 1993), with similar risks observed for early and problem substance use (Chassin et al., 1991; Lieb et al., 2002; Schuckit & Smith, 1996; Sher et al., 1991). Although the magnitude of effects is somewhat reduced for internalizing compared to externalizing outcomes, associations between parental alcoholism and depressive and anxious behaviors are also reported (Chassin et al., 1999; Kuperman et al., 1999; Sher et al., 1991). With approximately one-quarter of children exposed to alcoholism in the family (Grant, 2000), increased understanding of mechanisms underlying risks to COAs is critical for targeted preventive and intervention efforts.

Distinguishing among causal and correlated risks for problem behavior is an increasing focus of much theoretical and empirical work, and this is especially true of research on COAs (see Sher, 1991; Windle & Searles, 1990). Although widely believed, it was until recently difficult to demonstrate a causal effect of growing up in an alcoholic home. Family studies consistently document a range of adversities associated with rearing by an alcoholic parent, but leave herita-

ble influences uncontrolled. For biological offspring, exposure to parental alcoholism is confounded with genetic risks associated with problem and dependent drinking (for reviews, see Heath, 1995; McGue, 1999). Thus, it is impossible to determine from family studies alone whether risk to COAs is due to shared genes or exposure to alcoholic environments. Because genetic effects and gene by environment interaction (GxE) are confounded in classical twin designs (Eaves et al., 1977), estimates of shared environment from twin studies include only those influences that do not interact with shared genes. Adoption designs in theory should be ideal for demonstrating environmental effects, while controlling for genetic influences. However, selection for high risk genetic backgrounds but reduced environmental risk is a critical limitation, resulting in restriction of the range of environmental adversity to which adoptive offspring are exposed (Stoolmiller, 1999).

The goal of this study is to examine whether being raised by a parent with history of alcohol use disorder (AUD) increases risk of child behavior problems, over and above genetic influences. We use a Children-of-Twins (COT) design (Gottman & Bertelson, 1989; Heath et al., 1985; Nance & Corey, 1976), in which genetic and environmental risks are inferred from parent and co-twin history of AUD. By comparing outcomes of biological offspring of an alcoholic parent to biological offspring of the unaffected co-twin of the alcoholic parent, the COT design combines the advantages of both adoption and classical twin studies, while minimizing their respective limitations. In COT studies, outcomes of offspring from a minimum of four groups are compared, each with varying degrees of genetic risk and environmental

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exposure. In the context of the present study, these groups include: offspring whose parent is alcoholic (Group 1); offspring of an unaffected parent whose monozygotic (MZ) co-twin is alcoholic (Group 2); offspring of an unaffected parent whose dizygotic (DZ) co-twin is alcoholic (Group 3); and offspring from control families, where neither parent nor co-twin, regardless of zygosity, is alcoholic (Group 4).

Following from quantitative genetic theory, assumptions regarding environmental, genetic, and  $G \times E$  risks to offspring are summarized in Table 1, separately by risk group. If the association between parental alcoholism and child behavior problems results from exposure to alcoholic environments, that is, environmental transmission, offspring reared by an alcoholic parent should exhibit more behavior problems than offspring of unaffected parents (Group 1 > Groups 2–4), regardless of genetic risk. If the association results from genes shared between parents and their children, i.e., genetic transmission, offspring at high genetic risk should exhibit more behavior problems than offspring at intermediate genetic risk (Groups 1 and 2 > Group 3), regardless of environmental risk. Offspring at intermediate genetic risk in turn should exhibit more problems than offspring at low genetic risk (Group 3 > Group 4). A pattern consistent with  $G \times E$  is evident if offspring reared by an alcoholic parent exhibit more behavior problems compared to offspring of unaffected parents, with offspring of an unaffected parent whose co-twin is also unaffected exhibiting the fewest problems (Group 1 > Group 2 > Group 3 > Group 4).

To date, a handful of studies have capitalized on the COT design to examine causal consequences of exposure to a range of putative environmental risks, including parental alcoholism. Taken together, this research largely supports genetic over environmental transmission underlying the association between parental alcoholism and conduct disorder symptoms (Haber et al., 2005) and both diagnostic and symptom-count measures of ADHD (Knopik et al., 2006; Knopik et al., 2009), despite attenuation in models adjusting for important correlates, such as parental comorbid psychopathology and prenatal alcohol or nicotine exposure. For AUD assessed in older offspring, evidence of environmental transmission from parental alcoholism has been shown in some reports (Duncan et al., 2006; Jacob et al., 2003),

but not all (Slutske et al., 2008). Fewer COT studies of parental alcoholism and internalizing outcomes are available. The single report of offspring suicidal behavior was inconclusive with respect to the role of environmental, genetic or  $G \times E$  risks associated with paternal alcoholism (Glowinski et al., 2004), but likely suffered from limited power associated with low prevalence outcomes. The present study extends this growing body of work by using a COT design to examine associations between parental alcoholism and offspring internalizing as well as externalizing behavior during childhood.

## Methods

### Participants

Participants were drawn from ongoing studies of Australian children of twins selected from two broadly representative Australian volunteer twin panels maintained by the Australian National Health and Medical Research Council (NHMRC). The older panel (Cohort I) was born between 1893 and 1964 and ascertained as adults (Heath et al., 1997). The younger panel (Cohort II) was born between 1964 and 1971 and ascertained as children through their parents in response to flyers distributed throughout Australian schools (Heath, Howells et al., 2001; Knopik et al., 2004). Twins in both cohorts are of primarily European descent reflecting the predominantly Caucasian Australian population from which they were ascertained. Detailed characterization of each panel is described elsewhere (see Heath et al., 1997; Heath et al., 2001).

All twins completed diagnostic telephone interviews between 1992–1994 (Cohort I) or 1997–2002 (Cohort II). Twins in the current study were selected based on lifetime history of alcohol use disorder (AUD). Pairs where at least one twin had biological children ages 7–24 and one twin met DSM-IV criteria for AUD [operationalized for this study as alcohol dependence (AD) in male twins and either AD or alcohol abuse (AB) in female twins] were recruited for participation in one of two children of twins studies with coordinated assessment: *Mothers And Their Children* (MATCH) and *Parental Alcoholism & Child Environmental Risk* (PACER). A random sample of control pairs, where at least one twin had biological

**Table 1**

Risk Group by Environmental, Genetic, and  $G \times E$  Risks

| Risk Group                         | Environmental risk | Genetic risk | $G \times E$ risk |
|------------------------------------|--------------------|--------------|-------------------|
| 1. Parent AUD+                     | High               | High         | High              |
| 2. Parent AUD- and MZ co-twin AUD+ | Low                | High         | Low               |
| 3. Parent AUD- and DZ co-twin AUD+ | Low                | Intermediate | Low               |
| 4. Parent and co-twin AUD-         | Low                | Low          | Very low          |

Note: AUD = alcohol use disorder.

**Table 2**

Available Data by Risk Group: Combined MATCH and PACER Samples

| Risk group                 | <i>n</i> parents | <i>n</i> offspring |
|----------------------------|------------------|--------------------|
| 1. Parent AUD+             | 327              | 604                |
| 2. MZ co-twin AUD+         | 94               | 167                |
| 3. DZ co-twin AUD+         | 131              | 246                |
| 4. Parent and co-twin AUD- | 733              | 1475               |

**Table 3**

Sample Characteristics by Risk Group

| Risk group                 | Parent age<br>M ( <i>SD</i> ) | Child age<br>M ( <i>SD</i> ) | Child sex<br>(% Male) |
|----------------------------|-------------------------------|------------------------------|-----------------------|
| 1. Parent AUD+             | 41.30 (3.99) <sup>a</sup>     | 13.67 (3.64) <sup>a</sup>    | 52                    |
| 2. MZ co-twin AUD+         | 41.05 (4.28) <sup>a</sup>     | 13.94 (3.47) <sup>a,b</sup>  | 49                    |
| 3. DZ co-twin AUD+         | 41.36 (4.30) <sup>a</sup>     | 13.80 (3.81) <sup>a,b</sup>  | 52                    |
| 4. Parent and co-twin AUD- | 42.59 (3.98) <sup>b</sup>     | 14.40 (3.82) <sup>b</sup>    | 51                    |

Note: Means with different superscripts are significantly different from each other.

children ages 7–24, but neither twin met criteria for AUD, was also recruited.

Twins from the MATCH study were selected from female same-sex pairs from both cohorts. PACER twins were selected from male same- and opposite-sex pairs from Cohort II. Reassessment of twin parents and co-twins by telephone interview and self-report questionnaire began in 2000 and 2005 for MATCH and PACER studies, respectively. The present analyses are based on questionnaire data from 874 MATCH twins (from 337 MZ and 280 DZ pairs) reporting on 1643 offspring, and 411 PACER twins (from 116 MZ, 87 same-sex DZ, and 171 opposite-sex DZ pairs), of whom 367 provided reports on 756 offspring and 44 biological co-parents (mothers in the case of twin fathers) reported on 93 offspring. In all, questionnaire data were available from 1285 parents reporting on 2492 offspring. Combined samples sizes by risk group are shown in Table 2, with sample characteristics provided in Table 3. Average age of parents was 42.01 (*SD* = 4.08), ranging from 32 to 65 years. The average age of offspring was 14.14 years (*SD* = 3.77), with roughly equal proportion of male (51%) and female (49%) offspring.

### Measures

Twins completed adaptations of the Semi-Structured Assessment of the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994; Hesselbrock et al., 1999), which was developed for the Collaborative Study on the Genetics of Alcoholism (COGA) to assess physical, psychological, and social manifestations of alcoholism and related psychiatric disorders in adults. All interviews, both initial and follow-up, were administered by trained interviewers, who were supervised by a project coordinator and clinical psychologist. Interviews were tape-recorded, with a random sampling of tapes

reviewed for quality control and coding inconsistencies. Informed consent was obtained from all participants using procedures approved by the institutional review boards at both Washington University School of Medicine and Queensland Institute of Medical Research.

Much of the original SSAGA was retained at reassessment, with additional items from the Family History Assessment Module (FHAM; Rice et al., 1995) included to assess biological co-parent history of conduct problems, adult antisocial behavior, and alcohol and other substance use or disorder. To assess rearing history and offspring psychopathology, parent-report items adapted from the Diagnostic Interview for Children and Adolescents (DICA; Herjanic & Reich, 1982) and the Child SSAGA (C-SSAGA-P; Kuperman et al., 2001) were also incorporated. In addition, twins with age-eligible offspring completed a brief self-report questionnaire (SRQ), which included assessments of offspring medical history, school and neighborhood characteristics, parenting behaviors, parent-child relationship quality, and partner conflict. Included with the SRQ was the parent-report form of the Child Behavior Checklist for Ages 6–18 (CBCL; Achenbach, 2001).

**Parental alcohol use disorder (AUD).** For Cohort I, lifetime history of AD and AB were coded from DSM-IV symptoms based on a DSM-III-R assessment. The SSAGA administered to Cohort I predates DSM-IV; however, we applied an approximate algorithm for DSM-IV criteria without clustering that was developed by Heath and colleagues (see Heath, Whitfield et al., 2001). Consistent with other reports (Grant et al., 2007; Knopik et al., 2006; Waldron et al., 2008), twins in Cohort I were diagnosed with AD if they endorsed three or more DSM-IV AD symptoms. Female twins were diagnosed with AB if they reported at least one symptom of DSM-IV AB. For Cohort II, DSM-IV AD was directly assessed, as was DSM-IV AB for female twins.

**Offspring behavior problems.** Twin parents or biological co-parents completed the Child Behavior Checklist for Ages 6–18 (CBCL; Achenbach, 2001) reporting on up to three age-eligible offspring. The CBCL is a widely used parent report measure of child behavior problems, with well-established psychometrics (see Achenbach & Rescorla, 2001; Achenbach, 1991). Parents reported whether a given behavior described his or her child's behavior within the previous six months using a likert rating of 0 = *not true*, 1 = *somewhat or sometimes true*, or 2 = *very true or often true* for each item. For offspring ages 19–24, parents provided a retrospective report of offspring behavior at age 18. Scores from *internalizing* and *externalizing* scales of the CBCL were analyzed. Internalizing consists of items from anxious/depressed, withdrawn/depressed, and somatic complaints subscales, with rule-breaking and aggressive behavior subscales comprising externalizing. Scores from a combined index of

problem behavior, *total problems*, were also analyzed and include items from all subscales, including social problems, thought problems, and attention problems subscales. Given significant positive skew observed of raw CBCL scores, log-transformations of internalizing, externalizing, and total problems scales were analyzed. Consistent with Achenbach and Rescorla (2001), data were considered valid if fewer than 8 items were missing, excluding open-ended items. Valid mother and twin father reports were both available for 16 of 411 PACER families; for these families, mother report was used.

### Analytic Strategy

Regression models predicting CBCL internalizing, externalizing, and total problems scores from parent and co-twin AUD were conducted in STATA (StataCorp, 2005), with post-hoc tests for group differences. All models included three dummy variables coding for offspring at both high genetic and high environmental risk (Group 1: parent AUD+), offspring at high genetic risk but reduced environmental risk (Group 2: parent AUD-, MZ co-twin AUD+), and offspring at intermediate genetic risk but reduced environmental risk (Group 3: parent AUD-, DZ co-twin AUD+). Control families, where offspring are at low genetic and low environmental risk (Group 4: parent and co-twin AUD-), comprised the reference group. Analyses were conducted first without and then with adjustment for offspring age and gender. The Huber-White robust variance estimator option in Stata was used to correct for non-independence of twin-family data.

### Results

Preliminary analyses indicate significant differences in parental age by risk status,  $F(3, 1281) = 10.82, p < .0001$ . Differences in offspring age by risk status were also significant,  $F(3, 2488) = 6.30, p < .001$ . Results of post-hoc tests suggest that parents from control families (Group 4) are older on average than parents from families where either the parent or parent's MZ or DZ co-twin is AUD+ (Groups 1, 2 and 3). Although offspring in Group 4 are also older than offspring whose parent is AUD+ (Group 1), both groups are indistinguishable from offspring whose parent's MZ or DZ co-twin is AUD+ (Groups 2 and 3).

At the phenotypic level, parental AUD accounted for modest variation in offspring behavior problems. Associations between parental AUD and internalizing ( $r = .09, p < .0001$ ), externalizing ( $r = .11, p < .0001$ ) and total problems ( $r = .10, p < .0001$ ) were of similar magnitude. The strength of these associations did not differ as a function of maternal versus paternal AUD, nor offspring gender (all  $p$  levels  $> .30$ ).

Results of regression models predicting internalizing, externalizing, and total problems from parent and co-twin AUD are shown in Tables 4 to 6, respectively. Means and standard deviations of raw scores are also

shown. Offspring of an affected parent (Group 1) scored higher on internalizing compared to offspring from control families (Group 4). Differences between Group 4 and offspring of an unaffected parent with an alcoholic MZ or DZ co-twin (Groups 2 and 3) were nonsignificant. Differences between Group 1 and either of Groups 2 or 3 were also nonsignificant. This pattern was observed regardless of adjustment for offspring age and sex.

For externalizing problems, compared to offspring from control families (Group 4), offspring of an affected parent (Group 1) had higher scores, as did offspring of an unaffected parent with an alcoholic MZ co-twin (Group 2). Furthermore, Groups 1 and 2 did not differ in overall level of externalizing, with both groups differing significantly from offspring of an unaffected parent with an alcoholic DZ co-twin (Group 3). This pattern of Groups 1 and 2  $>$  Groups 3 and 4 held in models without and with adjustment for offspring age and sex. The same pattern was observed for total problems: Groups 1 and 2  $>$  Groups 3 and 4, regardless of covariate adjustment.

### Discussion

We examined whether being raised by an alcoholic parent increases risk of offspring behavioral problems over and above genetic influences. Using data from two ongoing studies of children of twins, we compared internalizing and externalizing behaviors of offspring, with genetic and environmental risks inferred from parent and co-twin history of alcohol use disorder (AUD). Consistent with previous research, results provide little evidence of environmental transmission of risk from parental alcoholism for internalizing, externalizing, or a measure of total problem behavior. In contrast, there was strong support for genetic transmission for externalizing and total problem behavior. Findings regarding genetic risks for internalizing behavior were inconclusive.

Although the pattern of internalizing scores by risk group was consistent with expectations assuming genetic transmission from parental alcoholism, differences between groups were small and mostly nonsignificant. Compared to offspring of control twins, offspring of an alcoholic parent exhibited greater internalizing behavior; however, offspring of an alcoholic parent were indistinguishable from offspring at low environmental risk but high or intermediate genetic risk, who were in turn indistinguishable from offspring of control twins. In contrast, offspring of an alcoholic parent and offspring of an unaffected parent at high genetic risk exhibited similar levels of externalizing, with both groups exhibiting greater externalizing than offspring at intermediate genetic risk or offspring of control twins. The same pattern was observed for a measure of total problem behavior.

Together, results are highly suggestive of genetic but not environmental transmission of risk from parental AUD for externalizing and total problem



**Table 4**  
Regression of Risk Status Predicting Internalizing Behavior

| Risk group                          | M (SD)      | Unadjusted       |     |      | Adjusted         |     |      |
|-------------------------------------|-------------|------------------|-----|------|------------------|-----|------|
|                                     |             | Coef             | SE  | p    | Coef             | SE  | p    |
| 1. Parent AUD+                      | 4.62 (5.76) | .21 <sup>a</sup> | .06 | .001 | .21 <sup>a</sup> | .06 | .001 |
| 2. MZ co-twin AUD+                  | 4.59 (6.19) | .18 <sup>a</sup> | .10 | .07  | .18 <sup>a</sup> | .10 | .07  |
| 3. DZ co-twin AUD+                  | 3.34 (3.76) | .04 <sup>a</sup> | .08 | .59  | .04 <sup>a</sup> | .08 | .57  |
| 4. Parent and co-twin AUD-/constant | 3.29 (4.22) | 1.08             | .03 | .00  | 1.13             | .09 | .00  |

Note: Coefficients with different superscripts are significantly different from each other.

**Table 5**  
Regression of Risk Status Predicting Externalizing Behavior

| Risk group                          | M (SD)      | Unadjusted       |     |      | Adjusted         |     |      |
|-------------------------------------|-------------|------------------|-----|------|------------------|-----|------|
|                                     |             | Coef             | SE  | p    | Coef             | SE  | p    |
| 1. Parent AUD+                      | 5.79 (6.74) | .27 <sup>a</sup> | .06 | .00  | .27 <sup>a</sup> | .06 | .00  |
| 2. MZ co-twin AUD+                  | 5.58 (6.20) | .28 <sup>a</sup> | .10 | .005 | .28 <sup>a</sup> | .10 | .004 |
| 3. DZ co-twin AUD+                  | 3.88 (4.20) | .05 <sup>b</sup> | .07 | .481 | .05 <sup>b</sup> | .07 | .48  |
| 4. Parent and co-twin AUD-/constant | 4.14 (5.77) | 1.22             | .03 | .00  | 1.09             | .09 | .00  |

Note: Coefficients with different superscripts are significantly different from each other.

**Table 6**  
Regression of Risk Status Predicting Total Problems

| Risk group                          | M (SD)        | Unadjusted       |     |      | Adjusted         |     |      |
|-------------------------------------|---------------|------------------|-----|------|------------------|-----|------|
|                                     |               | Coef             | SE  | p    | Coef             | SE  | p    |
| 1. Parent AUD+                      | 19.98 (20.75) | .28 <sup>a</sup> | .07 | .00  | .27 <sup>a</sup> | .07 | .00  |
| 2. MZ co-twin AUD+                  | 19.51 (20.09) | .32 <sup>a</sup> | .11 | .005 | .31 <sup>a</sup> | .11 | .006 |
| 3. DZ co-twin AUD+                  | 13.61 (12.45) | .06 <sup>b</sup> | .08 | .50  | .04 <sup>b</sup> | .08 | .60  |
| 4. Parent and co-twin AUD-/constant | 14.45 (16.36) | 2.25             | .04 | .00  | 2.40             | .11 | .00  |

Note: Coefficients with different superscripts are significantly different from each other.

behaviors, but there are several limitations of the present study to warrant cautious interpretation. First, the sample was drawn from predominantly Causation twin panels in Australia. Given reported differences by race in risk of alcohol use and disorder (Grant, 1997; Hasin et al., 2007), it is possible that observed patterns differ for other racial or ethnic groups. Observed patterns might also differ cross-nationally, further restricting generalizability. Another limitation pertains to sample ascertainment, with well-educated individuals over-represented in both panels (see Heath et al., 2001; Heath et al., 1998).

Perhaps most importantly, proximal causal mechanisms remain unknown. Behaviors or conditions genetically correlated with AUD, and not parental AUD per se, may explain the association between parental alcoholism and externalizing and total problem behaviors. Likely candidates include comorbid parental psychopathology, such as antisociality and depressive disorders, both of which show moderate to high genetic variation, and are strongly correlated with AUD (Fu et al., 2002; Prescott et al., 2000; Slutske et

al., 1998; Slutske et al., 2002). A related limitation concerns transmission of risks associated with *co-parent* AUD, which was unmeasured in the present analysis. To the extent that *co-parent* AUD, including *co-parent* comorbid psychopathology, are not included in COT models, this model-misspecification may lead to false inference (see Eaves et al., 2005).

Capitalizing on coordinated ascertainment and assessment protocols, we combined samples of offspring of maternal and paternal alcoholics and their co-twins. As a result, we are unable to examine separately the effects of maternal versus paternal alcoholism on offspring outcome within the COT design, owing largely to reduced statistical power from analyzing each sample separately. This is especially true for internalizing, where combined samples lack statistical power necessary to distinguish among genetic and environmental risks. In addition, we used mother report of offspring behavior problems if valid CBCL data were available from both a biological mother and twin father. Although reports from both parents were available from only a handful of families,

using a structural equation modeling rather than regression-based framework for data-analysis would enable incorporation of data from both parents, while allowing for potential rater biases associated with parental psychopathology.

### Conclusion

Overall, this study adds to a growing body of work using innovative research designs to address issues of causation in alcoholic families. Despite methodological limitations, the present findings offer important insight into risk mechanisms underlying the association between parental alcoholism and offspring behavior problems. Continued research on causal processes is necessary, including a more comprehensive analysis of the role of other correlated but yet unmeasured genetic and environmental risks. Co-parent history of AUD and comorbid psychopathology in both parents as well as offspring will be especially important to examine to ensure specificity of observed effects, but also to identify potential mechanisms of mediation or risk moderation.

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### References

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.

Achenbach, T. M., & Rescorla, L. A. (2001). *The manual for the ASEBA school-age forms and profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

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.

Chassin, L., Rogosch, F., & Barrera, M. (1991). Substance use symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology*, 100, 449–463.

Chassin, L., Pitts, S. C., DeLucia, C., & Todd, M. (1999). A longitudinal study of children of alcoholics: Predicting young adult substance use disorders, anxiety, and depression. *Journal of Abnormal Psychology*, 108, 106–119.

Duncan, A. E., Scherrer, J. S., Fu, Q., Bucholz, K. K., Heath, A. C., True, W. R., Haber, J.R., Howell, D., & Jacob, T. (2006). Exposure to paternal alcoholism does not predict development of alcohol-use disorders in offspring: Evidence from an offspring-of-twins study. *Journal of Studies on Alcohol*, 67, 649–656.

Eaves, L. J., Last, K., Martin, N. G., & Jinks, J. L. (1977). A progressive approach to non-additivity and genotype-environmental covariance in the analysis of human differences. *British Journal of Mathematical and Statistical Psychology*, 30, 1–42.

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.

Fu, Q., Heath, A. C., Bucholz, K. K., Nelson, E., Goldberg, J., Lyons, M. J., True, W. R., Jacob, T., Tsuang, M. T., & Eisen, S. A. (2002). Shared genetic risk of major depression, alcohol dependence, and marijuana dependence: Contribution of antisocial personality disorder in men. *Archives of General Psychiatry*, 59, 1125–1132.

Glowinski, A. L., Jacob, T., Bucholz, K. K., Scherrer, J. F., True, W., & Heath, A. C. (2004). Paternal alcohol dependence and offspring suicidal behaviors in a children-of-twins study. *Drug and Alcohol Dependence*, 76, S69–S77.

Gottesman, I. I., & Bertelsen, A. (1989). Confirming unexpressed genotypes for schizophrenia. *Archives of General Psychiatry*, 46, 867–872.

Grant, B. F. (2000). Estimates of U.S. children exposed to alcohol abuse and dependence in the family. *American Journal of Public Health*, 90, 112–115.

Grant, B. F. (1997). Prevalence and correlates of alcohol use and dependence in the United States: Results of the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Studies on Alcohol*, 5, 464–473.

Grant, J. D., Heath, A. C., Bucholz, K. K., Madden, P. A. F., Agrawal, A., Statham, D. J., & Martin, N. G. (2007). Spousal concordance for alcohol dependence: Evidence for assortative mating or spousal interaction effects? *Alcoholism: Clinical and Experimental Research*, 31, 717–728.

Haber, J. R., Jacob, T., & Heath, A. C. (2005). Paternal alcoholism and offspring conduct disorder: Evidence for the ‘common genes’ hypothesis. *Twin Research and Human Genetics*, 8, 120–131.

Hasin, D. S., Stinson, F. S., & Grant, B. F. (2007). Prevalence, correlates, disability and comorbidity of DSM-IV alcohol abuse and dependence in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, 64, 830–842.

Heath, A. C. (1995). Genetic influences on alcoholism risk: A review of adoption and twin studies. *Alcohol Health & Research World*, 19, 166–171.

Heath, A. C., Bucholz, K. K., Madden, P. A. F., Dinwiddie S. H., Slutske, W. S., 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 men and women. *Psychological Medicine*, 27, 1381–1396.
- Heath, A. C., Howells, W., Kirk, K. M., Madden, P. A. F., Bucholz, K. K., Nelson, E. C., Slutske, W. S., Statham, D. J., & Martin, N. G. (2001). Predictors of non-response to a questionnaire survey of a volunteer twin panel: Findings from the Australian 1989 twin cohort. *Twin Research*, 4, 73–80.
- Heath, A. C., Kendler, K. S., Eaves, L. J., & Markell, D. (1985). The resolution of cultural and biological inheritance: Informativeness of different relationships. *Behavior Genetics*, 15, 439–465.
- Heath, A. C., Madden, P. A. F., & Martin, N. G. (1998). Assessing the effects of cooperation bias and attrition in behavioral genetic research using data-weighting. *Behavior Genetics*, 28, 415–427.
- Heath, A. C., Whitfield, J. B., Madden, P. A. F., Bucholz, K. K., Dinwiddie, S. H., Slutske, W. S., Bierut, L. J., & Martin, N. G. (2001). Towards a molecular epidemiology of alcohol dependence: Analysing the interplay of genetic and environmental risk factors. *British Journal of Psychiatry*, 178, 33–40.
- Herjanic, B., & Reich, W. (1982). Development of a structured psychiatric interview for children: Agreement between child and parent on individual symptoms. *Journal of Abnormal Child Psychology*, 10, 307–324.
- Hesselbrock, M., Easton, C., Bucholz, K. K., Schuckit, M., & Hesselbrock, V. (1999). A validity study of the SSAGA — A comparison with the SCAN. *Addiction*, 94, 1361–1370.
- Jacob, T., Waterman, B., Heath, A., True, W., Bucholz, K. K., Haber, R., Scherrer, J., & Fu, Q. (2003). Genetic and environmental effects on offspring alcoholism: New insights using an offspring-of-twins design. *Archives of General Psychiatry*, 60, 1265–1272.
- Knopik, V. S., Heath, A. C., Jacob, T., Slutske, W. S., Bucholz, K. K., Madden, P. A., Waldron, M., & Martin, N. G. (2006). Maternal alcohol use disorder and offspring ADHD: Disentangling genetic and environmental effects using a children-of-twins design. *Psychological Medicine*, 36, 1461–71.
- Knopik, V. S., Heath, A. C., Madden, P. A. F., Bucholz, K. K., Slutske, W. S., Nelson, E. C., Statham, D., Whitfield, J. B., & Martin, N. G. (2004). Genetic effects on alcohol dependence risk: Re-evaluating the importance of psychiatric and heritable risk factors. *Psychological Medicine*, 34, 1519–1530.
- Knopik, V. S., Jacob, T., Haber, J. R., Swenson, L. P., & Howell, D. N. (2009). Paternal alcoholism and offspring ADHD problems: A children of twins design. *Twin Research and Human Genetics*, 12, 53–62.
- Kuperman, S., Schlosser, S. S., Kramer, J. R., Bucholz, K., Hesselbrock, V., Reich, T., & Reich, W. (2001). Risk domains associated with an adolescent alcohol dependence diagnosis. *Addiction*, 96, 629–636.
- Kuperman, S., Schlosser, S. S., Lidral, J., & Reich, W. (1999). Relationship of child psychopathology to parental alcoholism and antisocial personality disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38, 686–692.
- Lieb, R., Merikangas, K. R., Höfler, M., Pfister, H., Isensee, B., & Wittchen, H. U. (2002). Parental alcohol use disorders and alcohol use and disorders in offspring: A community study. *Psychological Medicine*, 32, 63–78.
- McGue, M. (1999). The behavioral genetics of alcoholism. *Current Directions in Psychological Science*, 8, 109–115.
- Nance, W. E., & Corey, L. A. (1976). Genetic models for the analysis of data from the families of identical twins. *Genetics*, 83, 811–826.
- Prescott, C. A., Aggen, S. H., & Kendler, K. S. (2000). Sex-specific influences on the comorbidity of alcoholism and major depression. *Archives of General Psychiatry*, 57, 803–811.
- Reich, W., Earls, F., Frankel, O., & Shayka, J. J. (1993). Psychopathology in children of alcoholics. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 995–1002.
- Rice, J. P., Reich, T., Bucholz, K. K., Neuman, R. J., Fishman, R., Rochberg, N., Hesselbrock, V. M., Nurnberger, J., Schuckit, M. A., & Begleiter, H. (1995). Comparison of direct interview and family history diagnoses of alcohol dependence. *Alcoholism: Clinical and Experimental Research*, 19, 1018–1023.
- Rutter, M. (2007). Proceeding from observed correlation to causal inference: The use of natural experiments. *Perspectives on Psychological Science*, 2, 377–395.
- Schuckit, M. A., & Smith, T. L. (1996). An 8-year follow-up of 450 sons of alcoholic and control subjects. *Archives of General Psychiatry*, 53, 202–210.
- Sher, K. J. (1991). *Children of alcoholics: A critical appraisal of theory and research*. Chicago, IL: University of Chicago Press.
- Sher, K. J., Walitzer, K. S., Wood, P. K., & Brent, E. E. (1991). Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. *Journal of Abnormal Psychology*, 100, 427–428.
- Slutske, W. S., D'Onofrio, B. M., Turkheimer, E., Emery, R. E., Harden, K. P., Heath, A. C., & Martin, N. G. (2008). Searching for an environmental effect of parental alcoholism on offspring alcohol use disorder: A genetically informed study of children of alcoholics. *Journal of Abnormal Psychology*, 117, 534–551.
- 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. (1998). Common genetic risk factors for conduct disorder and alcohol dependence. *Journal of Abnormal Psychology*, 107, 363–374.
- Slutske, W. S., Heath, A. C., Madden, P. A. F., Bucholz, K. K., Statham, D., & Martin, N. G. (2002). Personality

- and the genetic risk for alcohol dependence. *Journal of Abnormal Psychology*, 111, 124–133.
- StataCorp (2005). *Stata Statistical Software: Release 8.2*. College Station, TX: StataCorp LP.
- Stoolmiller, M. (1999). Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. *Psychological Bulletin*, 125, 392–409.
- Waldron, M., Heath, A. C., Bucholz, K. K., Madden, P. A. F., & Martin, N. (2008). Alcohol dependence and reproductive onset: Findings in two Australian twin cohorts. *Alcoholism: Clinical and Experimental Research*, 32, 1–10.
- Windle, M., & Searles, J. S. (Eds.) (1990). *Children of alcoholics: Critical perspectives*. New York: Guilford Press.
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