A Behavior Genetic Investigation of
Adolescent Motherhood and Offspring Mental Health Problems

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Abstract

The U.S. has the highest adolescent pregnancy rate in the industrialized world, and adolescent motherhood is associated with increased risk for child mental health problems. The present study examines the relations between adolescent motherhood and children’s behavior, substance use, and internalizing problems in a sample of 2,553 children of 887 twin pairs. Adolescent motherhood remained significantly associated with all mental health problems, even when using a quasi-experimental design capable of controlling for genetic and environmental confounds. In addition, the relation between adolescent motherhood and offspring behavior problems and substance use was partially confounded by family background variables that influence both generations. The results highlight the usefulness of behavior genetic designs when examining putative environmental risks for the development of psychopathology.

Key words: 1) Adolescent motherhood; 2) Children-of-Twins; 3) Teenage pregnancy; 4) Behavior genetics; 5) Internalizing; 6) Behavior problems; 7) Substance use
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and Offspring Mental Health Problems

The U.S. has one of the highest adolescent pregnancy rates in the industrialized world, primarily due to ineffective contraceptive use and lower abortion rates compared to other industrialized nations, rather than more frequent or precocious sexual activity (Coley & Chase-Lansdale, 1998; United Nations, 1991). One of out three girls experience a pregnancy before the age of 20 (National Campaign to Prevent Teen Pregnancy, 2004). Of these pregnancies, 15% are miscarried, over 30% are aborted, and over 50% are carried to term (Alan Guttmacher Institute, 1999). Perhaps the most obvious correlate of adolescent childbearing is privation: teenage mothers are twice as likely to be impoverished when adults (Hoffman, Foster, & Furstenberg, 1998) and more likely to receive welfare (Moore et al., 1993), although the extent to which these adverse economic outcomes are a product of adolescents’ disadvantaged background, versus the pregnancy itself, is debated (see Geronimus & Korenman, 1992). In addition to the negative socioeconomic correlates of adolescent childbearing, the children of adolescent mothers are at elevated risk for diverse forms of psychopathology, including depressive and anxiety disorders (Hofferth, 1987; Moore, Morrison, & Green, 1997), antisocial behavior and other externalizing disorders (Jenkins, Shapka, & Sorenson, 2006; Levine, Pollack, & Comfort, 2001; Nagin, Pogarksy, & Farrington, 1997; Spiiker, Larson, Lewis, Keller, & Gilchrist, 1999; Wakschlag, Gordon, Lahey, Loeber, Green, & Leventhal, 2000). The disparity in adjustment between children of adolescent and adult mothers seems only to widen over children’s lifespans, with the most dramatic disparities evident in adolescence and adulthood (Brooks-Gunn & Furstenberg, 1986; Furstenberg, Brooks-Gunn, & Morgan, 1987).
Consequently, several authors have concluded that a mother’s age at her first birth influences her child’s cognitive and psychosocial development and eventual adult adjustment. This has not remained a purely academic hypothesis: Organizations with disparate political ideologies, such as The Heritage Foundation (The Heritage Foundation, 2004) and Planned Parenthood (Planned Parenthood, 2006), publicly advocate that women should delay childbirth to improve their own and their children’s lives. The relation between adolescent motherhood and child adjustment, however, may not be as simple as commonly characterized. In this paper, we use a family design to consider how adolescent childbearing is related to child psychological adjustment, namely, the extent to which a teenage mother’s “background” variables – both environmental risks and genetic liabilities – account for her children’s higher risk for mental health problems.

Disentangling Maternal Age from Maternal Family Background.

There is an essential difficulty in resolving the extent to which adolescent childbearing per se causes offspring psychopathology: Adolescent mothers differ from adult mothers in many respects other than age. Obviously, one cannot randomly assign children to be born to adolescent versus adult mothers. Moreover, many of the “upstream” variables at play in a teenage woman’s life before her pregnancy predict both her age at first birth also and her children’s functioning, and thus may account for the relation between adolescent motherhood and child psychopathology. For example, impoverished women are more likely to give birth as adolescents (Geronimus, 2004), and poverty is associated with increased risk for antisocial behavior in childhood and adolescence (Christ, et al., 1990). In lieu of experimental control, researchers have largely attempted to measure and statistically control for potential environmental confounds, such as socioeconomic status (Christ et al., 1990), race (Macgregor,
2000), maternal education and welfare receipt (Vogel, 2000). While such epidemiological investigations have advanced our understanding of the roles that certain aspects of an adolescent mother’s background environment play in her children’s poorer adjustment, the use of statistical controls is necessarily limited by researchers’ inability to perfectly measure every possibly relevant covariate.

Rutter and his colleagues (Rutter, 2005; Rutter, Pickles, Murray, & Eaves, 2001; Rutter & Silberg, 2002) outlined several research strategies useful for this common methodological impasse. Primary among them was quasi-experimentation, to “pull apart” (i.e., reduce or eliminate covariation between) confounded variables. Geronimus, Korenman, & Hillemeier (1994) first used a quasi-experimental family design to pull apart adolescent childbearing from environmental background: comparing the firstborn offspring of biological sisters. In a comparison of the children of two sisters, one who gives birth as a teenager and one who delays childbearing, any differences between the children cannot be attributed to environmental variables shared by sisters, including sociodemographic characteristics. This comparison, therefore, is a rigorous test of whether a woman’s net of environmental background influences, rather than her age at birth per se, are responsible for the apparent consequences of teenage pregnancy. The results indicated that heightened risk for negative behavioral problems in offspring were common to the children of socioeconomically disadvantaged mothers in general, not specific to teenage mothers. In a replication of Geronimus et al. (1994), Turley (2003) found that when upstream environmental variables are controlled for by comparing the children of biological sisters discordant for early childbearing, the effect for early motherhood on child adjustment is greatly attenuated, if not eliminated. Mothers who delay childbearing, but who
share a similarly disadvantaged family background with teenage mothers, have children at equal risk for academic difficulties and behavior problems as the children of teenage mothers.

Comparing the children of biological sisters improves upon standard epidemiological studies by controlling for unmeasured environmental variables that are shared by sisters; nevertheless, this approach does not control for genetic background variables. A number of twin studies have demonstrated that genetic differences between individuals partly account for population variation in timing of first birth (Kohler, Rodgers, & Christensen, 2002; Neiss, Rodgers, & Rowe, 2002) and family size (Fisher, 1930, but see Williams & Williams, 1974). In addition, there are genetic influences on other milestones of sexual development predictive of age at first birth (Udry & Cliquet, 1982), including age at menarche (Chern, 1974; Kaprio, Rimpelä, Winter, Viken, Rimpelä, & Rose, 1995; Rowe, 2002) and age at initiation of sexual intercourse (Bailey, Kirk, Zhu, Dunne, & Martin, 2000; Doughty, 2000; Dunne, Martin, Statham, Slutske, Dinwiddie, Bucholz, et al., 1997; Martin, Eaves, & Eysenck, 1977; Lyons et al., 2004; Miller, Pasta, MacMurray, Chiu, Wu, & Comings, 1999; Rodgers, Rowe, & Buster, 1999). Previous research using the sample used in the current study found that genetic differences accounted for 33% of the variation in whether a woman gave birth as a teenager, while environmental influences shared by twins accounted for 27% and environmental influences not shared by twins accounted for 40% (Waldron, 2004). These results are consistent with a larger body of research demonstrating that differences in family and other social environments partly reflect genetic differences between individuals.

Not only do genetic differences between women influence the likelihood of adolescent childbearing, but children inherit these same genetic influences. The environmental experiences involved in having a teenage parent are thus correlated with children’s genetic liabilities, a
phenomenon referred to in the behavior genetics literature as passive gene-environment correlation (or rGE; Plomin & Bergeman, 1991; Rutter & Silberg, 2002). Moreover, genetic factors influence diverse forms of psychopathology, including internalizing problems (Haberstick, Schmitz, Young, & Hewitt, 2005), externalizing problems (Arsenault et al., 2003; Scourfield, Van den Bree, Martin, & McGuffin, 2004; Slutske, et al., 1997), and alcohol and substance use problems (Heath, Bucholz, Madden, et al., 1997; Heath & Martin, 1994). If the same genetic factors influence reproductive timing in women and risk for psychopathology in offspring (an example of pleiotropy, wherein a single set of genetic factors is related to multiple phenotypes), then the poorer adjustment seen in children of adolescent mothers may be mediated, to some extent, by common genetic liabilities. It should be noted that the origin of a psychosocial risk indicator is not necessarily the same as the mechanism of its effect, as made obvious by an analogy of smoking: the personality predictors of smoking initiation are completely independent of the mechanisms by which smoking causes lung cancer (Rutter, Silberg, & Simonoff, 1993). Genetic influence on adolescent motherhood, therefore, does not necessarily indicate that association between adolescent motherhood and offspring mental health is not environmentally mediated; rather, genetic effects remain a possibility to be examined empirically.

The Children-of-Twins Design.

The Children-of-Twins design (D’Onofrio et al., 2003; Heath, Kendler, Eaves, & Markell, 1985; Nance & Corey, 1976) improves upon the first cousin comparison design utilized by Geronimus et al. (1994) and Turley (2003), because it compares the children of twin sisters, who share a common family environment and a common set of genes. In a comparison of a pair of identical twin mothers and their respective children, in which Twin A is younger at the birth
of her first child than Twin B, and the firstborn child of Twin A demonstrates more mental health problems than the firstborn child of Twin B, the association cannot be attributed to any family background variables – either environmental or genetic in origin – that make twin mothers similar. In addition, the majority of previous studies have been limited to only one child per mother (but see Turley, 2003). Sampling multiple children per twin parent permits comparisons between siblings: if the child born when his or her mother is a teenager demonstrates more mental health problems than his or her siblings born later in the mother’s life, the association cannot be attributed to any environmental or genetic variables shared by siblings (Dick, Johnson, Viken, & Rose, 2000; Rodgers, Cleveland, van den Oord, & Rowe, 2000). Considered together, the family comparisons permitted by the Children-of-Twins design provide a rigorous test of whether a putative environmental risk – in this case, adolescent motherhood – causes adverse child outcomes. The Children-of-Twins design has been used previously to examine the processes by which marital conflict (Harden et al., 2005), step-fathering (Mendle et al., In press), harsh punishment (Lynch et al., In press), smoking during pregnancy (D’Onofrio et al., 2003), marital dissolution (D’Onofrio et al., 2005; In press), parental schizophrenia (Gottesman & Bertelsen, 1989), and parental alcohol problems (Jacob et al., 2003) are related to offspring adjustment.

In the current investigation, we examine the relation between adolescent motherhood and behavior, substance use, and internalizing problems in adolescent and adult offspring. Although our investigation uses twins and their offspring, and thus may be broadly characterized as “behavior genetic,” we wish to emphasize that our goal is not to investigate genetic processes per se. Rather, our goal is to discriminate possible causal relations between adolescent motherhood and offspring mental health problems, similar to both previous epidemiological
research using statistical controls (e.g., Christ et al., 1990) and to previous quasi-experimental family research (Geronimus et al., 2004; Turley, 2003). To that end, we capitalize on the clustering within families of potential confounding variables – both environmental and genetic in origin – by comparing biologically related children who differ in their exposure to adolescent motherhood.

Method

Participants

Participants included twins and their offspring from a volunteer twin register, formed in 1978 and maintained by the Australian National Health and Medical Research Council (NHMRC). Figure 1 illustrates the relationships among three Australian twin sub-samples. The primary sub-sample to be utilized in the present research is the Maternal Sub-Sample, but explaining the composition of this sub-sample necessitates describing all three.

First, in 1993-5, 5889 twins (86% response rate) were interviewed by telephone as part of an on-going investigation of the genetics of alcoholism (Genetics of Alcoholism Twin Sample; Heath, Bucholz, Madden, et al., 1997). Mean ages at the 1993 interview were 42.7 years for men (range = 28 – 89 years) and 44.8 years for women (range = 27 – 90 years). In keeping with the low proportion of ethnic minorities in the non-Aboriginal Australian population, the twins were almost exclusively of European ancestry. The sample mirrors other population demographics, as well. Previous analyses have found no effects of self-selection for marital status, religious affiliation, frequency of church attendance, personality traits, mental illness, and abnormal behavior (Heath, Bucholz, Madden, et al., 1997; Slutske et al., 1997). The Genetics of Alcoholism Sample does overrepresent monozygotic twins, overrepresent twins born before 1930, and underrepresent twins with less than an 11th grade education (Baker, Treloar, Reynolds,
Heath, & Martin, 1996; Slutske et al., 1997). Cooperation bias and the underrepresentation of poorly educated participants, however, have been shown to have negligible effects on behavioral genetic analyses of conduct disorder (Heath, et al., 1996). Overall, the Genetics of Alcoholism Sample can be considered broadly generalizable to the non-Aboriginal Australian population.

Between 1998 and 2001, investigators contacted a selected sub-sample of the 1993 interview sample’s offspring (Children-of-Twins Sample). Selection targeted offspring considered at-risk for conduct disorder, depressive disorder, alcohol dependence, and/or divorce, as well as a control group considered to be at low risk. In total, 2,554 offspring of 1,409 adult twins participated in the study (an 82% response rate). Of the children-of-twins, 51% came from nuclear families in which the twin parent did not have a history of psychopathology or divorce, and 24% came from nuclear families in which neither the twin parent nor the co-twin had a history of psychopathology or divorce.

The purpose of the present study is to examine early motherhood, thus the Maternal Sub-Sample was restricted to female-female twin pairs and their offspring. This sub-sample consisted of 1368 children (51.5% female, 48.5% male) of 230 complete pairs of twins and 252 individual twins. Of the twin pairs, 60.1% were monozygotic (MZ) and 39.04% were dizygotic (DZ). The number of children per nuclear family ranged from one to six; the mean number of siblings per nuclear family was approximately two. The offspring’s age at assessment ranged from 14 to 39 years ($M = 25.1; SD = 5.7$). The Maternal Sub-Sample was utilized in the present analyses.

**Measures**

Zygosity was determined by questionnaire responses concerning physical similarity and frequency of occasions where twins were mistaken for each other. When there was disagreement between co-twins about zygosity or when zygosity assignment was otherwise ambiguous, further
information, including photographs, was requested. Comparisons of these zygosity assignments with multilocus genotyping have shown the self-report questions to greater than 95% accurate (Eaves, Eysenck, & Martin, 1989). In addition, final zygosity assignments from questionnaire responses demonstrated perfect agreement with zygosity assignment based on DNA typing of eight polymorphic markers in a subsample of 190 twin pairs (Duffy, 1994).

Mothers’ age at each birth and children’s psychosocial adjustment were assessed using the SSAGA-OZ (Semi-Structured Assessment for the Genetics of Alcoholism – OZ version), a comprehensive psychiatric interview designed for genetic studies of alcoholism, modified for use over the telephone (Bucholz et al., 1994). The SSAGA is derived from the National Institutes of Mental Health Diagnostic Interview Schedule (DIS; Helzer & Robins, 1988), the Structured Clinical Interview for DSM-III-R (Spitzer, Williams, Gibbon, & First, 1992), the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978), the Composite International Diagnostic Interview (Robins et al., 1988), and the HELPER Interview (Coryell, Cloninger, & Reich, 1978). The SSAGA-OZ includes DSM-IV diagnostic items for oppositional defiant disorder, attention deficit hyperactivity disorder, conduct disorder, alcohol dependence and abuse, illicit drug use, and major depression. Additional items assess suicidal intent and self-injurious behavior, lifetime cigarette use, regular smoking, lifetime history of alcohol use, and regular alcohol use. Interrater reliability of the SSAGA has been shown to be excellent and did not appear to be compromised by telephone use (Bucholz et al., 1994; Heath, Bucholz, Madden, et al., 1997). Interviews were administered by trained lay interviewers who were supervised by a trained clinical psychologist. All interviews were audiotaped, and a randomly selected 5% of the interviews were reviewed for quality control and check of coding inconsistencies.
Correction for Sample Selection

Similar to many genetic epidemiological studies, the Maternal Sub-Sample is a product of deliberate selection on stratification variables (parental psychiatric history and marital status), in conjunction with potentially non-random self-selection. Without addressing this sample selection problem, our analyses may be biased. In general, sample selection may be considered a case of missing data, with data on the variables of interest only present in selected twins and their families (Bechger, Boomsma, & Koning, 2002). Accordingly, sample selection may be considered within Rubin’s model of missing data (Rubin, 1976; Little & Rubin, 1987). Missingness (i.e., selection) is considered ignorable not only when participants are a random sample from the general population (selected completely at random; SCAR), but also when selection depends on the values of other variables, related to the variables of interest, observed in both selected and unselected participants (selected at random; SAR).

As detailed above, the Maternal Sub-Sample is derived from a larger, population-representative twin sample (the Genetics of Alcoholism Twin Sample), in which multiple sociodemographic and psychiatric characteristics, including the deliberate selection variables, were observed for both selected and unselected twins. Therefore, the Maternal Sub-Sample may be considered SAR. Following Heath, Madden, & Martin’s (1998) procedure for developing and testing models of non-response in SAR data, we used multiple logistic regression to identify predictors of whether or not a female-female twin pair (i.e., at least one twin) from the Genetics of Alcoholism Twin Sample participated in the Maternal Sub-Sample. Pair-wise participation, rather than individual twin participation, was predicted because sample selection occurred on the pair level. Sociodemographic and psychiatric characteristics assessed using the SSAGA-OZ were used as predictors of selection into the Maternal Sub-Sample (see Bucholz et al., 1994 or
Heath et al., 1998 for more details). Propensity weights were then constructed as the inverse predicted probability of a pair participating in the Maternal Sub-Sample, as calculated from the logistic regression model (for details on standard methods of data weight construction, see Heath et al., 1998 or Lee, Forthofor, & Lorimor, 1989). Our model for selection was tested by comparing the unweighted and weighted frequency distributions of sociodemographic and psychiatric characteristics in the Maternal Sub-Sample with the distributions in the female-female twin pairs from the Genetics of Alcoholism Sample.

Table 1 summarizes the observed frequency distributions for sociodemographic characteristics, family history of alcohol use and depression, alcohol use behaviors, and psychiatric disorders of twin pairs participating in the Maternal Sub-Sample compared to non-selected female-female twin pairs from the population-representative Genetics of Alcoholism Sample. Based on univariate comparisons, selected twin pairs were different from non-selected female pairs in multiple respects, including birth cohort, family history of alcohol and depression, panic, phobias, high frequency drinking, suicidal ideation, major depression, alcohol dependence, conduct disorder, and migraine. Also summarized in Table 1 are partial odds ratios estimated from a multiple logistic regression predicting participation in the Maternal Sub-Sample. The following were significant predictors of sample selection: birth cohort; age; discordance for spouse alcohol use history, twin and spouse depression history, panic, agoraphobia, social phobia, major depressive disorder, alcohol dependence, conduct disorder; and concordance for paternal alcohol use history, high maximum drinking, migraine, and major depressive disorder. Most of these predictors were deliberate selection variables.

Finally, the right hand columns of Table 1 summarize the frequency distributions for the Maternal Sub-Sample when pair-wise propensity weights were used. A comparison of these
distributions may be used to examine whether data weighting removed differences between selected and non-selected participants. Of the significant predictors of sample selection identified by multiple logistic regression, differences in mean age, as well as frequency differences in birth cohort, discordance for panic, social phobia, major depressive disorder, and conduct disorder; and concordance for high maximum number drinks were removed by data weighting. In addition, frequency differences in discordance for spouse alcohol use history, twin and spouse depression history, and alcohol dependence; and concordance for migraine, major depression, and paternal alcohol use history were substantially reduced, although univariate comparisons remained statistically significant. Residual differences in the frequency of psychiatric disorders may be evident even after data weighting because the probability of selection was heavily driven by sociodemographic characteristics, particularly birth cohort, as illustrated by the extreme odds ratios associated with birth cohort dummy variables. These propensity weights were used in all subsequent analyses. Despite data clustering, all methods for single level data weighting are applicable, because weights are applied at the highest level of clustering only (Asparouhov, 2004).

Exploratory Factor Analysis of Offspring Adjustment

We performed an exploratory factor analysis (EFA) of dichotomous DSM-IV diagnostic symptoms endorsed by children from the entire Children-of-Twins sample during the SSAGA interview. Only one child per twin family from the entire Children-of-Twins sample was included to avoid bias introduced by response correlations between related members. One SSAGA item (forcing another into sexual intercourse) was dropped because of low endorsement frequency. All EFA were conducted in Mplus (Muthen & Muthen, 1998-2004) using Promax rotation. The EFA resulted in a three-factor solution with inter-factor correlations between .39
and .49. Each factor showed high internal consistency (α factor 1 = .90, α factor 2 = .87, α factor 3 = .91). Complete details of the exploratory factor analysis are available upon request from the first author or from D’Onofrio et al. (2005).

The first factor, Behavior Problems (BP), includes reports of oppositional defiant behaviors, attention problems, hyperactivity, conduct problems (excluding serious violations of rules), and report of recurrent legal problems due to alcohol use. The second factor, Substance Use Problems (SUP), includes cigarette use, alcohol use, alcohol abuse, alcohol dependence, drug use, problems associated with drug use, and serious violations of rules. The third factor, Internalizing Problems (IP), includes depressive episode criteria and items concerning suicide.

A 1-year follow-up of 176 of the offspring of the twins found the child adjustment factors to be reliable (κ = 0.78 for Behavior, 0.89 for Substance Use, and 0.74 for Internalizing). We summed the endorsed items loading on each factor to calculate unit-weighted factor scores for children’s BP (M = 3.52, SD = 4.42, range = 0 – 28), SUP (M = 10.26, SD = 6.02, range = 0 – 32), and IP (M = 4.03, SD = 4.39, range = 0 – 16). Symptom counts were used rather than clinical diagnoses because clinical-level pathology was rare; symptom counts better capture the range of outcomes expected in the general population.

Results

Descriptive Means Comparisons

We conducted five descriptive means comparison to illustrate the between-family, within-twin pair, and within-nuclear family associations. These comparisons of means were meant to be purely illustrative, thus no inferential statistics were computed. The role of sampling error will be assessed in the following, more rigorous, hierarchical linear modeling analyses. In
lieu of probability testing, we computed effect sizes ($d$; Cohen, 1988) for each comparison.\(^1\)

“Teen mothers” were defined as women who were $\leq 20$ years old at childbirth. The five comparisons were as follows:

1. All children born to teen mothers ($n = 91$) vs. all children born to non-teen mothers ($n = 1277$).

2. All children born to teen mothers vs. firstborn children of non-teen mothers ($n = 471$).

3. All children born to teen mothers vs. laterborn (i.e., not firstborn) children born to non-teen mothers who had their first child when still a teen ($n = 329$).

4. Laterborn siblings of children born to teen mothers vs. laterborn children born to non-teen mothers who never had a teen birth ($n = 477$).

5. Firstborn children classified into four groups: (a) children of non-teen mothers whose aunts never had a teen birth ($n = 238$); (b) children of non-teen mothers whose aunts had a teen birth ($n = 28$); (c) children of teen mothers whose aunts never had a teen birth ($n = 28$); and (d) children of teen mothers whose aunts also gave birth as a teen ($n = 14$). (Only firstborn children of complete twin pairs were included, because information on both twins is necessary to accurately classify children into groups.)

Table 2 shows the mean behavior, substance use, and internalizing problems of children grouped by the age of their mother. Comparing the first two columns on the left of Table 2, the children born to mothers $\leq 20$ years old demonstrated more BP ($d = 0.435$), SUP ($d = 0.414$), and

\[
d = \frac{\bar{x}_1 - \bar{x}_2}{\sqrt{\frac{(n_1 - 1)s_1^2 + (n_2 - 1)s_2^2}{n_1 + n_2}}} = \frac{\bar{x}_1 - \bar{x}_2}{s_p}.
\]

An effect size of 0.2 is considered small, 0.5 medium, and 0.8 large.
IP ($d = 0.251$) than children of older mothers. Comparing the first and third columns of Table 2, the children born to teenage mothers demonstrated higher BP ($d = 0.535$), SUP ($d = 0.513$), and IP ($d = 0.276$) than the firstborn children of older mothers. Of the 91 children born to teen mothers, the majority were firstborn children ($n = 76$) and all but one were second born at the latest. This comparison, therefore, partly eliminated the effects of birth order. These phenotypic associations, the latter partially controlling for birth order effects, are consistent with previous epidemiological research.

The third analysis compares the children of teenage mothers with their laterborn siblings, thus controlling for any genetic or environmental variables common to children in the same nuclear family. Comparing the first and fourth columns of Table 2, children born to teenage mothers show more BP ($d = 0.334$), SUP ($d = 0.292$), and IP ($d = 0.177$) than siblings born later in their mother’s development. This is consistent with a causal effect, although the effect sizes are attenuated compared to the between-family comparisons. The relation between adolescent motherhood and child mental health problems may be attenuated when comparing siblings rather than unrelated children because some of the epidemiological association is due to environmental or genetic variables common to children in a nuclear family, or because adolescent childbearing changes the nuclear family environment such that all subsequent children are also adversely affected (e.g., by reducing later socioeconomic resources). Caution in interpreting the sibling comparison is warranted.

The fourth analysis compares children who were all born to non-teen mothers ($M_{age} = 28.3$ years in the first group vs. $M_{age} = 28.9$ years in the second group), and who have similar birth orders (all non-firstborn), but who differ in whether they were born to a mother who had her first child as a teen and thus have different levels of environmental and genetic risk. In other
words, they only differ in whether they come from the type of nuclear family that is started when a woman is still a teenager. Comparing the fourth and fifth columns of Table 2, laterborn children whose mother had her first child when still a teen did not demonstrate substantially more BP \((d = -0.028)\), SUP \((d = 0.061)\), or IP \((d = 0.072)\). This, too, is consistent with a causal effect, because an increased risk of psychopathology was not evident for offspring not born when their mothers were teenagers.

The final means analysis (Table 3) compared children divided into groups based on their aunt’s and their mother’s teen birth status. The key comparison is between the children of discordant twin pairs (i.e., between first cousins in discordant twin families): Does the twin who gave birth earlier than her co-twin have a child who demonstrates poorer adjustment than his or her cousin? In other words, does being born into the type of extended family where women give birth as teenagers, even if one’s own mother delays childbearing, predict one’s adjustment as well as being born to a teen mother? Comparing the second and third columns of Table 3, children born to teenage mothers demonstrated substantially more BP \((d = 0.214)\), SUP \((d = 0.408)\), and IP \((d = 0.383)\), than their first cousins born to older mothers. This pattern of results, again, is consistent with causal effects of early motherhood, rather than the operation of genetic or environmental third variables.

*Hierarchical Linear Models*

The above means comparisons, while capitalizing on multiple family relationships, have three critical limitations. First, the inclusion of varying numbers of non-independent observations per nuclear family weights some families more heavily than others, precluding the correct estimation of standard errors. Second, the means comparisons do not capitalize on the difference in genetic relatedness between MZ and DZ twin pairs, and are thus incapable of
distinguishing between environmental and genetic confounds. Third, means comparisons, by
definition, illustrate the relations between adolescent childbearing and mean numbers of
behavior, substance use, and internalizing problems, but do not illustrate any relations with the
extent to which children in the same family differ in mental health problems. Hierarchical linear
modeling (HLM) is an analytic strategy capable of addressing the above limitations (see
Raudenbush & Byrk, 2002 for a review). First, HLM explicitly models non-independence
among observations by partitioning unaccounted for variance into that shared by members of a
cluster \( \text{(random effects)} \) and that unique to an individual \( \text{(residual)} \), thus permitting correct
calculation of inferential statistics. Second, the HLM approach allows the inclusion of zygosity
interactions, in order to examine to what extent family confounds of the association between
early motherhood and child adjustment are genetic versus environmental in origin. Third, the
residual and random effect variance components can be modeled as a function of another
covariate, thus allowing the examination of whether adolescent motherhood is associated with
greater differences among siblings in the same nuclear family.

As shown in Figure 2, the complex organization of our sample can be considered on three
levels: individual offspring comprise level one; clusters of offspring born to an individual
mother \( \text{(nuclear families)} \) comprise level two; and clusters of offspring born to both mothers in a
twin pair \( \text{(twin families)} \) comprise level three. The most basic HLM for our three-level data is an
unconditional model:

\[
y_{ijk} = B_{000} + r_{00k} + r_{0jk} + e_{ijk}
\]

\[
r_{00k} \sim N(0, \tau_{000}); \quad r_{0jk} \sim N(0, \tau_{111}); \quad e_{ijk} \sim N(0, \sigma^2).
\]

which models the adjustment of the \( i^{th} \) child of the \( j^{th} \) twin in the \( k^{th} \) twin pair as a function of an
overall intercept, variation shared by all children within a twin family \( (\tau_{00k}) \), variation shared by
all children within a nuclear family \( (\tau_{0jk}) \), and residual variation unique to individual offspring
(σ²). (Dividing the variation shared by a cluster (e.g., τ₀₀k) by the total variance yields the familiar intraclass correlation.)

Similar to traditional regression analysis, this model can be first expanded to include the effect of being born to a teenage mother, and subsequently expanded to include measured covariates as statistical controls:

\[ y_{ijk} = B_{000} + B_{1}x_{1ijk} + \ldots + B_{n}x_{nijk} + r_{00k} + r_{0jk} + e_{ijk}. \]  

(2)

In the above model, \( x_{1ijk} \) is a dichotomous variable representing whether a child was born to a teenage mother; other \( x \) variables include the effect of child gender and the linear and quadratic effects of child age.

The methodological controls afforded by the Children-of-Twins design may be modeled by including additional fixed effects. First, we may include a dichotomous variable representing whether a mother’s first child was born when she was a teenager (\( x_{0jk} \)):

\[ y_{ijk} = B_{000} + B_{1}x_{1ijk} + B_{2}x_{0jk} + \ldots + B_{n}x_{nijk} + r_{00k} + r_{0jk} + e_{ijk}. \]  

(3)

Notice that the inclusion of this level two covariate changes the interpretation of the \( x_{1ijk} \) effect: among children born to mothers who had their first child when still a teenager, does being born oneself to a teenage mother predict adjustment? This model more precisely recapitulates the descriptive means analysis comparing children of teenage mothers with their later-born siblings.

Second, we may include an ordinal variable (\( x_{00k} \)) representing how many women in a twin family gave birth to their first child while still a teen (0, 1, or 2):

\[ y_{ijk} = B_{000} + B_{1}x_{1ijk} + B_{2}x_{0jk} + B_{3}x_{00k} + \ldots + B_{n}x_{nijk} + r_{00k} + r_{0jk} + e_{ijk}. \]  

(4)

Again, the inclusion of this level three covariate changes the interpretation of the \( x_{0jk} \) effect: among children born into the type of extended family where women are at risk for teenage pregnancies, does being born to mother who gives birth as a teen predict adjustment? This
model more precisely recapitulates the final descriptive means analysis comparing the firstborn children of twins discordant for teenage childbearing.

Third, a significant effect of the level three covariate \((x_{00k})\) indicates that being born into an extended family where women give birth as teenagers, regardless of whether one’s own mother gave birth as a teenager, predicts poorer adjustment. Whether this effect is due to environmental or genetic variables can be estimated by including an interaction between the twin family covariate and zygosity (coded as 0 in MZ twin families and 1 in DZ twin families):

\[
y_{ijk} = B_{000} + B_1 x_{ijk} + B_2 x_{0jk} + B_3 x_{00k} + B_4 x_{00k} (zygo) + B_5 zygo + \ldots + B_n x_{njk} + r_{00k} + r_{0jk} + e_{ijk}.
\]

If, for example, the relationship between early motherhood and BP were entirely due to transmission of common genetic liabilities from parent to child, then children’s BP will be predicted equally well by their aunt’s teenage childbearing as their mother’s in MZ twin families. In DZ families, however, children’s BP will be better predicted by their mother’s teenaged childbearing because of genetic differences between sisters. By coding zygosity with 0 for MZ and 1 for DZ, the regression coefficient for \(x_{00k}\) \((B_3)\) represents the relation between number of teen births in the twin family and offspring mental health problems in MZ twin families, and the regression coefficient for the interaction term \((B_4)\) represents the difference in the effect of the twin family covariate between MZ and DZ twin families. A significant effect of the interaction term, i.e., a significant difference in the effect of the twin family covariate between MZ and DZ twin families, indicates that the influences confounding associations between early motherhood and adjustment are genetic in origin. (It is conventional to include the main effect of an interaction variable, but we do not expect overall adjustment to differ significantly between the children of MZ and DZ twin families.) All models were estimated using SAS. Model fit was
compared using differences in model $\chi^2$, which are themselves distributed as $\chi^2$, with $df$ equal to the difference between the models’ $df$.

**Behavior Problems**

Results from the unconditional model (not tabulated) indicate that children in the same twin family (i.e., cousins) are not reliably similar in their behavior problems ($\tau_{000} = 0$). This is consistent with previous analyses of Children-of-Twin analyses using this dataset (e.g. Lynch et al., In press). The intraclass correlation reflects the extent to which children in the same nuclear family are similar; it is calculated as the proportion of unaccounted for variance shared by members of a cluster. The nuclear family intraclass correlation for behavior problems was 0.189, indicating that there was only a small degree of similarity among siblings.

Model 1, shown in Table 4, showed there was a significant effect of being born to a teenage mother. Model 2 statistically controlled for the effect of child gender and the linear and quadratic effects of child age. Males reported significantly more behavior problems, consistent with previous research; however, there were no significant effects of age. Even after the addition of these covariates, being born to a teenage mother remained a significant predictor of behavior problems. Model 3 included the effect of being born to a mother who had her first child as a teen; this covariate had no significant effect. The effect of being born to a teen mother remained significant, consistent with a causal hypothesis. Model 4 included the effect of the number of teen births in the twin family. This covariate significantly predicted behavior problems, indicating that environmental or genetic variables shared by twin sisters were responsible for at least part of the association between early motherhood and behavior problems. Nevertheless, the effect of being born to a teen mother remained significant, again consistent with a causal hypothesis.
Next, whether the family level confounds were environmental or genetic in origin was tested with an interaction between the number of teen births in the teen family and the zygosity of the teen pair in Model 5. The interaction was in the predicted direction (larger for MZ twin families than DZ families) but was marginally non-significant ($p = 0.06$). The change in model fit from Model 4 to Model 5 was significant, however, suggesting that family-level influences were at least partly genetic in origin. Figure 3 illustrates the number of behavior problems estimated by Model 5 for offspring born into twin families with no adolescent childbearing, offspring whose first cousins were born to adolescents (in either DZ or MZ twin families), offspring born themselves to adolescents, and offspring who were born to adolescents and whose first cousins were born to adolescents (in either DZ or MZ twin families).

Overall, the results suggest that behavior problems are related to adolescent motherhood specifically and to genetic risk passed from mother to offspring. The relative importance of adolescent motherhood, *per se*, versus genetic risk can be quantified by comparing the proportional reduction of error in predicting an individual outcome (level-one $R^2$; Snijders & Bosker, 1999) for Model 2, which includes the direct effect of adolescent motherhood, with Model 4, which includes the direct effect of adolescent motherhood and the effects of being in the type of (nuclear or twin) family where women give birth as teens. Model 2 accounted for 6.12% of the variance in individual offspring’s behavior problems, whereas Model 4 accounted for 6.88%, indicating that family genetic risk explains relatively little of the association between behavior problems and adolescent motherhood.

*Substance Use Problems*

Results from the unconditional model (not tabulated) indicated that, unlike behavior problems, children in the same twin family were modestly similar in their substance use
problems (intraclass correlation = 0.195; \( \tau_{000} = 6.294 \)). Siblings were even more similar in their substance use problems (intraclass correlation = 0.249; \( \tau_{111} = 6.454 \)). Subsequent HLM, shown in Table 5, revealed a pattern similar to that seen for behavior problems. Male children reported more substance use problems. Older children reported more substance use problems, with the effect of age tapering as offspring reached middle age. Regardless of the statistical or methodological controls added, the effect of being born to a teenage mother remained significant. In addition, the number of teen births in the twin family predicted substance use problems. The change in model fit from Model 4 to Model 5 was marginally non-significant \((p = 0.06)\), as was the coefficient itself. The direction of the interaction term suggests that either the family-level influences are entirely environmental in origin or that the analyses lack the power to discriminate between environmental and genetic effects. Figure 4 illustrates the number of substance use problems estimated by Model 4 for offspring with increasing exposure to family background risks and exposure to adolescent motherhood per se. Comparing level-one \( R^2 \) for Model 2 (9.47%) with Model 4 (10.06%) suggests that family background risks, although significant, explain relatively little of the association between substance use problems and adolescent motherhood.

*Internalizing Problems*

Results from the unconditional model (not tabulated) indicated that there was little reliable similarity between children in the same twin family (intraclass correlation = 0.065; \( \tau_{000} = 1.04 \)) and between children in the same nuclear family (intraclass correlation = 0.183; \( \tau_{111} = 2.94 \)). Most of the variation in internalizing problems, then, exists among siblings. Subsequent HLM, shown in Table 6, revealed a pattern somewhat different than behavior problems and substance use. Male children reported *fewer* internalizing problems, consistent with previous
research. There were no effects of age. Regardless of the statistical or methodological controls added, the effect of being born to a teenage mother remained significant, consistent with the causal hypothesis. Unlike previous outcomes, the number of teen births in the twin family had no statistically significant effect and the magnitude of the parameter was quite small, indicating that genetic or environmental influences shared by twin sisters are not responsible for any of the association between early motherhood and IP. Figure 5 illustrates the number of internalizing problems estimated by Model 4 for offspring with increasing exposure to family background risks and to adolescent motherhood specifically.

Discussion

Results from both descriptive means comparisons and hierarchical linear models are consistent with the hypothesis that adolescent motherhood causes increased risk for behavior, substance use, and internalizing problems in offspring. Specifically, offspring born to adolescent mothers demonstrate higher numbers of mental health problems than their laterborn siblings and higher numbers than their first cousins, comparisons that control for genetic and environmental variables shared by family members. Previous studies of the relation between adolescent motherhood and offspring adjustment that have failed to find significant associations when comparing first cousins (Geronimus et al., 1992; Turley, 2003) have primarily used young children, whereas our study used adolescent and adult offspring aged 14 to 39 years. The adverse effects of adolescent motherhood for offspring mental health may be more evident as offspring themselves transition into adulthood, as has been previously suggested (Brooks-Gunn & Fustenberg, 1986). Overall, our results illustrate the usefulness of ostensibly “genetic” designs for elucidating putative environmental risks for psychopathology.
Adolescent childbearing was not significantly associated with elevated mental health problems for children born later in a mother’s life. This finding is consistent with Grogger (1997), who found that male children born to adolescent mothers had higher incarceration rates than their laterborn siblings. In contrast, Turley (2003) found that maternal age at first birth was more predictive of laterborn children’s cognitive test scores than maternal age at children’s own births, and Jaffee, Caspi, Moffit, Belsky, & Silva (2001) found that maternal age at first birth significantly predicted early school leaving, unemployment, early parenthood, and violent criminal offending in young adult offspring, above and beyond maternal age at offsprings’ own birth. The degree to which adolescent motherhood is generally predictive of compromised functioning for the entire nuclear family, versus specifically for the firstborn child, appears to differ between child outcomes.

Finally, being born into the type of extended family where women gave birth as teenagers, even if not born to a teenager oneself, was predictive of higher levels of behavior and substance use problems. It appears that the children of adolescent mothers would be at elevated risk for behavior and substance use, even if their mothers delayed childbearing, because of background risk variables. These results make intuitive sense, given that adolescent mothers frequently have histories of conduct problems (Bardone, Moffitt, Caspi, Dickson, & Silva, 1996; Woodward & Fergusson, 1998) and substance abuse (Ketterlinus, Lamb, & Nitz, 1994). Maternal antisocial behavior and substance use problems, in turn, are strongly predictive of similar problems in offspring (Frick & Loney, 2002; Jacob et al., 2003). To what extent the background variables confounding the association between adolescent motherhood and child behavior and substance use problems are genetic or environmental in origin remains a topic for future research. Hierarchical linear models suggest that the intergenerational association with
behavior problems is due, in part, to passive gene-environment correlation, whereas the
intergenerational association with substance use problems is due, in part, to environmental
confounds. Thus, it will be important to study further the underlying processes responsible for
each outcome associated with teenage motherhood. Explicit quantification of HLM in terms of
latent genetic and environmental components remains a difficult enterprise, although McArdle
and Prescott (2005) have recently detailed model fitting strategies that may be useful in this
regard.

Comparing Australia and the United States.

As mentioned previously, the U.S. has one of the highest teenage pregnancy rates in the
industrialized world (United Nations, 1991), primarily due to lower utilization of contraception
and abortion, not different patterns of adolescent sexual activity or proportions of ethnic
minorities. Approximately 5% of Australian adolescents experience a pregnancy each year
(versus 10% of U.S. adolescents each year), and over 50% of Australian pregnancies are aborted.
Adolescent motherhood, therefore, is a considerably less common phenomenon in Australia, and
its relative cultural infrequency may moderate the consequences for children’s adjustment.
Despite quantitative differences in base rates, there is little evidence for qualitative differences
between Australia and the U.S. in the biological, family, and sociocultural risk factors at play.
Extensive research on the correlates of adolescent motherhood is not available for Australian
populations; however, results from British and New Zealand populations are consistent with
American research (Waldron, 2004).

It is important to note one essential difference between Australia and the U.S.: the non-
Aboriginal population of Australia is almost exclusively Caucasian. This demographic
difference is important in two respects. First, the relative influence of genetic factors on
adolescent childbearing, as well as the processes by which adolescent motherhood is related to child functioning, may differ between racial sub-populations. Second, the existence of genetic influences on adolescent childbearing within a Caucasian sub-population cannot be used to infer the origin of differences in adolescent childbearing rates between ethnicities. Geronimus (2004) has suggested that the higher rates of teenage pregnancy seen among African-American and Latina women versus Caucasian women are driven entirely by cultural differences in perceived “optimal” fertility timing: among women experiencing structural constraints to economic success, higher health and mortality risks, and a normative multigenerational family structure, early childbearing may be correctly perceived as advantageous. Future genetically-informative research should use ethnically diverse American samples, in order to examine potential cultural differences between the U.S. and Australia and between ethnic groups in the U.S. in the processes relating adolescent motherhood with child functioning.

Limitations of the Children-of-Twins Design: What about the Fathers?

The Children-of-Twins design is described as quasi-experimental for good reason: Without random assignment of children to adolescent vs. adult mother conditions, we cannot prove that adolescent childbearing does or does not cause child psychopathology. The Children-of-Twins design controls for environmental and genetic variables shared by twin sisters, but variables that vary systematically with adolescent motherhood within twin pairs remain as uncontrolled confounds. Although this list of potential confounds is considerably shorter than traditional epidemiological studies, it still includes non-shared environmental influences on the twin mother and the genetic influences of the father (Eaves, Silberg, & Maes, 2005). Elevated rates of academic problems, school dropout, unemployment, delinquency, criminal behavior, alcohol and substance use are seen in the fathers of children born to adolescent girls (Tan &
Men with genetic liabilities for, among other behaviors, delinquency and substance use are thus more likely to impregnate adolescent girls, and their children inherited these same liabilities. Consequently, the higher numbers of behavior and substance use problems seen in the children of discordant twin pairs, a result superficially consistent with environmental causation, may at least partly be due to biological inheritance of paternal genetic liabilities. In other words, spousal genetic influence is confounded with the non-shared environmental variance in the twin parent, a notable limitation in using the Children-of-Twins design to examine the processes underlying associations between offspring functioning and any dyadic parental characteristic (Eaves, Silberg, & Maes, 2005). This limitation is mitigated, to some extent, by the inclusion of multiple children per twin parent. If siblings in the same nuclear family have the same father, then the test of whether the first born child of an adolescent mother fares worse than his or her siblings born later in the mother’s life controls for paternal genetic influence. Adolescent mothers, however, may be more likely than women who delay childbearing to have children by multiple partners, and thus differences among their children’s mental health problems may, at least in part, reflect inheritance of different parental genes. Unfortunately, we do not have sufficient information to ascertain the extent to which sibling offspring in our current study share paternity.

These limitations highlight several important points. First, it is critical for future research on adolescent childbearing to collect more information on fathers, even if they are non-residential. This need is not limited to the current line of research, but is common to research on families, especially high-risk families, in general. Second, controlling for the effects of genetic variation is a difficult task, even with complex behavioral genetic designs. Barring an
improbable sample of female identical twins having children by male identical twins, disentangling the environmental effects of maternal age from paternal genetic effects will remain a considerable challenge. Despite these limitations, the current study provides more rigorous control of environmental and genetic background variables than any previous investigation, and thus represents a stride forward in our understanding of the processes by which adolescent motherhood is related to child functioning.

Conclusions

In the current American political landscape, few topics galvanize such acrimonious debates as adolescent sexuality. The trend towards earlier initiation of sexual intercourse has been characterized as, among other things, an inevitable consequence of secular decreases in pubertal timing (e.g., Parent, Teilmann, Juul, Skakkebaek, Toppari, & Bourguignon, 2003), the liberation of youth from antiquated sexual mores (e.g., Reiss, 1990), or an indication of catastrophic moral decline (e.g., Popenoe, 1998). Unlike Western European nations, where delegating to public schools the task of educating adolescents about reproductive biology, contraceptive use, and sexually transmitted diseases is little contended, factions in the U.S. continuously debate not only what sexual education curricula should entail, but whether such education should be the proprietary domain of the family rather than schools. Perhaps not surprisingly, these debates have extended to adolescent childbearing: Many researchers and policymakers portraying the lives of adolescent mothers and their children as extremely bleak (e.g., Hayes, 1987), while others consider the idealization of delayed fertility as indicative of ethnic bias and emphasize potential advantages to early childbearing (e.g., Geronimus, 2004).

The current results, however, are consistent with a more nuanced view of adolescent motherhood. Far from being innocuous, adolescent motherhood was associated with elevated
risks for behavior problems, substance use problems, and internalizing problems in offspring, suggesting that delaying childbearing until adulthood may have long-term benefits for the psychological adjustment of offspring. However, a narrow focus on delaying fertility, without ameliorating the background risks experienced by adolescent mothers and their children, may not be fully effective in reducing offspring behavior and substance use problems. More generally, the current results highlight the usefulness of behavior genetic designs for elucidating the processes underlying associations between environmental risk factors and psychological outcomes, and for thus informing contentious public debates.
Acknowledgements

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Table 1.

Comparison of sociodemographic and psychiatric characteristics of female twins from the Genetics of Alcoholism Sample and the Maternal Sub-Sample, before and after data weighting.

<table>
<thead>
<tr>
<th>Sociodemographics</th>
<th>Genetics of Alcoholism Sample (%)</th>
<th>Maternal Sub-Sample (%)</th>
<th>Odds Ratios**</th>
<th>Weighted Maternal Sub-Sample (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cohort</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>26.80</td>
<td>9.54*</td>
<td>1.0</td>
<td>27.59</td>
</tr>
<tr>
<td>2</td>
<td>32.47</td>
<td>45.64*</td>
<td>15.16 (9.84 – 23.36)</td>
<td>26.51</td>
</tr>
<tr>
<td>3</td>
<td>40.73</td>
<td>44.81*</td>
<td>52.45 (27.08 – 101.59)</td>
<td>35.89</td>
</tr>
<tr>
<td>Age (Mean)</td>
<td>45.25</td>
<td>44.92</td>
<td>0.90 (0.88 – 0.93)</td>
<td>43.26</td>
</tr>
<tr>
<td><strong>Discordant Pairs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Family Alc. Use</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Twin</td>
<td>7.73</td>
<td>8.09</td>
<td>-</td>
<td>10.22</td>
</tr>
<tr>
<td>Father</td>
<td>10.30</td>
<td>9.75</td>
<td>-</td>
<td>12.24</td>
</tr>
<tr>
<td>Mother</td>
<td>2.68</td>
<td>2.49</td>
<td>-</td>
<td>2.25</td>
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<tr>
<td>Spouse</td>
<td>17.81</td>
<td>25.52*</td>
<td>1.71 (1.24 – 2.34)</td>
<td>19.17*</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Twin</td>
<td>32.93</td>
<td>40.46*</td>
<td>1.35 (1.01 – 1.80)</td>
<td>35.56*</td>
</tr>
<tr>
<td>Father</td>
<td>16.66</td>
<td>17.43</td>
<td>-</td>
<td>16.93</td>
</tr>
<tr>
<td>Mother</td>
<td>22.41</td>
<td>24.07</td>
<td>-</td>
<td>21.20</td>
</tr>
<tr>
<td>Spouse</td>
<td>22.79</td>
<td>31.95*</td>
<td>1.59 (1.20 – 2.11)</td>
<td>24.95*</td>
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<tr>
<td>Agoraphobia</td>
<td>6.52</td>
<td>6.64</td>
<td>0.45 (0.26 – 0.78)</td>
<td>6.11</td>
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<tr>
<td>Panic</td>
<td>6.85</td>
<td>11.62*</td>
<td>1.79 (1.10 – 2.92)</td>
<td>6.96</td>
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<td>Social phobia</td>
<td>3.62</td>
<td>5.39*</td>
<td>-</td>
<td>4.16</td>
</tr>
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<td>-</td>
<td>3.55</td>
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<tr>
<td>Abstain from alcohol</td>
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<td>3.73</td>
<td>-</td>
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</tr>
<tr>
<td>High frequency drinking</td>
<td>23.62</td>
<td>27.18*</td>
<td>-</td>
<td>23.21</td>
</tr>
<tr>
<td>High density drinking</td>
<td>12.82</td>
<td>12.03</td>
<td>-</td>
<td>13.87</td>
</tr>
<tr>
<td>High maximum number drinks</td>
<td>18.14</td>
<td>17.22</td>
<td>-</td>
<td>17.12</td>
</tr>
<tr>
<td><strong>Migraine</strong></td>
<td>38.58</td>
<td>35.48</td>
<td>-</td>
<td>39.04</td>
</tr>
<tr>
<td><strong>Suicidal ideation</strong></td>
<td>4.52</td>
<td>7.26*</td>
<td>-</td>
<td>5.99</td>
</tr>
<tr>
<td><strong>Major depression</strong></td>
<td>33.59</td>
<td>43.36*</td>
<td>2.05 (1.53 – 2.76)</td>
<td>34.18</td>
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<tr>
<td><strong>Alcohol dependence</strong></td>
<td>8.60</td>
<td>12.24*</td>
<td>1.92 (1.20 – 3.08)</td>
<td>10.64*</td>
</tr>
<tr>
<td><strong>Conduct disorder</strong></td>
<td>3.34</td>
<td>5.81*</td>
<td>2.92 (1.47 – 5.80)</td>
<td>3.24</td>
</tr>
</tbody>
</table>

Concordant Pairs

**Family Alc. Use**

History
<table>
<thead>
<tr>
<th>Predictor</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
<th>Univariate Comparison</th>
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</thead>
<tbody>
<tr>
<td>Twin</td>
<td>0.38</td>
<td>1.04*</td>
<td></td>
</tr>
<tr>
<td>Father</td>
<td>10.36</td>
<td>16.80*</td>
<td>1.76 (1.19 – 2.59)</td>
</tr>
<tr>
<td>Mother</td>
<td>2.08</td>
<td>3.32*</td>
<td></td>
</tr>
<tr>
<td>Spouse</td>
<td>1.75</td>
<td>2.70</td>
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<td>Family Depr. History</td>
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</tr>
<tr>
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<td>17.22*</td>
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</tr>
<tr>
<td>Father</td>
<td>6.90</td>
<td>10.37*</td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td>11.89</td>
<td>19.09*</td>
<td></td>
</tr>
<tr>
<td>Spouse</td>
<td>2.47</td>
<td>4.36*</td>
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</tr>
<tr>
<td>Agoraphobia</td>
<td>0.71</td>
<td>2.07*</td>
<td></td>
</tr>
<tr>
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<td>0.55</td>
<td>0.62</td>
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<td>Social phobia</td>
<td>0.11</td>
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<tr>
<td>Other phobias</td>
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<tr>
<td>Abstain from alcohol</td>
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<td>High maximum number drinks</td>
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<td>4.36</td>
<td>0.47 (0.22 – 0.995)</td>
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<td>Migraine</td>
<td>28.22</td>
<td>40.46*</td>
<td>1.59 (1.16 – 2.19)</td>
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<tr>
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<td>1.04*</td>
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<td>Major depression</td>
<td>11.51</td>
<td>19.92*</td>
<td>1.83 (1.15 – 2.92)</td>
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<tr>
<td>Alcohol dependence</td>
<td>1.10</td>
<td>1.87</td>
<td></td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>0.44</td>
<td>0.83</td>
<td></td>
</tr>
</tbody>
</table>

*Univariate comparisons with non-selected female participants from the Genetics of Alcoholism Sample are significant, $p < .05$.

**All listed odds ratios are significantly different from 1; $p < .05$. 95% confidence intervals are shown in parentheses. Non-significant odds ratios for remaining predictors are not shown.
Table 2.

*Mean Mental Health Problems in All Children of Teenage Mothers vs. All, Firstborn, and Laterborn Children of Older Mothers.*

<table>
<thead>
<tr>
<th>Problems</th>
<th>Teen Mother</th>
<th>Old Mother</th>
<th>1st Child when Teen</th>
<th>1st Child when Older</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All (n = 91)</td>
<td>All (n = 1277)</td>
<td>Firstborn (n = 471)</td>
<td>Laterborn (n = 477)</td>
</tr>
<tr>
<td>Behavior</td>
<td>4.92 (0.55)</td>
<td>3.21 (0.11)</td>
<td>2.85 (0.17)</td>
<td>3.37 (0.24)</td>
</tr>
<tr>
<td>Substance Use</td>
<td>11.79 (0.68)</td>
<td>9.67 (0.16)</td>
<td>9.04 (0.26)</td>
<td>10.23 (0.32)</td>
</tr>
<tr>
<td>Internalizing</td>
<td>4.56 (0.51)</td>
<td>3.56 (0.12)</td>
<td>3.43 (0.19)</td>
<td>3.79 (0.23)</td>
</tr>
</tbody>
</table>

*Note.* Teenage mothers were $\leq 20$ years old at time of childbirth. Standard errors of mean estimates are in parentheses.
Table 3.

*Mean Mental Health Problems of Firstborn Children by Mother’s and Aunt’s Teenage Birth.*

<table>
<thead>
<tr>
<th>Problems</th>
<th>No Teen Mother/ No Teen Aunt</th>
<th>No Teen Mother/ Teen Aunt</th>
<th>Teen Mother/ No Teen Aunt</th>
<th>Teen Mother/ Teen Aunt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behavior</td>
<td>3.00 (0.28)</td>
<td>2.84 (0.73)</td>
<td>3.60 (0.83)</td>
<td>2.00 (0.82)</td>
</tr>
<tr>
<td>Substance Use</td>
<td>9.60 (0.37)</td>
<td>8.95 (1.19)</td>
<td>11.16 (1.11)</td>
<td>11.44 (1.61)</td>
</tr>
<tr>
<td>Internalizing</td>
<td>3.81 (0.25)</td>
<td>5.26 (0.97)</td>
<td>6.95 (0.92)</td>
<td>3.37 (1.28)</td>
</tr>
</tbody>
</table>

*Note.* Teenage mothers and aunts were $\leq$ 20 years old at time of childbirth. Standard errors of estimates are in parentheses.
Table 4.

Hierarchical Linear Models of Early Motherhood and Behavior Problems.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Random Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Twin Family - τ₀₀₀</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Nuclear Family - τ₁₁₁</td>
<td>2.82 (0.54)</td>
<td>2.70 (0.52)</td>
<td>2.68 (0.52)</td>
<td>2.59 (0.51)</td>
<td>2.45 (0.51)</td>
</tr>
<tr>
<td>Children - σ²</td>
<td>12.80 (0.60)</td>
<td>12.15 (0.58)</td>
<td>12.13 (0.58)</td>
<td>12.14 (0.58)</td>
<td>12.19 (0.58)</td>
</tr>
<tr>
<td><strong>Fixed Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>3.23 (0.13)</td>
<td>3.88 (2.30)</td>
<td>3.67 (2.30)</td>
<td>3.94 (2.30)</td>
<td>3.78 (2.29)</td>
</tr>
<tr>
<td>Child Gender**</td>
<td>1.75 (0.22)</td>
<td>1.75 (0.22)</td>
<td>1.74 (0.22)</td>
<td>1.73 (0.22)</td>
<td></td>
</tr>
<tr>
<td>Child Age</td>
<td>-0.05 (0.18)</td>
<td>-0.03 (0.18)</td>
<td>-0.07 (0.18)</td>
<td>-0.05 (0.18)</td>
<td></td>
</tr>
<tr>
<td>Child Age Squared</td>
<td>&lt;.01 (&lt;.01)</td>
<td>-0.01 (&lt;.01)</td>
<td>&lt;.01 (&lt;.01)</td>
<td>&lt;.01 (&lt;.01)</td>
<td></td>
</tr>
<tr>
<td>Born to Teen x₁₀jk</td>
<td>1.66 (0.43)</td>
<td>1.84 (0.42)</td>
<td>1.60 (0.45)</td>
<td>1.37 (0.46)</td>
<td>1.43 (0.46)</td>
</tr>
<tr>
<td>First Child as Teen x₀jk</td>
<td></td>
<td>0.45 (0.29)</td>
<td>-1.01 (0.69)</td>
<td>-1.17 (0.69)</td>
<td></td>
</tr>
<tr>
<td>Teen Births in Twin Family - x₀₀k</td>
<td></td>
<td></td>
<td>1.36 (0.58)</td>
<td>1.79 (0.62)</td>
<td></td>
</tr>
<tr>
<td>Parent Zygosity***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;.01 (0.31)</td>
</tr>
<tr>
<td>Zygosity Interaction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-0.81 (0.45)</td>
</tr>
<tr>
<td>-2LL</td>
<td>7942.0</td>
<td>7913.0</td>
<td>7910.6</td>
<td>7905.3</td>
<td>7900.5</td>
</tr>
<tr>
<td>[Δ-2LL; Δdf]</td>
<td>[29; 3]</td>
<td>[2.4; 1]</td>
<td>[4.7; 1]</td>
<td>[4.8; 1]</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard errors of parameter estimates in parentheses. Bold face type: significant at \( p < 0.05 \). *Variances of random effects estimated; **Female = 0; Male = 1; ***MZ = 0; DZ = 1.
Table 5.

Hierarchical Linear Models of Early Motherhood and Substance Use Problems.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Random Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Twin Family - $\tau_{000}$</td>
<td>6.14 (1.73)</td>
<td>5.80 (1.55)</td>
<td>5.71 (1.56)</td>
<td>5.91 (1.53)</td>
<td>5.68 (1.53)</td>
</tr>
<tr>
<td>Nuclear Family - $\tau_{111}$</td>
<td>6.44 (1.64)</td>
<td>6.29 (1.47)</td>
<td>6.36 (1.47)</td>
<td>5.97 (1.44)</td>
<td>6.10 (1.45)</td>
</tr>
<tr>
<td>Children - $\sigma^2$</td>
<td>19.32 (0.97)</td>
<td>17.06 (0.85)</td>
<td>17.05 (0.85)</td>
<td>17.08 (0.86)</td>
<td>17.06 (0.85)</td>
</tr>
<tr>
<td><strong>Fixed Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>9.87 (0.21)</td>
<td>-12.99 (3.13)</td>
<td>-13.12 (3.14)</td>
<td>-12.89 (3.13)</td>
<td>-13.11 (3.14)</td>
</tr>
<tr>
<td>Child Gender**</td>
<td>2.83 (0.29)</td>
<td>2.83 (0.29)</td>
<td>2.81 (0.29)</td>
<td>2.82 (0.29)</td>
<td></td>
</tr>
<tr>
<td>Child Age</td>
<td>1.65 (0.25)</td>
<td>1.66 (0.25)</td>
<td>1.63 (0.25)</td>
<td>1.63 (0.25)</td>
<td></td>
</tr>
<tr>
<td>Child Age Squared</td>
<td>-0.03 (&lt;.01)</td>
<td>-0.03 (&lt;.01)</td>
<td>-0.03 (&lt;.01)</td>
<td>-0.03 (&lt;.01)</td>
<td></td>
</tr>
<tr>
<td>Born to Teen $x_{1ijk}$</td>
<td>1.93 (0.59)</td>
<td>1.69 (0.56)</td>
<td>1.51 (0.59)</td>
<td>1.31 (0.59)</td>
<td>1.37 (0.59)</td>
</tr>
<tr>
<td>First Child as Teen $x_{0jk}$</td>
<td></td>
<td>0.46 (0.43)</td>
<td>-1.52 (0.99)</td>
<td>-1.65 (1.00)</td>
<td></td>
</tr>
<tr>
<td>Teen Births in Twin Family - $x_{00k}$</td>
<td></td>
<td></td>
<td>1.87 (0.85)</td>
<td>2.44 (0.91)</td>
<td></td>
</tr>
<tr>
<td>Parent Zygosity***</td>
<td>0.31 (0.49)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zygosity Interaction</td>
<td></td>
<td></td>
<td></td>
<td>-1.26 (0.70)</td>
<td></td>
</tr>
<tr>
<td>$-2LL$</td>
<td>8809.2</td>
<td>8667.2</td>
<td>8666.1</td>
<td>8661.4</td>
<td>8657.9</td>
</tr>
<tr>
<td>$[\Delta-2LL; \Delta df]$</td>
<td>[142; 3]</td>
<td>[1.1; 1]</td>
<td>[4.7; 1]</td>
<td>[3.5; 1]</td>
<td></td>
</tr>
</tbody>
</table>

Note. Standard errors of parameter estimates in parentheses. Bold face type: significant at $p < 0.05$. *Variances of random effects estimated; **Female = 0; Male = 1; ***MZ = 0; DZ = 1.
Table 6.

*Hierarchical Linear Models of Early Motherhood and Internalizing Problems.*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Random Effects</strong>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Twin Family - (\tau_{000})</td>
<td>1.04 (0.76)</td>
<td>0.57 (0.73)</td>
<td>0.56 (0.73)</td>
<td>0.55 (0.73)</td>
<td>0.58 (0.72)</td>
</tr>
<tr>
<td>Nuclear Family - (\tau_{111})</td>
<td><strong>2.94 (0.91)</strong></td>
<td><strong>3.05 (0.89)</strong></td>
<td><strong>3.05 (0.89)</strong></td>
<td><strong>3.05 (0.89)</strong></td>
<td><strong>2.98 (0.88)</strong></td>
</tr>
<tr>
<td>Children - (\sigma^2)</td>
<td><strong>12.15 (0.59)</strong></td>
<td><strong>11.86 (0.57)</strong></td>
<td><strong>11.86 (0.57)</strong></td>
<td><strong>11.87 (0.57)</strong></td>
<td><strong>11.87 (0.57)</strong></td>
</tr>
<tr>
<td><strong>Fixed Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>3.78 (0.14)</td>
<td>0.73 (2.36)</td>
<td>0.81 (2.36)</td>
<td>0.85 (2.36)</td>
<td>0.69 (2.36)</td>
</tr>
<tr>
<td>Child Gender**</td>
<td>-1.12 (0.22)</td>
<td>-1.12 (0.22)</td>
<td>-1.12 (0.22)</td>
<td>-1.12 (0.22)</td>
<td>-1.12 (0.22)</td>
</tr>
<tr>
<td>Child Age</td>
<td>0.17 (0.19)</td>
<td>0.17 (0.19)</td>
<td>0.16 (0.19)</td>
<td>0.16 (0.19)</td>
<td></td>
</tr>
<tr>
<td>Child Age Squared</td>
<td>&lt;.01 (&lt;.01)</td>
<td>&lt;.01 (&lt;.01)</td>
<td>&lt;.01 (&lt;.01)</td>
<td>&lt;.01 (&lt;.01)</td>
<td>&lt;.01 (&lt;.01)</td>
</tr>
<tr>
<td>Born to Teen</td>
<td>(x_{iijk})</td>
<td><strong>1.54 (0.44)</strong></td>
<td><strong>1.23 (0.43)</strong></td>
<td><strong>1.32 (0.46)</strong></td>
<td><strong>1.29 (0.47)</strong></td>
</tr>
<tr>
<td>First Child as Teen</td>
<td>(x_{ijk})</td>
<td>-0.18 (0.30)</td>
<td>-0.43 (0.72)</td>
<td>-0.55 (0.72)</td>
<td></td>
</tr>
<tr>
<td>Teen Births in Twin Family</td>
<td>(x_{00k})</td>
<td></td>
<td>0.24 (0.61)</td>
<td>0.62 (0.65)</td>
<td></td>
</tr>
<tr>
<td>Parent Zygosity***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.19 (0.33)</td>
</tr>
<tr>
<td>Zygosity Interaction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-0.77 (0.48)</td>
</tr>
</tbody>
</table>

\[-2LL\] 7971.2 7923.7 7923.4 7923.2 7920.5

[\(\Delta -2LL; \Delta df\)] [47.5; 3] [0.3; 1] [0.2; 1] [2.7; 1]

**Note.** Standard errors of parameter estimates in parentheses. Bold face type: significant at \(p < 0.05\). *Variances of random effects estimated; **Female = 0; Male = 1; ***MZ = 0; DZ = 1.
Figure Captions.

Figure 1. Relations among three Australia Twin Register sub-samples.

Figure 2. Organization of the Maternal Sub-Sample.

Figure 3. Number of behavior problems by gender, twin family zygosity, and family births to teenage mothers.

Figure 4. Number of substance use problems by gender and family births to teenage mothers.

Figure 5. Number of internalizing problems by gender and family births to teenage mothers.
Genetics of Alcoholism Sample
Population Based

5,889 Twin individuals
86% Response Rate
Selected Completely at Random

Selection for twins with history of psychopathology or divorce.

Children of Twins Sub-Sample

1,490 Twin individuals
2,554 Children of Twins
82% Response Rate
Selected at Random

Selection for female-female twin pairs only.

Maternal Sub-Sample

712 Female Twin individuals
1,368 Children of Twins
Selected at Random
Note. Circles represent female individuals; squares represent male individuals. Number of offspring per mother varies across nuclear families.
Who in Your Extended Family was Born to a Teenager?

Note. Mean number of behavior problems, as estimated by Model 5 (Table 5).
Substance Use Problems

Who in Your Extended Family was Born to a Teenager?

Note. Mean number of substance use problems for 26 year old offspring (median sample age), as estimated by Model 4 (Table 6).
Internalizing Problems

Who in Your Extended Family was Born to a Teenager?

Note. Mean number of internalizing problems for 26 year old offspring (median sample age), as estimated by Model 4 (Table 7).