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Intergenerational Consequences of Adolescent Substance Use: Patterns of Homotypic and Heterotypic Continuity

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Abstract

Does substance use run in families? In this paper, we examine both homotypic continuity in substance use – the impact of a parent’s adolescent substance use on their child’s adolescent substance use – and heterotypic continuity – the impact of a parent’s adolescent substance use on their child’s involvement in other adolescent problem behaviors. The analysis is based on data from the Rochester Youth Development Study and its intergenerational component, the Rochester Intergenerational Study. The initial study began with a representative sample of 7th and 8th grade students followed until age 31, and the intergenerational study is currently following their oldest biological child from childhood through adolescence. The final sample size in the current analysis consists of 341 parent-child dyads. For fathers, their adolescent substance use predicts both homotypic and heterotypic outcomes of their child. For mothers though, there is no evidence of intergenerational continuity for either homotypic or heterotypic outcomes. In contrast, when the parent’s adult substance use is examined, the opposite pattern emerges. The mother’s adult substance use is a more consistent predictor of child behavioral outcomes, but there is little evidence that the father’s adult behavior matters. Thus, it appears that the answer to the question of whether or not substance use runs in families is more nuanced than typically thought. Based on these results, continuity depends both on the sex of the parent and when in the parent’s life course substance use occurs.

Keywords

substance use; intergenerational continuity; homotypic continuity; heterotypic continuity

There is an old notion that substance use runs in families. That is, parental substance use is viewed as a significant risk factor for, and potential cause of, elevated involvement in substance use by offspring, and many studies find a significant relationship between parent and child substance use (e.g., Biederman et al., 2000; Capaldi et al., 2016; Chassin et al., 1991; Chassin et al., 1996; Cranford et al., 2010). These studies focus on contemporaneous, or *concurrent*, similarity in substance use, investigating the parent’s adult substance use (or a

retrospective report of earlier substance use) as it relates to the child's substance use. For example, Cranford et al. (2010) found that parental alcohol involvement during their children's childhood was related to their children's adolescent alcohol use. Identifying parental substance use as a significant risk factor for child substance use is an important contribution to our understanding of the origins of adolescent substance use and aids in the development of effective prevention and treatment programs (e.g., Dishion et al., 2012; Spoth et al., 2008).

Recently, attention has begun to focus on a related but distinct aspect of the extent to which "substance use runs in families." *Intergenerational continuity* is similarity in behavior between a parent and child that is exhibited during the same developmental stage for each generation (e.g., Kaplan & Tolle, 2006; Knight, Menard, & Simmons, 2014; Thornberry, 2016). Unlike concurrent similarity, which refers to substance use that occurs during the *same time period* for each generation, intergenerational continuity refers to substance use that occurs at the *same developmental stage*, but in different time periods, for each generation. A central question of intergenerational study is the extent to which a parent's history of involvement in a particular behavior – in our case, substance use – is related to the child's behavior. In investigating this issue, it is important to control for concurrent similarity to show that the link between parental adolescent substance use and child adolescent substance use is not simply explained by intra-individual stability in the parent's substance use. That is, if intergenerational continuity were important in its own right, we would expect the parent's history of adolescent substance use to have a significant impact on child behavior independent of the concurrent relationship.

There are several theoretical reasons for why one would expect to observe intergenerational continuity in substance use. Perhaps the most parsimonious is a purely genetic model in which adolescent substance use is caused by genetic risk that is passed from parent to child. Indeed, there is ample evidence for genetic influences on substance use (Polderman et al., 2015), with estimates of heritability averaging about 40 percent (Rhee & Waldman, 2002). There is also clear evidence of gene-environment interactions, and they may be particularly important for adolescent substance use (Dick et al. 2016). For example, Dick et al. (2007) found the impact of genetic risk to be enhanced by the presence of substance using peers and weak parental monitoring.

Environmental influences are also important. Recent explanations based on the life-course approach (e.g., Bailey et al., 2013; Capaldi et al., 2012; Conger et al., 2012; Thornberry, 2009) hypothesize that intergenerational continuity may be explained, in part, by the negative intra-generational consequences of adolescent substance use which can disrupt the normal course of adolescent development, increase premature and disorderly transitions from adolescence to adulthood, and diminish the acquisition of social and human capital. Because of these developmental cascades, some parents are less likely to be able to provide safe, stable, and nurturing environments for their children, increasing the likelihood of the child's substance use. Life-course models also suggest that intergenerational continuity is more likely to occur when the parent's adolescent substance use is more frequent, prolonged, and problematic (Thornberry, 2005). It is unlikely that short-term, experimental use that is somewhat normative in American society will have the long-term negative

consequences just described. More frequent, problem substance use, however, is more likely to engender these negative developmental consequences that may ultimately lead to a less effective socializing environment for the child¹.

Although there is good reason to expect intergenerational continuity in adolescent substance use, there are surprisingly few empirical studies of this issue, especially when prospective data from each generation are used. That is important, in part, because intergenerational continuity cannot be inferred from the larger literature on concurrent similarity, and vice versa (see Huesmann et al., 1984; Thornberry et al., 2006; Wu & Kandel, 1995). These are independent effects, and either can be present without the other (Thornberry, 2016).

Previous studies have produced mixed results. Thornberry et al. (2006) found that maternal adolescent substance use increased the chances that children would have an early onset of drug use, but this relationship was not found for the fathers. Others have found that father adolescent alcohol use predicted early use of alcohol among their children (Kerr et al., 2012), and parental adolescent tobacco use predicted child tobacco use (Chassin et al., 1998; Vuolo, 2013). Knight et al. (2014) found a significant relationship for parental marijuana use during adolescence and their child's marijuana use during adolescence.

Still, not all studies report an intergenerational link. Knight et al. (2014) report that parents' use of alcohol and of "other drugs" during adolescence did not predict their children's use of those same substances². Kerr et al. (2015) reported no significant direct effect from a parent's adolescent marijuana use to the child's marijuana use onset, although they did report a marginally significant relationship between parental adolescent marijuana use and child alcohol use.

None of these studies controlled for concurrent similarity in substance use while investigating these intergenerational relationships. Thus, it is possible that the link between a parent's adolescent substance use and a child's adolescent substance use is simply explained by intra-individual stability in the parent's substance use. We have identified only three studies that have examined intergenerational continuity and concurrent similarity in substance use at the same time, albeit for different substances. The results are not very consistent. Wu and Kandel (1995) report that maternal concurrent smoking significantly increased their child's involvement in smoking but a measure of past smoking did not. In contrast, Thornberry et al. (2006) report significant effects between maternal adolescent substance use and child early onset substance use, but they did not find significant concurrent similarity. Neither study found significant effects for fathers and their children. As noted above, Kerr et al. (2012) found a direct effect of father adolescent alcohol use on child adolescent alcohol use. When father and mother adult alcohol use were included in the equation, however, the intergenerational effect of the parent's adolescent use was no longer significant.

¹We recognize that the child's socializing environment is also heavily influenced by concurrent factors including the parent's adult substance use. Intergenerational models, however, suggest that those socializing environments may have longer developmental roots that extend back to the parent's adolescence (or beyond). Indeed, even without concurrent parental substance use, an earlier history of serious adolescent substance use, and the consequences that it can create, can disrupt later family functioning. How those distal and proximal influences are interrelated is an important issue for future research.

²Knight et al. (2014) did not examine continuity across different substances e.g., alcohol to marijuana.

Clearly, it is difficult to draw firm conclusions from these few and inconsistent studies, but understanding intergenerational continuity more fully is important as it extends our understanding of the negative consequences that are associated with a person's adolescent substance use. Adolescent substance use has serious intra-generational consequences such as impaired cognitive ability, school failure, adult substance abuse and dependence, and so forth (Meier et al., 2012; Lynskey & Hall, 2000; Dewit et al., 2000). Intergenerational continuity examines whether a parent's adolescent substance use also has negative consequences that ripple across to the next generation, increasing the odds that their children will be involved in substance use. If found, this impact would be independent of concurrent similarity and therefore identify a different, earlier risk factor that is associated with a child's involvement in substance use. Thus, the first purpose of this study is to investigate whether a parent's adolescent substance use has a significant direct effect on offspring adolescent substance use using prospective data for both parent and child generations and while also examining concurrent similarity.

Heterotypic Continuity

When discussing the intergenerational continuity of substance use, we may also want to consider heterotypic continuity. Here, heterotypic continuity refers to the extent to which a parent's involvement in adolescent substance use increases the likelihood that the child will be involved in *different* problem outcomes such as adolescent delinquency, depression, and risky sex behaviors. The same theoretical arguments that suggest the likelihood of homotypic continuity also suggest that it is plausible, even likely, that there are significant levels of heterotypic continuity. Many of the genes that influence substance use are nonspecific and are related to a range of other problem outcomes, including externalizing behaviors (Dick et al. 2016). Life-course theories hypothesize that the cascading consequences of a parent's adolescent substance use that ultimately impacts the family environment provided for their children are likely to be associated with elevated involvement in a range of problem outcomes, not just substance use. This view is consistent with the empirical observation that substance use and other problem outcomes often co-occur (Huizinga et al., 1993) and that they have many common risk factors (Hawkins et al., 1992).

Studies find that parental adult substance use affects child outcomes for a wide range of behaviors (e.g., Hussong et al., 2010, Hussong et al., 2008; Chassin et al., 1999). For instance, Chassin et al. (1999) found that not only does parental alcoholism increase substance use in their children, but it also may negatively affect their mental health. However, the few intergenerational studies examining heterotypic continuity provide mixed results. Kerr et al. (2012) did not find father alcohol use in adolescence to be significantly related to child externalizing problems. Similarly, Pears, Capaldi, and Owen (2007) did not find a direct link between parental adolescent substance use and child inhibitory control. On the other hand, Brook et al. (1996) found parental alcohol involvement to be indirectly linked to difficult temperament in toddlers, and Brook et al. (2012) found parental tobacco use during early adulthood to be significantly related to their child's externalizing behavior. In addition, earlier parent substance use is related to an increased risk of emotional disorders in early childhood (Leventhal et al., 2011).

Based on these studies, it is difficult to come to any firm conclusions concerning heterotypic continuity across the generations. Thus, our second purpose is to examine whether parental adolescent substance use is significantly related to the child's involvement in other problem outcomes during adolescence. In assessing this question, we control for the parent's adolescent involvement in the behavior of interest, as it is reasonable to expect that continuity in the same behavior may outweigh continuity across different problem outcomes. Investigating this issue further extends our understanding of the consequences of a parent's adolescent substance use by indicating the range of problem outcomes to which it may be related. In fact, it is possible that there could be significant levels of heterotypic continuity but not homotypic continuity.

We also recognize that there are other patterns of heterotypic continuity that could be investigated. For example, one could focus on the child's adolescent substance use as the primary outcome and examine the extent to which it is influenced, not only by parental adolescent substance use, but by a range of other adolescent problems that the parent might have experienced as an adolescent (delinquency, aggression, internalizing problems, risky sex behaviors, truancy, etc.). Indeed, the array of heterotypic relationships if multiple parental behaviors and multiple child behaviors are considered is dizzying and extends beyond the scope of a single paper. Our aim in this paper is much more focused: to start with a parent's earlier involvement in adolescent substance use and see if it is significantly related to their child's substance use as well as related problem outcomes. Other patterns of heterotypic relationships, each important in its own right, await future investigation.

Sex Differences

Finally, the third purpose of this study is to contribute to our understanding of father influences on child behavior, as much less is known about paternal effects on child behavior compared to what is known about maternal effects (Phares et al., 2005). Different patterns of contact and supervision for mothers versus fathers have substantive implications for the investigation of intergenerational continuity in substance use. Mothers are almost always the primary caregiver and the major socializing presence in the child's life (Doherty et al., 1998). In contrast, fathers are more apt to be absent and to have less involvement with their children. Moreover, fathers play more diverse roles within the family (Lamb & Tamis-LeMonda, 2004), and the impact of fathers on child behavior is more strongly shaped by contextual influences (Apel & Kaukinen, 2008; Doherty et al., 1998). As a result, there may well be different patterns of homotypic and heterotypic continuity for mothers versus fathers. There is little previous investigation of this issue and, as noted in the earlier literature reviews, the results that are available are inconsistent. The following analysis will therefore examine whether intergenerational continuity in substance use differs for mothers and fathers.

The Present Study

The present study is designed to examine intergenerational continuity in substance use across generations. In doing so, we use prospective data, self-reported by members of each generation, to assess substance use during adolescence, a developmental period when

substance use starts and begins to escalate. We also examine both homotypic and heterotypic continuity, and, for the latter, examine indicators of both externalizing and internalizing problems for the children. We investigate the level of intergenerational continuity when concurrent similarity in substance use is controlled for and when it is not. Finally, we investigate these relationships by parent sex.

Based on this approach, we test three hypotheses:

1. The frequency of parent adolescent substance use significantly increases the likelihood of offspring adolescent substance use (homotypic continuity) and other problem outcomes (heterotypic continuity).
2. Parent adolescent problem substance use significantly increases the likelihood of offspring adolescent substance use (homotypic continuity) and other problem outcomes (heterotypic continuity). Parent problem substance use is more likely to yield significant intergenerational relationships as compared to the general frequency measure.
3. Significant intergenerational relationships will continue to be observed when parent's adult substance use (concurrent similarity) is controlled.

Methods

To test these hypotheses, we use data from the Rochester Intergenerational Study (RIGS), an extension of the original Rochester Youth Development Study (RYDS). These companion longitudinal studies are based on a community sample designed to investigate the origins and consequences of drug use and related problem behaviors. These research studies were approved by the IRB at the University at Albany.

Sampling—The original study, RYDS, began in 1988 with a probability sample of 1,000 adolescents (Generation 2 or G2) from Rochester, New York, a city with high rates of crime, poverty, and distressed neighborhoods. The sample is based on the public school population of all 7th and 8th graders in 1988, selected so that a) youth at risk for delinquency and drug use are overrepresented and b) the findings can be weighted to represent the full cohort of all 7th and 8th graders. Overrepresentation of high-risk youth was accomplished by disproportionately stratifying on sex (75% males) and proportionately stratifying on residence in high-crime areas (Krohn & Thornberry, 1999). These stratifying variables are included as control variables in the analyses reported below. The RYDS sample was interviewed 14 times from age 14 to 31 – 9 times at 6-month intervals between ages 14 and 18 and then annually at ages 21, 22, 23, 29 and 31. A parent (G1) was interviewed 11 times (until G2 was 23). The sample is 68% African American, 17% Hispanic, and 15% white.

In this analysis we use data from the adolescent phase of the study, from Wave 1, average age 14.0, to Wave 9 average age 17.9. Data for this phase was collected between 1988 and 1992. At Wave 9 the retention rate was 88%. Comparisons of retained and non-retained G2 participants show that there are no significant differences between these groups on a host of variables, including 9 indicators of substance use, and that those retained still represent the original sample (Krohn & Thornberry, 1999; Thornberry, 2013).

The intergenerational study, RIGS, began in 1999 to examine intergenerational continuity and discontinuity in problem behaviors. The G3 focal subjects are the oldest biological children of the original G2 RYDS participants. In Year 1 we enrolled children age 2 or older and, in each subsequent year, we enrolled first-born children as they turned 2. Overall, the average age of first assessment is 4.8 years. By project Year 15 (2013), there were 624 eligible G3 children: 192 of RYDS mothers and 432 of RYDS fathers. Of the families of RYDS mothers 97% (186 of 192) participated in the study. Although less than a third of G2 fathers lived with the G3 child, 79% (343 of 432) enrolled in the study. Participating and non-participating fathers do not differ significantly by such variables as G2 adolescent drug use and delinquency, race/ethnicity, age at the birth of G3, high school dropout status, or history of child maltreatment. Retention has been high; 88% in Year 15 for the children of G2 fathers and G2 mothers. There is no evidence of differential subject loss. In the RIGS, we conducted annual interviews with all G2 fathers and G2 mothers (these are not couples, but rather the original male and female RYDS participants) and G3 children age 8 and older.

The analytic sample in this study includes all G3 adolescents for whom we have self-reports on substance use between the ages of 14 and 18. This age period was chosen as it is when children enter high school and begin to experiment with alcohol and drugs, and it is also the same period for which we have G2 adolescent substance measures. We include all G3 participants interviewed at ages 14 to 18 regardless of the year in which the interview occurred (the majority of interviews for G3 took place between 2007 and 2012), provided they had valid data for at least 4 of these 5 ages. Also included is their G2 parent. The final sample size consists of 341 parent-child dyads (one G2 parent and one G3 child). A total of 368 G3 participants were interviewed at these ages; the difference between this number and the 341 used in the analysis is because of missing data on one or more of the study variables.

Measurement

Independent variables

G2 Adolescent Substance Use: During adolescence, G2 participants self-reported how often they drank beer, wine, or hard liquor without their parents' permission as well as how often they smoked marijuana since their last interview. As both alcohol and marijuana use are highly skewed, each measure was top-coded at 26, equivalent to weekly use³. Our measure uses self-reports from Waves 2 through 9. Because G2 was interviewed at 6-month intervals at these ages, pairs of waves during the same academic year were summed together (i.e., Waves 2–3, Waves 4–5, etc.) to create an annual estimate comparable to the annual measures available for G3. Finally, the newly created annual measures were summed and averaged across the four years to create *frequency of G2 adolescent substance use*. We note that for some of the G2 mothers, adolescent substance use could include use while they were pregnant with the G3 child, although a clear measure of substance use during pregnancy is not available in the data set.

³Top coding reduces skew by re-coding extreme values to a point in the distribution where there is a higher density of observations, typically between the 95th and 99th percentiles. This is a common practice to reduce the influence of unrealistic or spurious outliers and, in count models such as the ones estimated here, it has the added value of improving the conditional variance while retaining as many observations in the data as possible (Nagin & Smith, 1990).

We hypothesized that parental substance use during adolescence will have a greater impact on offspring substance use if the parental use is more frequent and problematic (Thornberry, 2005). A second variable indicates whether G2 experienced problems associated with the use of alcohol or marijuana during Waves 2–9. These questions were only asked if the respondent indicated using each substance at least once a month. If they did, for each of these two substances, G2 was asked a series of 8 questions indicating problem substance use. Sample questions include, “Have you gotten into trouble with the police because of your drinking?” and “Have you tried to cut down on marijuana use and found that you could not?”. *Parental adolescent problem substance use* is coded as ‘1’ if the participant reported any problem substance use or ‘0’ if no problem use was reported.

G2 Adult Substance Use: The annual parent interviews in the RIGS included G2 self-reports about drinking five or more alcoholic drinks in one sitting⁴ and about the frequency of marijuana use. These substance use measures are skewed and were top-coded at 52. To annualize these substance use measures, the frequencies were summed together and then averaged across the 2 years when G3 was age 12 and 13. We selected the two years immediately before the measurement of the dependent variable to maintain temporal order between the independent and dependent variables.

In addition, a *G2 adult problem substance use* variable was created, also measured at G3 ages 12 to 13. The available measure indicates whether G2 experienced problems associated with the use of alcohol, as G2 problem marijuana use was not measured in the RIGS. These questions were only asked if the respondent indicated that they had been drunk or had drunk five or more drinks in one sitting at least 5 times since the last interview. Sample questions include, “Have you had problems with your health because of your drinking?” and “Have you gotten into physical fights because of your drinking?”. *G2 adult problem substance use* is coded as ‘1’ if the participant reported any problem substance use or ‘0’ if no problem use was reported.

Dependent variables

G3 Substance Use: The G3 substance use variables are based on the annual self-reports of G3 from ages 14 to 18. To measure alcohol use, G3 was asked how often they drank beer, wine, or hard liquor without their parents' permission within the last year. To measure marijuana use, G3 was asked how often they used marijuana within the last year. Each type of substance use was top-coded at 52 incidents per year to reduce skew. The frequencies of alcohol and marijuana use were summed together at each observation period to create an annual substance use measure. Finally, the annual frequency measures were averaged across G3 ages 14 to 18.

G3 Delinquency: Delinquent behavior is based on the annual self-reports of G3 from ages 14 to 18. The *delinquency* measure includes 35 acts of delinquency that range from minor offenses, such as running away from home, to serious crime, such as robbery. As the

⁴In the RIGS we did not ask about the simple prevalence and frequency of alcohol use because of the age of the sample.

measures were skewed, the annual measures were top coded at 52 incidents per year. The frequency of self-reported general delinquency was then averaged across ages 14 to 18.

G3 Depressive Symptoms: The measure of depressive symptoms, developed for the original Rochester project, is adapted from the Centers for Epidemiological Studies Depression Scale (Radloff, 1977). It uses 13 of the 20 items from the original scale and was reformatted to conform to the style of the RYDS face-to-face interview rather than the paper and pencil style of the original scale. Since it was used to measure G2 depressive symptoms, it was also used in the RIGS to assess G3 depressive symptoms. Sample items are “Since the last interview, how often did you feel scared or afraid”, “how often did you feel depressed or very sad?”, etc. A four-point response set ranging from ‘0’, never, to ‘3’, often, was employed. The score from each year was averaged across ages 14 to 18.

G3 Number of Sexual Partners: At each annual interview, G3 was asked to report the number of individuals they had sexual intercourse with during the previous year. The highest number of partners reported in any given year, between the ages 14 to 18, is used as our measure. To reduce skew, it was top-coded at the 95th percentile, or 5 sexual partners. Although almost one third of the participants were not sexually active, slightly over one third reported having 2 or more partners, and the average for those who were sexually active is 2 partners.

Control variables—*G3 sex*, *G2 sex*, *G2 socioeconomic status*, and *G2 neighborhood arrest rate* were included as control variables. *G3 sex* and *G2 sex* are binary variables with ‘1’ indicating male. *G2 socioeconomic status* is also a binary variable based on the socioeconomic status of G2’s parents at Wave 1. Based on G1 reports, ‘1’ indicates that the principal wage earner is unemployed, receives welfare, or the household is at or below the federally established poverty level. The *arrest rate* for G2 neighborhood of residence at the beginning of RYDS, a stratifying variable, was also included. Finally, in assessing the impact of G2 substance use on the other problem outcomes (delinquency, depressive symptoms, and number of sexual partners), the respective measure of G2 involvement in that behavior during adolescence, ages 14 to 18, was also controlled. The project used essentially the same measures for these behaviors in the RIGS as in the original RYDS, and these 3 measures were created in comparable fashion to the measures for G3 just described. *G2 sexual partners* was top-coded at the 95th percentile, which was 10 sexual partners. Only 16% of the G2 respondents reported not being sexually active at these ages and 27% of the sample reported having 3 or more partners.

Analytic Plan

Given the distribution of the dependent variables, we use negative binomial regression models when we are examining G3 substance use, delinquency and number of sexual partners^{5, 6}. For the last outcome of interest, depressive symptoms, an OLS regression is used as that variable is normally distributed.

⁵In order to facilitate interpretation of the coefficients, the G2 frequency of substance use and delinquency measures were rescaled using the interquartile range, or the distance between this 25th and 75th percentile. The interquartile range is appropriate when variables are highly skewed, as well as when there is no clear theoretical basis for selecting a scaling value (Babyak, 2009).

The analysis unfolds in 3 steps.

1. We first examine whether the *frequency* of G2 adolescent substance use is related to the G3 substance use, that is, whether there is homotypic continuity. We next examine whether the frequency of G2 adolescent substance use is related to any of the other G3 outcomes, that is, whether there is heterotypic continuity.
2. We examine whether G2 *problem* substance use is related to G3 substance use and the other G3 outcomes.
3. Finally, we re-estimate the models described in steps 1 and 2 but controlling for the G2 parent's *adult* involvement in substance use, allowing us to observe whether intergenerational relationships are altered once concurrent similarity is taken into account.

Results

Descriptive Statistics—Descriptive statistics for G2 measures are presented in the top panel of Table 1. During adolescence, the mean frequency of substance use for fathers is 11.81 times per year, and for mothers it is 6.42 times per year. In addition, 34.0% of G2 fathers and 19.3% of mothers reported some problem substance use during adolescence. In adulthood, fathers again reported higher levels of use than mothers. The mean frequency of substance use for fathers is 14.34 times per year, and for mothers it is 5.15 times per year; 13.9% of the fathers report problem substance use during adulthood compared to 5.5% of the mothers. Turning to G3 adolescent substance use (Table 1, bottom panel), the mean frequency of substance use is 4.27 times per year. G3 also self-reported an average of 5.91 delinquent acts per year. Finally, the average score for depressive symptoms, on a scale from 0 to 3.0, was 1.0, and the average maximum number of sexual partners reported by G3 is 1.44 per year.

The data presented in Table 1 indicate a secular decline in these problem outcomes from G2 to G3. It is difficult to tell how much this reflects an actual change in behavior across the generations and how much it reflects changes in design. The G2 data were collected at six-month intervals and adjacent waves were combined to create the annual estimates used here. In contrast, the G3 data were collected annually. Self-report data collected at shorter intervals are likely to be more precise and to uncover greater involvement in problem behaviors than self-report data collected at longer intervals (Thornberry & Krohn, 2000). Thus, part of the decline may be more apparent than real. Nevertheless, the involvement of the G3 adolescents in these outcomes is less than that observed for the G2 parents. Interestingly though, a direct comparison of the impact of core family, school, and peer risk factors on adolescent substance use indicates that these basic relationships are virtually identical for the G2 respondents and the G3 respondents (Krohn et al. 2016).

⁶Robustness checks were run for all the models. In addition to using the negative binomial, models were run using OLS and Tobit with a lower limit of zero. There was general agreement across all three methods.

Intergenerational continuity—We first examine the extent to which a parent's substance use during adolescence is predictive of their child's adolescent substance use, as well as related problem behaviors, some 20–25 years later.

Parental frequency of substance use: Table 2 presents the results when we use the more general measure of G2 adolescent substance use – the frequency of use between ages 14 and 18 – as the independent variable. The left-hand panel examines intergenerational continuity⁷. The right hand panel, which examines intergenerational continuity plus concurrent similarity, will be discussed in the next section.

The results in the first column focus on homotypic continuity in substance use. As can be seen, there are no significant effects. The frequency of parental adolescent substance use does not increase the likelihood of offspring adolescent substance use. The next 3 columns present data on heterotypic continuity. In each of these equations, we control for G2 adolescent involvement in the problem behavior that is the dependent variable. There is no evidence of heterotypic continuity between the frequency of a parent's adolescent substance use and the child's delinquency, depressive symptoms, or number of sexual partners. In the latter case though, the relationship is marginally significant ($p = .07$). The only significant effects observed in these equations are for G3 depressive symptoms. In these OLS models there is a significant effect of the parent's adolescent depressive symptoms on the child's depressive symptoms for all parents and for G2 fathers, as well as a marginally significant relationship for G2 mothers.

Parental problem substance use: Table 3 presents the results when the more serious measure of G2 adolescent substance use – involvement in problem use between ages 14 and 18 – is the independent variable. This provides data relevant to hypothesis 2: intergenerational continuity is more likely to be evident when we focus on more serious involvement in substance use by the G2 parent. As in the previous table, the left-hand panel (discussed below) presents results for intergenerational continuity while the right hand panel presents results for intergenerational continuity plus concurrent similarity.

Starting with the former relationships, for all G2 parents there is a marginally significant impact of problem substance use on G3 substance use. This relationship though is driven entirely by the G2 fathers. For the G2 fathers, their problem substance use during adolescence significantly increases substance use by their G3 children. The incident rate ratio of 2.26 suggests a moderately strong relationship. In contrast, the relationship is not significant for G2 mothers.

A similar picture emerges with respect to heterotypic continuity. For G2 fathers, their adolescent problem substance use significantly increases the likelihood of G3 delinquency (incident rate ratio = IRR = 2.34) and number of sexual partners (IRR = 1.65), even when the effect of G2 involvement in those behaviors during adolescence are, respectively, controlled. These relationships are not significant for the G2 mothers, however. Once again,

⁷Within Table 2 and Table 3, incident rate ratios are included as an indicator of effect size.

the relationships that are observed for all parents –significant for general delinquency and number of sexual partners – are driven entirely by father effects.

For depressive symptoms there is no evidence of heterotypic continuity from parental problem substance use to child depressive symptoms. There is again evidence of intergenerational continuity specific to depressive symptoms since G2 depressive symptoms during adolescence significantly increase G3 adolescent depressive symptoms in the equations for all parents, G2 mothers, and G2 fathers.

Overall, there is no support for hypothesis 1 when substance use is measured by the frequency of parental substance use during adolescence. That is, there is no evidence of homotypic or heterotypic continuity. When problem substance use is used as the independent variable, however, the results are quite different. In this case, there is evidence of homotypic and heterotypic continuity for G2 fathers, but not for G2 mothers.

Intergenerational continuity and concurrent similarity—The results just described do not include G2 adult substance use in the assessment of intergenerational continuity. It is possible that the association between a parent's adolescent substance use and their child's substance use is fully mediated by the parent's adult substance use and the association is really a product of intra-individual stability in substance use over time (Kerr et al. 2012). The models in the right-hand panels of Tables 2 and 3 account for this possibility.

Parental frequency of substance use: We again begin by using G2 frequency of adolescent substance use as the independent variable, adding G2 frequency of adult substance use as the primary control variable (the right-hand panel of Table 2). As in the previous results, there is no evidence of intergenerational continuity in substance use. There are, however, significant levels of concurrent similarity. For all parents, *adult* drug use significantly increases the likelihood that the G3 child will also engage in substance use. In this case, the overall relationship is driven entirely by the G2 mothers. The mother's adult substance use significantly increases the chances of G3 substance use, but there is no significant relationship for G2 fathers.

A similar picture emerges for delinquency and depressive symptoms. G2 adult substance use is significantly related to general delinquency for G2 mothers, but not for G2 fathers. G2 adult substance use is marginally related to G3 depressive symptoms for G2 mothers, but not for G2 fathers. In addition, the impact of the parent's adolescent depressive symptoms on G3 depressive symptoms remains significant for G2 mothers and G2 fathers.

The pattern of results for number of sexual partners is somewhat different. The frequency of G2 adult substance use significantly increases the number of sex partners reported by G3 in the equations for G2 fathers, but not for G2 mothers.

Parental problem substance use: The right hand panel of Table 3 contains the models when G2 adolescent problem use and G2 adult problem use are used as the independent variables. Unfortunately, this model cannot be estimated for G2 mothers, as their rate of problem drug use during the adult years is too low to provide stable estimates.

The results for the G2 fathers are presented in the bottom rows of Table 3. The pattern in the right-hand panel of Table 3 is identical to that presented in the left-hand panel of Table 3. That is, including adult problem use in the model does not change the relationship between the G2 father's adolescent problem substance use and G3 substance use. Paternal adolescent problem use still significantly increases the chances of the child's adolescent substance use, delinquency, and number of sexual partners. Interestingly, G2 adult problem substance use is not significantly related to any outcomes. Also, the incidence rate ratios in the right-hand panel are virtually identical in magnitude to those in the left-hand panel indicating that G2 adult problem substance use does not operate as an important mediator. G3 depressive symptoms is again related to the parent's adolescent depressive symptoms but not to either substance use measure.

Discussion

The answer to the question of whether there is homotypic and heterotypic continuity in substance use across the generations turns out to be somewhat complex. It depends, at least in part, on whether we are examining intergenerational continuity versus concurrent similarity, whether we are using a general measure of parental frequency of adolescent substance use versus problem substance use, and whether we are examining relationships for mothers versus fathers.

For G2 mothers and their offspring, there is no evidence of intergenerational continuity in substance use, either homotypic or heterotypic. Moreover, the results are the same whether one uses the frequency of adolescent substance use or problem substance use as the independent variable. Maternal involvement in adolescent substance use is not significantly related to G3 adolescent substance use, delinquency, depressive symptoms, or number of sexual partners.

For G2 fathers and their offspring, however, the measure of adolescent substance use is important. There are no significant relationships observed when the frequency of adolescent substance use is the predictor. But, consistent with our second hypothesis, when problem substance use is employed, there is evidence of both homotypic and heterotypic continuity. Paternal problem substance use is significantly related to G3 substance use, delinquency, and number of sexual partners, albeit not depressive symptoms. Moreover, these relationships are still observed when G2 father adult substance use is controlled and, for the heterotypic relationships, when the parent's adolescent involvement in the behavior of interest is also controlled. Overall, the father's involvement in more serious patterns of adolescent substance use appears to be a significant risk factor for their child's involvement in a variety of problem behaviors some 20 to 25 years later. Moreover, when looking at the impact of adolescent problem substance use on the child's outcomes, there is no reduction in statistical significance and virtually no reduction in the risk ratios when adult problem substance use is controlled, suggesting that the relationship is not mediated by the parent's adult substance use.

Turning to the impact of the parent's concurrent substance use, the results are quite different. In this case, maternal involvement in substance use is the more important risk factor for

child behavior. The frequency of maternal adult drug use significantly increases the child's substance use, delinquency, and, marginally, their depressive symptoms. Although the level of maternal problem substance use during the adult years was too low to support estimation, it appears that general involvement in substance use during the adult years is a significant risk factor for offspring substance use and other problem behaviors.

In contrast, for the G2 fathers the frequency of their adult substance use only increases the number of sexual partners reported by the child but not G3 substance use, delinquency, or depressive symptoms. Moreover, when the father's adult problem substance use is examined, none of the relationships are statistically significant. In general, the father's adult substance use is not a potent risk factor for child involvement in substance use and other problem behaviors.

Overall, the analysis reported here suggests that parental substance use is an important risk factor for child substance use, but not universally so. More serious patterns of adolescent substance use by fathers are significantly related to their children's involvement in adolescent substance use and other problem behaviors. For mothers, however, it is their adult substance use that creates risk for their children.

These findings remind us that parental substance use is not necessarily a risk factor for all adolescent problem behaviors. By and large, parental substance use, for both fathers and mothers, is not related to G3 depressive symptoms. In contrast, parental adolescent depressive symptoms are significantly and consistently related to the child's depressive symptoms. In combination with the findings summarized above, this suggests that parental substance use may be more strongly related to externalizing problems than internalizing problems, or at least depressive symptoms.

Perhaps the most intriguing finding concerns the interaction between parental sex and the timing of substance use. First, there are significant levels of *intergenerational continuity* for fathers but not for mothers. One possible explanation for this difference stems from the greater involvement in adolescent substance use and related problem behaviors by males as compared to females (Johnston et al., 2014). Life-course theories of intergenerational continuity (e.g., Capaldi et al., 2012; Thornberry, 2005) hypothesize that it is the cascading intra-generational consequences of adolescent substance use that ultimately lead to elevated risk for offspring. As adolescent boys are more extensively involved in adolescent substance use and related problem behaviors, these negative consequences may be more likely for fathers and, thus, may lead to a greater transmission of risk. In contrast, since adolescent girls have lower involvement in substance use and related problem behaviors, their adolescent substance use may be a less potent influence both on their later development and on their child's chances for successful development.

In contrast, the mother's *concurrent substance use* is more consistently related to the child's adolescent problem behaviors. Given the larger and more enduring role of mothers in the lives of their children as the primary parent and socializing agent, it is plausible that their contemporary behavior would have a larger influence on the behavior of their children than that of the father (see also, Cranford et al., 2010; Jester et al. 2000).

Our findings also highlight the importance of two methodological issues related to the design of intergenerational studies – the advantages of having both second-generation mothers and fathers and the advantages of long-term follow-up of both generations (Thornberry, 2016). If we only had a sample of G2 mothers, we would have concluded that there is no intergenerational continuity; if we only had G2 fathers, we would have concluded that there is virtually no concurrent similarity. Both conclusions are misleading as they do not tell the whole story. Similarly, if we had only examined intergenerational continuity, we would have concluded that only substance use by the father is important and that substance use by the mother is unimportant. If we had only examined concurrent similarity we would have concluded that only substance use by the mother is an important influence. Based on our results, however, the more complete answer is that substance use by *both* mothers and fathers is a significant risk factor for offspring substance use, but the timing of parental substance use is quite important to uncovering that relationship.

Although several significant relationships were uncovered, they only identify parental substance use as a risk factor for child outcomes and not necessarily as a cause. It is possible that these relationships are spurious – driven by the same influences in each generation. There could be shared genetic risk for substance use for parents and children (Polderman et al., 2015) or exposure to the same types of stressful environments such as residence in areas of concentrated poverty. Future research should address issues of causality more directly, for example, by the use of propensity score modeling and similar approaches to the investigation of causal relationships with observational data. Another important task for future research is to identify the mediating processes that help explain how a parent's involvement in substance use increases the risk that their children will also be involved in substance use. Doing so will advance our understanding of how parental substance use, both during adolescence and adulthood, influences their offspring's involvement in substance use and related problem behaviors.

The present study has its limitations. For example, it is based on a single G2 cohort selected from a single city. Replicating these findings with other samples drawn from other settings is crucial, especially given the paucity of multi-generation studies that have investigated this issue with prospective data. Also, there are many ways to measure parental substance use that take into account the heterogeneity that is encompassed by the concept “substance use”. We looked at only two and found somewhat different effects. Obviously, other approaches are available and it would be important to examine how robust these findings are to alternative measures. Relatedly, it would also be wise to examine specific types of substance use, e.g., alcohol use versus hard drug use, to see whether different patterns emerge across different types of substance use. This is consistent with the primary findings of this analysis that the level of intergenerational continuity, both homotypic and heterotypic, is likely to be more nuanced than universal. Also, we have looked at several G3 outcomes but certainly not the entire range. There are other ways of assessing substance use and there are other adolescent problem behaviors, both externalizing and internalizing. No analysis can include all, and future research should expand the range and type of child problem behaviors that are investigated. Finally, there are many other ways in which heterotypic continuity can be investigated. There are multiple problem behaviors in which parents can be involved and multiple problem behaviors in which children be involved. How individual behaviors, as

well as clusters of behaviors, are interrelated across the generations is an important question. We chose to focus on a more specific and direct issue in this analysis – the extent to which a parent's involvement in adolescent substance use increase the chances of child substance use, as well as three other problem outcomes. Even with this rather focused question, the answers are somewhat complicated. Future studies will need to expand our understanding of patterns of heterotypic continuity.

Although the present analysis has its limitations, we believe it has made substantial contributions to our understanding of whether parental adolescent substance use is a risk factor for offspring involvement in adolescent substance use and related problem behaviors. The analysis is based on a representative community sample, uses prospective reports of substance use in each generation, covers the same developmental stage in each generation, incorporates the influence of concurrent substance use while investigating the level of intergenerational continuity, investigates both homotypic and heterotypic continuity, and examines both G2 mothers and fathers. Our findings suggest that paternal adolescent substance use is an important risk factor for offspring problem behaviors. For mothers, however, adult substance use but not adolescent substance use is an important risk factor. The results of previous studies investigating intergenerational continuity are somewhat mixed and often report rather modest relationships. They often combine both mothers and fathers and examine either adolescent or adult substance use, but not both. As a result, countervailing influences may cancel each other out, providing somewhat muted results. By looking at mothers and fathers separately and by looking at their developmental stages separately, we hope this investigation provides a fuller and clearer picture of when intergenerational continuity (and concurrent similarity) in substance use is likely to be observed.

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Table 1

Descriptive Statistics for G2 Substance Use and G3 Outcome Variables (mean, standard deviation; adolescent measures, for both generations, are measured at ages 14 to 18.)

	All G2 Parents (N=341)	G2 Mothers (N=145)	G2 Fathers (N=196)
Descriptive Statistics for G2 Substance Use			
Substance Use Frequency			
Average Annual Adolescent Substance Use	9.52 (15.22)	6.42 (10.81)	11.81 (17.48)
Average Annual Adult Substance Use	10.43 (20.40)	5.15 (12.50)	14.34 (23.96)
Problem Substance Use			
Adolescent Problem Substance Use Prevalence	27.7%	19.3%	34.0%
Current Problem Substance Use Prevalence	10.3%	5.5%	13.9%
Adolescent Problems			
Average Annual General Delinquency	15.61 (22.26)	11.59 (16.38)	18.59 (25.40)
Average Annual Depressive Score	2.10 (.37)	2.16 (.39)	2.06 (.35)
No. of Sexual Partners	2.25 (2.63)	1.05 (1.03)	3.14 (3.07)
Descriptive Statistics for G3 Outcomes			
Average Annual Substance Use	4.27 (10.82)	3.23 (9.27)	5.05 (11.80)
Average Annual General Delinquency	5.91 (14.27)	4.78 (13.76)	6.74 (14.62)
Average Annual Depressive Score	1.00 (.50)	.95 (.51)	1.04 (.49)
No. of Sexual Partners	1.44 (1.44)	1.34 (1.44)	1.50 (1.43)

Intergenerational relationships with G2 frequency of substance use as the independent variable (rate ratio, unstandardized coefficient, standard error).

Table 2

G2 independent variables	A. G3 adolescent problem outcomes predicted by G2 adolescent substance use				B. G3 adolescent problem outcomes predicted by G2 adolescent substance use and G2 adult substance use			
	Substance use	Delinquency	Depressive symptoms	Sexual partners	Substance use	Delinquency	Depressive symptoms	Sexual partners
All G2 parents								
Frequency of G2 adolescent substance use	1.11 .11 (.10)	1.07 .07 (.11)	-0.03 (.02)	1.08 .08 [†] (.04)	1.06 .06 (.10)	1.06 .06 (.11)	-0.04 [†] (.02)	1.05 .05 (.05)
Frequency of G2 adult substance use	-	-	-	-	1.12 .11 [*] (.05)	1.10 .09 [*] (.04)	.02 [†] (.01)	1.04 .04 [*] (.02)
Control variable: G2 adolescent problem behavior ^a	-	1.07 .07 (.11)	.30 ^{**} (.07)	1.01 .01 (.02)	-	1.06 .05 (.11)	.30 ^{**} (.07)	1.01 .01 (.02)
G2 mothers								
Frequency of G2 adolescent substance use	1.19 .17 (.20)	1.01 .01 (.19)	-0.05 (.05)	1.11 .11 (.10)	1.07 .07 (.19)	.98 -.02 (.17)	-.08 (.05)	1.11 .11 (.11)
Frequency of G2 adult substance use	-	-	-	-	1.37 .32 ^{**} (.11)	1.33 .29 ^{**} (.09)	.05 [†] (.03)	.10 -.00 (.06)
Control variable: G2 adolescent problem behavior ^a	-	1.04 .04 (.21)	.21 [†] (.11)	1.04 .04 (.08)	-	1.14 .13 (.10)	.22 [*] (.11)	1.04 .04 (.08)
G2 fathers								
Frequency of G2 adolescent substance use	1.13 .12 (.13)	1.13 .12 (.15)	-0.02 (.02)	1.08 .08 (.05)	1.09 .09 (.13)	1.12 .12 (.15)	-.03 (.03)	1.05 .04 (.05)
Frequency of G2 adult substance use	-	-	-	-	1.05 .05 (.06)	1.02 .02 (.05)	.01 (.01)	1.05 .05 [*] (.02)
Control variable: G2 adolescent problem behavior ^a	-	1.09 .08 (.13)	.37 ^{**} (.09)	1.01 .01 (.02)	-	1.07 .07 (.13)	.37 ^{**} (.09)	1.00 .00 (.02)

Note. Each equation also includes the following control variables: G3 sex, G2 SES, G2 neighborhood arrest rate. The equation for All G2 Parents also includes G2 sex as a control.

^aG2 Adolescent Problem Behavior corresponds to the same behavior as the G3 dependent variable. For example, G2 Adolescent Delinquency is used in the G3 Adolescent Delinquency column.

[†] $p < .10$.

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Table 3

Intergenerational relationships with G2 problem use as the independent variable (rate ratio, unstandardized coefficient, standard error).

G2 independent variables	A. G3 adolescent problem outcomes predicted by G2 adolescent substance use				B. G3 adolescent problem outcomes predicted by G2 adolescent substance use and G2 adult substance use			
	Substance use	Delinquency	Depressive symptoms	Sexual partners	Substance use	Delinquency	Depressive symptoms	Sexual partners
All G2 parents								
G2 adolescent problem substance use	1.66 .50 [†] (.27)	1.68 .52 [*] (.26)	-.06 (.06)	1.32 .28 [*] (.12)	1.54 .43 (.28)	1.65 .50 [†] (.26)	-.08 (.06)	1.27 .24 [†] (.12)
G2 adult problem substance use	-	-	-	-	1.45 .37 (.41)	1.16 .15 (.33)	.10 (.09)	1.28 .25 (.17)
Control variable: G2 adolescent problem behavior ^a	-	1.03 .03 (.10)	.29 ^{**} (.07)	1.01 .01 (.02)	-	1.02 .02 (.10)	.29 ^{**} (.07)	1.01 .01 (.02)
G2 mothers								
G2 adolescent problem substance use	1.22 .20 (.49)	.96 -.04 (.39)	-.14 (.11)	.86 -.15 (.25)	-	-	-	-
G2 adult problem substance use	-	-	-	-	-	-	-	-
Control variable: G2 adolescent problem behavior ^a	-	1.05 .05 (.18)	.23 [*] (.11)	1.07 .07 (.08)	-	-	-	-
G2 fathers								
G2 adolescent problem substance use	2.26 .81 [*] (.33)	2.34 .85 ^{**} (.32)	-.03 (.07)	1.65 .50 ^{**} (.14)	2.05 .72 [*] (.34)	2.34 .85 ^{**} (.32)	-.05 (.07)	1.57 .45 ^{**} (.14)
G2 adult problem substance use	-	-	-	-	1.63 .49 (.48)	1.13 .12 (.40)	.14 (.10)	1.32 .28 (.18)
Control variable: G2 adolescent problem behavior ^a	-	1.00 .00 (.12)	.35 ^{**} (.10)	.99 -.01 (.02)	-	.99 -.01 (.12)	.34 ^{**} (.10)	.99 -.01 (.02)

Note. Each equation also includes the following control variables: G3 sex, G2 SES, G2 neighborhood arrest rate. The equation for All G2 Parents also includes G2 sex as a control.

^a G2 Adolescent Problem Behavior corresponds to the same behavior as the G3 dependent variable. For example, G2 Adolescent Delinquency is used in the G3 Adolescent Delinquency column.

[†] $p < .10$.

^{*} $p < .05$.

$p < .10$
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