This study examines intergenerational continuity in drug use across three generations of respondents in the same family. The data are from the Rochester Intergenerational Study, an ongoing, prospective, multi-generational investigation using a community-based sample. Our findings indicate that there is intergenerational continuity in drug use for Generation 2, or G2 daughters, but not sons, of G1 mothers. Use by G3 is significantly influenced by both G2 mothers and G1 grandmothers. However, for children of G2 fathers, neither prior generation's substance use is significantly related to G3 use. There is some indication that the absence of an effect from G2 fathers to G3 drug use is due to the number of nonresident fathers in the sample.

Adolescent drug use is a serious and persistent public health problem in the United States, a problem that appears rather impervious to massive efforts to bring it under control. Although the prevalence of adolescent drug use has shown a slight drop in the past few years (Johnston, O'Malley, Bachman, & Schulenberg, 2004), the long-term trend remains alarming: “In the last third of the twentieth century, young Americans reached extraordinary levels of illicit drug use, either by historical comparisons in this country or by international comparisons with other countries” (Johnston et al., 2004, p. 8). Continuing high levels of use exact a heavy toll on the individuals involved, their families, and, more broadly, society. Understanding the origins of adolescent drug use is a crucial step to developing successful intervention programs to reduce the burden of drug use. It is particularly important to identify the factors associated with the early onset of drug use given the association between early onset and more persistent and serious deviant careers (Jackson, Henriksen, Terence P. Thornberry, Ph.D., is the director of the research program on problem behavior at the Institute of Behavioral Science and a professor of sociology, University of Colorado. He is also principal investigator of the Rochester Youth Development Study. Marvin D. Krohn, Ph.D., is a professor of criminal justice at the University of Albany and a co-principal investigator of the Rochester Youth Development Study. Adrienne Freeman-Gallant, Ph.D., is a statistician and data manager for the Rochester Youth Development Study at the Hindelang Criminal Justice Research Center, University at Albany, State University of New York.
Dickinson, & Levine, 1997; Kaplow, Curran, Dodge, & The Conduct Problems Prevention Research Group, 2002; Krohn, Thornberry, Rivera, & LeBlanc, 2001). As Glantz and Pickens have noted:

Vulnerability to drug problems is greater the earlier involvement begins; it is especially unfortunate that some degree of involvement with illegal psychoactive substances (including underage alcohol use) is currently prevalent among adolescents in our society because this early exposure increases the potential for substance abuse problems for this population. (1992, pp. 1-2)

Fortunately, substantial progress has been made in our understanding of drug use, especially adolescent drug use. Risk factors for drug use in such areas as family, school, peers, neighborhood, and individual characteristics have been clearly identified. (See, for illustrations, Clayton, 1992; Fergusson & Horwood, 1997; Glantz & Pickens, 1992; Hawkins, Catalano, & Miller, 1992; Simons, Conger, & Whitbeck, 1988). Although this knowledge base has expanded considerably over the past few decades, there are still important gaps where our empirical foundation is, relatively speaking, weak.

For example, there are relatively few prospective studies of risk factors or antecedents for early onset drug use (see, for exceptions, Costello, Erkanli, Federman, & Angold, 1999; Kaplow et al., 2002; Masse & Tremblay, 1997). Related to this, there are surprisingly few comprehensive studies of the intergenerational transfer of risk for drug use, that is, the extent to which earlier parental drug use increases the risk of drug use by their adolescent children. Although seemingly a fundamental question about the origins of drug use, prior work on this question has been hampered by two research design limitations.

First, there are few prospective investigations of intergenerational continuity in drug use. That is, there are few study designs that are based on multiple, successive generations from the same families, with prospective data on drug use collected directly from members of each generation. As we show below, much prior work has been hampered by reliance on a single reporter, i.e., an individual reporting on his or her own drug use and the drug use of his or her parents, and by the use of retrospective measures. Thus, while there is a general presumption of a parent-to-child effect in this area, there is less than general empirical evidence demonstrating it.

Second, prior longitudinal studies are typically able to address only a specific aspect of the overall parent-to-child effect. Unless they rely on retrospective measures, prior studies usually examine whether a parent's adult drug use is concurrently associated with their child's adolescent drug use. While an important issue, this
represents only part of the broader issue of the intergenerational transfer of risk. Such a study fails to address whether a parent’s adolescent drug use is associated with the child’s adolescent drug use, that is, whether there is intergenerational continuity in drug use at the same developmental stage. Yet, as Cairns and colleagues point out: “[intergenerational] ...investigations presuppose the study of people who are observed at approximately the same age (or developmental stage) in two or more successive generations…” (1998, p. 1163; see also Huesmann, Eron, Lefkowitz, & Walder, 1984). This is particularly important in studies of drug use because most parents will have terminated their use by the time their children reach adolescence. However, parental use in their own youth may either be a genetic marker of a predisposition to drug use or may be indicative of a family environment conducive to drug use. By adding a subsequent generation to a prospective longitudinal study, this question, and related ones, can be addressed. Using the typical notational system of G1, G2, and G3 to refer to the successive generations, these additional questions include the following impacts:

1. G2 adolescent drug use on G3 adolescent drug use
2. G2 young adult drug use on G3 adolescent drug use
3. G1 adult drug use on G3 adolescent drug use, i.e., the grandparental impact
4. G1 plus G2 drug use on G3 adolescent drug use, i.e., the cumulative impact.

There are several conceptual models that lead to an expectation of significant levels of intergenerational continuity in drug use. They include genetic models, focusing on the impact of genetic risk and its interaction with environmental risk, in explaining the onset of drug use (Caspi et al., 2002; Hopfer, Stallings, Hewitt, & Crowley, 2003; Lynskey et al., 2003; Rhee et al., 2003). They also include mediational models in which adolescent drug use has negative consequences on the person’s life-course development (Krohn, Lizotte, & Perez, 1997), including a negative impact on later family formation and parenting skills that are associated with drug use and other problem behaviors in the next generation (Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003). Intergenerational similarity in drug use could also be produced simply by shared environments, with successive generations experiencing the same type of environmental influences that are linked to drug use, for example, neighborhood or peer influences. Understanding which combination of these influences accounts for intergenerational continuity is a central scientific question. A logically prior question, however, is to determine, with scientific rigor, the presence and magnitude of intergenerational similarity across
successive generations. This more descriptive question is the focus of the current investigation.

In addressing this issue, it is important to identify the specific pattern of intergenerational continuity. For example, to know that the parent's adult drug use is concurrently related to the child's drug use does not inform our understanding of how the parent's earlier history of drug use may additionally be related to the child's drug use. Moreover, the absence of an effect from the parent's adult use to the child's use does not imply that the parent's earlier adolescent use is not a risk factor for the child's use. Each of these effects can be independently related to the child's use and needs to be independently evaluated.

Moreover, these different patterns of intergenerational linkages imply somewhat different mediating pathways. For example, if the only significant transfer of risk comes from the parent's concurrent use, direct modeling and reinforcement may play a central role in accounting for it. On the other hand, if only the parent's earlier adolescent use is a risk factor, then genetic influences or more indirect processes, e.g., via life-course disruption, might be more central. Thus, the correct identification of these different patterns of intergenerational relationships has more than simple descriptive value. Correctly identifying the empirical linkages has important implications for the development of conceptual models.

In the remainder of this paper, we try to identify both the magnitude and pattern of the intergenerational transfer of risk for drug use, relying on data from the Rochester Intergenerational Study, an ongoing, multi-generational investigation of drug use and related problem behaviors. Importantly, members of three successive generations prospectively report on their own involvement with drugs. We begin with a brief review of previous studies.

The Impact of Parent's Drug Use

There is a rather large literature that addresses intergenerational linkages in the use of drugs and other substances. The majority of these studies, however, suffer from a set of methodological weaknesses that greatly limit their ability to adequately test the intergenerational transfer hypotheses. Prominent among these limitations are the following: Many studies are based on a select sample of drug users, for example, a clinic sample (Chassin, Pitts, DeLuria, & Todd, 1999; Merikangas et al., 1998); results from these studies do not generalize to definable community populations and are likely to overestimate the intergenerational effect. Some studies only collect data from a sample member in a single generation, relying on that person to report on the drug use of their parents (or of their children). Thus, the data are affected by common-reporter bias. Other studies rely on retrospective accounts by one (or all) generation(s). Yet other studies combine these design characteristics in a
single investigation. The problems that these design features generate for properly estimating the level of intergenerational continuity, or discontinuity, are well known and have been documented in such areas as alcohol use (Velleman, 1992), child abuse (Kaufman & Zigler, 1992), and violence (MacEwen, 1994). Based on these critiques, we do not review studies of this nature here. Suffice it to say, they generally suggest some degree of intergenerational continuity in substance use. We focus instead on studies that have community samples and prospective data from multiple generations.

Prospective studies of the influence of parental substance use assess parental use by asking about either lifetime use or use within the past year. These parental assessments are typically conducted when their children are in their adolescent years.

There are more studies that examine intergenerational linkages either for cigarette smoking or for alcohol use/alcoholism than focus specifically on drug use. In general, these studies are supportive of a link for both substances, either directly or indirectly through family functioning variables. For example, Wu and Kandel (1995) identified 12 studies that collected independent data on parental and adolescent smoking. Of those studies, 11 found that smoking by either or both parents had an effect on their child’s smoking. In their own study, Wu and Kandel examined the effect of 201 mother-father dyads on adolescents aged 9 to 17. Mothers’ concurrent smoking had a stronger effect on adolescent smoking than did fathers’ smoking. Both mothers’ and fathers’ smoking had a stronger effect on daughters’ smoking than on sons’ smoking. Much of the research on alcohol use has focused on alcoholism with the initial samples selected on the basis of a clinical diagnosis of alcoholism (Velleman, 1992). As with cigarette use, however, parental alcohol use, especially abuse or problem use of alcohol, is related to their children’s use.

We turn now to the literature that focuses on either drug use or some combination of drug use and alcohol use. These studies are also supportive of a link between concurrent parental use and adolescent use, although studies that contain independent reports by parents and children are quite limited (Kandel, Griesler, Lee, Davies, & Schaffer, 2001). Gfroerer (1987) examined the intergenerational link in the use of tobacco, alcohol, and cannabis. Use by either parent substantially increased the odds that the child also used substances. Adult marijuana use by both mother and father further increased the risk of use by children. These relationships held even after incorporating a number of control variables in the equation.

Hops and colleagues (1996) found that both mothers’ and fathers’ use of cannabis independently predicted adolescents’ cannabis use one year later. Andrews and colleagues (1993) explored a number of relationships between both parental use and parental attitudes toward drugs and use by their children. Parental cannabis use
and favorable attitudes toward use significantly influenced adolescent initiation of cannabis. Maternal variables had a stronger effect on the behavior of adolescent girls than they did for boys.

Newcomb and colleagues (1983) found that mothers’ use of cannabis directly impacted children’s use. Interestingly, mothers’ use of alcohol and pills was only indirectly related to children’s use through the child’s perception that adults use these substances regularly. Newcomb and colleagues did not find that mothers have a different effect by the gender of their child.

Kandel and Andrews (1987) found that parent use has a greater impact on initiation of drug use, while peer use becomes more important once drug use is initiated. More recently, Kandel and colleagues (2001) focused on the influence of parents born during the baby boom on their children’s use of marijuana. The proportion of youth having ever used marijuana was 40% higher when parents reported having ever used. When parents reported using marijuana within the last year, twice as many of the children had also used marijuana as compared to those whose parents had not used. In addition, parental use of other substances predicted child use of marijuana. There was some suggestion that mothers’ use was more predictive of children’s use than was fathers’, but the difference was not statistically significant.

Hill and colleagues (2004) reported preliminary findings using intergenerational data from the Seattle Social Development Project (SSDP). Parental use of tobacco, alcohol (binge drinking), and marijuana (assessed when their children were 13 to 14) predicted children’s use at 15 to 18 years of age. However, parental use did not predict continued use by their children at later ages (early to mid 20s) once use at age 15 to 18 was held constant. In addition, they found that the effect of parental use was completely mediated by variables measuring parenting, family conflict, and G2 problem behavior.

While the evidence for a relationship between parents’ concurrent substance use and substance use by their children is impressive, having been found over a range of studies, there are several related questions that have not been addressed in these studies. They include (1) whether there is an independent link between parents’ use when they were teenagers and use by their adolescent children, (2) whether grandparental use also increases risk for adolescent substance use, and (3) whether use in prior generations is related to early onset substance use. Addressing these questions requires a prospective assessment of use in prior generations, including assessments when the parents were teenagers. Although a number of studies are now collecting data on three generations and have or will have the capacity to do this, such as the projects in the special issue of the *Journal of Abnormal Child Psychology* (Capaldi, Conger, Hops, & Thornberry, 2003), we have been unable to identify any
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study that has completed such analysis. The purpose of this paper is to begin to fill in this gap in the literature. In particular, we will investigate intergenerational continuity in substance use across three successive generations. Because of the age distribution of the G3 sample in our study, we focus on early onset substance use in the youngest generation.

METHODS

To address these issues, we rely on both the Rochester Youth Development Study, for data on G1 and adolescent G2 respondents, and the Rochester Intergenerational Study, for data on adult G2 and G3 respondents. We begin with a brief description of each study.

SAMPLING

The Rochester Youth Development Study (RYDS), a longitudinal study designed to investigate the development of drug use and other problem behaviors in children and adolescents, began in 1988. The initial RYDS sample consisted of 1,000 7th and 8th graders selected from the Rochester, New York, public schools in 1988. Subjects were selected to overrepresent high-risk youth in an urban community, ensuring that serious, chronic offenders and drug users were included in the sample. Overrepresentation was accomplished by stratifying the sample on two dimensions. First, males were oversampled (75% vs. 25%) because they are more likely than females to engage in problem behaviors (Moffitt, Caspi, Rutter, & Silva, 2001). Second, students residing in areas of the city with a high resident arrest rate were oversampled since they are at greater risk for involvement in a variety of problem behaviors than those living in areas with fewer active offenders.

Phase 1 of the study covered the adolescent years, from approximately age 14 to 18. The G2 adolescents were interviewed every six months between spring 1988 and spring 1992 (Waves 1 to 9). After a two-year gap in data collection, annual interviews resumed in 1994 (Phase 2, Waves 10 to 12). Subjects were in their early adult years at this collection period (20 to 22). Throughout this period, with the exception of Wave 9, we also interviewed the adolescent's primary caregiver. These G1 respondents were overwhelmingly female; 85% were the biological mother and 8% were other female caregivers, e.g., grandmother, adoptive mother, etc. At Wave 12, 85% and 83% of the G2 and G1 respondents, respectively, were reinterviewed. These retention rates compare favorably to other panel studies of antisocial behavior, especially since we oversampled high-risk youth. There is no indication of selective subject loss and detailed descriptions of the sample and of the attrition analysis are presented in Krohn and Thornberry (1999) and Thornberry, Krohn, Lizotte, Smith, & Tobin (2003).
The Rochester Intergenerational Study (RIGS) started in 1999 and added a third generation to the overall design. The G3 focal children in the study are comprised of the first biological child of each of the original G2 subjects who was two years of age or older at the initiation of the study. The actual age range at Year 1 is 2 to 13, although 93% are younger than 10. In addition, in subsequent years we added first-born children who turned two as we move toward the sampling goal, all first-born children.

Of the 1,000 original G2s, 527 (176 mothers and 351 fathers) had a child who was eligible to participate in the Intergenerational Study as of Year 5. G2 mothers are almost always the child’s primary caregivers, and 95% (168 of 176) agreed to participate in the RIGS. Only 30% of the RYDS fathers lived with G3 at Year 1, and many of the nonresident fathers have infrequent contact with G3. Despite this, 78% (275 of 351) of the original male participants with biological children are included in the study. The primary reasons for noninclusion of RYDS fathers are that the other caretaker refused participation (35.5% of nonparticipants), the father lost contact with the child and his or her mother (22.6%), or the fathers refused to participate (33.9%). We compared the G2 fathers who enrolled in the study with those who did not on race/ethnicity, age at the birth of G3, high school dropout status, history of maltreatment, number of caretaker changes during adolescence, adolescent drug use, and delinquency. None of the differences are statistically significant ($p<.05$).

Attrition has been exceptionally low. Focusing on the Year 1 sample ($n=371$), 98% (365 of 371) remained in the study at Year 5. The overall loss rate, only six cases, is not differentially distributed. Of the 436 who entered the study between Years 1 and 4, 98% ($n=427$) were retained at Year 5. These retention rates far exceed the target rates that Hansen and colleagues (1990) recommend for longitudinal studies of three or more years.

The urban school district from which the sample was drawn yielded a panel composed primarily of people of color. The original G2 sample was 68% African American, 17% Hispanic, and 15% White. Given the somewhat higher early birth rate among minority groups, the G2 sample in the current analysis is 79% African American, 16% Hispanic, and 5% White. The distributions for the other generations are quite similar: for G1 the percentages are 76, 15, and 9 and for G3 they are 81, 12, and 7 for African Americans, Hispanics, and Whites, respectively.

**WEIGHTING**

The original RYDS sample is a simple stratified sample with different sampling rates within strata designed to represent a cohort of students in the Rochester public schools when appropriately weighted. The RIGS focuses on the first-born children of the original study participants and, therefore, can be weighted to be representative of the original cohort of Rochester 7th and 8th graders who have since
Becomes parents. Sample weights have been constructed as the inverse of the original selection probabilities with minor adjustments to account for attrition and have been normalized to have a mean of one. All analyses presented here are weighted to account for the RYDS sampling design.

Based on this weighting, the results reported here technically generalize to the cohort of 7th and 8th graders in Rochester schools in 1988 and their families. More generally, results from the Rochester project appear to generalize to the population of adolescents residing in mid-sized North American cities circa the late 1980s. The Rochester project has two companion studies, the Denver Youth Survey and the Pittsburgh Youth Study, with slightly different sampling designs and different racial/ethnic distributions. Despite this, the degree of replication across the three studies is noteworthy (e.g., Huizinga, Loeber, & Thornberry, 1995; Huizinga, Loeber, Thornberry, & Cothern, 2000; see also the respective chapters in Thornberry & Krohn, 2003). There is also close replication of Rochester results with projects in Seattle (Battin-Pearson, Thornberry, Hawkins, Krohn, 1998); Eugene, Oregon (compare Krohn, Lizotte, Thornberry, Smith, & McDowall, 1996, with Dishion & Owen, 2002; Smith, 1997, with Capaldi, Stoolmiller, Clark, & Owen, 2002); and Montreal (compare Thornberry, Krohn et al., 2003 and LaCourse, Nagin, Tremblay, Vitaro, & Claes, 2003). The extent to which the results reported herein directly generalize to adolescents from our largest cities, e.g., New York or Los Angeles, or to other time periods is less certain. The available evidence on replication, however, suggests that it is unlikely that our findings are unique to Rochester or to a very narrow portion of the population.

Measurement

The G2 parents are interviewed at all five years of the intergenerational study. The G3 children are interviewed starting at age 8 and, as a result, the number of interviews varies. The oldest cohorts were interviewed at all five years and the youngest, the birth cohort of 1995, was interviewed once. Based on these interviews, we can estimate the lifetime prevalence of substance use up to their age at Year 5. Table 1 presents the age distribution at that point. Clearly, this is a very young sample with respect to the onset of substance use. Two thirds (69%) are under 12 and only a minority have reached middle school when the onset of dmg use begins to escalate. As a result, our investigation focuses on the impact of intergenerational influences on very early onset substance use in G3.

The measure of G3 substance use is based on their self-reports. In annual interviews starting at age 8, they were asked if they drank beer, wine, or hard liquor without parental permission, or if they had smoked marijuana in the past 12 months (or since the prior interview). Given the low frequencies of reported use at these
Table 1
Age Distribution of G3 Respondents at Year 5

<table>
<thead>
<tr>
<th>Age</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>32</td>
<td>11</td>
</tr>
<tr>
<td>9</td>
<td>43</td>
<td>15</td>
</tr>
<tr>
<td>10</td>
<td>67</td>
<td>23</td>
</tr>
<tr>
<td>11</td>
<td>59</td>
<td>20</td>
</tr>
<tr>
<td>12</td>
<td>33</td>
<td>11</td>
</tr>
<tr>
<td>13</td>
<td>32</td>
<td>11</td>
</tr>
<tr>
<td>14</td>
<td>20</td>
<td>7</td>
</tr>
<tr>
<td>15+</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>293</td>
<td>100</td>
</tr>
</tbody>
</table>

ages, we create a dichotomous measure indicating any use. The prevalence of use for the full G3 sample is 7%. Of the users, two thirds report only alcohol use and one quarter report use of both alcohol and marijuana. The prevalence rate is similar to those reported in other studies of childhood drug use (e.g., Costello et al., 1999; Dishion, Capaldi, & Yoerger, 1999).

Although a wider range of substance use indicators is available for the prior generations, we use measures that are comparable to the prevalence measure created for G3. We experimented with a number of different measures of use for each of the prior generations and found the results to be quite consistent with those reported here.

For G1, we asked if they used marijuana and if they used alcohol frequently (three or more times per week) at each interview from Waves 2 to 12. At that point, the G1 respondents were adults ranging in age from their mid-20s to their 60s. During this time, G2 was between 14 and 22 years of age. The measure we employ reflects the prevalence of frequent drinking or marijuana use.

For G2, we created measures at three developmental stages: adolescence (Waves 2 to 9; ages 14 to 18), emerging adulthood (Waves 10 to 12; ages 20 to 22), and adulthood (Years 1 to 4 of the intergenerational study; ages 25 to 28). Measures in the first two periods reflect the prevalence of marijuana use or frequent drinking (being in the upper quartile of the cumulative frequency of alcohol use). For the intergenerational study, the measure reflects the prevalence of drug use and binge drinking.
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Overall, all measures for each generation are based on that generation's reports of current use. There is no reliance on retrospective recall, nor do we rely on the same reporter for multiple generations.

RESULTS

The central questions in this analysis are as follows: Does substance use in a prior generation increase the risk of substance use in subsequent generations? If so, what is the intergenerational pattern across three generations? We begin by examining the linkages across the first two generations.

THE IMPACT OF G1 ON G2

Table 2 presents the relationship between GI substance use as an adult and G2 substance use at each of three developmental stages. All results are presented separately by G2 gender, since some prior research has demonstrated that mothers' use has a greater impact on their children (especially daughters) than does fathers' use (Andrews et al., 1993; Kandel et al., 2001; Wickrama, Conger, Wallace, & Elder, 1999; Wu & Kandel, 1995). We use odds ratios to test the magnitude and significance of the effect and also present the prevalence of substance use in the younger generation for the children of nonusers in the prior generation and then for the children of substance users in the prior generation.

For the total sample, there is evidence of intergenerational continuity in substance use from GI to G2. We begin with the top row of Table 2, the impact of GI adult use on G2 adolescent use. If GI reports use, 65% of the G2 respondents report adolescent use, but if GI does not report use, only 47% of the G2 respondents report use. The odds ratio is 2.1 (p<.01). This intergenerational impact is totally driven by the G2 females, however. Recall that, in 93% of the cases, GI is also female. For the G2 females, there is a strong association between GI use and G2 daughter use. If GI was a substance user, 67% of the G2s report adolescent use; if GI was not a user, only 41% report use. The odds ratio is 2.9 (p<.01). In contrast, there is no link between GI mothers and G2 sons. The G2 prevalence rates are 58% and 60% for the sons of GI nonusers and GI users, respectively, with an odds ratio of 1.2 (ns).

Table 2 also examines the impact of GI substance use at two later developmental stages. The first is during emerging adulthood, ages 20 to 22. The second is at ages 25 to 28; at this point, the G3 child had already been born. Again, the relationship is gender specific. For the G2 females, the odds ratios are 3.3 (p<.01) for substance use in emerging adulthood and 3.6 (p<.01) for the adult years. The odds ratios are not significant for the G2 males. While the associations for the total sample are significant, they are entirely produced by the relationships for G2 females.

WINTER 2006
Table 2: Relationship Between G1 Adult Substance Use and G2 Substance Use

<table>
<thead>
<tr>
<th></th>
<th>All G2 (n=293)</th>
<th>G2 Females (n=127)</th>
<th>G2 Males (n=166)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>G2 Substance Use at:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adolescence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>2.1**</td>
<td>2.9**</td>
<td>1.2</td>
</tr>
<tr>
<td>G2 Prevalence (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G1 Nonuser</td>
<td>47</td>
<td>41</td>
<td>58</td>
</tr>
<tr>
<td>G1 User</td>
<td>65</td>
<td>67</td>
<td>60</td>
</tr>
<tr>
<td>Emerging Adulthood</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>2.4**</td>
<td>3.3**</td>
<td>1.3</td>
</tr>
<tr>
<td>Prevalence (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G1 Nonuser</td>
<td>45</td>
<td>33</td>
<td>67</td>
</tr>
<tr>
<td>G1 User</td>
<td>65</td>
<td>61</td>
<td>73</td>
</tr>
<tr>
<td>Adulthood</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>2.5***</td>
<td>3.6**</td>
<td>1.5</td>
</tr>
<tr>
<td>Prevalence (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G1 Nonuser</td>
<td>39</td>
<td>26</td>
<td>62</td>
</tr>
<tr>
<td>G1 User</td>
<td>61</td>
<td>56</td>
<td>71</td>
</tr>
</tbody>
</table>

*p<.05; **p<.01; ***p<.001

G2 INTRAGENERATIONAL STABILITY IN SUBSTANCE USE

For the G2 generation, there is strong stability in substance use across the life course and, in this case, it is observed for G2 males as well as females (Table 3). For the longest link, from adolescent substance use to adult substance use, the odds ratio is 6.8 for the total sample, 5.8 for the females and 10.5 for the males (all p<.001). The odds ratios for the other links are all large and highly significant as well.

This pattern of intragenerational stability suggests that G1’s impact on G2 at later ages may be produced entirely by its impact on G2 adolescent substance use and then the ensuing stability in substance use we just observed. In other words, G1 substance use would have no independent effect on G2 adult substance use once adolescent substance use is controlled, a pattern of findings recently observed by Hill et al. (2004). To test this, we regressed G2 substance use on G1 substance use plus prior measures of G2 substance use (Table 4).

For the females, the impact of G1 substance use on G2 use in emerging adulthood (OR=2.6; p<.05) remains statistically significant even when prior use by G2 is controlled. G1 use also impacts G2 adult use (OR=2.6; p<.05) when G2 use in both
SUBSTANCE USE ACROSS GENERATIONS

### Table 3

**Intragenerational Stability of Substance Use for G2, Odds Ratios**

<table>
<thead>
<tr>
<th>G2 Substance Use from:</th>
<th>All G2 (n=293)</th>
<th>G2 Females (n=127)</th>
<th>G2 Males (n=166)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adolescence to Emerging Adulthood</td>
<td>5.9***</td>
<td>5.3***</td>
<td>7.3***</td>
</tr>
<tr>
<td>Adolescence to Adulthood</td>
<td>6.8***</td>
<td>5.8***</td>
<td>10.5***</td>
</tr>
<tr>
<td>Emerging Adulthood to Adulthood</td>
<td>10.9***</td>
<td>9.0***</td>
<td>10.7***</td>
</tr>
</tbody>
</table>

***p<.001

### Table 4

**Intergenerational Effects from G1 to G2, Controlling for Intragenerational Stability, Odds Ratios**

<table>
<thead>
<tr>
<th>Substance Use by:</th>
<th>All G2 (n=293)</th>
<th>G2 Females (n=127)</th>
<th>G2 Males (n=166)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outcome = G2 Emerging Adulthood Substance Use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G1 Adult</td>
<td>1.9*</td>
<td>2.6*</td>
<td>1.2</td>
</tr>
<tr>
<td>G2 Adolescent</td>
<td>5.5***</td>
<td>4.6**</td>
<td>7.1***</td>
</tr>
</tbody>
</table>

| Outcome = G2 Adult Substance Use                            |                |                    |                  |
| G1 Adult                                                    | 1.9*           | 2.6*               | 1.3              |
| G2 Adolescent                                               | 4.2***         | 3.4**              | 7.0***           |
| G2 Emerging Adult                                           | 7.1***         | 5.6***             | 6.3***           |

*p<.05; **p<.01; ***p<.001

prior developmental stages is controlled. While the odds ratios for G1 are reduced somewhat in magnitude (compare Table 2), they remain significant.

For the G2 males, the inclusion of prior levels of substance use does not alter the basic picture. As in the bivariate results (Table 2), G1 substance use does not significantly impact their use. There continues to be strong intragenerational stability, however.
To this point, we have seen that G1 adult substance use is related to G2 substance use at all three developmental stages, but only for the G2 females. We have also seen high stability in drug use across the life course for G2 males and females. Do these levels of substance use in prior generations generate risk for early onset substance use in G3?

**The Impact of G2 on G3**

We begin by examining intergenerational continuity in the adjacent generations: G2 and G3 (Table 5). These results also differ by G2 gender.

For G2 mothers, their earlier substance use significantly increases the chances that their children will experience an early onset of substance use. If G2 mothers report adolescent substance use, 14% of their children report early substance use, but if the G2 mother was not an adolescent substance user, only 3% of their children report use (OR=4.9; p<.05). Significant relations are also observed from G2 emerging adult substance use to G3 use. Interestingly, the weakest effect is for the concurrent relationship. The odds ratio for G2 substance use during their mid-20s, while they are raising G3, is 3.5 (p<.06) and the prevalence rates are least dispersed, 5% to 15%. While the effect is in the expected direction, it does not reach conventional standards for statistical significance.

For the G2 fathers, earlier substance use does not significantly increase the likelihood that their children will report an early onset of substance use. One odds ratio, for G2 emerging adulthood substance use to G3 use, is large (6.5), but it is not statistically significant (p=.13). Splitting the sample by G2 gender with only a moderate number of G3 substance users raises the issue of power. However, it is important to note that the significant results are observed for the smaller sample, G2 mothers.

**Grandparental Effects: G1 to G3**

Given intergenerational continuity for adjacent generations, G1 to G2 and G2 to G3, is there also an impact from G1 to G3? Table 6 indicates that there is, but again only for the children of G2 mothers. For these children, the overall prevalence of G3 substance use is 7%; that rate is 20% if G1 reports substance use, as compared to 3% when G1 is not a substance user. The odds ratio is 7.2 (p<.01). For the children of G2 fathers, the impact of G1 substance use is in the expected direction, 4% vs. 10%, but not statistically significant.

**Exploring the Grandparental Effect**

The impact of G1 substance use on G3 for the children of G2 mothers is rather strong. Indeed, it appears to be slightly larger than the impact of G2. For example,
### Relationship between G2 Substance Use and Early Onset of G3 Substance Use

<table>
<thead>
<tr>
<th></th>
<th>All G2 (n=293)</th>
<th>G2 Mothers (n=127)</th>
<th>G2 Father (n=166)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>G2 Adolescent Use</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>to G3 Use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>3.4*</td>
<td>4.9*</td>
<td>1.5</td>
</tr>
<tr>
<td>Prevalence (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G2 Nonuser</td>
<td>4</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>G2 User</td>
<td>11</td>
<td>14</td>
<td>7</td>
</tr>
<tr>
<td><strong>G2 Emerging Adult Use</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>to G3 Use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>6.3**</td>
<td>7.8**</td>
<td>6.5</td>
</tr>
<tr>
<td>Prevalence (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G2 Nonuser</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>G2 User</td>
<td>13</td>
<td>17</td>
<td>8</td>
</tr>
<tr>
<td><strong>G2 Adult Use</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>to G3 Use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>2.3</td>
<td>3.5</td>
<td>1.1</td>
</tr>
<tr>
<td>Prevalence (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G2 Nonuser</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>G2 User</td>
<td>11</td>
<td>15</td>
<td>6</td>
</tr>
</tbody>
</table>

*p<.05; **p<.01; ***p<.001

for the G2 mothers who used substances, the highest prevalence of G3 substance use is 17% (see Table 5); if G1 reported substance use, it is 20%. To explore this effect further, we divided the G1 grandmothers into four categories based on their substance use and their level of contact with G3. The latter placed G1 grandmothers who live with G3 or who frequently visit and supervise the child in the high contact category. Table 7 shows the association between this variable and G3 substance use.

Clearly, grandmother contact is a strong mediator of intergenerational continuity. If G1 uses substances and has high contact with G3, the prevalence of substance use for G3 is 21%. In the other three categories, the prevalence of G3 substance use varies from 3% to 4%.

The level of G1 contact also helps account for the differential effect, by G2 gender, of G1 substance use on G3 substance use. Not surprisingly, the level of current contact between G1 and G3 is much higher in the families of G2 mothers.
TABLE 6
RELATIONSHIP BETWEEN G1 ADULT SUBSTANCE USE AND G3 EARLY ONSET USE

<table>
<thead>
<tr>
<th>G1 Adult Use</th>
<th>All G2 (n=293)</th>
<th>G2 Mothers (n=127)</th>
<th>G2 Fathers (n=166)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Odds Ratio</td>
<td>Prevalence (%)</td>
<td></td>
</tr>
<tr>
<td>Gl Nonuser</td>
<td>5.1***</td>
<td>4</td>
<td>17</td>
</tr>
<tr>
<td>Gl User</td>
<td>7.2**</td>
<td>3</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>2.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p<.05; **p<.01; ***p<.001

TABLE 7
IMPACT OF G1 SUBSTANCE USE AND CONTACT WITH G3 ON G3 EARLY ONSET SUBSTANCE USE

<table>
<thead>
<tr>
<th>G3 Substance Use</th>
<th>G1 Use and Contact</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No Substance Use</td>
</tr>
<tr>
<td></td>
<td>Low Contact</td>
</tr>
<tr>
<td>Prevalence of G3 Use (%)</td>
<td>3</td>
</tr>
</tbody>
</table>

n=293, χ²=20.7, df=3; p<.001

(87%) than in the families of G2 fathers (62%; χ²=22 df=1; p<.001). So, when G1 has a history of substance use and when G1 has frequent contact with G3, G3 risk for substance use increases, an effect that is larger for the children of G2 mothers because of G1’s more frequent involvement with them.

THE CUMULATIVE IMPACT OF G1 AND G2 ON G3

Substance use in each of the prior generations independently increases the risk of early onset substance use for G3 children, at least in the families of G2 mothers. We address two final questions that flow from these findings. First, does G1 substance use continue to have an impact on G3 use once G2 use is held constant? In other words, does G2 substance use mediate the impact of G1 use? Second, does substance use in both prior generations further increase the risk of G3 substance use? In other words, is there a cumulative effect?
To address the first question, we regress G3 substance use on G1 substance use plus G2 substance use (Table 8). Because of the low frequency of G3 substance use at these ages, we can only present multivariate results for the total sample. Dividing the sample by G2 gender places more demands on the data than acceptable.

Even when G2 substance use is taken into consideration, G1 substance use has a significant impact on G3 substance use. Indeed, it has a more consistently significant impact than the measures of G2 use. In each of the three equations in Table 8, the odds ratio for G1 substance use is statistically significant and virtually identical, ranging from 4.4 to 4.6. In contrast, the impact of G2 substance use is somewhat weakly and erratically related to G3 use, once G1 use is considered. The odds ratio for the emerging adult years is significant ($OR=4.9$), for the adolescent period it is marginally significant ($OR=2.7, p<.10$), and for the adult years it is not significant.

In exploratory analyses, we reestimated these equations by G2 gender. Although, as pointed out earlier, these results must be considered highly tentative, we do see very similar results to those in Table 8 for the G2 mothers. For the G2 fathers, none of the odds ratios are significant.

### Table 8
**Impact of G1 Substance Use and G2 Substance Use on G3 Early Onset Substance Use, All G2**

<table>
<thead>
<tr>
<th>Substance Use by:</th>
<th>G3 Substance Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1 Adult</td>
<td>4.5**</td>
</tr>
<tr>
<td>G2 Adolescent</td>
<td>2.7</td>
</tr>
<tr>
<td>Model $\chi^2$</td>
<td>16.6***</td>
</tr>
<tr>
<td>G1 Adult</td>
<td>4.4**</td>
</tr>
<tr>
<td>G2 Emerging Adult</td>
<td>4.9**</td>
</tr>
<tr>
<td>Model $\chi^2$</td>
<td>21.9***</td>
</tr>
<tr>
<td>G1 Adult</td>
<td>4.6***</td>
</tr>
<tr>
<td>G2 Adult</td>
<td>1.7</td>
</tr>
<tr>
<td>Model $\chi^2$</td>
<td>13.8***</td>
</tr>
</tbody>
</table>

$n=293$, df=2 for all $\chi^2$; *p<.05; **p<.01; ***p<.001
Overall, the findings in Table 8 suggest that there is little mediation of the effect of either prior generation on G3 substance use. Including G2 use does not alter the impact of G1 use: compare the odds ratio for G1 in Tables 6 and 8 (5.1 vs. 4.5). Similarly, including G1 use does not alter the impact of G2; compare the odds ratios in Tables 5 and 8 (3.4 and 2.7; 6.3 and 4.9; and 2.3 and 1.7 across the three developmental periods). Each prior generation seems to have an independent impact on G3 substance use.

The final issue we examine is the cumulative impact of use in both prior generations. We create a variable which indicates whether both G1 and G2 self-report involvement in substance use. The measure for G2 is based on substance use at any of the three developmental stages examined here. This variable has the strongest link to G3 early onset substance use (Table 9). For children of G2 mothers, if both G1 and G2 report substance use, the prevalence of G3 use is 24% with an odds ratio of 9.3 ($p<.001$). Consistent with the pattern of results throughout this analysis, the impact of this cumulative variable is not significant for the children of G2 fathers.

<table>
<thead>
<tr>
<th>G1 plus G2 Substance Use</th>
<th>All G2 (n=293)</th>
<th>G2 Mothers (n=127)</th>
<th>G2 Fathers (n=166)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Odds Ratio</td>
<td>6.5***</td>
<td>9.3***</td>
<td>2.8</td>
</tr>
<tr>
<td>Prevalence (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Both G1 and G2 Users</td>
<td>19</td>
<td>24</td>
<td>11</td>
</tr>
<tr>
<td>Other</td>
<td>4</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

*p<.05; **p<.01; ***p<.001

**DISCUSSION**

This paper addresses a simple but fundamental question about the origins of substance use: Does substance use in prior generations increase risk for substance use in subsequent generations? Despite the basic nature of this question, and the commonsense notion that it should be answered in the affirmative, prior work has only examined the association between parental use as adults and offspring’s adolescent use, finding an intergenerational association. We could find no prior study that extended this analysis to a consideration of parental adolescent use or of grandparental use when utilizing prospective data from multiple generations. Studies have addressed these issues using retrospective measures and data from the
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same reporter, but the validity of such data is suspect (Velleman, 1992). The present analysis attempted to go beyond prior work by using data from three generations of the families in the Rochester Youth Development Study. Given the ages of the third generation, we focused on early onset substance use, an important issue in its own right given the deleterious impact of early onset on later development (Glantz & Pickens, 1992).

Perhaps the most striking finding is that we observed intergenerational continuity in substance use only for the G2 females and their children. Recall that virtually all G1 respondents in the Rochester study are female. This means that the substance use of G1 mothers increases the risk of use for their daughters, but not their sons. The greater impact for daughters may imply that they are more likely than sons to model the behavior of their mothers and that they are more involved with and influenced by the family (Heimer, 1996; Wickrama et al., 1999). It is also consistent with the findings of some prior intergenerational studies (Andrews et al., 1993; Kandel et al., 2001). In contrast, sons may be less likely to model the behavior patterns of their mothers and sons’ drug use may be more influenced by extrafamilial influences, such as peer associations (Cairns & Cairns, 1994).

The gender-specific patterns continue as the analysis moves to a consideration of early onset substance use in the third generation. Use by the children of G2 mothers is significantly influenced by both G2 substance use and by G1 substance use. For the children of G2 fathers, however, neither prior generation’s substance use is significantly related to G3 use. The pattern of these findings strongly suggests that continuing contact with the child is almost essential for the intergenerational transfer of risk.

First, the impact of G1 substance use is only evident if G1 either lives with or frequently visits and supervises G3; absent this ongoing contact, G1 use has little impact on G3 use. Grandparental contact is significantly higher for the families of G2 mothers than G2 fathers. Similarly, G2 mothers are virtually always (95%) the child’s primary caregiver and live with the child. In this case, the parent’s substance use, both current and developmentally earlier use, appears to create risk for the child. In contrast, only about a quarter of the G2 fathers live with G3, and the rest have varying degrees of contact with G3. In addition, their mothers (G1) have much less frequent contact with G3 than is the case for G2 mothers. Not surprisingly, in light of this distance by both G1 and G2 fathers, their substance use has little impact on G3.

One way of examining the impact of continuing contact is to look at the children of resident and nonresident fathers. Unfortunately, there are too few G3 drug users at these ages to support this analysis. In other analyses, however, we have looked at other G3 problem behaviors that have a younger onset, such as externalizing
behaviors. When this is done, we see results that are quite consistent with the expectation that ongoing contact is a key ingredient in the intergenerational transfer of risk. For example, for resident G2 fathers, both their adolescent delinquency and their adolescent substance use are related to maternal reports of G3 externalizing problems (r=.32 and .34, respectively). For nonresident fathers, however, neither correlation is statistically significant.

Several other findings about the pattern of intergenerational influences for G2 mothers are also of interest. The impact of G2 on G3 is not limited to concurrent associations of mothers’ adult use on child use. Indeed, in the findings presented here, G2 current substance use was somewhat more weakly related to G3 use than was G2 earlier use in adolescence and emerging adulthood. This is consistent with our observation in the introduction that the absence of a link between the parent’s adult use and the child’s use does not imply that the parent’s earlier use is also unrelated. These effects can be, and in our case apparently are, independent. Failure to consider the parent’s history of drug use, as well as current drug use, may underestimate the level of intergenerational continuity.

Relatedly, our results indicate that the intergenerational transmission of risk is not limited to the immediately prior generation. Substance use by grandmothers exerts a sizeable impact on early onset substance use by G3. Importantly, this impact is not mediated by G2’s substance use; when the impact of G2 use is held constant, G1 substance use still exerts a powerful influence. In other words, assuming that an estimate of the total effect of G2 use would include the impact of G1 use is wrong. Each prior generation exerts an independent effect on G3 use, at least for early onset use by G3.

The pattern of these intergenerational effects has both methodological and substantive implications. Methodologically these results suggest that multi-generational study designs have the capacity to add uniquely to our understanding of the origins of drug use. Measures of current parental use do not fully capture the intergenerational transfer of risk from prior generations to subsequent ones. Prospective data, including data on G2’s history and adolescent development, contribute to an understanding of early onset substance use. This information can be collected most rigorously and validly by ongoing prospective assessments in each generation.

The findings also indicate the importance of having a sample large enough to separate by G2 mothers and G2 fathers. Based on our results, if only the total sample is used, one would overestimate the impact of G2 fathers and underestimate the impact of G2 mothers.

These findings also have substantive implications. The strong role of grandparental contact, and apparently father contact, in explaining intergenerational
substance use across generations

Continuity in drug use suggests that psychosocial factors may play a prominent role in mediational processes. In other analyses based on the Rochester Intergenerational Study focusing on general externalizing and internalizing problems, we have begun to explore mediating pathways (e.g., Krohn et al., 2004; Thornberry, Freeman-Gallant et al., 2003). G2 poverty and structural adversity, parenting styles, and stress and depression have all been shown to play a mediating role in accounting for linkages from G1 and G2 to G3 behavior. The pattern of linkages in this analysis suggests they are also likely to be influential in accounting for intergenerational continuity in substance use.

The intergenerational discontinuity in drug use observed for G2 fathers requires further attention. If it is maintained as G3 is followed into adolescence, sources of discontinuity need to be identified. Obviously, paternal involvement, or lack of involvement, is likely to be a crucial factor. But the behaviors and characteristics of G3’s mother are also likely to be influential.

The present descriptive analysis is not without its limitations. The most obvious one is the necessity of only examining early onset substance use by G3. Another concerns our current inability to separate the sample by G3 gender as well as G2 gender. In preliminary analysis (not reported), we looked at G3 boys and girls separately, combining G2 gender. Results are quite similar for G3 boys and girls. We need to see if these and the other results reported here change when measures of G3 adolescent substance use become available and the prevalence and frequency of use increases. At that point, we can examine intergenerational linkages for specific substances, for example, alcohol and drug use, separately. We can also see if the impact of G2 use increases in magnitude, compared to the impact of G1, as we assess use in both G2 and G3 during the same developmental period, adolescence. We will also be able to examine the differential impact of resident and non-resident fathers specifically for the intergenerational transfer of risk for drug use. All of these await continued prospective data on G3.

In the meantime though, the present results move our understanding of the intergenerational transfer of risk for substance use forward. To our knowledge, this is the first investigation of this issue using prospective data collected directly from three successive generations. The findings indicate strong intergenerational linkages across three generations, but linkages conditioned by gender and continuing contact.

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