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# Linking Substance Use and Problem Behavior Across Three Generations

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This study examined patterns of between-generation continuity in substance use from generation 1 (G1) parents to generation 2 (G2) adolescents and from G2 adult substance use and G1 substance use to generation 3 (G3) problem behavior in childhood. Structural equation modeling of prospective, longitudinal data from 808 participants, their parents, and their children showed low levels of G1 to G2 cross-generational continuity in the general tendency to use drugs. This effect was fully mediated by G2 early adolescent behavior problems. Drug-specific residual effects were observed across generations for cigarette smoking. Once established in adolescence, substance use in G2 showed stability over time. G2 substance use at age 27 significantly predicted G3 problem behavior. G1 substance use also was related to G3 problem behavior indirectly. These findings highlight the importance of interrupting intergenerational cycles of substance use and problem behavior.

**KEY WORDS:** substance use; problem behavior; intergenerational.

For the past several decades, researchers have investigated the effects of parental substance use on children. One of the most widely studied outcomes associated with parental substance use has been substance use in the subsequent generation. In general, researchers have found that parent substance use is a risk factor for substance use among offspring, but that this relationship is far from deterministic (e.g., Hawkins, Catalano, & Miller, 1992; McGue, Sharma, & Benson, 1996; Walters, 2002). A wide variety of samples and methods have been used to examine this link, including: cross-sectional studies of adults who report on their own substance use and, sometimes retrospectively, that of their parents; family history studies in which sample members and their first-degree relatives report on their own substance use (e.g., Merikangas, Dierker, & Szatmari, 1998), adoption studies, in which children of substance abusers are raised by nonbiologically-related parents (e.g., McGue et al., 1996); and studies using selected samples of, for example, alcoholic parents and their children (e.g., Chassin & Barrera, 1993; Chassin, Pillow, Curran, Molina, & Barrera, 1993; Moss, Vanyukov, Yao, & Kirillova, 1999). One powerful method for understanding the familial transmission of substance use that has been under-utilized is the prospective, longitudinal, intergenerational study.

Intergenerational studies examine between-genera tion continuity and discontinuity in behavior and link developmental outcomes in one generation to developmental outcomes in the next. These studies aid in the creation and enhancement of preventive interventions targeted at breaking cycles of intergenerational transmission of problem behaviors such as substance use by identifying the factors that disrupt or facilitate the transmission of behavioral and health problems across generations. Thus, intergenerational studies may be able to inform questions of *primordial prevention*. Whereas primary prevention aims to head off the development of problem behaviors by reducing risk factors and enhancing protective factors, *primordial prevention* aims to head off the development of the risk factors themselves (Leupker & Leyasmeyer, 1999, see special issue of Preventive Medicine, v 29, 1999).

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## INTERGENERATIONAL TRANSMISSION OF SUBSTANCE USE

Empirical evidence is clear that parent substance use predicts substance use among offspring. Past research has established that children of alcoholics are at increased risk for using alcohol in adolescence (Chassin, Rogosch, & Barrera, 1991; Leib et al., 2002), for initiating alcohol use earlier than their peers (Hawkins et al., 1997; Merikangas & Avenevoli, 2000; Obot, Wagner, & Anthony, 2000), and for abusing or becoming dependent on alcohol during the life course (Chassin & Barrera, 1993; Jennison & Johnson, 1998; Leib et al., 2002; Merikangas & Avenevoli, 2000). Children of parents who smoke cigarettes are more likely to smoke (Andrews, Hops, & Duncan, 1997; Chassin, Presson, Rose, Sherman, & Prost, 2002; Foshee & Bauman, 1992; Kandel & Wu, 1995), and children of parents who use marijuana are more likely to use marijuana (Andrews et al., 1997; Merikangas & Avenevoli, 2000).

Individuals who use one drug often use other drugs as well (Brook, Whiteman, Finch, & Cohen, 1996; Kendler, Jacobson, Prescott, & Neale, 2003; Tsuang, Bar, Harley, & Lyons, 2001). In both the Harvard Twin Study (Tsuang et al., 2001) and in analyses based on the Virginia Twin Registry (Kendler et al., 2003), comorbidity in adult males in the abuse of different types of illicit drugs was attributed to a general liability toward substance use. Brook and colleagues found that indicators of the frequency of use of hard liquor, marijuana, and other illicit drugs formed unitary latent constructs among both male and female adolescents and young adults ranging in age from 13 to 27 (Brook et al., 1996). Other researchers also have found that co-occurring use or abuse of or dependence on different drugs may reflect a common tendency toward substance use among adolescents (Han, McGue, & Iacono, 1999).

Evidence that this general tendency to use drugs may be transmitted across generations is accumulating. In one study, children of parents with a DSM-III-R substance abuse diagnosis were more likely to develop both alcohol and other drug dependence during adolescence (Hoffmann & Cerbone, 2002). Family history studies report familial aggregation, or clustering within families, of a general tendency toward substance abuse and dependence (Merikangas & Avenevoli, 2000; Merikangas et al., 1998). Behavior genetic studies also suggest that there may be common genetic factors underlying the use of various substances (Kendler et al., 2003; Tsuang et al., 2001).

The degree to which intergenerational transmission of substance use is due to substance-specific transmission versus transmission of a general tendency toward use is unknown. As discussed above, many studies have examined parent-child concordance in the use of specific substances, especially alcohol, and there is a growing body of research on parent-child similarity in the use of substances in general. Few studies, however, look at both types of transmission simultaneously, and fewer still institute controls for one type of transmission while examining the other. Understanding whether parent-child resemblance in substance use is primarily due to transmission of a general tendency to use or to substance-specific transmission will inform intervention and prevention efforts. For example, if the source of intergenerational continuity is substance-specific, prevention and intervention strategies would likely need to be substance-specific as well. Further, general versus substance-specific transmission may implicate different mechanisms. Transmission of a general propensity to use drugs may point to broader mechanisms, such as socioeconomic and neighborhood variables and inherited personality traits like risk-seeking, whereas substance-specific transmission may implicate more narrow mechanisms such as availability and inherited physiological responses to specific substances. In this study, we investigate intergenerational continuity in both the general tendency toward substance use and in the use of specific substances, including both in statistical models in order to tease apart their relative contributions.

## PARENT SUBSTANCE USE AND CHILD PROBLEM BEHAVIOR

Several theoretical models address the links between parent and child substance use. Two models of the intergenerational transmission of substance use that have received support include genetic theories (e.g., McGue, Elkins, & Iacono, 2000; Tsuang et al., 2001; Walters, 2002) and theories, such as the Social Development Model, that focus on parenting and other socialization practices (e.g., Catalano & Hawkins, 1996; Hawkins et al., 1992). Genetic theories identify a range of potential mechanisms to explain links between parent and child substance use, including physiological responses to substances of abuse, predisposing temperament and personality traits, and impaired neuropsychologic function inherited by children from their parents (Johnson & Leff, 1999; McGue, 1994). The Social Development Model and other socialization theories suggest that parent substance use negatively affects parental monitoring and discipline practices, family bonding, socioeconomic status, and other social contextual variables that increase the risk of substance use among children (Catalano & Hawkins, 1996; Hawkins

et al., 1992; Hill, White, Chung, Hawkins, & Catalano, 2000).

Genetic and social development theories converge in implicating child problem behavior as a potential mechanism in the intergenerational transmission of substance use. Specific behavior problems noted among offspring of substance users include conduct disorder (Merikangas et al., 1998), hyperactivity and impulsivity (Kuperman, Schlosser, & Lidral, 1999; Milberger, Biederman, Faraone, Chen, & Jones, 1996; Stein, Newcomb, & Bentler, 1993), oppositional-defiant disorder (Kuperman et al., 1999), and delinquency (Catalano, Haggerty, Gainey, & Hoppe, 1997). Problem behavior in childhood and adolescence has been identified repeatedly as a predictor of later substance use (Colder & Stice, 1998; Hawkins et al., 1992; McGue, Iacono, Legrand, Malone, & Elkins, 2001; Milberger, Biederman, Faraone, Chen, & Jones, 1997; Neumark-Sztainer, Story, French, & Resnick, 1997).

Geneticists point to inherited difficulties in behavioral inhibition and attention (Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Tarter et al., 1999) as central in the link between parent and child substance use. Essentially, parents who have a genetic liability toward problem behavior, and thus, toward substance use, pass this liability on to their children. These children, in turn, are at increased risk of developing problem behavior and substance use. Researchers who study socialization mechanisms note that parent substance use affects both parenting practices and the child's developmental environment in ways that increase child behavior problems (Barnard & McKeganey, 2004; Hawkins et al., 1992). Thus, both theory and empirical evidence suggest that the transmission of substance use between generations may be mediated in part by child problem behavior, such that parental substance use is associated with the development of child problem behavior, which, in turn, promotes the development of substance use among children as they move into adolescence and adulthood.

## COMPETING EXPLANATIONS

Problem behavior theory and ample research suggest that substance use and problem behavior are strongly correlated within person. Jessor and Jessor (1977) have suggested that substance use, delinquency, and other problem behaviors are simply manifestations of a general tendency to engage in deviant behavior. Thus, if parents are using substances, they are also likely to engage in antisocial behavior (e.g., Kendler, Prescott, Myers, & Neale, 2003; Menard, Mihalic, & Huizinga, 2001). It is possible, then, that child problem behavior results not from parent substance use, but from parents' general pattern of antisocial behavior that is correlated with substance use. This hypothesis is consistent with a large body of research linking parent antisocial behavior and child problem behavior (e.g., Farrington, Jolliffe, Loeber, Stouthamer-Loeber, & Kalb, 2001; Fuller et al., 2003; Rhee & Waldman, 2002).

Socioeconomic status variables, such as parent marital status, parent education, and neighborhood disorganization, have been linked both to parent substance use (Brook, Richter, Whiteman, & Cohen, 1999; Hill et al., 2000) and child problem behavior (Aneshensel & Sucoff, 1996; Hawkins et al., 1992). Consequently, they also represent plausible, alternative hypotheses to explain parent-child resemblance in substance use. The present study includes these variables as controls when examining the relationship between parent substance use and child problem behavior.

## THE ROLE OF GRANDPARENTS

Both genetic and socialization theories suggest that grandparent substance use may predict substance use and problem behavior among grandchildren. The influence of grandparents may be direct, by way of their potential role as caretakers who provide models and norms for behavior, or indirect, by way of their contribution of genetic material to G2 and the models of parenting and contexts for development they provide for G2 mothers and fathers. Another way in which G1 substance use may affect G3 problem behavior indirectly is by increasing G2 adolescent substance use, which demonstrates a high degree of within-person stability from adolescence into adulthood (Brook et al., 1996; Labouvie, 1996; Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001). Very little prospective, longitudinal research has explored the role of grandparent substance use in predicting substance use and problem behavior among grandchildren. Chassin and colleagues found that G1 grandparents' cigarette smoking, as reported by G2 mothers, was indirectly related to their grandchild's cigarette smoking via increased maternal smoking (Chassin, Presson, Todd, Rose, & Sherman, 1998). Stein and colleagues found that grandparent drug use, as reported by G2 mothers, was associated with behavior problems among both male and female grandchildren (Stein et al., 1993). The present study provides an important opportunity to examine both the direct and indirect effects of grandparent substance use on grandchild problem behavior using G1 self-reported substance use and a prospective, longitudinal design.

## STUDY QUESTIONS

Prospective studies of the intergenerational transmission of problem behavior involving more than two generations are rare, particularly in the substance use literature. This study extends prior research on the intergenerational continuity of substance use by using data from two prospective, longitudinal studies linking three generations. Five main research questions are addressed: (a) Is there a general tendency toward substance use that is transmitted across generations? (b) Is there substance-specific transmission of use over and above the general tendency to use substances? (c) Does problem behavior serve as a mechanism for the transmission of substance use across generations? (d) Does parent substance use predict child problem behavior when competing explanatory variables are controlled? (e) Does grandparent substance use affect problem behavior among grandchildren, either directly or indirectly?

## METHODS

### Sample and Procedure

The present analyses draw data from two closely related research projects: the Seattle Social Development Project and The Intergenerational Project. The Seattle Social Development Project (SSDP) is a longitudinal study of youth development and pro- and antisocial behavior. Participants were recruited from 18 Seattle public elementary schools that served high crime areas of the city during a period of mandatory busing. From the population of 1,053 students entering Grade 5 (age 10) in participating schools in the fall of 1985, 808 students (76.7% of the population) consented to participate in the longitudinal study and constitute the SSDP sample. The sample has been interviewed 11 times over a 17-year period through 2002 when G2 participants were 27 years old; interviews were conducted yearly from ages 10 to 16, at age 18, and every three years thereafter. Data were obtained through the administration of parent (G1) and student (G2) questionnaires. Student (G2) interviews were conducted in person, and were administered by trained interviewers. Parent (G1) interviews were administered over the telephone. When respondents did not have a telephone or when their number was unlisted, in-person interviews were conducted (O'Donnell, Hawkins, & Abbott, 1995). Only student surveys were administered after age 18.

Seven hundred seventy one G1 parents completed at least one of the 7th and 8th Grade (G2 ages 13 and 14) interviews. At the Grade 7 interview, 86% of G1 parents were mothers or acting as mothers. Eighty-six percent of these women were biologically related to the G2 participant, and 64% had live-in partners or spouses, of whom 60% were biologically related to the G2 child. About 9% of G1 parents were fathers or acting as fathers. Seventy percent of these men were biologically related to the G2 participant, and 75% had live-in partners or spouses, of whom 65% were biologically related to the G2 child. The remaining G1 "parents" were other adults acting as parents.

The G2 SSDP sample included about equal numbers of males ($n = 412$) and females ($n = 396$) and was ethnically diverse. About 47% were Caucasian, 26% were African American, 22% were Asian American, and 5% were Native American. Of these groups, 5% were Hispanic. A substantial proportion of the participants were from low-income families. About 52% of the G2 participants were from families in poverty as evidenced by participation in the National School Lunch/School Breakfast Program between the ages of 10 and 12. These 808 participants constitute the available sample for analyses of the effects of G1 substance use on G2 substance use and problem behavior.

About 81% of the original 808 participants completed the age 13 interview, 96% completed the age 14 interview, and 97%, 95%, and 94% completed the ages 15, 16, and 18 interviews, respectively. Ninety-three percent and 95%, respectively, completed the age 21 and 24 interviews, and 92% completed the age 27 interview. Nonparticipation at each assessment wave was not consistently related to ethnicity or alcohol, tobacco, marijuana, or other illicit drug use by age 10. At age 27, slightly more women than men participated (94.7% vs. 90.3%).

In 2002, The Intergenerational Project (TIP) began intensive data collection on SSDP participants and their children (G3). TIP uses an accelerated longitudinal study design to examine the consequences of parental and grandparental substance use on child development. SSDP sample members were invited to participate in TIP if they had a biological child with whom they had face-to-face contact, at minimum, on a monthly basis. In cases where there were multiple biological children, the oldest child was selected. There were 305 SSDP participants who reported having a biological child at the time TIP began. Of these G2 parents, 281 (92%) met eligibility criteria. Eligible G2 parents and those who had a biological child but were not eligible (did not have face-to-face contact at least once a month, $n = 24$) were compared on a wide range of factors. Eligible G2 parents (those who had face-to-face contact with their child once a month) lived in less disorganized neighborhoods when they were children [$t(20.7) = 2.14$, $p = .04$], came from homes where G1 reported binge

drinking significantly more frequently [$t(21.6) = -2.33$, $p = .03$], reported significantly higher educational attainment at age 24 [$t(295) = -2.17$, $p = .03$], and were more likely to be female [$\chi^2(1,305) = 15.43$, $p = .00$] than parents who did not have sufficient contact with their child to meet eligibility requirements. Eligible and ineligible parents did not differ in terms of childhood poverty (eligibility for free/reduced-price lunch in 5th thru 7th grade); adolescent problem behavior; binge drinking, cigarette use, or marijuana use in adolescence or at age 21–24; marital status at age 27; welfare receipt at age 27; G1 cigarette use or marijuana use; or G1 educational attainment.

Of the 281 eligible families 208 (74%) consented to be in the study. Eligible SSDP G2 participants who chose to participate in TIP ($n = 208$) were compared to those who declined participation ($n = 73$). G2 parents who participated in TIP reported binge drinking significantly more frequently in high school [$t(123.9) = 2.09$, $p = .04$], were more likely to have received welfare in the past year at age 27 [$\chi^2(1,273) = 4.02$, $p = .05$], and came from families where G1 reported significantly higher educational attainment [$t(215) = 2.13$, $p = .03$]. Recruited parents did not differ from those eligible but not recruited in terms of gender; childhood neighborhood disorganization; childhood poverty; adolescent problem behavior; cigarette use or marijuana use in adolescence; binge drinking, cigarette use, or marijuana use at age 21–24; educational attainment at age 24; marital status at age 27; or G1 binge drinking, cigarette use, or marijuana use.

The TIP sample in Wave 1 consisted of 208 SSDP families: 144 SSDP mothers, 64 SSDP fathers, their oldest biological child (99 boys and 109 girls), and 122 other caregivers (52 fathers, 48 mothers, and 22 non-parental caregivers, most of whom were grandparents or other relatives). The children in the total TIP sample ranged in age from 1 to 13 years (median age = 6 years). Seventy percent ($n = 145$) of the children were being raised by two parents (95% of whom lived together), 10% ($n = 22$) by one parent and a non-parental caregiver (14 of whom lived with the parent), and 20% ($n = 41$) by one parent alone. The median family income in 2002 was $33,000. Twenty-eight percent of the families received benefits from TANF, AFDC or food stamps in the past year in 2002. Similar to the original SSDP G2 sample from which the parents were recruited, the TIP sample is ethnically diverse. Both the children and parents in the TIP sample are mostly non-Hispanic (84% and 82%, respectively, as reported by the parents). Of the non-Hispanic sample members, the largest proportion of children and parents are white (33% and 36%, respectively), African American (21% and 23%, respectively), or multiethnic (37% and 14%, respectively). The remainder is Asian, Native American, or Pacific Islander.

Parent, alternate caregiver, and child interviews were conducted in person by trained interviewers. In addition, questionnaires were mailed to teachers of 105 children age 6 and older. Ninety-seven teachers (92%) returned the teacher survey. Because the present analyses make use of teacher-reported child behavior problems, these 97 school-aged children constitute the available sample for analyses of the effects of G1 and G2 substance use on G3 problem behavior. The present study uses data from the first wave of data collection; four waves of data collection are planned.

## Measures of Substance Use and Problem Behavior

### *Grandparent Substance Use*

Grandparents (G1) reported on the frequency of their own and their live-in partner's binge drinking (five or more drinks per occasion), marijuana use, and cigarette use when G2 were in seventh and eighth grades (average ages 13 & 14). Each of the substance use questions asks about their current patterns of use (e.g., "Do you currently drink beer, wine, or liquor?" followed by "When you do drink, how often do you have as many as five or six drinks at one time?"). Binge drinking, marijuana use, and cigarette use frequencies were averaged across partners and grades. When there was no live-in partner, only the respondent's substance use was used. Because the distributions of these variables were highly skewed, frequency scores for each substance were grouped into three categories that still captured meaningful levels of use: binge drinking—0 (*never*), 1 (*once in a while*), 2 (*less than half of the time* or *more than half of the time*); marijuana use—0 (*never*), 1 (*less than once per month*), 2 (*2-3 times or more per month*); cigarette use—0 (*never* or *quit*), 1 (*less than 1 pack per day*), 2 (*1 pack or more per day*). These three category variables were used in the structural equation model (SEM) analyses described below. Substance use items for G1 and G2 were drawn from the Social Development of Youth Project (Hawkins & Catalano, unpublished survey), the Denver Youth Study and National Youth Study (Browning & Huizinga, 1999; Elliott & Huizinga, 1987; Elliott, Huizinga, & Menard, 1989; Esbensen & Elliott, 1994), the Longitudinal Research Instrument used in the Oregon Youth Study (Dishion, Patterson, Stoolmiller, & Skinner, 1991; Fagot, Pears, Capaldi, Crosby, & Leve, 1998), and the Monitoring the Future Study (Bachman, Wadsworth, O'Malley, Johnston, & Schulenberg, 1997).

*Parent Substance Use in Adolescence*

Parents (G2) reported on their own binge drinking (5 or more drinks per occasion), marijuana use, and cigarette use when they were in 9th, 10th, and 12th grade (average ages 15, 16, & 18). At each wave, respondents were asked how many times they had engaged in each of these behaviors during the month prior to the interview. Average frequency of binge drinking, marijuana use, and cigarette use scores were obtained by averaging use at 9th, 10th, and 12th grade. As with G1 substance use, the distributions of these variables were highly skewed, and frequency scores for each substance were grouped into three categories that still captured meaningful levels of use: binge drinking and marijuana use—0 (*never*), 1 (*once or less per week*), 2 (*more than once per week*); cigarette use—0 (*never*), 1 (*less than 1 pack per day*), 2 (*1 pack or more per day*). These three category variables were used in the SEM analyses described below.

*Parent Substance Use in Adulthood*

Parents (G2) reported on their own binge drinking (5 or more drinks in a two-hour period), marijuana use, and cigarette use when they were 21, 24, and 27 years old. At each wave, respondents were asked how many times they had engaged in each of these behaviors during the month prior to the interview. The frequency of use at age 21 and age 24 for each substance was averaged. Frequency variables were categorized in the following way: binge drinking and marijuana use—0 (*never*), 1 (*once or less per week*), 2 (*more than once per week*); cigarette use—0 (*never*), 1 (*less than 1 pack per day*), 2 (*1 pack or more per day*). Current substance use at age 27 was not averaged with use at any other age. These three category variables were used in the SEM analyses described below.

*Parent Problem Behavior in Adolescence*

When parents (G2) were in seventh and eighth grade (ages 13 & 14), their teachers completed the Teacher Report Form of the Child Behavior Checklist (Achenbach & Edelbrock, 1986). Responses to individual items were averaged across the two years and standardized. A Conduct Disorder score was obtained by averaging the following 10 items: cruelty, bullying, or meanness to others; destroys others' things; destroys his or her own things; gets in many fights; physically attacks people; hangs out with kids who get in trouble; runs away from home; steals; sets fires; threatens others. The internal consistency of items in the Conduct Disorder scale was good (Cronbach's $\alpha = .89$). An Attention Problems score was created by averaging the following 3 items ($\alpha = .80$): restless, can't sit still; impulsive; has trouble concentrating. An Oppositional Defiant score was obtained by averaging the following 5 items ($\alpha = .89$): argues; disobedient at school; defiant, talks back; stubborn, sullen, or irritable; has a hot temper. The use of these items and scales is consistent with work by Lengua, Sadowski, Friedrich, and Fisher (2001) and by Achenbach and Rescorla (2001).

*Child Problem Behavior*

During the first wave of TIP, teachers completed the Teacher Report Form of the Child Behavior Checklist (Achenbach & Rescorla, 2001) about the G3 children attending school (i.e., age 6+). Because past research indicates that behavior problems change as children age (e.g., Bongers, Koot, van der Ende, & Verhulst, 2003) and because participating children spanned a wide age range, items were standardized within 2- to 4-year age group ranges prior to scaling (ages 6–7, 8–9, and 10–13). Thus, children's behavior problems are scored relative to their same-aged peers. Similar to the G2 problem behavior scales, we created Conduct Disorder (Cronbach's $\alpha = .78$), Attention Problems ($\alpha = .76$), and Oppositional Defiant scores ($\alpha = .91$) for the G3 children. Substance use among G3 children was exceedingly rare, as would be expected given their young age ($M = 8$ years). Consequently, this construct was not included in the present study.

## Measures of Control Variables

*Sociodemographic Variables*

G1 marital status and highest educational attainment were based on G1 self-reports obtained when G2 were in seventh grade (age 13). When there was a G1 live-in partner, the educational attainment of both partners was averaged. G2 marital status was self-reported at age 27, and G2 educational attainment was self-reported at age 24. The measure of neighborhood disorganization in the G1-G2 neighborhood was based on G2 reports of their perceived levels of neighborhood crime, drug selling, poverty, gangs, and undesirable neighbors, obtained when G2 were in eighth grade (age 14, Cronbach's $\alpha = .88$). Neighborhood disorganization in the G2–G3 neighborhood was similarly measured using reports by G2 parents at age 27.

*Grandparent Antisocial Behavior*

Grandparents (G1) reported on whether they had ever been incarcerated and whether they had ever "been in trouble with the law for something other than minor traffic violations" when G2 were in seventh and eighth grades (average ages 13 & 14). If either the respondent or her/his partner had been incarcerated or in trouble with the law, this was taken as evidence of G1 antisocial behavior. Responses were dichotomized—0 (*no antisocial behavior*), 1 (*yes, antisocial behavior*).

*Parent Antisocial Behavior in Adulthood*

At age 27, parents (G2) reported on how many times they had been involved in any of 15 different criminal activities in the year prior to the interview. Crimes ranged from property crime and fraud to violent crime. Questions were drawn from the Social Development of Youth Project (Hawkins & Catalano, unpublished survey) and the Denver Youth Study and National Youth Study (Browning & Huizinga, 1999; Elliott & Huizinga, 1987; Elliott et al., 1989; Esbensen & Elliott, 1994), and included: How many times in the past year have you . . . "broken into a house, store, school, or other building without the owner's permission?"; "tried to use credit cards without the owner's permission?"; "taken something worth more than $50?"; "hit someone with the idea of seriously hurting them?"; "used a weapon or force to get money or things from people?". Responses to the crime questions were averaged to obtain the mean frequency of crime in the year prior to the survey. Because this measure is an index of criminal behaviors as opposed to a scale, calculation of Cronbach's alpha is not appropriate.

## Analytic Strategy

We evaluated the hypothesized within- and between-generation relationships between G1, G2, and G3 substance use and problem behavior in a latent variable framework. We estimated measurement and structural equation models (SEMs) using Mplus Version 3.0 (Muthén & Muthén, 1998–2004). Modeling was done in two stages. We first evaluated the measurement model by conducting a confirmatory factor analysis of the G1 Substance Use, G2 Adolescent Substance Use, G2 Adolescent Problem Behavior, G2 Early Adult Substance Use, G2 Age 27 Substance Use, and G3 Problem Behavior latent factors. Next, we analyzed the hypothesized structural relationships between the G1, G2, and G3 latent factors. Mplus Version 3.0 uses Full Information Maximum Likelihood (FIML) estimation in the presence of cases with missing data on both continuous and categorical outcome variables. FIML was employed to utilize all available information from the larger SSDP ($n = 808$) and the smaller TIP sample ($n = 97$). In the present study, questions about child behavior problems were not applicable to those SSDP participants who did not have children. Current methodological research on analysis in the presence of missing data suggests that FIML provides unbiased parameter estimates when some questions are not applicable to all sample members ("out-of-scope missingness," Schafer & Graham, 2002). In this study, data from the full SSDP sample provide parameter estimates for G1-G2, and G2-G2 links, and parameter estimates of the effects of G1 and G2 substance use on G3 problem behavior are based only on the 97 cases for which relevant variables are not missing (Muthén & Muthén, 1998–2004; Schafer & Graham, 2002).

An additional benefit of FIML is that it produces parameter estimates that are unbiased with relation to any potential correlates of missingness that are included in the model (Collins, Schafer, & Kam, 2001; Graham, Cumsille, & Elek-Fisk, 2003; Schafer & Graham, 2002). Thus, parameter estimates obtained here are unbiased with respect to variables included in the model that may be related to whether G2 had become parents by age 27, such as G1 substance use, antisocial behavior, marital status, and education, and G2 problem behavior, substance use, antisocial behavior, neighborhood disorganization, marital status, and education. To the extent that pertinent correlates are included in the model, the parameter estimate obtained for the relationship between G2 substance use and G3 problem behavior is generalizable to those G2's who have not yet had children. Bias is still possible if missingness is related to variables that are not included in the model (Collins et al., 2001; Graham et al., 2003; Schafer & Graham, 2002).

In order to further support the validity of parameter estimates obtained in this study, we conducted a multigroup SEM comparing those SSDP G2 participants who were and were not in TIP (i.e., those with children compared to those without or who did not participate in TIP). The goal of the multigroup SEM was to determine measurement invariance and invariance in structural parameters related to G1 and G2 substance use and G2 problem behavior. A finding of invariance in measurement and structural parameters would further support the generalizability of parameters obtained here by demonstrating that the factor structure of substance use and patterns of

covariance between G1 and G2 variables were the same for those SSDP G2's who were in TIP as for those who were not.[3] Because the models included categorical outcome variables, the WLSMV estimator was used throughout. The difference in chi-square values estimated with WLSMV is not distributed as chi-square. Therefore, we used the mean-adjusted robust chi-square difference test implemented in Mplus (DIFFTEST) to get correct chi-square difference values when comparing the fit of nested models (Muthén & Muthén, 1998–2004).

## RESULTS

### Descriptive Analyses

There was a substantial amount of substance use reported in the sample (Table I). Table II shows descriptive statistics for key study variables and controls. About 5% of G2's scored in the clinical range for Conduct Disorder, 7% scored in the clinical range for Oppositional Defiant, and 8% scored in the clinical range for Attention Problems (Achenbach & Rescorla, 2001). Among G3's, no children scored in the clinical range for Conduct Disorder, 1% scored in the clinical range for Oppositional Defiant, and 1% scored in the clinical range for Attention Problems. These percentages are approximations, because we used the scales recommended Lengua and colleagues (2001), not the versions used by Achenbach and Rescorla on which clinical cutoffs are based.

### Confirmatory Factor Analysis

Confirmatory Factor Analysis (CFA) of latent constructs revealed support for the hypothesized measurement model. Model fit was acceptable ($\chi^2[39, 806] = 54.90$, $p = .05$, CFI = .99, TLI = .99, RMSEA estimate = .02). All indicators loaded significantly on their

**Table I.** Prevalence of Substance Use Among G1 and G2

| | G1 (G2 age 13–14)[a] | G2 Age 15–18[b] | G2 Age 21–24[b] | G2 Age 27[b] |
|---|---|---|---|---|
| Binge drinking | | | | |
| 0 | 72% | 77% | 60% | 73% |
| 1 | 24% | 21% | 33% | 21% |
| 2 | 4% | 3% | 7% | 7% |
| Cigarette use | | | | |
| 0 | 57% | 72% | 56% | 68% |
| 1 | 31% | 22% | 26% | 14% |
| 2 | 12% | 7% | 19% | 18% |
| Marijuana use | | | | |
| 0 | 89% | 75% | 69% | 80% |
| 1 | 9% | 18% | 18% | 11% |
| 2 | 2% | 7% | 13% | 9% |

*Note*. percentages may sum to more than 100% due to rounding.
[a]Binge drinking categories: 0 (never), 1 (once in a while), 2 (less than half of the time or more than half of the time); marijuana use categories: 0 (never), 1 (less than once per month), 2 (2–3 times or more per month); cigarette use categories: 0 (never or quit), 1 (less than 1 pack per day), 2 (1 pack or more per day).
[b]Binge drinking and marijuana use categories: 0 (never), 1 (once or less per week), 2 (more than once per week); cigarette use categories: 0 (never), 1 (less than 1 pack per day), 2 (1 pack or more per day).

**Table II.** Descriptive Statistics for Study Variables

| Variable | % *M* (SD) | Range |
|---|---|---|
| G1 (when G2 age 13–14) | | |
| Completed high school | 61% | |
| Completed 4 year college | 22% | |
| Neighborhood disorg. | 1.71 (.66) | 1–4[a] |
| G2 Middle school (13–14) | | |
| Conduct disorder | .15 (.27) | 0–2[b] |
| Attention problems | .39 (.48) | 0–2 |
| Oppositional defiant | .28 (.43) | 0–2 |
| G3 (Ages 6–13) | | |
| Conduct disorder | .19 (.29) | 0–2 |
| Attention problems | .70 (.49) | 0–2 |
| Oppositional defiant | .60 (.42) | 0–2 |
| G2 Early adult (21—24) | | |
| Completed high school | 51% | |
| Completed 4 year college | 21% | |
| G2 Age 27 | | |
| Neighborhood disorg. | 1.65 (.56) | 1–4 |
| Married | 31% | |

[a]Response categories: "Are these things [crime/drug selling, fights, abandoned buildings, etc.] found in your neighborhood?" 1 (*not at all*), 2 (*not much*), 3 (*pretty much*), 4 (*a lot*).
[b]Response categories: "How true of [child] are the following statements?" 0 (*not true*), 1 (*somewhat or sometimes true*), 2 (*very true or often true*.).

[3] A portion of the sample was exposed to a multicomponent preventive intervention in the elementary grades, consisting of teacher training, parenting classes, and social competence training for children (Hawkins, Catalano, Kosterman, Abbott, & Hill, 1999). While differences in prevalences and means have been observed between intervention and control groups, prior analyses have shown few differences in the covariance structures of the groups (Catalano, Kosterman, Hawkins, Newcomb, & Abbott, 1996; Huang, Kosterman, Catalano, Hawkins, & Abbott, 2001). Similarly, analyses for this report were based on the full sample after examining possible differences in measurement and covariance structures between a group that received all of the intervention components and a control group of participants who received no intervention. Previous analyses have shown that this "full" intervention group was most likely to demonstrate significant intervention effects on mean levels of behavior (Hawkins et al., 1999). The control group was compared to the full intervention group using multiple group structural equation modeling. Nonsignificant results of the DIFFTEST procedure in Mplus (Muthén & Muthén, 1998-2004) support measurement invariance and invariance in structural parameters between the control and full intervention groups.

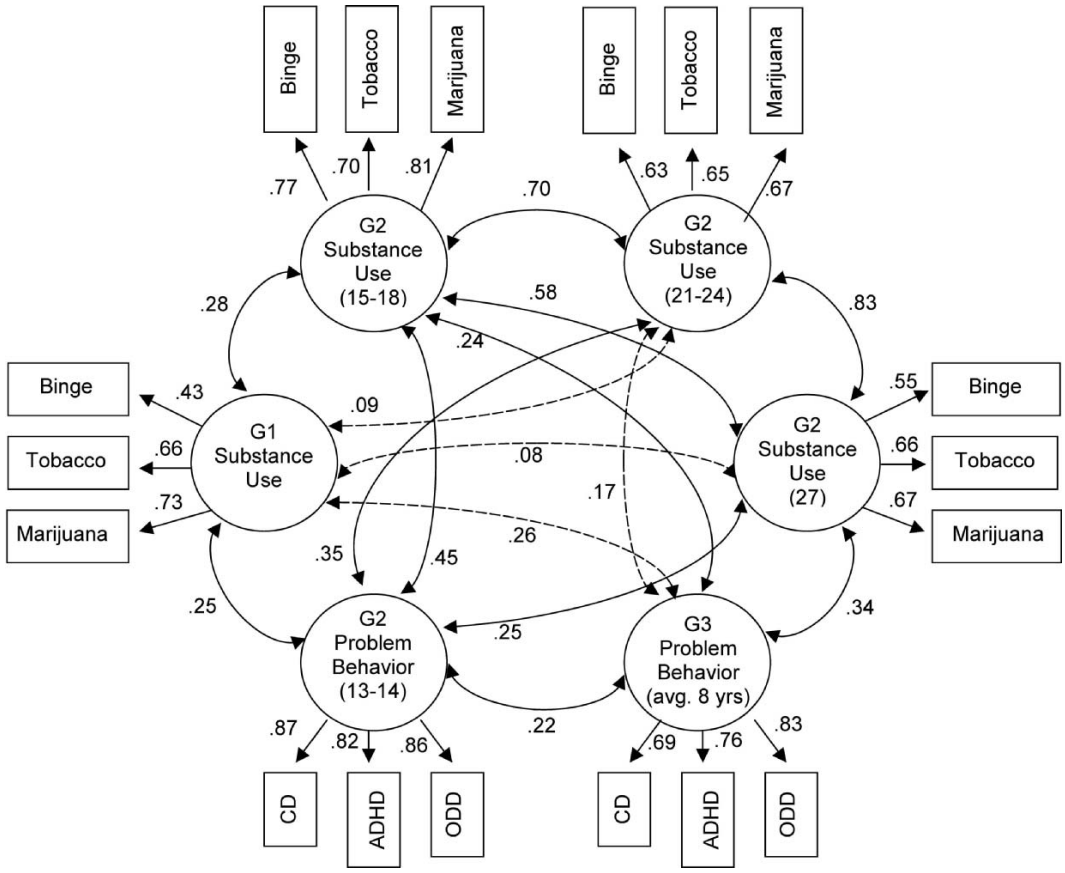


**Fig. 1.** Confirmatory factor analysis of hypothesized latent constructs. Correlated, substance-specific residual error terms are not shown, but were included in the analysis (age at measurement in parentheses). Dashed lines indicate nonsignificant paths.

respective latent factors, and all loadings were .43 or higher (see Fig. 1). The G1 latent substance use factor was significantly correlated with the G2 adolescent substance use ($r = .28$, $p < .001$) and problem behavior factors ($r = .25$, $p < .001$). G1 substance use was not significantly correlated with G2 early adult or age 27 substance use or G3 problem behavior. G2's early adolescent problem behavior (ages 13–14) was significantly correlated with their later substance use in high school (ages 15–18; $r = .45$, $p < .001$), in early adulthood ($r = .35$, $p < .001$), and at age 27 ($r = .25$, $p < .001$). Intercorrelations among the G2 adolescent, early adult, and age 27 substance use factors were significant and ranged from .58 to .83 (all $p$'s $< .001$). Significant correlations were observed between G2 substance use in adolescence and at age 27 and G3 problem behavior ($r = .24$, $p = .04$ and $r = .34$, $p = .03$, respectively). G2 problem behavior in early adolescence was significantly correlated with G3 problem behavior in childhood ($r = .22$, $p = .04$).

## Intergenerational Continuity in General Substance use

Analyses addressing our first question showed evidence of intergenerational transmission of a general tendency to use substances. One way we looked at continuity in drug use across generations was by examining G1–G2 correspondence in having used substances at all. Based on preliminary analyses shown in Table III, G1 use of any substance was significantly related to G2 use of any substance in both adolescence and early adulthood. Another way we looked at continuity in drug use across generations was through SEM. Figure 2 displays results of the SEM used to test for intergenerational continuity in general substance use. Model fit was acceptable ($\chi^2$ [20, 805] = 31.81, $p = .05$, CFI = 1.0, TLI = .99, RMSEA estimate = .03). Results suggest that G1 general substance use was significantly related to G2 general substance use in adolescence after accounting for unique transmission of specific substances, but the magnitude of the

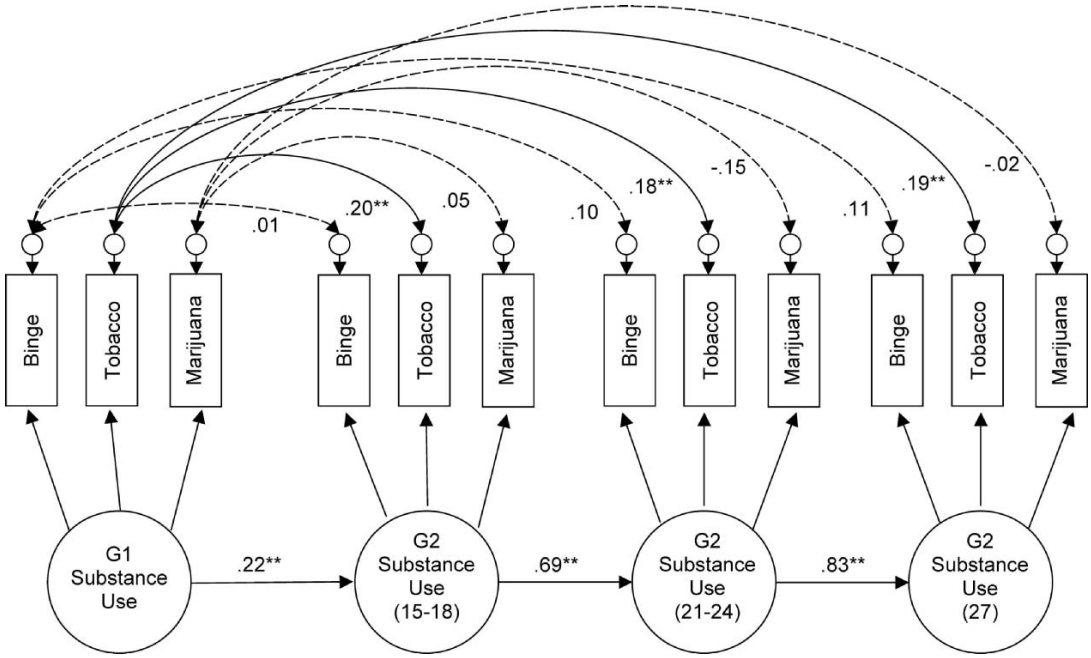


**Fig. 2.** Intergenerational substance use model. Correlated, substance-specific, within-person residual error terms for G2 are not shown, but were included in the analysis (age at measurement in parentheses). Dashed lines indicate nonsignificant paths. $^{*}p < .05;^{**}p < .01,^{***}p < .001$.

relationship was modest (beta = .22, $p < .01$). In this model, G1 general substance use explained 5% of the variance in the G2 adolescent substance use factor.

An alternative model including effects of G1 Substance Use on G2 age 21–24 Substance Use and G2 age 27 Substance Use was tested (not shown, $\chi^2[18, 805] = 29.69$, $p = .04$, CFI = 1.0, TLI = .99, RMSEA estimate = .03), but these paths were not significant. Comparing these models suggested that eliminating nonsignificant paths did not significantly reduce model fit ($\chi^2[2, 805] = 2.00$, $p = .36$). In other words, we found that the effects of G1 general substance use on G2 general substance use at ages 21–24 and 27 were fully mediated by G2 adolescent substance use at ages 15–18.

## Intergenerational Continuity in Substance-Specific Use

Analyses addressing our second question showed some evidence of substance-specific transmission across generations. We addressed this question in three ways: using $\chi^2$ tests, using correlation, and using SEM. We first ran preliminary $\chi^2$ and correlation analyses that did not control the general tendency to use substances. Table III

**Table III.** Correspondence Between G1 and G2 Substance Use Across G2 Developmental Periods

| | *G2 Adolescence* (15–18) (% Yes) | *G2 Early Adulthood* (21–24) (% Yes) | *G2 Age* 27 (% Yes) |
|---|---|---|---|
| Any substance use | | | |
| G1 yes (57%) | 49% | 67% | 51% |
| G1 no (43%) | 35% | 59% | 45% |
| | $\chi^2 = 13.44^{*}$ | $\chi^2 = 4.33^{*}$ | $\chi^2 = 2.35$ |
| Binge drinking | | | |
| G1 yes (28%) | 27% | 47% | 34% |
| G1 no (72%) | 21% | 38% | 27% |
| | $\chi^2 = 3.17$ | $\chi^2 = 5.64^{*}$ | $\chi^2 = 3.34$ |
| Marijuana use | | | |
| G1 yes (11%) | 36% | 28% | 23% |
| G1 no (89%) | 23% | 32% | 19% |
| | $\chi^2 = 5.91^{*}$ | $\chi^2 = .50$ | $\chi^2 = .61$ |
| Cigarette use | | | |
| G1 yes (43%) | 36% | 51% | 39% |
| G1 no (57%) | 23% | 40% | 27% |
| | $\chi^2 = 14.00^{*}$ | $\chi^2 = 9.32^{*}$ | $\chi^2 = 10.02^{*}$ |

*Note*. Table created from dichotomous variables indexing *any* reported use. For G2, use was defined as any binge drinking, cigarette smoking, or marijuana use in the past month. For G1, use was defined as any "current" binge drinking, cigarette smoking, or marijuana use, but a timeframe was not specified.
$^{*}p < .05$.

displays the correspondence between G1 and G2 use of each substance. G1 smoking was significantly related to G2 smoking in each developmental period. G1 binge drinking was significantly related to G2 binge drinking during early adulthood, and G1 marijuana use was related to G2 marijuana use during adolescence. Table IV shows a correlation matrix of variables included in analyses. These correlations reveal a similar pattern of results; G1 cigarette use is consistently significantly related to G2 cigarette use, but there is less evidence for the substance-specific transmission of binge drinking and marijuana use.

Figure 2 shows results of the SEM testing the substance-specific continuity in binge drinking, cigarette use, and marijuana use after accounting for the general latent tendency to use substances. Although only the G1 to G2 substance-specific, correlated error terms are shown in the figure, within-generation, substance-specific, correlated residual error terms for G2 were included in the model (see Table V). Results suggest that cigarette use is correlated across generations above and beyond a general tendency to use drugs. G1 cigarette use residuals were related to G2 cigarette use residuals in adolescence, early adulthood, and at age 27 ($r = .18$ to .20). Binge drinking and marijuana use, however, did not show evidence of specific continuity across generations once general substance use was accounted for.

## Child Problem Behavior as a Mechanism for Transmission

Our third question asked whether problem behavior serves as a mechanism for the transmission of substance use across generations. We addressed this question in two ways: using correlation and using SEM. As shown in Table IV, G2 problem behavior was significantly correlated with G1 substance use and G2 substance use variables, consistent with the hypothesis that problem behavior may be a mechanism for the intergenerational transmission of substance use. Results in Fig. 3 further suggest that G2 problem behavior in early adolescence mediated the relationship between G1 substance use and G2 high school substance use. Adding G2 adolescent problem behavior to the model depicted in Fig. 2 reduced the G1–G2 general substance use relationship to nonsignificance (from .22 to .12).

The model in Fig. 3 also adds G3 problem behavior, and shows that, as in the G1–G2 relationship, G2 age 27 substance use was significantly related to G3 problem behavior (ages 6–13). Thus, parent substance use was associated with increased child problem behavior in both generation pairs. A path linking G2 adolescent problem behavior and G3 problem behavior was tested, and was not significant (parameter estimate = .17). Overall, the pattern of results depicted in Fig. 3 suggests that parent substance use predicts child problem behavior. For G2, problem behavior predicted later substance use. These findings suggest that problem behavior does serve as a mechanism for the transmission of substance use across generations.

To address the possibility that the relationship among parent substance use and problem behavior is different for those who go on to become parents (SSDP parents in the TIP sample) and those who do not (SSDP participants without children and not in TIP), a multiple group analysis excluding the G3 problem behavior factor was conducted. Muthen and Muthen (2003a, 2003b) recommend a series of three steps when testing factorial invariance for latent variables with categorical outcomes. First, the hypothesized model should be run separately on each group. Second, a model in which all parameters are free (except scale factors, which are fixed to 1 in all groups, and factor means, which are fixed to 0 in all groups) should be estimated. Finally, a model should be tested in which factor loadings and thresholds are held constant across groups, scale factors are fixed at 1 in the first group and free in other groups, and factor means are fixed at 0 in the first group and free in other groups. Table VI shows fit statistics for steps 1 through 3, as well as the results of the chi-square difference test assessing measurement invariance. In addition to testing measurement invariance in step 3, we also tested invariance in the structural parameters. Results indicated that the same measurement and structural models for the G1–G2 section of the model fit the data for members of the SSDP sample who had a child by age 27 and participated in TIP and those who did not participate in TIP (most of whom did not have children).

## Competing Hypotheses

Our fourth research question asked whether parent substance use predicts child problem behavior when competing explanatory variables are controlled. Several competing hypotheses for observed intergenerational continuity in substance use and for the relationship between parent substance use and child problem behavior were tested using SEM, but not supported. The inclusion of G1 marital status, G1 education, G1 antisocial behavior, and contemporaneous neighborhood disorganization into the model shown in Fig. 3 did not alter substantive findings about the interrelationships among G1 substance use, G2 problem behavior, and G2 adolescent substance use. The

**Table IV.** Correlation Matrix of Substance Use and Problem Behavior Variables

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| G1 substance use | | | | | | | | | | | | | | | | | | |
| 1. Binge drinking | — | | | | | | | | | | | | | | | | | |
| 2. Cigarette use | .20* | — | | | | | | | | | | | | | | | | |
| 3. Marijuana use | .20* | .26* | — | | | | | | | | | | | | | | | |
| G2 Adolescent substance use (Age 15–18) | | | | | | | | | | | | | | | | | | |
| 4. Binge drinking | .07 | .10* | .02 | — | | | | | | | | | | | | | | |
| 5. Cigarette use | .07 | .22* | .14* | .45* | — | | | | | | | | | | | | | |
| 6. Marijuana use | .03 | .15* | .08* | .45* | .45* | — | | | | | | | | | | | | |
| G2 Early adult substance use (Age 21–24) | | | | | | | | | | | | | | | | | | |
| 7. Binge drinking | .10 | −.02 | −.01 | .32* | .18* | .20* | — | | | | | | | | | | | |
| 8. Cigarette use | .03 | .14* | .08* | .21* | .53* | .25* | .29* | — | | | | | | | | | | |
| 9. Marijuana use | .06 | −.03 | −.05 | .20* | .16* | .34* | .40* | .28* | — | | | | | | | | | |
| G2 Substance use age 27 | | | | | | | | | | | | | | | | | | |
| 10. Binge drinking | .15* | −.02 | −.03 | .20* | .15* | .12* | .52* | .26* | .26* | — | | | | | | | | |
| 11. Cigarette use | .04 | .15* | .06 | .17* | .44* | .21* | .22* | .72* | .21* | .29* | — | | | | | | | |
| 12. Marijuana use | .06 | .01 | −.02 | .13* | .11* | .20* | .23* | .24* | .58* | .22* | .24* | — | | | | | | |
| G2 Adolescent problem behavior (Age 13–14) | | | | | | | | | | | | | | | | | | |
| 13. Conduct problems | .07 | .13* | .09* | .25* | .28* | .33* | .19* | .20* | .20* | .10* | .15* | .11* | — | | | | | |
| 14. Opposition al defiant | .05 | .15* | .14* | .22* | .22* | .26* | .13* | .16* | .16* | .02 | .11* | .14* | .78* | — | | | | |
| 15. Attention problems | .07 | .16* | .10* | .20* | .24* | .24* | .16* | .23* | .17* | .07 | .15* | .14* | .69* | .71* | — | | | |
| G3 Problem behavior (Age 6–13) | | | | | | | | | | | | | | | | | | |
| 16. Conduct problems | −.06 | .36* | .11 | −.08 | .07 | .11 | .02 | .06 | −.05 | −.12 | .22* | .24* | .02 | .17 | .06 | — | | |
| 17. Opposition al defiant | .12 | .25* | .21† | .10 | .19† | .14 | .07 | .06 | .01 | .02 | .15 | .13 | .13 | .28* | .19† | .66* | — | |
| 18. Attention problems | .05 | .07 | .08 | .04 | .09 | .15 | .22* | .15 | .09 | .18† | .23* | .14 | .14 | .22* | .10 | .55* | .53* | — |

*$p < .05$.
†$p < .10$.

**Table V.** Within Generation Correlations of Residual Variance Terms from Model Shown in Fig. 2: Substance-Specific Correlations Over Time

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|
| Binge drinking | | | | | | | | | |
| 1. G2 adolescent | — | | | | | | | | |
| 2. G2 early adult | .16* | — | | | | | | | |
| 3. G2 age 27 | .07 | .30* | — | | | | | | |
| Cigarette use | | | | | | | | | |
| 4. G2 adolescent | | | | — | | | | | |
| 5. G2 early adult | | | | .44* | — | | | | |
| 6. G2 age 27 | | | | .38* | .52* | — | | | |
| Marijuana use | | | | | | | | | |
| 7. G2 adolescent | | | | | | | — | | |
| 8. G2 early adult | | | | | | | .17* | — | |
| 9. G2 age 27 | | | | | | | .14* | .39* | — |

*$p < .05$.

inclusion of G2 marital status at age 27, G2 education as of age 24, G2 antisocial behavior, and contemporaneous neighborhood disorganization into the model shown in Fig. 3 resulted in a parameter estimate linking G2 age 27 substance use to G3 problem behavior that is virtually identical to that presented in Fig. 3.

### G1 Substance Use and G3 Problem Behavior

The SEM presented in Fig. 3 also allowed us to examine the final research question: Does earlier grandparental substance use affect problem behavior among their grandchildren, either directly or indirectly? There

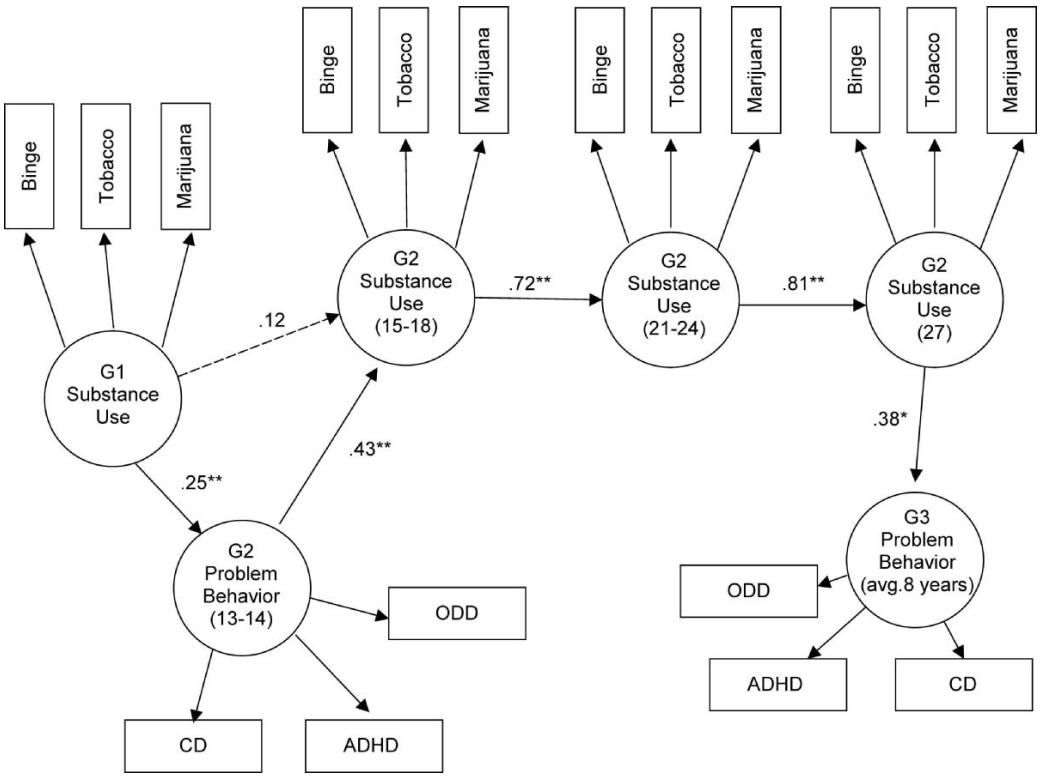


**Fig. 3.** Final structural model. Correlated, substance-specific residual error terms are not shown, but were included in the analysis (age at measurement in parentheses). Dashed lines indicate nonsignificant paths. *$p < .05$;**$p < .01$,***$p < .001$.

**Table VI.** Model Fit Statistics for Steps in Measurement and Structural Invariance Testing

| Step | $\chi^2$ (*df*, N) | *p* | CFI | TLI | RMSEA |
|---|---|---|---|---|---|
| **1** | | | | | |
| TIP participants only | 21.36 (26, 160) | .72 | 1.0 | 1.0 | .00 |
| Non-TIP participants only | 35.06 (31, 400) | .28 | 1.0 | 1.0 | .02 |
| **2** | | | | | |
| All parameters free across groups | 66.76 (61, 560) | .29 | 1.0 | 1.0 | .02 |
| **3** | | | | | |
| Measurement and structural parameters fixed across groups | 64.56 (59, 560) | .29 | 1.0 | 1.0 | .02 |
| DIFFTEST results comparing models from steps 2 and 3 | 6.86 (6, 560) | .33 | — | — | — |

*Note*. Degrees of freedom and RMSEA are approximated when the WLSMV estimator is used.

was clear evidence that G1 substance use when G2 was age 13–14 indirectly affected G3 problem behavior thirteen years later. G1 substance use was significantly related to G2 problem behavior, which was, in turn, significantly related to G2 adolescent substance use. Within-generation continuity in G2 general substance use from adolescence to age 27 was strong. The model explained 48% of the variance in the age 21–24 substance use factor and 68% of the variance in the age 27 substance use factor. There was also evidence of within-person continuity in the use of specific substances over time (see Table V). For G2, the tendency to continue using a specific substance from adolescence into adulthood (controlling general substance use) was strongest for cigarettes ($r = .38$ to .52) and significant but smaller for marijuana use ($r = .14$ to .39) and binge drinking ($r = .07$ to .30). This within-generation continuity in G2 drug use from adolescence into adulthood was a mechanism for the effect of G1 substance use on G3 problem behavior; G2 substance use at age 27 was significantly related to G3 problem behavior.

There was less evidence for direct effects of G1 substance use on later G3 problem behavior. As shown in Table IV, grandparent cigarette use was significantly correlated with conduct problems and oppositional defiant behavior in G3 thirteen years later. The correlation between G1 marijuana use and G3 oppositional defiant behavior was marginally significant. In the CFA (Fig. 1), the latent G1 general substance use factor was correlated with the G3 problem behavior factor at .26. This value was not significant, perhaps due to the sample size of 97 cases on which this estimate was based, but was quite similar in magnitude to the significant correlation between G1 substance use and G2 problem behavior. Finally, in the structural model (Fig. 3) a path from the G1 general substance use factor to the G3 problem behavior factor was tested (not shown, $\chi^2[44, 805] = 60.79$, $p = .05$, CFI = .99, TLI = .99, RMSEA estimate = .02). This path was estimated at .28, but was not significant. This is similar to the estimate of the relationship between G2 substance use and G3 problem behavior (.38) and to the estimate of the influence of G1 substance use on G2 problem behavior (.25), which is based on 771 participants. Results of the DIFFTEST procedure, however, showed that removing this nonsignificant path from G1 substance use to G3 problem behavior did not reduce model fit ($\chi^2[1, 805] = 2.53$, $p = .11$), so the more parsimonious model presented in Fig. 3 was retained as the final model.

## DISCUSSION

The present study examined the extent to which general substance use and substance-specific use are perpetuated across generations, whether child behavior problems are a key mechanism of the intergenerational link, and whether grandparent substance use affects grandchild problem behavior directly. This study extends prior research on the intergenerational continuity of substance use and problem behavior by including three generations and considering continuity in both general substance use and the use of specific drugs. Five main findings related to our five research questions emerged: (a) cross-generational continuity in the general tendency toward substance use was small, but significant (controlling continuity in specific substances); (b) cigarette use showed moderate specific continuity across generations (controlling general use); (c) G2 adolescent problem behavior fully mediated the effects of G1 general substance use on G2 substance use in high school and G2 adult substance use was related to G3 child problem behavior, a precursor of possible future G3 substance use; (d) these relationships persisted in the presence of competing explanatory variables; (e) grandparent substance use was indirectly related to G3

problem behavior thirteen years later, as mediated through G2 problem behavior and substance use.

Several limitations should be kept in mind when interpreting findings presented here. First, measures of G1 substance use and antisocial behavior were not optimal. Although G1 substance use was assessed prospectively and using self-reports of current use, measures could have been improved by reference to a specific time period and response scales yielding a more detailed picture of the frequency of use. The measure of G1 antisocial behavior reflects only behavior severe enough to have resulted in police contact or detention, and may have missed less severe expressions of antisocial behavior. In addition, the measure does not capture the frequency of antisocial behavior. Nevertheless, the inclusion of a more comprehensive measure of G2 antisocial behavior did not eliminate the relationship between G2 substance use and G3 problem behavior, suggesting that this limitation in the data on G1 antisocial behavior did not greatly influence observed findings. Second, findings about substance-specific correlation across generations (and within-person) should be interpreted with caution, because residual error terms may contain method variance or other correlated error variance that may increase the correlation coefficients. Third, findings about the influence of G1 and G2 substance use on G3 problem behavior are based on 97 cases; analyses based on this small sample may have resulted in overly large standard error estimates, which increase the likelihood of Type II error. It is possible that we have committed a Type II error by rejecting the model in which G1 substance use affected G3 problem behavior directly. Findings about the relationship between G1 and G2 substance use and G3 problem behavior need to be replicated with a larger sample. Fourth, some research indicates that the predictors of problem behavior may vary with child age (Frick, Christian, & Wootton, 1999), making the broad age range of G3's in this sample potentially troublesome. Parent substance use, however, has been linked with problem behavior among children of varying ages, from toddlerhood (Brook, Tseng, & Cohen, 1996; Brook, Whiteman, Shapiro, & Cohen, 1996) to adolescence (Chassin et al., 1991; Clark et al., 1997), and we standardized problem behavior scores within 2–4 year age groups. Fifth, although our finding that child problem behavior mediated the relationship between parent substance use and later child substance use is consistent with both genetic and social development approaches, the presently available data did not permit us to investigate the relative contributions of genetic and social development influences on problem behavior. Sixth, we hypothesized that parent substance use leads to child problem behavior, but these constructs were, in fact, measured concurrently in both generation pairs. It is possible, though we think unlikely, that the causal direction is reversed. It is also possible that parent substance use and child problem behavior are reciprocally related (Pelham & Lang, 1999; Pelham et al., 1997). Finally, we tested several competing explanations of the relationship between parent substance use and child problem behavior, but other plausible confounds exist. For example, psychopathology, especially depression, often co-occurs with substance use in adults (Biederman et al., 2005; Kessler et al., 1994; Merikangas et al., 1998; Zilberman, Tavares, Blume, & el-Guebaly, 2003), and parental psychopathology, particularly maternal depression, has been linked to child problem behavior (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005; Marmorstein, Malone, & Iacono, 2004). Parental depression cannot be ruled out as a potential explanation of the observed relationship between parent substance use and child problem behavior.

Results examining our first question about the intergenerational transmission of general substance use were consistent with existing research suggesting that there is a general latent tendency to use drugs that shows continuity across generations (Hoffmann & Cerbone, 2002; Kendler et al., 2003; Merikangas & Avenevoli, 2000; Merikangas, Stolar et al., 1998; Tsuang et al., 2001). Intergenerational continuity in general substance use, however, was small in magnitude (.22) at the zero order, and descriptive statistics showed a high rate of substance use among G2s whose parents did not use substances (59% in early adulthood), suggesting that there is also a great deal of intergenerational *dis*continuity in substance use in this sample. The small magnitude of the observed relationship between G1 and G2 substance use is not clearly consistent with previous research that has suggested stronger effects of genetic influence and parent substance use (e.g., Chassin et al., 1991; Kendler, Karkowski, Neale, & Prescott, 2000; Leib et al., 2002; Merikangas et al., 1998; Walters, 2002). The present study looked at the frequency of substance use in a community sample, but elevated levels of use, especially abuse and dependence, tend to show more evidence of intergenerational continuity than more normative levels of use (McGue, 1994; Walters, 2002). The small effect of parent substance use observed here may be due to a lower prevalence of severe substance use problems in the G1 and G2 samples. Alternatively, the small observed relationship between parent and child substance use may be due to the fact that the majority of G1 parents were mothers, and the effects of paternal substance use may be underestimated. Finally, other research suggests that parent influences on child substance use, be they due to genetic influence, availability, socialization practice, or other mechanisms, are only part of the story. Peer,

school, neighborhood, and other factors are also important determinants of adolescent substance use (Hawkins et al., 1992), especially initiation of use (Han et al., 1999; McGue et al., 2000).

Our second research question asked whether there was continuity in the use of specific substances between parents and children. We found evidence for substance-specific intergenerational continuity in cigarette smoking. This suggests that parts of the variance in cigarette use not associated with general substance use show substance-specific continuity across generations. Why this may be remains to be studied. Chassin and colleagues suggest that maternal smoking leads to less discussion of smoking with children and less punishment of smoking when it occurs (Chassin et al., 1998). Decreased smoking-specific discussion and decreased punishment of smoking were linked to increased smoking among children. Thus, smoking-specific parenting practices may explain the link between parent and child smoking. Other possible explanations include exposure to second-hand smoke in the home, possibly leading to addiction, genetic liability for cigarette use, greater availability of cigarettes in the home for child access, modeling, or perhaps greater involvement of the child in the parent's smoking behavior (Hill, Hawkins, Catalano, Abbott, & Guo, 2005). Finally, prenatal exposure to cigarette smoke has been linked to later problem behavior, especially attention problems (Linnett et al., 2003) among children. Results from this study and others (Colder & Stice, 1998; Hawkins et al., 1992; McGue et al., 2001; Milberger et al., 1997; Neumark-Sztainer et al., 1997) suggest that problem behavior increases the risk for later substance use. Attention problems, specifically, have been linked to early cigarette use (Milberger et al., 1997). Prenatal nicotine exposure is another possible explanation for the observed parent-child continuity in cigarette use. More research is necessary to investigate mechanisms in the substance-specific transmission of cigarette use.

We found no evidence for drug-specific intergenerational continuity in binge drinking and marijuana use. This is somewhat surprising given the strength of the association between parent and child alcohol abuse in existing literature (e.g., Chassin et al., 1991; Leib et al., 2002; McGue, 1994; Merikangas et al., 1998). Again, prior research suggests that parent alcohol use may be more predictive of child alcohol use as the severity of use increases, with the strongest relationships being observed for abuse and dependence (McGue, 1994; Walters, 2002). It may be that participants in this study reported more normative, non-clinical patterns of binge drinking that do not demonstrate a high degree of intergenerational continuity. In addition, the effects of paternal drinking may be underestimated in this sample, as may genetic influence, given that some G1 parents were not biologically related to G2 participants. Previous findings on the substance-specific transmission of marijuana use have been mixed. Some studies have found evidence for this type of transmission (e.g., Andrews et al., 1997; Merikangas et al., 1998). These studies, however, did not control for the general tendency to use substances in examining the cross-generation variance specific to marijuana use. Our results are in line with those of Tsuang and colleagues (2001), who found that a large majority of the variance in marijuana use was accounted for by genetic, family environmental, and nonfamily vulnerabilities common to the use of many substances.

Our third question asked whether child problem behavior was a key mechanism in the relationship between parent and child substance use. Consistent with prior research and with genetic and social development theories, problem behavior among members of G2 in early adolescence mediated the effects of G1 substance use on G2 high school substance use in our sample, even in the presence of competing explanatory variables (our fourth research question). Further, the relationship between parent substance use and child problem behavior observed in G1 and G2 was replicated in G2 and G3, also in the presence of competing explanatory variables. These findings should be generalized with caution until replicated. In particular, the parameter estimate of the effects of G2 substance use on G3 problem behavior may not be generalizable to families in which childbearing occurs after age 27. However, the current data suggest the generalizability of these findings to older parents and their children. First, though G1 parents varied widely in age, and were older, on average, than were G2 parents at the point of this study (age 27) the relationships between G1 substance use and G2 problem behavior were similar in magnitude to those between G2 substance use and G3 problem behavior. Second, the G2 substance use to G3 problem behavior parameter estimate should be unbiased with relation to all variables included in the models tested here. Third, invariance in measurement and structural parameters for the G1 and G2 variables was demonstrated when TIP participants were compared to those participants who were not in TIP. We found some evidence of a relationship between G2 problem behavior and G3 problem behavior.

From a genetic standpoint, the observed association between parent substance use and child problem behavior may be due to inherited genetic liability common to both substance use and other forms of behavioral undercontrol (McGue, 1994). Social development factors that may be important environmental influences on this link between parental substance use and child problem behavior include suboptimal parenting, such as low monitoring

and harsh discipline practices, low family bonding, and family norms that increase problem behavior (Catalano & Hawkins, 1996; Hawkins et al., 1992; Hill et al., 2000). Marital aggression also has been linked to both parent substance use and child problem behavior (Fuller et al., 2003). Future multigenerational studies of the relationship between parent substance use and child problem behavior should include measures of these potential genetic and environmental mechanisms.

Very little prospective, longitudinal research has explored the role of grandparent substance use in predicting substance use and problem behavior among grandchildren. We found that G1 substance use affected G3 problem behavior indirectly by increasing G2 adolescent substance use, which showed considerable within-person stability over time and was, in turn related to G3 problem behavior. Again, these findings may be due to transmission of a genetic predisposition toward problem behavior that is passed from G1 to G3 via G2. In addition, these findings may be due to intergenerational transmission of parenting practices, such as low bonding, harsh discipline, and parent to child aggression (Capaldi, Pears, Patterson, & Owen, 2003; Conger, Neppl, Kim, & Scaramella, 2003; Hops, Davis, Leve, & Sheeber, 2003; Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003). Because these parenting practices also have been linked to parent substance use (see Barnard & McKeganey, 2004 for a review; Kandel, 1990), they constitute potential pathways for the indirect influence of G1 substance use on G3 problem behavior that should be explored. Evidence for the direct effect of G1 substance use on G3 problem behavior was less clear. Additional research is necessary to examine the potential direct influences of G1 substance use on G3 problem behavior.

## CONCLUSIONS

The present findings highlight several implications for substance use prevention as well as the importance of primordial prevention aimed at interrupting intergenerational cycles of substance use and problem behavior. First, intergenerational continuity in substance use appeared to be largely due to the transmission of a general tendency to use substances, rather than substance-specific mechanisms. This suggests that prevention and intervention programs taking a general, as opposed to substance-focused, approach may be beneficial. Cigarette use, however, did show evidence of specific transmission over and above the general tendency to use substances. Efforts directed at reducing cigarette use among parents and preventing the onset of smoking during adolescence should be continued, as should research aimed at understanding the parent-child transmission mechanisms at work in order to improve intervention and prevention strategies. Second, the observed within-person stability in general substance use over time suggests that efforts directed at preventing adolescent substance use should be continued, because this stability constitutes a mechanism for intergenerational transmission. Cigarette use also showed a high degree of within-person continuity, which was likely due to addiction (Cohen, Kodas, & Griebel, 2005; Mansvelder & McGehee, 2002), and points to the need for further, substance-specific prevention programs and support for cessation. Third, prevention programs that seek to reduce chronic and severe conduct problems for high-risk children (Conduct Problems Prevention Research Group, 2002a, 2002b, 2002c; Greenwood, Model, Rydell, & Chiesa, 1996; Webster-Stratton et al., 2001) may also aid in breaking cycles of intergenerational transmission of substance use. Finally, in the present study, negative outcomes associated with grandparent substance use are observed not only in their children, but are transmitted to their grandchildren as well. Thus, the benefits of successful intervention may also echo across generations. Successful preventive interventions may not only reduce conduct problems and substance use and put youth on a positive track towards adult development, but may also affect positive development in the next generation.

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