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Age of Onset of First Alcohol Intoxication and Subsequent Alcohol Use Among Urban American Indian Adolescents

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Abstract

The objective was to assess the effect of early onset intoxication on subsequent alcohol involvement among urban American Indian youth. The data come from the American Indian Research (AIR) project, a panel study of urban Indian youth residing in King County, Washington. Data were collected annually from the adolescent and his/her primary caregiver from the 1988–89 school year to the 1996–97 school year, providing a total of nine waves of data. Early intoxication (by age 14) was related to delinquency, family history of alcohol abuse or dependence, poverty, broken family structure, less family cohesiveness, and more family conflict. The effects of these characteristics were, therefore, partialed out in testing effects of early intoxication on later alcohol involvement. Two-part latent growth models of alcohol use and alcohol problems were specified. Effects of early onset intoxication on these trajectories, as well as lifetime alcohol abuse or dependence by the transition to young adulthood, were examined. Findings indicate that adolescents who experienced their first intoxication early (by age 14), used alcohol more heavily from the ages of 16 to 18, experienced more problems related to the alcohol's use from the ages of 16 to 18, and were more likely to have a diagnosed alcohol disorder by the final wave of data collection. Congruent with similar studies in the general population, early intoxication appears to be associated with a deleterious course of alcohol involvement during adolescence and into the

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Keywords

American Indian adolescents; alcohol use; alcohol problems; early onset; two-part model

Alcohol abuse and dependence create serious health problems for American Indians. A national survey indicates that American Indian youth are more likely to qualify for a recent (i.e., past year) alcohol disorder diagnosis (8.5% of American Indian youth compared to 5.8% of non-American Indian youth; Office of Applied Studies, 2006). This health disparity extends beyond adolescence and into young adulthood. Among individuals aged 18 to 25, 20.8% of American Indians compared to 17.4% of non-American Indians qualify for a recent alcohol disorder diagnosis (Office of Applied Studies, 2006).

Similar to the general population, American Indian youth suffer consequences related to alcohol use. Adolescent American Indians who drink, rather than abstain, are more likely to be involved in alcohol related accidents, to have problems with their parents, to be arrested (Beauvais, 1992a), to be convicted, to run away, to smoke, to use more drugs, to have sex (Mitchell, Beals, Whitesell, & the Voices of Indian Teens and Pathways of Choice Teams, 2008), to engage in risky sexual behaviors, to engage in delinquent behaviors, and to ride with a driver who has been drinking (Potthoff et al., 1998). Research also indicates that American Indian youth who consistently consume alcohol throughout adolescence report heavier use of alcohol in adulthood (Mitchell et al., 2008).

These negative consequences of alcohol use extend into adulthood for American Indians and reports indicate that alcohol-related consequences are elevated among American-Indians as compared to the general population. For example, the Indian Health Service (1996) estimates that alcohol-related death rates may be from 5 to 7 times higher than they are for other Americans, liver disease nearly 4 times higher, deaths in alcohol related car crashes 3 times higher, and alcohol-related suicides and homicides occur more frequently. The rate of alcohol use varies greatly across tribes, but May (1991) found that many tribes had rates of Fetal Alcohol Syndrome that were as much as four times higher than national rates.

There is little question that alcohol use is a significant problem for many American Indian tribes, but Beauvais (1992a, 1992b) warns against stereotyping and points out that Indians show many different patterns of alcohol use and, in fact, there are both high rates of alcohol abstinence and high rates of alcohol abuse and dependence in many tribes (May, 1991; Spicer et al., 2003). Recognizing this heterogeneity, research is needed to study the process of alcohol use over the life course *within* American Indian populations in order to reduce the prevalence of problematic use of alcohol and its consequences.

The Role of Adolescent Alcohol Use in the Life-Course of Alcohol Abuse And Dependence

The role that alcohol use plays in the lives of adolescents and young adults has been the focus of a great deal of research. This period of time in a young person's life is critical to the study of alcohol behavior as it represents the developmental time frame in which most individuals begin experimenting with alcohol. For many youth, alcohol experimentation is limited and few short or long-term consequences are suffered. However, for others, adolescent alcohol use marks the beginning of a path leading to deleterious consequences and long-term abuse.

Zucker, Fitzgerald, and Moses (1995) described the mechanisms by which adolescent alcohol experimentation may lead to problematic outcomes. They pointed to evidence suggesting that adolescence is a time of development of independence and includes many behavioral and attitudinal changes such as increased rebelliousness, heightened acceptance for deviant behavior, and greater alcohol and other drug involvement. They indicate that this period of time can be contrasted with the expectations of young adulthood, which include prosocial/conventional behavior, career success, and entry into marriage. Based on the role and expectation differences between adolescence and young adulthood, Zucker and colleagues suggest the following:

If one makes it through adolescence without drug involvement, one misses the window of exposure, availability, and peer pressure which drives onset of the phenomenon. Thereafter, without earlier use, even if the biopsychological structure is appropriate for a pattern of abuse, the environmental triggers and the significant substance availability are absent—hence the notion of a critical period for risk based upon the mediating effects of stage-specific contextual factors (p. 686).

In other words, Zucker and colleagues suggest that an individual who initiates alcohol use during adolescence is more likely to progress to more problematic patterns of use than an individual who initiates use later in life because of the role and expectation differences between adolescence and young adulthood.

Building on this premise, age of onset of alcohol use has become one of the variables of interest that may help to differentiate between adolescents who go on to be heavy users of alcohol and experience alcohol-related consequences and those who do not. Many existing empirical studies have reported a prospective association between early onset of alcohol use and subsequent heavy alcohol use and/or alcohol problems (Chassin, Pitts, & Prost, 2002; Hawkins et al., 1997; Pedersen & Skrondal, 1998; Thomas, Reifman, Barnes, & Farrell, 2000; Warner & White, 2003). In one of the earlier papers on this topic, Grant and Dawson (1997) found that individuals who initiated drinking at age 14 or younger were 4 times as likely to become alcohol-dependent (as defined by the criteria of the DSM-IV) as those who initiated use at age 20 or older. Extending this work, Odgers and colleagues (2008) utilized a propensity score model (i.e., causal inference model) to assess the role of early onset of alcohol and other drug use on adult substance dependence. Matching on a host of salient potential confounders (i.e., shared risk factors that likely lead to both early onset and adult

dependence), the results of their study indicate that the deleterious effect of early onset substance use is likely to be causal. As a result of evidence linking early age of onset to subsequent alcohol problems, the U.S. Surgeon General stated that preventing early alcohol use is a public health priority (U.S. Department of Health and Human Services, 2007).

Besides heightening the risk for problematic use and alcohol consequences, the deleterious effects of early onset use surface in other areas as well. For example, adolescents who demonstrate an early debut to alcohol use are also more likely to perform poorly in school, engage in delinquency, and commit criminal acts (Ellickson, Tucker, & Klein, 2003; Peleg-Oren, Saint-Jean, Cardenas, Tammara, & Pierre, 2009).

Research examining the role of age of initiation on short- and long-term outcomes among American Indian youth has, unfortunately, been sparse. This is critically important work given that American Indian adolescents appear to initiate alcohol use earlier than other adolescents (Roski, Perry, McGovern, Veblen-Mortenson, & Farbaksh, 1998). We identified only two studies that utilized a sample of American Indian individuals and treated age of initiation as a major variable. The results of these studies do indeed indicate that early initiation is likely to be a problem among Indian youth.

First, Deardorff, Gonzales, Christopher, Roosa, and Millsap (2005) determined that age of first alcohol use (as well as age of first intercourse) mediated the relationship between age of menarche and age of first pregnancy among Native American mothers, suggesting that early alcohol initiation may be an important factor in early pregnancy. Second, Ehlers, Slutske, Gilder, Lau, & Willhelmsen (2006) surveyed American Indian adults on reservations in southwest California regarding their drinking histories, and found that those who had become intoxicated at an earlier age were more likely to become dependent on alcohol later (as defined by DSM-III-R criteria). While both studies relied on cross-sectional data and retrospective reporting of age at onset, these papers suggest that early alcohol debut may play an important role in the progression of alcohol problems and other precocious outcomes. Further research is needed to better understand these processes and relationships.

The goal of the current study is to fill a critical gap by assessing the effect of early onset of alcohol intoxication (defined here as "drank enough so that it was hard to walk, talk, remember or decide what to do" by age 14) on the subsequent course of alcohol use and alcohol related problems through adolescence and into young adulthood. Data from a 9-year longitudinal study of urban American Indian youth were utilized (Walker et al., 1996). The covariates, early onset status, and subsequent alcohol use and related consequences were all collected prospectively.

Hypotheses for the Current Study

H1: Individuals who experience first intoxication by age 14 will be more likely to use alcohol and will use alcohol more heavily at ages 16, 17, and 18 than those who experience first intoxication later.

H2: Individuals who experience first intoxication by age 14 will be more likely to experience alcohol problems and experience more problems related to alcohol use (measured by the

Rutgers Alcohol Problem Index or "RAPI") at ages 16, 17, and 18 than those who experience first intoxication later.

H3: Individuals who experience first intoxication by age 14 will be more likely to qualify for a lifetime diagnosis of alcohol abuse or dependence (DSM-IV criteria) by the transition to adulthood (i.e., the final wave of data collection) than those who experience first intoxication later.

Method

Data

The data for this study come from the American Indian Research (AIR) project, a panel study of urban Indian youth residing in the King County Washington metropolitan area (Walker et al., 1996). Research participants are 263 urban American Indian youth recruited in the 1988–89 school year and followed annually through the 1996–97 school year. Overall, the retention rate at the end of the study was 93%. Following Institutional Review Board approval from the school district boards, the Indian Health Board, and the University of Washington, youth were recruited from the rosters of students at two schools with high Indian enrollment, (n = 207) and age-matched youth recruited from rosters of patients at an urban Indian health clinic (n = 56). These 56 participants did not differ from the school sample on age, gender, degree of Indian blood, Indian lineage, or welfare subsidy. Students from this group had resided in Seattle for fewer years and had a family income that was lower than the students recruited from schools.

Participants were classified as "urban" based upon King County, Washington, zip codes and the U.S. Census definition of 2500 or more persons per designated place (United States Bureau of the Census, 1995). Based on 1990 Census data, at recruitment, participants represented an estimated 58% of the 11–14 year-old American Indian youth of Seattle, and 34% of the 11–14 year-old American Indian youth of King County. At the initial wave, the average age of these adolescents in the sample was 11.1 (SD = .8), they were nearly equally distributed by gender (52% female) and 65% lived above the poverty line. Walker and colleagues (1996) reported that participants recruited for the AIR project represented over 50 tribes and nine cultural areas. The highest representation was Tlingit (11.4%) Cherokee (10.2%), Blackfeet (7.3%), Ojibwa (6.8%), and Sioux (6.5%).

Measures

The dependent variable of interest in this study is adolescent alcohol use at ages 16, 17, and 18. We chose age 16 to begin in order to allow a delay between the age of onset and subsequent use (thus maintaining the temporal ordering) and we chose age 18 because many participants in the dataset were not observed after age 18. In the first set of analyses, an alcohol use index measured at ages 16, 17, and 18 is considered. This index score is made up of the sum of three items, all self-reported by the adolescent: 1) number of times he/she was intoxicated from alcohol in the past year; 2) number of times he/she was intoxicated from alcohol in the past year; 30 number of times he/she drank five or more drinks in one sitting during the past year. These items are highly correlated with one another; Cronbach's

alpha equals .83 at age 16, .90 at age 17, and .89 at age 18. In the second analysis, the adolescent's score on the Rutgers Alcohol Problem Index (RAPI) measured at ages 16, 17, and 18 is considered (White & Labouvie, 1989). Students indicated how often they experienced 23 different alcohol-related consequences during the previous year. These consequences range from relatively minor (did not complete homework) to serious (passed out or fainted). Each item was reported on a 5-point scale: 0 = never; 1 = 1 to 2 times; 2 = 3to 5 times; 3 = 6 to 10 times; and 4 = more than 10 times. Cronbach's alpha equals .93. In the third set of analyses a positive lifetime diagnosis of an alcohol abuse or dependence disorder (American Psychiatric Association's DSM-IV, 1994), measured with the Semi-Structured Assessment for the Genetics of Alcoholism, version II, diagnostic interview (Bucholz et al., 1994) at the final wave of the study (wave 9, when the average age of the participants was 19.1, SD = .8) is considered. Used in the NIAAA-funded multisite Collaborative Study on the Genetics of Alcoholism, the SSAGA reliability and validity is reported by Bucholz and colleagues (1994). Interviewers were trained in administration of the diagnostic instrument in compliance with SSAGA protocols and procedures and underwent intensive collaborative interviewing and independent coding to assure validity of the data obtained.

The primary predictor of interest is early onset of alcohol use. Although there are a variety of ways to define early alcohol debut, the AIR project emphasized clinically meaningful measurement (Walker et al., 1996), and we define it in this paper as first intoxication at or before age 14. This is in line with a study by Grant and Dawson (1997). We used the prospective reports from the youth at each wave to determine if he/she experienced his/her first intoxication from alcohol by age 14. Seventy-nine of the students (or 30% of the sample) were classified as early onset users.

A series of potential confounding and control variables is also considered. These include several demographic variables: gender, family structure (comparing adolescents who lived with both biological parents at wave 1 to all others), and family poverty (comparing adolescents who were living at or below the poverty line at wave 1 to those living above). The primary caregiver completed the Child Behavior Checklist (CBCL; Achenbach, 1991) at wave 1 and we consider the delinquency subscale in this study (Cronbach's alpha equals. 75). The substance use items were excluded from the delinquency scale for the purpose of the current analysis (i.e., to avoid controlling for a form of the variable of interest-early alcohol intoxication). Family functioning at wave 1 was measured with two variables reported by the adolescent, family cohesion and family conflict, using the Moos Family Relationships Index (FRI, Moos & Moos, 1986). Family cohesion (e.g., family members really help and support one another) and family conflict (e.g., we fight a lot in our home) each consist of nine items. Cronbach's alpha for cohesion equals .70 and Cronbach's alpha for conflict equals .61. Lifetime prevalence of alcohol abuse/dependence disorders was measured at wave 5 for the mother and father. Diagnostic data were obtained by administering the alcohol Family History Assessment Modules (FHAM; Janca, Bucholz, Janca & Jabos-Laster, 1991) for surrogate reports on family history or in the case of selfreport, the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994). We combined this information to form a binary indicator that differentiates between adolescents whose biological mother or biological father tested positive for a

lifetime diagnosis of alcohol abuse or dependence compared to adolescents whose biological mother and biological father tested negative for both.

Analyses

The first set of analyses considers the effect of early intoxication (i.e., intoxication by age 14) on the subsequent course of alcohol use and alcohol problems from ages 16 to 18. Both the alcohol index score and the alcohol problems score are positively skewed. To account for this, we took two steps. First, we log transformed the alcohol index and alcohol problems score (using a natural log transformation). Second, we modeled the data using a two-part growth model. Olsen and Schafer (2001) initially proposed this model in order to account for the preponderance of zeroes often encountered in developmental outcomes of problem behaviors. In these semi-continuous distributions there is a pile up of zeroes (i.e., those not exhibiting the behavior) and a continuous, positive distribution of values among those scoring a value higher than zero. A two-part model simultaneously estimates two separate, but correlated, growth models. The first growth model captures change in the probability of the outcome (e.g., alcohol use) at each measurement occasion. Here, binary indicators of use vs. no use at each wave are specified as a random coefficient, logistic growth model (Hedeker & Gibbons, 2006). The second growth model captures change in the level of the outcome (e.g., frequency of use) at each measurement occasion, conditional on some use (or some problems for the problem model). For example, in the alcohol use model students who demonstrate no use at a particular measurement occasion are coded as missing on the corresponding frequency of use measure and a random coefficient growth model for lognormal data is specified (Willett & Sayer, 1994). Once the unconditional growth models are specified, the growth parameters from each process may be regressed on the covariates of interest. For example, on early intoxication and the control/confounding variables.

Examination of growth processes via growth models first requires careful examination of model fit in order to best model change in the processes of interest over time. With only three waves of data, we are limited to simple functional forms (i.e., linear growth). We plotted the data for both alcohol use and alcohol problems for each adolescent in the sample against a linear process, examined residual plots and r-squared statistics, and assessed the assumptions of the random effects. These evaluations indicated that linear growth models provided a reasonable representation of change in these processes over time. In all models, random effects were estimated for all growth parameters and the growth parameters were specified to covary within process and across process.

In addition to the assessment of early intoxication on change in alcohol use and alcohol problems in late adolescence, we also consider the effect of early intoxication on the probability of a lifetime diagnosis of an alcohol abuse or dependence disorder by the final, wave 9 interview. Here, the log odds of diagnosis were regressed on early intoxication, the potential confounders, and age at the wave 9 interview. All models were estimated in Mplus, Version 5.21 (Muthen & Muthen, 1998–2007), using full information maximum likelihood and robust standard errors.

Although retention in the study was high, there are some missing data. Across the variables of interest, the percent of missing data on a particular variable ranges from 0% (for gender,

CBCL, family structure, and early onset intoxication) to 10% (for Father's alcohol/abuse dependence). There are very little missing data on the longitudinal assessments of drinking and problems. Among these variables, the highest number of missing cases is for the RAPI score at age 18, 5% of the cases are missing. While the percentage of missing cases is quite low for any one variable, if listwise deletion was employed in this study (i.e., any case with a missing value on one or more of the items was deleted), 74 of the 263 (28%) cases would be excluded. A wealth of research indicates that listwise deletion is likely to bias parameter estimates and significantly reduce statistical power (Schafer & Graham, 2002). Therefore, to appropriately handle missing data for the covariates and alcohol abuse/dependence we employed multiple imputation. The imputation was carried out using IVEware (Raghunathan, Solenberger, & van Hoewyk, 2002), a program which allows for the imputation of mixed, count, categorical, and continuous variables. A rich imputation model was specified that included all variables in the final models. Post-imputation diagnostics indicated that the imputation model was successful. In total, 10 imputed datasets were created. All analyses were performed on each of the imputed datasets, and the parameter estimates were then combined using the procedures outlined by Rubin (1987). Missing data for the growth model outcomes (i.e., alcohol use and alcohol problems) were handled using full information maximum likelihood during model estimation.

Results

Assessment of Potential Confounders

In all models, we sought to assess the relationship between early first intoxication and the alcohol outcomes after adjusting for a set of potential confounders. Table 1 presents descriptive statistics for these potential confounders as a function of early intoxication. Statistical comparisons conducted for each variable revealed that all identified potential confounders differ as a function of early onset status except for gender. That is, adolescents who experienced their first intoxication by age 14 demonstrated more delinquency, were more likely to have a family history of alcohol abuse or dependence, were more likely to live in poverty, were less likely to live with both biological parents, reported less family cohesiveness, and reported more family conflict. Therefore, all subsequent models included these variables as controls.

The Effect of Early Intoxication on Alcohol Use Trajectories

Hypothesis 1 considers the relationship between early intoxication and alcohol use from age 16 to age 18. To assess the unique effect of early intoxication status, we regressed the growth parameters for the binary and continuous growth models (i.e., the intercepts centered at age 16 and slopes) on early intoxication and the potential confounders. First consider the binary growth model for alcohol use. The results indicate that after adjusting for relevant covariates, the log odds of use were significantly higher at age 16 among adolescents who were intoxicated by age 14 (b = 2.39, SE = .63, p < .01). The effect of early intoxication on the rate of change (i.e., binary slope) in the probability of use is not statistically significant, indicating that early onset intoxication did not significantly influence the rate of change in the probability of use over time (b = -.40, SE = .40, p > .05). By varying the centering point of the intercept, we can directly assess the effect of early onset intoxication on the log odds

of use at each age (after adjusting for the potential confounders). Using this method, we find that the log odds of use were significantly higher for early onset adolescents at age 17 (b = 1.97, SE = .44, p < .01) and age 18 (b = 1.54, SE = .54, p < .01).

Next consider the continuous growth model for alcohol use. Congruent with the binary growth model, the unique effect of early intoxication on level of alcohol use at age 16 is statistically significant. Early onset adolescents used more alcohol than non-early onset adolescents at age 16 (b = 1.39, SE = .28, p < .01). Also similar to the binary growth model, the effect of early intoxication on the rate of change (i.e., continuous slope) in the frequency of use is not statistically significant (b = -.32, SE = .18, p > .05). We varied the centering point of the intercept to directly assess the effect of early intoxication on frequency of use at each age (after adjusting for confounders). Results of the models indicate that early onset adolescents used more alcohol at each subsequent age: age 17 (b = 1.08, SE = .24, p < .01) and age 18 (b = .76, SE = .32, p < .05).

Of all the estimates for the potential confounders, only two were statistically significant. First, girls had a lower intercept (when centered at age 16) for frequency of alcohol use (b = -.82, SE = .28, p < .01), indicating that female users used less alcohol than male users. Second, family conflict had a negative effect on the slope of the frequency of alcohol use model (b = -.13, SE = .05, p < .05), indicating that higher conflict was associated with a shallower increase in the frequency of use from age 16 to age 18.

To give a clearer description of the effects, Figure 1 presents the marginalized probability estimates (Hedeker & Gibbons, 2006) for the probability of use (top plot) and the frequency of use estimates conditional on some use (bottom plot) as a function of early intoxication status (holding all potential confounders at their mean). First consider the top plot. At age 16, those with an early intoxication debut had a much higher probability of use, and while the gap between early and non-early adolescents decreased over time, a substantial difference in the probability of use remained at age 18. On the bottom plot, the continuous growth models as a function of early intoxication status are presented. Again we see that youth who were intoxicated by age 14 used more alcohol at age 16 than the non-early adolescents, and their frequency of use, on average, remained high throughout late adolescence. Similar to the probability of use plot, the non-early adolescence, but those who had been intoxicated by age 14 remained significantly heavier users at age 18.

The Effect of Early Intoxication on Alcohol Problem Trajectories

For hypothesis 2, the same set of analyses were conducted to assess the relationship between early intoxication and alcohol problems from age 16 to age 18. We regressed the growth parameters for the binary and continuous growth models (i.e., the intercepts and slopes) on early intoxication debut and the potential confounders. First consider the binary growth model. The results indicate that after adjusting for potential confounders, the log odds of reporting alcohol problems were significantly higher at age 16 among adolescents who had been intoxicated by age 14 (b = 3.11, SE = .85, p < .01). The effect of early intoxication on the rate of change (i.e., binary slope) in the probability of problems is not statistically significant (b = -.63, SE = .56, p > .05). However, by varying the centering of the intercept,

we can directly assess the effect of early onset intoxication on the log odds of experiencing one or more problems at each age (after adjusting for confounders). Using this method, we find that the log odds of problems were significantly higher for youth who had been drunk by age 14 at age 17 (b = 2.49, SE = .61, p < .01) and age 18 (b = 1.80, SE = .77, p < .05).

Next consider the continuous growth model for alcohol problems. Congruent with the binary growth model, the unique effect of early intoxication on frequency of problems at age 16 is statistically significant. Adolescents who experienced their first intoxication by age 14 reported more alcohol-related problems at age 16 (b = 1.04, SE = .19, p < .01). The effect of early intoxication on the rate of change (i.e., continuous slope) in the frequency of problems is not statistically significant (b = -.22, SE = .12, p > .05). Those who had been drunk by age 14 continued to experience more problems than later debut adolescents at each subsequent age: age 17 (b = .82, SE = .15, p < .01) and age 18 (b = .61, SE = .19, p < .01).

Of all the estimates for the potential confounders, only one was statistically significant. Girls had a lower intercept (when centered at age 16) for frequency of alcohol problems (b = -.36, SE = .17, p < .05), indicating that female users experienced fewer problems than male users.

Figure 2 depicts the effect, using the same methodology as described for Figure 1. The figure shows that youth who experienced their first intoxication by age 14 were more likely to experience problems (top plot) and experience a greater frequency of problems (bottom plot) throughout late adolescence as compared to those who hadn't been drunk by age 14.

The Effect of Early Intoxication on Alcohol Abuse or Dependence by the Transition to Adulthood

We now turn to the final hypothesis, which considers the effect of early intoxication on the odds of a lifetime diagnosis of alcohol abuse or dependence by the transition to adulthood (the final wave). Since lifetime diagnosis is assessed here as a single binary indicator (lifetime diagnosis = 1, no lifetime diagnosis = 0) rather than a growth model, the logistic regression estimate is expressed in terms of the log odds of a diagnosis. The effect of early intoxication on a positive diagnosis is significant. Adjusting for the confounders, the odds of a diagnosis are about three times higher for adolescents with an early intoxication debut (OR = 3.12, 95% CI = 1.59, 6.12, p < .01). Holding all other covariates at their mean in the sample, the predicted probability of a positive diagnosis among youth who experienced their first intoxication by age 14 is .40 while the predicted probability of a positive diagnosis among youth who had not yet been drunk through age 14 is .17. In other words, about 40% of the adolescents with an early intoxication debut went on to test positive for an alcohol abuse or dependence diagnosis, while about 17% of the adolescents who hadn't been drunk through age 14 went on to test positive for an alcohol abuse or dependence diagnosis. Gender was the only significant potential confounder (OR = .51, 95% CI = .27, .98, p < .05), indicating that the odds of diagnosis were about 49% lower for young women than for young men.

Discussion

While epidemiology estimates over the last decade show that alcohol abuse is a serious problem for American Indians, nearly all of the research on alcohol abuse has involved Indians living on reservations or served by Indian Health Services where reservation based Indians are large parts of the clientele. This study involves urban Indians only. They are from a number of different tribes, most of them located in the Northwest coastal areas near Seattle, although a few are from far flung tribes across the country. While epidemiological data are not available on alcohol problems in these specific tribes, the parents of the youth involved in this study show high rates of alcohol problems, 40% of the fathers and 37% of the mothers qualified for a lifetime DSM-III-R diagnosis of alcohol abuse or dependence at the wave 5 interview. In 19% of the families, both father and mother qualified for abuse or dependence. As such, this study provides a unique look at adolescent alcohol trajectories among a population that is at risk but not often studied.

As with other Americans (Zucker et al., 1995), alcohol use among American Indians is largely initiated and to a considerable extent exacerbated during adolescence. This study explores the role played by early first intoxication on later alcohol use and alcohol problems. As described in the introduction, the existence of a relationship between early onset alcohol use and the subsequent onset of alcohol abuse and dependence, has been supported in studies of the more general population (Grant & Dawson, 1997), but has received only minimal examination among American Indians. Furthermore, other major consequences of early onset alcohol use such as increased subsequent drinking and greater incidence of alcohol related problems have received even less attention in samples of American Indian adolescents.

Several primary hypotheses were tested and confirmed. When compared to participants who hadn't yet been drunk through age 14, individuals who experienced their first intoxication early (by age 14) used more alcohol throughout late adolescence, experienced more alcohol-related consequences throughout late adolescence, and were more likely to have an alcohol use disorder (abuse or dependence) by the final wave of data collection (mean age 19, range 17 to 21 years). Perhaps most striking, about 40% of the adolescents with an early intoxication debut, compared to about 17% of adolescents who hadn't been drunk through age 14, went on to test positive for an alcohol abuse or dependence diagnosis (holding potential confounders at their mean). Indeed, the relationship between early onset intoxication and subsequent ill outcomes throughout late adolescence and into the transition to young adulthood is robust in this sample.

The study also found that a number of different factors were significantly related to early intoxication. Children who experienced their first intoxication by age 14 were also more likely to demonstrate early delinquency (as measured by the CBCL), more likely to have a biological parent with an alcohol abuse or dependence diagnosis, to live in poverty, to not live with both biological parents, to have higher family-based conflict, and to have less family-based cohesiveness. All of these findings would be expected, since these factors have been shown to relate to alcohol and drug problems among other youth and prior research with Indian youth has suggested that there are likely to be similar correlates of alcohol and

drug problems (Beauvais & Oetting, 2002). Even after all of these factors are taken into account, though, early onset intoxication is still highly related to later alcohol use, later alcohol problems, and the likelihood of developing an alcohol abuse or dependence disorder. In fact, controlling for early intoxication, very few of the potential confounders are related to subsequent alcohol outcomes.

This is a very important finding. Jessor and Jessor (1977) describe problem prone behavior in adolescents. The description relates to a group of adolescents who are likely to engage in multiple behaviors and have a multiplicity of problems that are related to deviance, alcohol use, and use of other drugs. That problem prone pattern likely exists within this group of urban Indian youth; the correlations in Table 1 show that early onset of alcohol intoxication is related to multiple family problems and early signs of delinquency. But the results show that, even after these factors are taken into account, early onset intoxication still predicts subsequent alcohol outcomes. That is, regardless of these risk factors, children who experience their first intoxication early have an increased likelihood of demonstrating problematic trajectories of alcohol involvement during late adolescence and into the transition to adulthood.

Limitations

Before considering implications for prevention, it is important to recognize that the results of this study are not without limitations. First, data collection spanned only nine years so that no data are available past the participants' transition into young adulthood. Therefore, this study cannot speak to effects of early onset intoxication past the transition to adulthood.

Second, the sample was limited to American Indian youth from the King County statistical metropolitan area. This raises valid questions regarding the generalizability of the study's findings to urban American Indians living in other regions of the United States. Spicer, Novins, Mitchell, and Beals (2003) compared the use of alcohol by adolescents from four major American Indian cultural groups ("Northern Plains, Southeast, Southwest-1" and "Southwest-2") using data from the Voices of Indian Teens Project, and found that group membership accounted for an extremely small percentage of the variance in alcohol use behaviors, and ceased to be significant when other factors were added to their model. Nonetheless, it cannot be assumed that there are no cultural differences in the onset of alcohol dependency or the experience of alcohol related problems. For example, O'Neill and Mitchell (1996) argue that what is considered a "problem" within one cultural group may not be perceived as a problem for another.

Third, intoxication ("drank enough so that it was hard to walk, talk, remember or decide what to do") used as the measure of onset for this study is a perception. Mail (1995) reported that American Indian elementary age Indian youth mimic drunken behavior with little or no alcohol intake. However, the validity of the concept of drunkenness has been demonstrated to be highly correlated with estimated blood alcohol levels in 14-year-old Finnish adolescents (Linton & Rimpella, 2001). The concept is also likely to be clearly conceptualized in 14-year-old American Indian adolescents, as supported by adverse outcomes that include binge drinking (five or more drinks) and alcohol use disorders.

Fourth, this study focused on *urban* American Indian adolescents. It may be that age of alcohol use onset has different consequences for rural American Indian adolescents or those living on reservations. The results of the study show very powerful and logical effects of early exposure and similar effects seem likely to be present in other groups as well.

Fifth there is no comparison group, for example, non-American Indian students from the same region. However, given the limited studies on American-Indian adolescents and the benefit of conducting within ethnic group studies (Graham, 1994), we believe that this manuscript makes a substantial contribution to the literature.

Finally, there was a small amount of missing data across the longitudinal assessments for the variables considered in this study. However, we employed appropriate missing data strategies to account for the missing values.

Implications for Prevention

Although causation cannot be implied based on these analyses, the results of this study may suggest that interventions which successfully delay first intoxication of alcohol use could have considerable benefit. On average, those children who hadn't been drunk through age 14 did not develop the level of problems that were apparent among those with early intoxication exposure. It is possible that simply delaying onset of intoxication will reduce the amount of heavy drinking during adolescence, the number of alcohol use disorder by the transition to young adulthood. Although the prospective nature of this study and the partialing out of many of the well known correlates of alcohol problems suggests there may be a causative relationship, determining whether the effect of delaying onset actually *causes* less drinking, fewer alcohol-related consequences, and a lower probability of abuse/dependence is critically important work for future intervention studies.

This study also found that there are a cluster of family, social, and economic problems that are associated with early drinking. Basic prevention programs should consider these issues, and prevention might start with attempting to reduce the effects of poverty, prejudice, family disruption, and family conflict. Culturally appropriate interventions that attempt to improve family status and parenting in disadvantaged Indian families may be particularly appropriate, since most Indian cultures include a strong emphasis on the family. But this study also makes it clear that, even after these factors are considered, early intoxication, in and of itself, may be a critical factor in producing later problems. Prevention programs aimed directly at delaying first intoxication in the urban setting should probably be general programs that do not specifically target Indian youth, since targeted programs may encourage already existing strong stereotypes about Indian alcohol use. They should, typically, be aimed at and available to all students, and not use Indian images, icons, or constructs, although Indian youth should certainly be included in all illustrative materials, as should other ethnic groups in the population. However, if they are to be effective with Indian youth, the programs must be congruent with tribal cultural values and beliefs and should have the approval and active support of tribal leaders and community leaders. This approach is consistent with community based participatory research (Israel, Eng, Schulz, & Parker, 2005).

It may be possible to establish targeted programs on nearby Indian reservations. Since many of these urban Indian families still have strong ties to nearby reservations, and family relationships and friendships are likely to involve people on the reservations, programs aimed at Indian youth on those reservations may also have an influence on these urban adolescents. Reservation-based programs should have strong cultural aspects and content and be viewed as culturally congruent and appropriate by tribal leaders. While cultural congruence is essential, on-reservation programs cannot, however, consist only of attempts to increase identification with Indian culture. Cultural identification, by itself, does not protect against substance abuse, although it does tend to support the family and increase the family's influence on a child's behaviors (Beauvais, Jumper-Thurman & Plested, 2002; Brady, 1995).

In summary, this study points to the potential benefit of delaying the onset of first intoxication among American Indian youth. Based on the series of studies that indicate the importance of delayed onset among youth in general, the U.S. Surgeon General indicated that preventing early alcohol use is a public health priority (U.S. Department of Health and Human Services, 2007). The results presented in this paper contribute to the literature by demonstrating that early onset intoxication has similar deleterious associations among urban American Indian youth specifically. Further work is needed to determine if programs designed to delay the onset of alcohol use among American Indian youth could result in improved developmental outcomes during the course of adolescence and into adulthood.

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Figure 1.

Two-part growth model for alcohol use as a function of early intoxication status (holding potential confounders at their mean).

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Figure 2.

Two-part growth model for alcohol problems as a function of early intoxication status (holding potential confounders at their mean).

Table 1

Potential Confounders as a Function of Early Onset Status

	Early		Not early	
	Mean	SD	Mean	SD
Female	0.48		0.54	
CBCL - delinquency scale	0.25 **	0.26	0.15	0.20
Family history of alcohol abuse/dependence	0.78 **		0.51	
Family lives at or below poverty line	0.43*		0.30	
Adolescent lives with both biological parents	0.20**		0.41	
Family cohesiveness	6.66*	2.13	7.23	1.69
Family conflict	3.22**	1.98	2.43	1.85

Note. Logistic regression was used to compare dichotomous outcomes and t-tests were used to compare continuous outcomes across early onset status. Variables without a standard deviation (*SD*) are dichotomous.

* Significantly different from not early group, p < .05.

** Significantly different from not early group, p < .01.