

The Effects of Parental Diagnosis and Changing Family Norms on Alcohol Use and Related Problems among Urban American Indian Adolescents

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This study investigated the role of parental diagnosis of alcohol abuse/dependence and perceived family norms for adolescent drinking on alcohol use and alcohol-related problems among urban American Indian youth. A total of 251 urban, American Indian youth and their parents/caregivers were followed from ages 13 to 18. Perceived family norms against alcohol decreased and alcohol use increased from ages 13 to 18. Relative to no parental diagnosis, youth with one or two parents diagnosed with alcohol abuse/dependence were less likely to perceive family norms against alcohol use. Youth with two parents diagnosed were more likely to report alcohol-related problems at age 18 compared to no parental diagnosis. Faster rates of decrease in perceived family norms against alcohol use were associated with faster increases in alcohol use over time. Higher rates of perceived family norms against alcohol use protected youth from high rates of use at age 13, but higher rates of alcohol use at age 13 predicted more alcohol-related problems at age 18. These results suggest that both family history and family behaviors in the form of communication of norms for adolescent alcohol use are likely to impact both rates of use and eventual alcohol-related problems. (Am J Addict 2011;20:212–219)

Damaging outcomes of alcohol use among American Indians are well documented for adult^{1,2} and adolescent populations.³ Among adults, Spicer et al.⁴ reported rates of alcohol dependence that were 50% higher for American Indian men compared to men in the National Comorbidity Survey. Alcohol and other drug use are among three leading causes of death among American Indian youth,⁵ and alcohol dependence is the most commonly diagnosed psychiatric condition.⁶ American Indian youth initiate alcohol

earlier compared to other ethnic/racial groups,⁷ and their rates of use are consistently higher compared to other ethnic/racial groups.^{8–10} Among 651 indigenous adolescents from a single cultural group spanning the United States and Canada, Whitbeck et al.¹¹ found that lifetime rates of substance use disorders were three times higher than rates reported in the 2004 National Survey on Drug Use and Health.

Given the high rates of alcohol and other drug use among both adult and adolescent populations among American Indians, this raises key questions regarding genetic susceptibility,¹² as well as the communication of attitudes and behaviors related to substance use^{13,14} through processes of familial influence. It is clear that in families in which one or both parents are heavy drinkers, risk is increased for alcohol use in offspring.^{15,16} Parental alcoholism increases risk for early onset drinking and trajectories of persistent alcohol use among offspring.^{17–19} Furthermore, the increased risk of alcoholism in offspring of drinking parents is four to nine times for males and two to three times for females.²⁰ The effects of parental alcoholism can be partially explained due to genetic influences,²¹ and there is evidence from twin studies that genetic influences are substantial in both non-Indian²² and Indian populations.¹² However, there is also support for environmental influences after control for genetic effects.²³

Consistent with social cognitive theory,²⁴ the environmental effects of parental alcoholism occur through social influence processes such as modeling of drinking behavior and the establishment of family norms for drinking. Various studies have found that families exert strong influences on offspring through these social influence processes.²⁵ Among 7th and 8th grade students in the Northeast, Callas et al.²⁶ found that after control for smoking and marijuana use, alcohol expectancies, peer norms, and family norms contributed independently to the prediction

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of alcohol use in the last month. In this study, norms referred to both peer and family attitudes toward drinking. In a similar study among 5th grade students, after control for sex and prior exposure to parental modeling of alcohol, the odds of frequency of alcohol use increased for those youth whose parents allowed them to try alcohol.²⁷ Sieving et al.²⁸ reported on results from Project Northland in which parental norms for adolescent alcohol use were the strongest prospective predictor of alcohol use for 7th and 8th grade students, compared to other parent variables.

While these studies indicate that parental socialization influences are potent predictors of alcohol and drug use among the general population of youth, similar findings have been reported for American Indian youth. Among 11th and 12th grade Plains and southwest American Indian students, family sanctions against substance use were related to levels of use in the students.²⁹ A disturbing report of the direct effects of modeling was reported by Mail¹³ in which reservation-based American Indian elementary school children reported mimicking the drunken behavior of reservation adults. Among urban, public school American Indian adolescents, family modeling of substance use predicted age of initiation after controlling for key covariates.³⁰ However, contrary findings were reported among another sample of reservation-based 6th through 12th grade students for whom parental disapproval of adolescent alcohol use was unrelated to their levels of use.³¹

THE CURRENT STUDY

Nearly all investigations of parental influences on drinking have utilized either cross-sectional or longitudinal data that treated effects as constant over time. Newer methods, however, allow for predictors and covariates to change over time. Using a parallel process latent growth curve model, Mitchell, Beals, and Kaufman³² demonstrated among a sample of American Indian high school students that, in addition to alcohol use, positive expectancies (positive attitudes regarding use and effects of alcohol) exhibited growth over time, but a lower risk group of students demonstrated slower rates of growth in these variables. The current study focuses on the specific influences of parental diagnosis of alcohol abuse/dependence and parental norms for alcohol use, on the alcohol use and alcohol-related problems of offspring among an urban sample of American Indian youth.

The great majority of investigations of American Indian youth have been conducted on reservation-based samples, and few of these have contained multiple observations over time. The current study is unique in that it was based on observations of urban American Indian youth, with multiple observations from ages 13 to 18, a period of time during which alcohol use and alcohol-related problems are likely to escalate.^{33,34} Use of simultaneous latent growth modeling was employed to consider changes over this 5-year span

in both perceived norms against alcohol use, and number of days alcohol was used in the past month. Number of biological parents diagnosed in their lifetime with alcohol abuse/dependence was dummy coded in order to consider the differential effects of having one or two parents diagnosed with alcohol abuse/dependence, relative to no parent diagnosis. Alcohol-related problems at age 18 (Rutgers Alcohol Problems Index [RAPI]) were treated as a model outcome.

The model accounts for several factors. First, by considering the number of parents with a lifetime diagnosis of alcohol abuse or dependence, genetic influences based on first-degree relatives are considered. However, this variable also captures, to some degree, social influences that are likely to occur based on parental modeling of drinking behaviors and other observational learning. In addition to these social influences, the specific effect of perceived changes in parental/caretaker attitudes toward drinking by offspring was captured based on the growth parameters for family norms for alcohol use. It should be noted that we did not measure actual parent-child communications regarding alcohol use. It can be argued, however, that perceived norms are as influential, and perhaps more so than actual parental behavior. Perceived parental norms among adolescents have been demonstrated to be a potent predictor of adolescent use,³⁵ even after control for actual norms.³⁶

The following hypotheses were evaluated. It was predicted that one and two parents diagnosed with alcohol abuse/dependence would predict lower family sanctions against alcohol use (FMNORM) at initial status and a faster rate of decline in this variable. It was also predicted that parental diagnosis would be associated with higher number of days alcohol was used in the last month (ALC30) at initial status and a faster rate of increase. It was further hypothesized that higher FMNORM at initial status would predict lower alcohol use at initial status and a slower rate of increase in ALC30, and that a faster rate of decrease in FMNORM would be associated with a faster rate of increase in ALC30. Finally, initial status and rate of change in ALC30 was hypothesized to predict more alcohol-related problems at age 18 (RAPIT8).

METHODS

Dataset and Subjects

This study is based on secondary analysis of substance use and related data obtained from 1988–1989 through 1996–1997 among an urban sample of American Indian adolescents, by Walker and Silk-Walker. Several American Indian groups reviewed and approved all procedures for the original study. This included the Seattle Indian Health Board, Seattle School Districts, and the Internal Review Boards of all involved agencies. Furthermore, many members of the Indian community-at-large aided the efforts of this project including extended family members and

various agency contacts. In addition, both the Principal Investigator and Co-Principal Investigator of this study are of American Indian descent (Cherokee). Thus, this study received substantial input from the local American Indian community. Further information regarding the original study can be obtained in Walker et al.³⁷ The current analysis focused on the growth of alcohol use from time 3 (age 13) to time 8 (age 18) and its relationship to other model variables. The overall sample (time 1 through time 9) began with recruitment of a sample of 11-year-old American Indian youth from the Seattle metropolitan area. Student ethnic identity, for recruitment, was provided by parents/legal guardians on Department of Education Form 508 (Indian Student Certification). Ethnicity was validated with data from a number of parameters of tribal heritage, citizenship status, cultural identity, and cultural activity participation.

The current sample consisted of participants from the combined first and second cohort. The first of these cohorts ($n = 224$) was recruited from schools in the Seattle area with high American Indian enrollments, and the second cohort ($n = 66$) was recruited from the roster of age appropriate (11–12 years old) American Indian patients of the urban Indian clinic. This clinic provided general health services; enrollment was not necessarily related to a substance use disorder by either parents or youth. In order to limit this study to urban youth, only adolescents whose home address was located in the Seattle metropolitan area were used, resulting in a sample size of 251 for this analysis. In addition to data obtained from the youth, collateral data were obtained from parents/guardians at each time point. Personalized introductory letters inviting participation in the study were mailed to the parents/careproviders of eligible students. The letter explained how students' names were obtained and that the University of Washington Human Subjects Review Committee had reviewed and approved the research protocol.

Assessment Procedures

Youth and parents/caretakers participated in annual, face-to-face interviews conducted by trained research personnel. The majority of the interviewers were of American Indian descent. Following time 1, a small percentage of participants who no longer resided in the immediate area completed interviews by telephone. Face-to-face assessments of youth and parents/guardians were conducted separately in private rooms. The majority (60%) of interviewers were American Indian, interviews averaged 2.5 hours, and participants were remunerated for their time.

Instrument

As part of an extensive assessment battery, the current study focused on four variables: (1) number of biological parents with an alcohol abuse/dependence diagnosis, (2) family norms against adolescent alcohol use (FMNORM), (3) number of days alcohol used in last month (ALC30), and

(4) number of alcohol-related problems (RAPIT8). These measures and others similar to them have been used successfully among both minority and nonminority populations of adolescents, including American Indians.^{38–40} FMNORM and ALC30 were each measured on six occasions over a period of 5 years. Parental diagnosis of any lifetime alcohol abuse or dependence of the target adolescent was obtained at time 5. Biological parents interviewed as collaterals provided self-report, using the Semi-Structured Assessment for the Genetics of Alcoholism diagnostic instrument,⁴¹ which was also collected at time 5. Data for the noncollateral parent were provided by the collateral adult, using the Family History Assessment Module alcohol module data, developed by NIAAA's COGA study.⁴² Parental diagnosis was dummy coded with no diagnosis by either parent serving as the referent. FMNORM was measured using a three-item scale administered to the youth. The scale measured the youth's perceived parental/caretaker norms regarding adolescent alcohol use. Response categories ranged from 0 to 3 (0 = no, 1 = not much, 2 = some, 3 = a lot) and items asked, "Would your family care if you drink?", "Would your family stop you from drinking?", and "Would your family stop you from getting drunk?". Cronbach alpha reliabilities from time 3 through time 8 ranged from .87 to .91. ALC30 was based on the self-reported number of days youth reported using alcohol in the last month, ranging from 0 to 30. RAPIT8 was measured at time 8 using the RAPI.⁴³ The standard 23-item version asks respondents to report the number of times in their lifetime that specific events occurred while drinking alcohol. With four response categories ranging from 0 to 3, scores range from 0 to 69 with higher scores reflecting more alcohol-related problems. For this study, respondents were asked at time 8 to report the number of alcohol-related problems in the previous 12 months. Therefore, this variable reflected the number of current alcohol-related problems at age 18. Due to the nonnormality of FMNORM, ALC30, and RAPIT8, these variables were log transformed. FMNORM was negatively skewed and was first reflected before the log transformation was conducted. It was then rereflected following transformation.

Data Analysis

The primary method of analysis was parallel process latent growth modeling (LGM) within a structural equation modeling framework.⁴⁴ This method allows for the simultaneous estimation of growth parameters (initial status, rate of change) of multiple latent variables, as well as consideration of the effects they may have on each other. In addition, manifest antecedent and outcome variables can be specified. In the current study, parental diagnosis was included as an exogenous antecedent variable, and RAPIT8 was treated as an outcome variable.

The first step in the LGM process was the assessment of the nature of growth for each latent variable

individually. First, an unconditional LGM model was specified for FMNORM and ALC30, with the repeated measures from time 3 through time 8 serving as indicators. This time period that corresponds to age 13 to age 18 was selected due to the changes in adolescent alcohol use that occur during this developmental period. Growth was estimated as a linear function, comprised of two parameters including the intercept (initial status) and the rate of change (linear slope). Factor loadings for the intercept were fixed at 1 and equally spaced factor loadings (0, 1, 2, 3, 4, 5) identified a pattern of linear growth. This process was repeated for the six repeated measures for ALC30 using the same assumptions for the growth parameters. All models were estimated with EQS6.1,⁴⁵ using robust estimation. Adequacy of model fit was assessed using the comparative fit index (CFI) and the root mean square error of approximation (RMSEA). Once the individual models were adequately estimated, the latent variables were estimated simultaneously, with inclusion of parental diagnosis as an exogenous antecedent, and RAPIT8 as an outcome variable.

Missing Data

The frequency of missing data was relatively small for most model variables, particularly given the length of the original study that spanned 9 years. Follow-up rates were 94–95% for participants included in the current analysis. While there was some mobility of participants from urban to reservation locations, this occurred relatively infrequently. The persistent efforts to locate and interview participants over the 9-year period of the entire original grant were responsible for the high retention rates of the overall study. For time 3 through time 8 FMNORM and ALC30, the frequency of missing data ranged from 2.37% to 5.93%. For parental diagnosis, 8.7% of data were missing. The largest percentage of missing data was for the 23 RAPIT8 items, with percentages ranging from 29.64% to 30.04%. Missing data were handled using maximum likelihood estimators, available in EQS6.1. With the inclusion of robust estimation, model parameters and standard errors are correctly estimated with this method.⁴⁶

RESULTS

Descriptive Data

The sample was evenly distributed by sex with 127 participants (50.6%) self-identifying as male. The mean age

of students at time 3 was 13.7 years and the mean age at time 8 was 18.6 years. A total of 34% of families reported no lifetime diagnosis of alcohol abuse or dependence, 39% reported one parent having a lifetime diagnosis, and 18% reported both parents having a diagnosis, with 9% missing. The mean RAPIT8 score was 5.704 with a range from 0 to 55 and a standard deviation of 8.069. The means and standard deviations for repeated measures model variables are presented in Table 1. As shown in the table, FMNORM gradually decreased over time, while ALC30 increased.

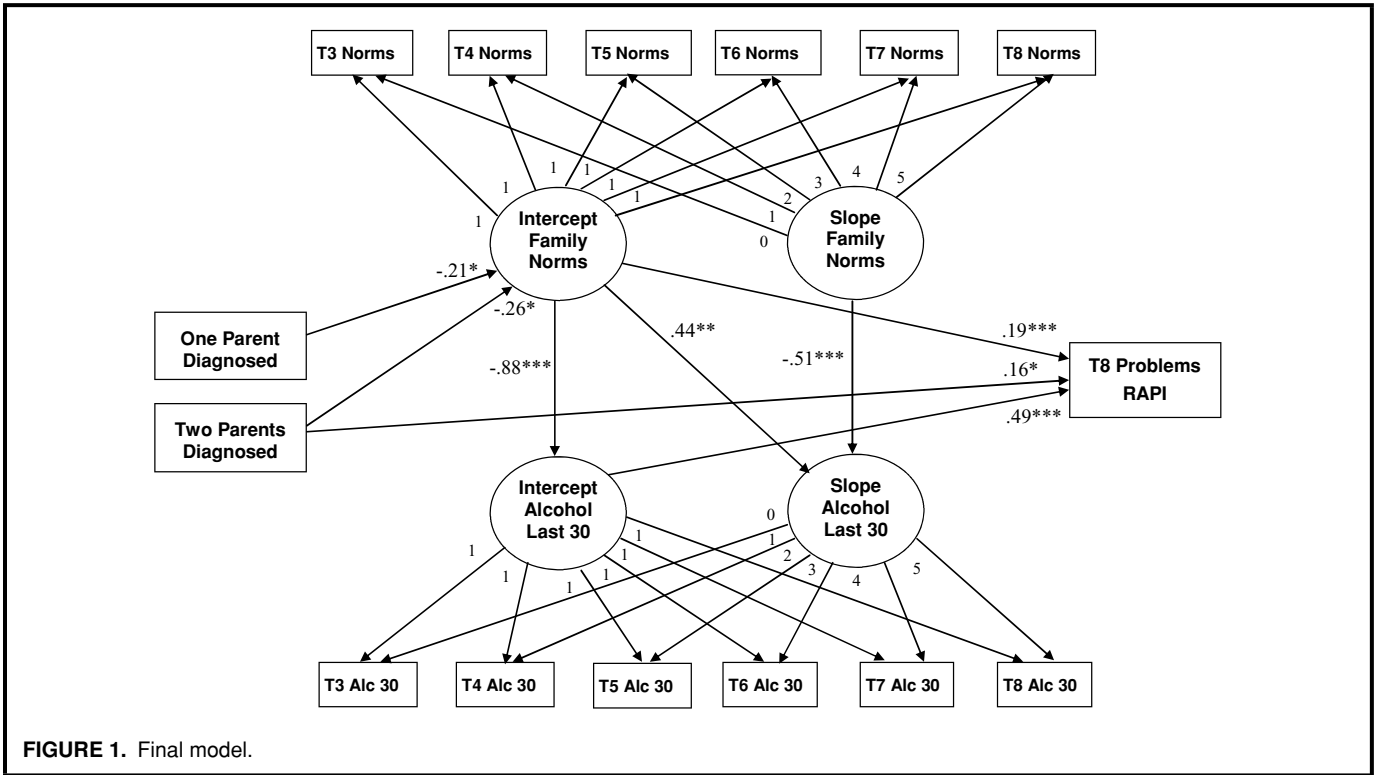
Latent Growth Models

Individual, unconditional growth models specified for each repeated measures variable (FMNORM, ALC30), with parameters for the intercept and linear growth resulted in adequate fit. For FMNORM, the model resulted in a Satorra-Bentler chi-square of 38.73 ($df = 16, p = .001$). The CFI was .89 and the RMSEA was .07. This process was repeated for ALC30. The Satorra-Bentler chi-square was 32.02 ($df = 16, p = .01$). The CFI was .91 and the RMSEA was .07.

The final step was to include both growth factors in a simultaneous LGM and to include parental diagnosis as an exogenous predictor variable and RAPIT8 as an outcome variable. The final model is depicted in Figure 1. Parameter estimates in the model are from the standardized solution. The hypotheses based on the effects of parental diagnosis were partially supported. As predicted, parental diagnosis was negatively related to initial status of family norms. Youth who had one or two parents diagnosed with alcohol abuse/dependence were more likely to report lower parental sanctions against alcohol use at age 13. Contrary to our hypotheses, parental diagnosis was not related to the rate of change in FMNORM, nor to the initial status or rate of change in ALC30. One unexpected finding was the direct effect of two parents diagnosed on RAPIT8. Relative to having no parents diagnosed, a family in which both parents had been diagnosed increased risk for alcohol-related problems at age 18. As predicted, the initial status of FMNORM at age 13 was significantly related to the initial status and rate of change of ALC30. Youth who perceived strong family sanctions against alcohol use were less likely to be using alcohol at age 13. The positive relationship between family norms at age 13 and the rate of change in alcohol use was positive. The hypothesis that rate of change of FMNORM would be associated with the rate of change

TABLE 1. Means and (standard deviations) by time for perceived family norms and alcohol use last 30 days

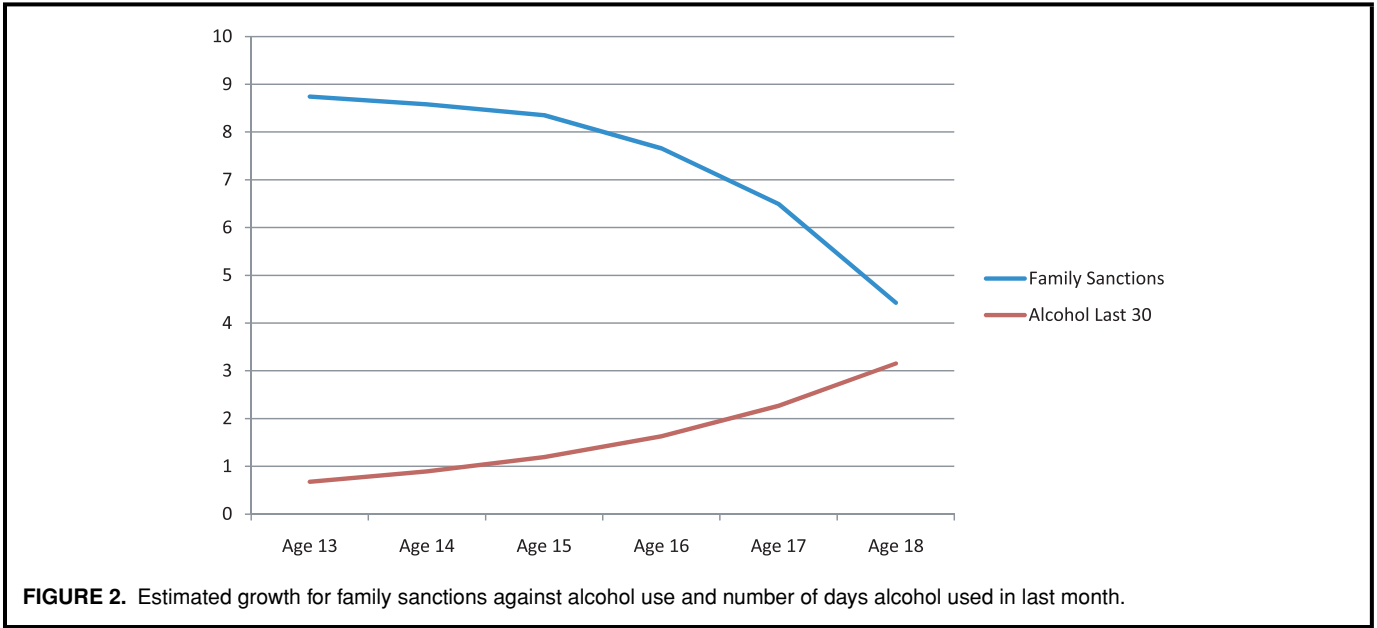
Variable	Time 3	Time 4	Time 5	Time 6	Time 7	Time 8
Perceived family norms	8.584 (1.352)	8.363 (1.577)	8.130 (1.783)	7.696 (2.161)	7.142 (2.584)	6.413 (2.814)
Number of days alcohol used last month	0.630 (2.629)	0.809 (2.264)	1.083 (2.711)	1.421 (3.349)	1.678 (3.714)	2.335 (4.211)



for ALC30 was supported. A faster rate of decline in perceived norms was associated with a faster rate of increase in alcohol use. An additional unexpected finding was the direct relationship between initial status of FMNORM at age 13 and RAPIT8. The initial status of ALC30 was significantly related to RAPIT8 as hypothesized, but rate of change was not. Fit of the final model depicted in Fig. 1 was

satisfactory. The Satorra-Bentler chi-square was 148.96 ($df = 00, p < .001$). The CFI was .91 and the RMSEA was .049.

Estimates of the growth parameters for FMNORM and ALC30 from the unconditional growth models are presented in Fig. 2. The initial status and the growth in each variable are depicted over time from ages 13 to 18. It should



be noted that while a single ordinate is used, the two variables were measured on different scales as described earlier. The predicted mean initial status was 8.58 for FMNORM. The original variable ranged from 0 to 9. Thus, perceived family norms against alcohol use were very high at age 13. The predicted mean initial status was .528 for ALC30. A large percentage of youth at age 13 reported no use of alcohol in the last 30 days. The linear pattern of both variables indicates a gradual decline in perceived family norms, and a gradual increase in the number of days alcohol was used over the 5-year period.

DISCUSSION

As expected, and consistent with prior research among non-Indian and American Indian youth, use of alcohol as measured by number of days used in the last month, increased over time from ages 13 to 18.^{3,33,34} Early adolescence for this sample of urban Indian youth was a key period in the escalation of alcohol use. While a number of youth reported no use of alcohol in the previous 30 days at age 13, mean number of days increased throughout the 5-year period. This is consistent with results reported by Mitchell et al.³² who reported growth for alcohol use among a reservation-based sample of Indian youth. However, the Mitchell et al. study spanned a longer time frame from age 14 through age 24, with results that indicated rates of use began to decline at age 20. It would appear from this study and the Mitchell results that the pattern of growth among Indian adolescents is characterized by increasing levels of use from ages 13 to 20. This analysis of urban youth did not extend beyond age 18, so we cannot predict whether the increase of use observed to age 18 would be followed by a period of deceleration as found by Mitchell et al. It would appear that the characteristics of growth in alcohol use among urban- and reservation-based Indian youth share similar features.

Concurrent with increasing alcohol use was a corresponding decrease in perceived family norms against alcohol use. While it is tempting to conclude this was due to a causal process whereby decreased family norms resulted directly in increased alcohol use, the relationship reported here could be spurious, caused by a third variable, either unmeasured or not included in the current analysis. We can conclude, however, that important changes in both the perceptions of family attitudes and alcohol use behaviors occurred from ages 13 to 18.

The hypotheses related to parental diagnosis were only partially confirmed. The results suggest that among urban American Indian youth, the effects of family history for alcohol abuse/dependence would appear to have a strong social influence component. While direct effects from parental diagnosis to both the initial status and rate of change in alcohol use were expected, these effects were indirect through family norms. This suggests an additional social influence

component beyond the direct effects of parental diagnosis. This is in contrast to results reported by Duncan et al.⁴⁷ among a non-Indian sample in which higher levels of parental alcohol use related directly to higher initial status of alcohol use. Their model, however, did not include family norms, so it is not possible to determine whether this indirect relationship between parental diagnosis, family norms, and alcohol use is unique to our sample.

The effects of family norms on alcohol use were complex. Higher family norms against alcohol use at age 13 served as a potent buffer against early alcohol use. However, higher family norms at age 13 also predicted a faster rate of growth in alcohol use. This is likely due to a floor effect in which youth who were using little alcohol at age 13 would be more likely to increase use at a faster rate than those youth who were already using at higher rates. Another important effect of parental diagnosis was the relationship of two parental diagnoses on alcohol-related problems at age 18. Having one parent diagnosed did not result in more alcohol-related problems than having no parents diagnosed. However, risk for alcohol-related problems was higher in families with two parental diagnoses.

Consistent with our hypothesis, the initial status in alcohol use was associated with an increased number of alcohol-related problems at age 18. Those youth who reported higher rates of use at age 13 were more likely to report alcohol-related problems at age 18. This finding is supported by past research that shows early alcohol initiation as a key predictor of subsequent alcohol-related problems.^{48–50} However, the rate of change in alcohol use did not predict more alcohol-related problems. This is inconsistent with findings of others in investigations of non-Indian youth. For example, Colder et al.⁵¹ identified different classes of growth trajectories for adolescent alcohol use from grades 7 through 12. Those youth classified as rapid escalators (those whose drinking began relatively low in quantity and frequency, but increased rapidly) were most likely to experience alcohol-related problems. In addition, while not predicted, a direct relationship was found between parental diagnosis and alcohol-related problems. Specifically, those youth with two parents diagnosed with alcohol abuse/dependence were more likely to experience alcohol-related problems at age 18 than those youth with no parental diagnosis. We expected that parental diagnosis would be indirectly related to alcohol-related problems through both initial status and rate of change in alcohol use. However, there is prior support for a direct relationship between family drinking and alcohol-related problems. Warner et al.⁵⁰ found among a sample of 12-year-olds, followed over a 5-year period, that classification in a problem-drinking group compared to a no or low problem group was predicted by family history of alcoholism. The relationship we found between parental diagnosis and RAPIT8 was not strong. One potential explanation for this may be due to our inclusion of diagnoses both for alcohol abuse and dependence, rather than dependence alone.

In order to place the results in the appropriate context, several limitations must be considered. First, this sample was limited to urban American Indian youth. As noted earlier, this sample is unique in that it is one of very few longitudinal studies of American Indian youth. However, we were not able to directly compare our sample to a reservation-based sample of youth. The sample investigated by Mitchell et al.³² provides a useful comparison, but identical measures were not used in the two studies.

A second limitation relates to the measurement of family norms. Our measure was based on the perceived family norms as reported by the adolescents. Thus, it is possible that youths' perceptions of their family's attitudes toward adolescent drinking would not match the parent/caretaker report of family attitudes. However, Brody et al.¹⁵ demonstrated that the relationship between parental norms and adolescent drinking are mediated by the internalization of parental norms. Although we did not model this mediational process directly, we can conclude from Brody et al.'s results that a similar progression of effects may be active among urban Indian youth. Furthermore, other research has demonstrated that the perception of parental attitudes is a strong determinant of adolescent behavior in relation to risky behaviors.^{52,53} Even when there is a discrepancy between actual parental expectations and adolescent perceptions of those expectations, adolescents make decisions and act on their perceptions.

CONCLUSIONS

Many of the findings presented here replicate those of other studies. First, alcohol use rates increase throughout adolescence. Second, early use of alcohol leads to more alcohol-related problems later in adolescence. Third, at least at the younger ages, parental norms against alcohol use reduce levels of use. Fourth, there is a relationship between parental diagnosis of alcohol disorders and rates of alcohol-related problems at age 18. But in this sample of urban American Indian adolescents, effects of parental diagnosis on alcohol use of offspring were indirect through the effects of family norms.

While these findings are not new, they establish that urban Indian youth do not constitute a unique group of adolescents with respect to perceived parental norms and alcohol-related problems at age 18. From a prevention point of view, programs that have been found effective in reducing alcohol use in other populations may be effective with urban Indian youth. Research into the efficacy of evidence-based prevention methods in American Indian populations is indicated. These findings make it clear, as others have pointed out, that prevention must begin early in order to meet the needs of those at highest risk. Certainly prevention must begin before age 13 to delay the onset of use and adverse consequences associated with higher levels of alcohol use. It should also be noted that more intensive prevention efforts are indicated for those

youth whose parents have been diagnosed with alcohol problems.

Perhaps the most important finding here is the increasing disconnect between parental sanctions against alcohol use and the use of alcohol. While perceived family sanctions against alcohol use decrease during adolescence, alcohol use increases. It is well established that peers become important influencers during adolescence as the impact of families lessens. Despite this, investigations into new methods to prolong the protective effects of parental norms against alcohol use as long as possible are needed. The usual practice is to shift the responsibility for drug education and prevention to the schools, as children get older. Including Indian community families in prevention aimed at a delayed age of initiation for alcohol use and reduced levels of consumption may reduce morbidity and mortality among Indian youth. Such a family approach could capitalize on strong cultural values of caring and cohesion for at-risk youth and adults.

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Declaration of Interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

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