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Alcohol Use Trajectories and Problem Drinking Over the Course of Adolescence: A Study of North American Indigenous Youth and their Caretakers*

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Abstract

This study investigated the links between alcohol use trajectories and problem drinking (DSM-IV abuse/dependence) using five waves of data from 727 North American Indigenous adolescents between 10–17 years from eight reservations sharing a common language and culture. Growth mixture models linking fundamental causes, social stressors, support, and psychosocial pathways to problem drinking via alcohol use trajectories over the early life course were estimated. Results indicated that 20% of the adolescents began drinking at 11–12 years of age and that another 20% began drinking shortly thereafter. These early drinkers were at greatly elevated risk for problem drinking, as were those who began drinking at age 13. The etiological analysis revealed that stressors (e.g., perceived discrimination) directly and indirectly influenced early and problem alcohol use by decreasing positive school attitudes while increasing feelings of anger and perceived delinquent friendships. Girls were found to be at risk independently of these other factors.

Although alcohol abuse has been studied for decades in North American Indigenous (NAI) communities (for reviews see Beauvais 1998; Herring 1994) there have been no large etiological developmental trajectory studies from early experimentation to regular use over the early life course among NAI youth. While this type of research has been ongoing for other groups for years (e.g., Li, Duncan and Hops 2001), NAI adolescent alcohol use development has been largely overlooked even though numerous studies indicate that youth in some NAI communities initiate drinking and transition to regular alcohol use earlier than their counterparts in other ethnic groups (Bachman et al. 1991). To address this gap we use longitudinal growth mixture models (Muthén 2001, 2004) to study how the early transition to regular alcohol use is influenced by background factors and how this process leads to problem drinking in adolescence.

Because early drinking can have negative health consequences over the life course (e.g., Dewitt et al. 2000; Sartor et al. 2006), we begin by describing the timing and shape of alcohol use patterns among adolescents from ages 10–14 years using a unique longitudinal

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sample of NAI youth and their families. We then use individual trajectories to determine whether and the extent to which early drinking leads to problem drinking – measured using alcohol abuse and/or dependence (DSM-IV, lifetime) criteria – in late adolescence when the youth were 15–17 years. This first set of goals is essential for understanding the timing, patterns and consequences of drinking over the early life course in this NAI population.

Next, we estimate etiological models predicting early use trajectories and then situate these drinking trajectories into a mediational model of problem use. This etiological model is guided by overlapping risk and protection domains based on structural inequalities reflecting the fundamental causes of disease (Link and Phelan 1995), stress processes (e.g., Pearlin 1989), social supports (e.g., Turner 1999), and intermediate psychosocial developmental pathways (e.g., Agnew and White 1992). Thus, our second set of research goals involves building a developmental model of alcohol use over the course of adolescence to better understand how social factors lead to risky pre-adult drinking behaviors in a rarely studied and highly at-risk NAI population.

1 Literature Review

1.1 Background

There has been so much research and media attention on alcoholism and alcohol abuse among North American Indigenous (NAI) people that a stereotypic image of the “drunken Indian” has emerged (see May 1994 and Gray and Nye 2001 for reviews of these myths). Yet the National Household Survey on Drug Abuse (NHSDA) report on substance use indicates that while NAI heavy alcohol use rates are higher than for African Americans and Asians, they are similar to those for European Americans and Hispanics (DASIS 2003). Moreover, European Americans must be constantly reminded that there is no single “Indian” culture. There are more than 561 federally recognized tribal governments in the U.S (Bureau of Indian Affairs 2007) and variation in alcohol abuse and dependence prevalences vary substantially between cultures and among communities within cultures (Whitbeck et al. 2006).

In some communities, however, NAI adolescents appear to begin drinking earlier and to progress more rapidly to regular use than non-Indigenous youth (Bachman et al. 1991; Beauvais 1998; Blum et al. 1992), resulting in earlier-onset of substance use disorders than in the general population (Whitbeck et al. 2008). Longitudinal studies indicate that early initiating adolescents are more likely than later starters to increase use in high school and early adulthood (Chassin, Pitts, and Prost 2002; Li, Duncan and Hops 2001) and are at dramatically increased risk for developing drinking problems later in life (Dewitt et al. 2000). Moreover, age at first use is associated with a broad list of negative outcomes, including greater lifetime impairment and social problems (Chou and Pickering 1992), lifetime psychopathology (Sartor et al. 2006), nicotine dependence, and illicit drug use (McGue et al. 2001).

Given the potentially life-long consequences of early drinking, we first describe alcohol use development trajectories during the transition to adolescence amongst a sample of NAI youth using semi-parametric growth modeling techniques (e.g., Muthén 2001, 2004). This step is essential for understanding the timing and patterns of early drinking along with the consequences over the early life course in an at-risk NAI population. Next, we characterize the extent to which subsequent problem drinking (DSM-IV alcohol abuse and/or dependence) is related to early alcohol use trajectory. In other words, does early drinking lead to increased problem drinking as youth age into middle- and later-adolescence shortly before beginning the transition to adulthood? Given the evidence that youth in some NAI populations are early starters, while the adults in the population comprising this study have

high rates of drinking problems themselves (Whitbeck et al. 2006), we seek to characterize the consequences of early alcohol use for later problem drinking.

Theoretical Frameworks—The analyses we present below are guided by a life course perspective (e.g., Elder 1998) viewing developmental alcohol use trajectories early in adolescence as important antecedents for drinking problems going into adulthood (Crosnoe and Riegle-Crumb 2007). This conception, reflected in our first research goals, echoes the life course view that the developmental impact of life events depends upon their timing (Elder 1998). With respect to our second set of research goals, our etiological model is composed of (a) fundamental causes reflecting structural constraints and circumstances affecting local environments and the people within them (Link and Phelan 1995), (b) stress processes that link these external factors to individuals' inner lives (e.g., Pearlin 1989), and (c) the social supports that mediate these processes (Turner 1999). In addition, individual biographies are composed of multiple overlapping behavioral and psychological trajectories reflecting how people feel about and engage with their social worlds, so we also consider (d) intervening psychosocial pathways.

Fundamental Social Causes: The history of oppression (e.g., Americanization “civilizing” policies), loss of land, culture, economic marginalization, and political disenfranchisement of NAI peoples has been an engine for inequality within and across communities (Duran and Duran 1995). Social inequality is of considerable importance in the study of health because social conditions put individuals at “risk of risks” (Link and Phelan 1995: 80). In their seminal paper, Link and Phelan (1995) described the social conditions contextualizing risk factors as “fundamental causes of disease.” Social conditions in this perspective (see also Glass and McAtee 2006) are thought to shape risks over the life course by affecting a diverse range of mechanisms including the built environment, human capital formation, social networks, access to economic resources, and the cultural norms and mores shaping the social meanings that underlie proximal lifestyle and biological mechanisms (Pescosolido et al. 2008).

Disorganization is endemic in some NAI communities (Indian Health Services, 1997; Sandefur, Rindfus and Cohen 1996), as are poor economic opportunities (Gregory, Abello and Johnson 1996; Trosper 1996), low educational attainment, crime, risk of victimization (Smith 1999), and widespread alcohol, drug, and mental health problems (Beals et al. 2005; Beauvais 1998). Many of these factors are present in the communities in this study (Whitbeck et al. 2006). Because our research design has only a very limited ability to study how variations in these factors across places influence adolescent alcohol use, we follow Pescosolido et al. (2008) and McLeod and Nonnemaker (1999) in viewing fundamental social causes as familial stratification (i.e., parent education, income, and employment) and ascribed characteristics (i.e., gender and age). For example, there is evidence that middle school NAI girls begin using substances earlier than boys (Walls 2008). These stratification characteristics are important because they can directly increase negative coping behaviors like alcohol use (Mirowsky and Ross 2003) and indirectly by increasing exposure to stressors (Pearlin et al. 1981), lowering social support (Turner and Marino 1994), and increasing distress (e.g., Kessler and Neighbors 1986).

Stress Process: We view the stress resulting from fundamental social and other causes as a process by which external factors become embodied within individuals (see Pearlin 1999). As Pescosolido et al. (2008: S176) note, “the stress process connects individuals to their inner selves (i.e., identity), to the rhythms of their daily lives, and to the larger social contexts in which they are embedded.” Central to our goals is the idea that proximal chronic and transitory (e.g., financial strain, discrimination, recent negative events) and parenting stressors (e.g., parent history of drug abuse and poor mental health) lead to negative coping

behaviors such as increased alcohol consumption at earlier ages in order to reduce stress or alleviate strain (e.g., Lindenberg et al. 1994; Agnew and White 1992).

Reservation NAI adolescents often experience chronic financial strain (Gregory, Abello and Johnson 1996; Trosper 1996) and on some reservations children are exposed to especially high rates of violence and negative life events (Bachman 1992). In addition, discrimination induces stress effects on physical and mental health among minority group members (Kessler, Mickelson, and Williams 1999; Williams, Yu, and Jackson 1997). Evidence from the NAI culture comprising this study indicates that perceived discrimination is an important contributor to alcohol abuse among adolescents and to depressive symptoms among adolescents and adults (Whitbeck et al. 2002). Moreover, parent substance abuse may be a particularly potent source of stress because it leads to ineffective parenting (Conger 1997) and decreases social support. The youth in this study have parents with high rates of alcohol abuse and dependence who, in many cases, also have mental health problems as well (Whitbeck et al., 2006).

Social Support: Social support perspectives are based on the observation that many coping resources buffering the negative consequences of stress occur in relationships with significant others such as family members, friends, and for adolescents, possibly school personnel (see Thoits 1995). There is evidence that single-parent families are viewed by adolescent children as less warm and supportive than two-parent families (McLanahan and Sandefur, 1994) while those in two-parent families report higher levels of school adjustment (Astone and McLanahan 1991). Moreover, family processes may be stronger deterrents to substance abuse among NAI than European American children (Swaim et al. 1993). Therefore, we include family structure as a sociodemographic indicator for support along with parent warmth and approval and positive school-based experiences both as direct protective factors and potential sources of mediation of both fundamental causes and social stressors (see Ensel and Lin 1991).

Intermediate Psychosocial Factors: Part of the reason alcohol use is expected to respond to social conditions reflecting fundamental causes, stressors, and social supports (e.g., Stockdale et al. 2007), is because drinking can be a negative coping behavior that adolescents use to reduce stress (Powers 1987) or alleviate strain (Agnew 1992). We view the consequences of stressors and strains to be *psychosocial* rather than *psychological* to clearly situate individual psychological states within the social environmental model reflected in the frameworks guiding our etiological analysis. Both contemporaneous stressors such as negative life events and cultural stressors such as discrimination and cultural losses are associated with NAI substance abuse (Walters, Simoni, and Evans-Campbell, 2002). Stress and strain can also manifest in individual psychologies as distress and externally in social attachments (e.g., attachment to school; DuBois et al. 1992). Since the school can provide social support, facilitating school attachment may be a protective mediating pathway (e.g., Crosnoe and Riegle-Crumb 2007) while depression and feelings of anger may indicate manifestations of distress that lead to negative health behaviors like alcohol use (e.g., Deas and Thomas 2002).

Finally, we view delinquent peer affiliations as a risky psychosocial factor situated at the intersection of individual agency and friendship opportunities reflecting local social structural constraints. Thus, we include a measure of perceived friends' delinquency since peers are considered a major factor in early alcohol and drug use (Conger 1997; Dinges and Oetting 1993). In some NAI cultures alcohol use is considered indicative of adulthood (Topper 1980) while active refusal or rejection of alcohol and drug using friends can be viewed as rude and confrontational (Beauvais 1980; Weibel-Orlando 1982).

2 Methods

2.1 Sample

We note, at the outset, that this project arose in partnership with the participating communities and that our agreement specifies confidentiality of culture, reservation, and reserve. The data were collected as part of a multi-year lagged sequential study currently underway on four American Indian reservations in the Northern Midwest and four Canadian First Nation reserves. Three of the Canadian Reserves are classified as “remote” in that they are at considerable distances from even small towns and are accessed by non-paved roads. The culture is one the most populous Indigenous cultures in the United States and Canada. All of the study reserves and reservations share a common cultural tradition and language with minor regional variations in dialects. The recruitment procedure, detailed in Whitbeck et al. (2008), resulted in an overall response rate of 79.4% and the wave to wave retention rates were more than 90% at each year.

Our analyses are based on five waves of data. Measures of alcohol use collected in Waves 1, 2, 3 and 5 were used to develop the growth mixture models and a diagnostic interview schedule at Wave 6 was used to construct the alcohol abuse and dependence measures. The baseline sample was 747 cases, but was reduced to 727 after dropping cases with fewer than two observations for the repeated alcohol use measures. We chose to eliminate those youth with fewer than two repeat observations to ensure that each case was able to provide trajectory information. Because there was also missingness on the independent variables, which were all collected at Wave 1, we used multiple imputation to maintain the covariance structure of the data¹ (Little and Rubin 2002). Using imputation raised our baseline listwise deletion sample size across the full covariate list from 511 to 727 (the listwise N for M-4 in Table 2 is 603). The proportion of nonmissing cases for the full 727-case sample is reported along with the descriptive statistics in Table 1.² These descriptive results indicate that only small amounts of missingness generally needed to be imputed for the independent variables (12%, for the parent discrimination measure, was the largest).

2.2 Measures

The analysis makes use of two dependent variables. The first is a grouped frequency of alcohol use over the previous year with values for never (0), one or two times (1), less than once a month (2), once a month (3), every week (4), nearly every day (5), and every day (6). The adolescents were 9–13 years of age (there were 20 13 year olds) at the first assessment and up to 14 years at the final assessment included in the analysis. Over time the wave-specific alcohol use frequency average rose from approximately 0 (range = 0–5) at age 11 years to approximately 1 at age 14. We chose not to use a longer timeline for the growth mixture models because (a) capping at 14 lead to a model series spanning 10–14 for early use and primarily 15–17 for problem drinking; and (b) because the number of alcohol use classes increases with increasing years leading to a more complicated model structure as more youth transition to drinking regularly. Given the relatively small sample size and concerns about power with these models, the choice to restrict the age-range had the benefit of producing a more straightforward model structure that conveniently delineates age in the

¹The multiple imputation model included the independent variables used in the analysis, the probability of trajectory classification from the growth mixture models, the repeated alcohol use measures, and indicators for alcohol dependence and abuse diagnosis. We used a chained equations approach implemented in ICE for Stata so that the imputations reflect the appropriate measurement level so that, e.g., the abuse/dependence measure could be modeled using logistic regression (see Royston 2004).

²The N=481 for alcohol use at age 11 in Table 1 reflects the research design. Some youth were older than 11 at Wave 1 and thus are missing by definition so the %N column overstates missingness. Moreover, we deterministically or probabilistically impute for age at Wave 6 has no impact on our results. Because the actual N for the problem drinking models is 657 there are only six relevant missing cases as far as the parameter estimation is concerned.

total model (the 20 Wave 1 13 year olds contributed two observations, 12 year olds up to three, and 10–11 year olds up to four).

The second dependent variable, problem drinking, is a combined measure of alcohol abuse and/or dependence³ based upon DSM-IV (APA, 2000) criteria from a diagnostic inventory administered to the adolescents at Wave 6 when the youth were aged 14–18 years (there were only 8 14 year olds and 41 18 year olds) so that this variable measures whether drinking is causing problems in late adolescence. At Wave 6, over 31% of the sample met the joint abuse/dependence criteria (206 of 657) and approximately 56% of the youth who met criteria for alcohol dependence also met criteria for abuse (18% who did not meet dependence criteria met abuse criteria).

2.3 Parent and Child Variables

The independent variables were all drawn from the questionnaire administered at Wave 1 and the scales generally have high reliabilities and, unless otherwise noted, are standardized (*z*-scores).⁴

Fundamental causes—We include status characteristics for *age* in our model series to account for additional heterogeneity not captured in the growth mixture model structure, and an indicator for *female* adolescents because previous studies have found the transition to regular alcohol use in this population to be gendered (Walls 2008). Additional fundamental cause indicators reflect class-based structural inequalities. We include a measure for family *Income below \$25000* and parent education is entered into our equations as two dummy variables, *Less than a high school education* and *Some college or degree attained*, with high school graduates omitted. Finally, parent employment is included as three dummy variables reflecting *Part time employment*, *Unemployed*, and *Other Employment* (disabled, retired, student, homemaker) with full-time employed caretakers comprising the reference category.

Stress processes—Proximal stress process variables include *Financial strain* ($\alpha=.73$), both parent and child experiences of *Discrimination* ($\alpha=.93$, $\alpha=.92$), and the child's *Negative life experiences* ($\alpha=.83$) scales. The financial strain measure was created from 16 items (e.g., we have enough money to afford the kind of food we need, etc.). The parent measure of discrimination was created from 11 ordinal items (e.g., how often someone yelled a racial slur or insult at you, etc.) and the child measure was constructed from 12 ordinal items (e.g., how often has store owner or clerk treated in a disrespectful way because you are [NAI], etc.). The measure of negative events in the child's life was constructed from 20 dichotomous items (e.g., friend died in past 6 months, etc.). Measures reflecting parenting stressors used DSM-III lifetime criteria for *Parent alcohol abuse (ever)* and *Parent major depression (ever)*.

Social support—*Family structure* is included as a sociodemographic proxy for social support. The variable is entered into the equations as dummy variables indicating whether

³Although we have combined measures of abuse and dependence this is not a problem because our predictors had similar effects across categorizations (see May 1984 for an example combining diagnostic criteria). We assessed whether the predictors had differential effects on each categorization by constructing a 4-category nominal variable and estimated multinomial logit models predicting no classification versus each classification (abuse, dependence, both). Next, we conducted Wald χ^2 tests to assess whether the 3 coefficients across categories for each independent variable jointly differed statistically from each other ($df=2$ for a single covariate; results available upon request). Finally, because of low power, we also assessed the direction and magnitude of effects subjectively for concordance. These results indicated that our covariates had similar associations with abuse and/or dependence measure of problem drinking so we have chosen to use the simpler none vs. 1+ diagnoses dichotomy.

⁴Full variable lists are available upon request. In addition, many of the measures we employ are scales intended to reflect latent or difficult to measure characteristics (e.g., depressive symptoms). Our strategy made use of latent variable model posterior factor score methods (see Muthén and Muthén, 2006). Further details are available in a Technical Appendix from the first author.

the parent/guardian is *Partnered* but unmarried, *Never married*, and *Separated/divorced/widowed*, with *married* as the omitted category. Direct measures of social support include both parent and child perceptions of *Parental warmth and support* ($\alpha=.73$, $\alpha=.82$ correlation=.17), and *Positive experiences* ($\alpha=.58$). The parent parental warmth and supportiveness measure was created from 6 ordinal items (e.g., when [child] does something you like or approve of, how often do you let (him/her) know you are pleased?, etc.) and the child measure was created from 12 ordinal items (e.g., when you do something good, how often does someone in your family let you know they are proud of you?, etc.). The positive life events scale, which primarily references positive school-based events, employed 6 dichotomous items (e.g., won an award or recognition for school work in the last 6 months, etc.).

Intermediate psychosocial characteristics—Because we view alcohol use as a developmental process, we also adjust for intermediate risky/protective developmental outcomes. The measures included in the analysis are *Depressive symptoms* ($\alpha=.83$), *Positive school attitudes* ($\alpha=.75$), *Feelings of anger* ($\alpha=.84$), and *Friend delinquency* ($\alpha=.75$) scales. The depressive symptoms scale was based on 20 ordinal items from the CESD (e.g., the number of days in the last week including today you felt sad, etc.) and the positive school attitudes scale was created from 11 ordinal items (e.g., you like school a lot, etc.). Finally, a measure of subjective friend delinquency was created from 7 ordinal items (e.g., how many of your three best friends drink alcohol, etc.).

2.4 Analytic Plan

We model adolescent alcohol use development patterns using growth mixture models^{5,6} (GMM). Cheadle, Amato, and King (2010) provide a technical discussion and substantive example of Poisson GMMs that are very similar to our models. This approach is conceptually similar to the standard latent growth curve model (LGCM) and may be thought of as an extension to multigroup LGCM (e.g., separate growth curves for males and females) where group membership is unknown and inferred from the data (Muthén 2001, 2004). Where the LGCM assumes a prototypical pattern of change for a single population (e.g., Singer and Willett 2003), GMM attempts to uncover sub-populations with discretely different change patterns. So while the LGCM captures heterogeneity around a prototypical pattern of change with random effects, GMMs first capture heterogeneity by identifying latent groups with similar change patterns, estimating multiple growth curves simultaneously (it is also possible to include random effects to capture additional heterogeneity). This approach is sometimes called group-based (Nagin 2005) or typological because cases are grouped and clustered together based on similarities in their change trajectories. Thus, multiple growth curves, rather than a single curve for everybody, are estimated (see Figure 1).

The probability that individuals are in specific groups or latent classes is modeled using logistic (when # groups = 2) or multinomial logistic regression equations (when # groups > 2; see Table 2). These latent trajectory classes are then used as mediating independent variables to predict abuse/dependence (see Table 3). Because group membership is probabilistic – it is latent and must be inferred – it is important that estimation be done jointly so that uncertainty in classification and parameter estimates is reflected in the

⁵Additional details are available from the first author in a Technical Appendix.

⁶Although we included the dependent variables and GMM classification in our imputation model, we used the un-imputed variables with missing values in the analysis, thus making use of full information maximum likelihood to model the longitudinal process and multiple imputation to preserve correlations with the independent variables. This means that the working N for the GMM models was 727 and 657 for the diagnostic outcome. Results do not change substantively when the analysis is limited to the 657 cases with diagnostic information.

standard errors. Thus, all our results are based on simultaneous estimation of the regression parameters and the GMM.

In order to determine the optimal number of classes we first estimated Poisson quadratic latent growth curves followed by four sets of GMMs. The first specification assumed only a linear time slope, the second added a random intercept to capture additional heterogeneity beyond the latent classes, the third incorporated a quadratic term for time, and the fourth added a random intercept. Using the BIC (Raftery 1995) for model comparisons, we selected a 3-class quadratic growth curve (Figure 1 below) without random intercept variation (from model set 3) since this was the simplest model with most favorable BIC relative to the other baseline LGCMs and GMMs.

3 Results

3.1 Sample Description

Descriptive statistics for the sample are presented in Table 1. The adolescents in this sample are relatively disadvantaged – nearly 45% live in families with incomes below \$25,000, approximately 20% reside with a parent/guardian with less than a high school education, and close to 20% live with an unemployed focal parent. The youth in this sample also live in a diverse range of family structures. Only 32% reside in two-parent homes, 27% live with a partnered but unmarried guardian, 22% with a never married parent, and 19% with a separated or divorced (or widowed) parent. Over 60% of youth live with a parent/caretaker who has ever met lifetime criteria for alcohol abuse and nearly 20% of caretakers have experienced a major depression episode. Taken together, these findings indicate that the young people in this indigenous population are exposed to a variety of risk factors which may lead to early alcohol use and later problem drinking.

3.2 Classification of Individuals

The average levels of alcohol use over the previous year for the three trajectory classes are plotted in Figure 1 for ages 10–14 years. The legend contains (a) the N and % classified in each group, (b) the odds-ratio for later problem drinking diagnosis comparing the early drinking classifications to abstainers (c) and the percentage that meet the abuse/dependence (problem drinking) DSM-IV criteria for each latent class. The majority of youth, about 64%, largely abstained from drinking through age 14. Nearly 17% of these *abstainer* youth, however, met the abuse/dependence criteria by late adolescence when most were between 15 and 17 years of age. The first group of drinkers is labeled *early-onset* because those in this class, about 20% of this NAI population, are characterized by increasing use after 11–12 years of age. Nearly 70% of these youth met criteria for abuse/dependence when they were older and the odds were nearly 9 times larger than for the youth who abstained. A similar percentage of the NAI youth in this sample, 18%, began transitioning to fairly regular drinking in early adolescence and half of them later met the diagnostic criteria. The odds for this group of *adolescent-onset* drinkers were over 5 times larger than for those who abstained. Figure 1 shows that nearly 40% of the youth in this population begin drinking, at least occasionally, by age 14 and that these youth have markedly increased risks for problem drinking by late adolescence.

3.3 The Etiology of Early Alcohol Use Trajectories

Exponentiated logistic regression coefficients (relative risk ratios⁷ [RRR]) and absolute t-values for the etiological models predicting membership in the early-onset and adolescent-

⁷The relative risk is the ratio of the probability for one group to the probability for another. The RRR is a ratio of these relative risks so is conceptually similar, though not identical, to an odds ratio.

onset classes relative to abstaining are presented in Table 2. There are a few important things to note in this table. First, the full covariate list reported in Table 1 does not appear in this table. We have excluded covariates not related to early drinking in order to (a) conserve space and (b) to simplify the model specification. Thus, a variable omitted from the table indicates that it was not associated with classification and has been dropped from the analysis. Second, before moving to the multinomial logistic regressions, we tested whether or not classification could be treated as an ordinal outcome: abstainer → adolescent-onset → early-onset with respect to each predictor. This proportionality assumption⁸ proved valid for some covariates (e.g., child-parent warmth/approval) but not for others (e.g., negative life events). For those covariates with proportional effects, the fixed coefficients are denoted with leftward arrows (←) indicating that the coefficient, standard error, and statistical significance in the adolescent-onset column (Ad.) is the same as that in early-onset column (Early). The first column of “Bivariate” results presents estimates for variables entered singly (e.g., female, or the parent education indicators), M1 adds the stressors to the fundamental causes equation, M2 adds the support variables, and M3 adds all of the psychosocial variables except friend delinquency, which is included in M4.

The results in the bivariate column indicate that females, despite having similar levels of average alcohol use (see Table 1), are more likely to regularly drink early than boys – the RR (relative risks) is approximately 60% higher across specifications (M1-M4). A number of stressors are also related to early drinking. Financial strain, parent alcohol abuse, and parent major depression all predict early drinking and their influences are statistically similar for both early-drinking classifications. Other stressors including negative life events and adolescent perceived discrimination are also associated and the results indicate that the effects are stronger for the early-onset than adolescent-onset group. Of the social support variables only child-parent warmth and approval and positive school events are protective, while positive events are not, perhaps because of the timing at which the variable was measured. For example, it is possible that the protective influence of positive events decays rapidly with time if not reinforced (e.g., Cornwell 2003). Of the intermediate psychosocial variables related to early drinking classification, positive school attitudes and feelings of anger are related only to early-onset drinking and depressive symptoms is only marginally associated with adolescent-onset alcohol use. These results indicate that positive school attitudes are protective with respect to early-onset drinking while feelings of anger and depressive symptoms, indicators of distress, increase risk. Notably, each standard deviation increase in delinquent friendships increase the RR of early-onset drinking by a factor of 2.6 and increase the risk of adolescent-onset by 50%.

Model M1 adds the stressors in the bivariate column to the significant fundamental cause indicator, female. When the stressor variables are included jointly financial strain and the negative events coefficient predicting adolescent-onset drinking become non-significant. Overall, however, the general pattern of findings indicates increased risk of early drinking for females, those who experience more negative events, are exposed to more discrimination, and who have parents with problem histories of alcohol abuse and depression. Parent alcohol abuse, however, does not remain significant in model M2 when the support variables are included, indicating that parent drinking behaviors are negatively correlated with the support provided to offspring. Overall, the results from this model suggest relatively independent effects of the stressor and support variables when the coefficients are compared across the model series. The coefficients for the support variables, which are smaller than one in magnitude, indicate that child perceptions of parent warmth

⁸The regression parameter is fixed to be equal for the early- and adolescent-onset equations.

and approval are protective – each standard deviation increase lowers the RR by 20%, and that positive (school-based) experiences protect youth from early-onset drinking.

Model M3 adds the intermediate psychosocial outcomes to M2. Of these coefficients, only those for positive school attitudes and feelings of anger predicting early-onset drinking are significant. Moreover, neither the influences of negative life events, child-parent warmth and approval, or positive events remain statistically significant with the psychosocial mediators included because these variables operate via orientations towards school and feelings of anger. This pattern of findings is consistent in M4 when perceived friend delinquency is added. Indeed, friendships with delinquent peers are another mediational avenue through which social support influences early drinking. The coefficients for delinquent friendships are large in magnitude and indicate that each standard deviation increase more than doubles the RR that an adolescent is an early-onset drinker and increases the odds of adolescent-onset drinking by over 40%.

3.4 The Etiology of Alcohol Dependence and/ or Abuse in Late Adolescence

Odds ratios and t-ratios for coefficients predicting DSM-IV alcohol abuse and/or dependence are reported in Table 3. As with the results reported in Table 2, the coefficients in Table 3 are drawn from models for which the reported variable was statistically associated with diagnosis in at least one model. In other words, variables from Table 1 which have been omitted indicate that the variable is not associated with problem drinking in this population. The “Bivariate” column presents findings for covariates singly, model N1 includes the fundamental causes and stressor variables, model N2 adds the psychosocial mediators (there were no statistically significant social support variables) with the exception of friend delinquency, which is included in N3, and N4 incorporates the early trajectory classifications, treating these early drinking trajectories as mediators of the fundamental causes, stressors, and psychosocial influence pathways.

The early- and adolescent-onset coefficients confirm the results reported in Figure 1 with odds nearly 9 and over 5 times larger, respectively. Two fundamental cause variables are positively related to alcohol abuse/dependence – age at the time of assessment and female (Bivariate column). A number of stress variables including negative events, perceived discrimination, and parent major depression also increase risk. Although none of the support variables are related to problem drinking, positive school attitudes is protective while feelings of anger, depressive symptoms, and friend delinquency are all positively correlated with abuse/dependence.

Model N1 incorporates the fundamental cause and stressor variables jointly into the prediction equation. Parent major depression does not remain statistically significant when included with the other covariates, but the remaining variables do. This finding indicates that the bivariate parent depression finding reflects correlations with the other stressors and not a direct effect. Moreover, the influence of negative life events appears to increase risk by leading to negative school attitudes (N2). In fact, in model N2 positive school attitudes is the only psychosocial variable related to problem drinking with a marginally significant effect indicating that each standard deviation increase decreases the odds of abuse/dependence diagnosis by 15%. This protective effect is mediated in N4 when friend delinquency is included in the model – each standard deviation increase on this variable increases the odds of abuse/dependence diagnosis by approximately 40%.

Only age at the time of assessment (Wave 6), perceived discrimination, and parent alcohol abuse remain statistically significant in N4 when the early drinking classifications are included in the model. The variable for female, for example, is greatly reduced in magnitude with the inclusion of the latent trajectory classes, indicating that the statistically marginal

association between female and problem drinking is the culmination of risky drinking patterns beginning before or during the early teenage years. The measure of friend delinquency is mediated as well, which suggests that friendships influence problem drinking indirectly through individual alcohol use biographies. Both perceived discrimination and parent alcohol abuse have independent effects. Controlling for early drinking classification, each standard deviation increase in perceived discrimination increases the odds of abuse/dependence diagnosis by 22% and having a parent with a history of alcohol abuse increases the odds by over 50%.

The coefficient for early-onset alcohol use classification diminishes between the bivariate estimates and the full model estimates reported in N4, although the odds ratio indicates that these youth have odds 6.75 times larger than their abstainer peers while the odds ratio for adolescent-onset drinkers remains large with a value of approximately 5. Moreover, they point to the consequences of individual drinking biographies in early adolescence for problem drinking in late adolescence, on the one hand, and the importance of these biographies in explaining the influences of background on problem drinking in late adolescence, on the other.

4 Discussion

With our first set of research goals we sought to delineate the timing, patterns, and consequences of alcohol use over the early life course for problem drinking in later adolescence. From the ages 10–14 years adolescent alcohol use in this particular NAI population is characterized by early-onset, adolescent-onset, and abstainer groups. The early-onset group (20%) reflected youth with increasing alcohol consumption after age 11 and the adolescent-onset group (20%) was characterized by sharply increasing drinking around age 13. The early-onset drinkers were far more likely to meet DSM-IV criteria for alcohol abuse and/or dependence, which we have viewed as an indication of problem drinking reflecting negative contemporary and possibly long-term consequences (Rohde et al. 2001). In fact, 70% of the early-onset and 50% of the adolescent-onset groups met the abuse and/or dependence criteria while only 17% of the abstainers did. This indicates that early drinkers are at substantially elevated risk for drinking problems and that a sizeable proportion of those youth who abstain early are also likely to show signs of problem drinking by late adolescence. In fact, 30% of all youth in this study did so.

In addition to these descriptive questions, we motivated an etiological analysis of drinking over the early life course guided by research into structural characteristics reflecting the fundamental causes of disease (Link and Phelan 1995), stress processes (e.g., Pearlin 1989), social support (Turner 1999), and intermediate psychosocial developmental pathways. This part of the analysis was oriented around questions of whether family and child risk/protection factors distinguish early drinking patterns and later problem drinking, and whether early drinking trajectories explain the relationships between later alcohol abuse/dependence and background.

The global picture portrayed in the analysis depicts a developmental process whereby stress processes lead to increased anger and friendships with delinquent peers, which then lead to early drinking. At the same time, gender appears to structure the risk of early drinking largely independently of the other factors. Perceived parental warmth was protective through the psychosocial pathways but did not strongly mediate stress effects. Early drinking was then found to play a key role in problem drinking by approximately age 16 – increasing the odds of alcohol abuse/dependence criteria by nearly 9 times for the earliest drinkers and 5 times for the adolescent-onset group. Although these drinking trajectory classifications provided indirect pathways through which stress and psychosocial pathways influenced

problem drinking, they played only a small role in mediating the two stress process indicators associated with diagnosis, perceived discrimination and parent alcohol abuse. Taken together, these results paint a dramatic picture of the role of stress in promoting negative affects which lead to behaviors that substantially increase the risk of harmful patterns of alcohol consumption over the early life course. Future studies will be able to address how these processes during the transition to adulthood.

The etiological analysis of early drinking trajectory and diagnosis classification indicated that features of fundamental causes of disease were not the primary motivators of early drinking, with gender providing a notable exception. Neither parent education, income, nor employment were related to these outcomes. This finding should not be taken as evidence against the fundamental social causes perspective since we compared variation on these characteristics within a single NAI culture and over a small set of communities. The influence of fundamental causes may be most visible when comparing across population groups because of restricted heterogeneity and because relative disadvantage within communities may be mediated by community-wide absolute disadvantage diffusion processes (see, e.g., Boardman et al. 2001). This may also be reflected in the fact that stress process indicators such as financial strain and social support variables like family structure also were not associated with our outcomes.

Two of our indicators of fundamental causes were associated with our outcomes (females→early-onset; age→abuse/dependence). The finding for age, which likely represents both fundamental and other developmental factors, indicated that older youth were more likely to meet diagnostic criteria and suggests that many of the younger youth are likely to show signs of problem drinking as they age into later adolescence and early adulthood. To qualify the importance of this age effect, 64% of these adolescents' parents meet lifetime criteria for alcohol abuse (Whitbeck et al. 2006). Moreover, the parent history of alcohol abuse was the one predictor of problem drinking that was not mediated by early drinking trajectories. This latter finding points to the complicated intergenerational cycle of influence that persists in many NAI communities and may reflect the complex interplay between genetic risk, risky environments, and risky substance use behaviors (e.g., Pescosolido et al. 2008).

The finding that the girls in this NAI population have odds of early-onset drinking 60% higher than the boys is a striking and troubling finding that we were not able to fully address in this analysis. The results indicated that the female-male gap is not the product of psychosocial pathways such as higher friend delinquency, although it should be noted that we were not able to account for the *age* of these friends. Thus, it is possible that girls do not have more delinquent friends (as is shown in Table 1) so much as *older* friends with greater access to alcohol and higher substance use. Moreover, the results of a supplemental *ad hoc* interaction analysis indicated that the female-male gap was not moderated by other characteristics included in our analysis.⁹

Our models also indicated that perceived discrimination and parent major depression are important stressors predicting early drinking that have both direct and indirect effects by decreasing positive school attitudes, generating feelings of anger, and increasing associations with deviant peers. Thus, this analysis contributes to the growing evidence that discrimination is a pervasive and important generator of negative health (Kessler et al. 1999), in this case by leading to thoughts, feelings, and associations leading to risky alcohol use. Not all of our indicators promoted risk for early drinking, although our analysis uncovered no protective abuse/dependence factors. Child perceptions of parent warmth and

⁹We tested a number of models interacting the variable for female with the other covariates in Table 1.

approval served to foster positive school attitudes which increased the probability of abstaining. Unfortunately, these positive influences are overwhelmed for far too many youth by the broader gestalt of negative life experiences, perceived discrimination, adverse parent characteristics such as past depression and histories of alcohol abuse and, finally, the direct consequences of drinking in early adolescence.

4.1 Limitations

This study has a number of important limitations. First, it was based upon a single NAI culture and may not be generalizable to other NAI populations. Second, the adolescents constituting the sample live either on or close to rural reservations, so these results may not translate to NAI youth living in urban areas even if they share the same cultural background. Third, this study explored a single dependent variable, alcohol, and it is not clear how the results generalize to other substances or health outcomes that may have different developmental courses and etiologies. Fourth, the age-range of this study covers only a single cohort of youth over a few years. Implications over the life course, including the transition to adulthood, have yet to be assessed. Fifth, there may be some concern that our measure of abuse and/or dependence mixes two different concepts that should be treated independently. In supplemental analyses we found that the influence of individual covariates did not differ across classifications (abuse, dependence, abuse and dependence), suggesting that the fundamental, stress, social support, and psychosocial pathways similarly influence these diagnostic categorizations. Finally, we found peer delinquency to be an important component of early drinking. Our measure, however, was perceptual so our coefficients may be biased upwards by the reflection problem, though the finding of friend effects is consistent with findings from numerous social network studies (see Haynie and Osgood 2005).

5 Conclusions

We have documented that approximately 40% of NAI youth from a single culture in the Northern Midwest and Canada begin drinking at a young age with sharply increasing rates of consumption over early adolescence. Moreover, these adolescents are at substantially elevated risk for meeting diagnostic criteria for alcohol abuse and/or dependence – a serious indicator of problem drinking by ages 15–17 years. Although 17% of youth who abstained from drinking between ages 10–14 later showed signs of problem drinking, most of the problem drinkers had begun drinking when they were quite young – around 11–13 years of age. This substantial effect strongly suggests that intervention efforts need to grapple with children's alcohol use before 11 years of age in this NAI population. Approximately 10 years of age may be optimal since the most at-risk NAI children will begin drinking with greater frequency shortly after. Later interventions may be too late since approximately 40% of children on these reservations will already have begun drinking, placing them at greatly elevated risk for alcohol use problems while still in adolescence.

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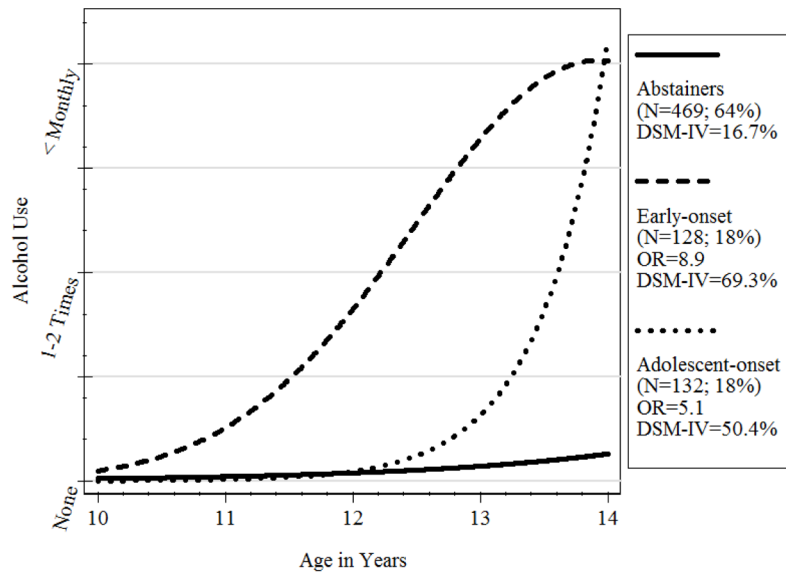


Figure 1. Predicted alcohol use frequency development patterns for the 3-class quadratic growth mixture model without intercept heterogeneity ($N=727$)

Table 1

Descriptive statistics for the complete covariate list (N 727)

Variables	N	% N	Mean	Sd	Min	Max	Male	Female
<i>Background</i>								
Age at wave 1	726	100	11.09	[0.82]	9	13	11.13	11.04
Age at wave 6	651	89	16.23	[0.89]	14	18	16.27	16.19
Female respondent	729	100	0.50	[0.50]	0	1	0.00	1.00
Education: < high school	726	100	0.17	[0.38]	0	1	0.16	0.19
Education: high school	726	100	0.36	[0.48]	0	1	0.38	0.34
Education: some college/degree	726	100	0.47	[0.50]	0	1	0.46	0.47
Employment: full time	727	100	0.58	[0.49]	0	1	0.57	0.59
Employment: part-time	727	100	0.10	[0.30]	0	1	0.10	0.10
Employment: unemployed	727	100	0.19	[0.39]	0	1	0.18	0.20
Employment: Other	727	100	0.14	[0.34]	0	1	0.16	0.12
<i>Stressors</i>								
Financial strain (z)	670	92	0.00	[1.00]	-1	4	0.02	-0.01
Negative events (z)	720	99	0.00	[1.00]	-2	4	-0.02	0.02
Perceived discrimination (z)	717	98	-0.01	[1.00]	-1	4	0.04	-0.05
Parent discrimination (z)	637	87	0.02	[1.00]	-2	3	-0.05	0.08
Parent alcohol abuse (ever)	722	99	0.64	[0.48]	0	1	0.65	0.62
Parent major depression (ever)	722	99	0.19	[0.39]	0	1	0.17	0.21
<i>Support</i>								
Family structure: married	725	99	0.32	[0.47]	0	1	0.34	0.30
Family structure: partnered	725	99	0.27	[0.44]	0	1	0.24	0.29
Family structure: never married	725	99	0.22	[0.41]	0	1	0.18	0.25
Family structure: sep/div/widowed	725	99	0.19	[0.39]	0	1	0.23	0.15
Child-parent warmth/approval (z)	726	100	0.00	[1.00]	-4	2	-0.03	0.03
Parent-parent warmth/support (z)	727	100	-0.01	[1.00]	-4	2	-0.13	0.11
Positive experiences (z)	721	99	0.00	[1.00]	-2	2	0.09	-0.09
<i>Psychosocial</i>								
Positive school attitudes (z)	724	99	0.00	[1.00]	-3	1	-0.05	0.04
Feelings of anger (z)	727	100	0.00	[0.99]	-2	3	0.00	-0.01

Variables	N	% N	Mean	Sd	Min	Max	Male	Female
Depressive symptoms (z)	710	97	0.00	[1.00]	-2	3	-0.07	0.07
Friend delinquency (z)	674	92	0.00	[1.00]	-1	3	0.00	0.00
<i>Outcomes^a</i>								
Alcohol use at age 11 ^b	481	66	0.07	[0.33]	0	3	0.09	0.05
Alcohol use at age 14	679	93	0.95	[1.35]	0	5	0.87	1.03
Alcohol abuse/dependence	657	90	0.31	[0.46]	0	1	0.28	0.34

^aThese values were not imputed. Instead, the estimation routines used full information ML for the outcomes across the MI data sets.

^bThe N=481 for alcohol use at age 11 reflects the fact that youth were different ages at Wave 1, some older and some younger, so that the %N column is misleading here.

Table 2

Relative risk ratios from multinomial logistic regression equations predicting early and adolescent-onset alcohol trajectory classification versus abstaining ($N=727$). Results are based on a multiple imputation analysis using 10 replicate data sets

Variables	Bivariate		M1		M2		M3		M4	
	Early	Ad.	Early	Ad.	Early	Ad.	Early	Ad.	Early	Ad.
<i>Fundamental Causes^b</i>										
Female respondent	1.55 (2.80) **	← ^a	1.60 (2.91) **	←	1.57 (2.76) **	←	1.58 (2.76) **	←	1.59 (2.74) **	←
<i>Stressors^c</i>										
Financial strain (z)	1.17 (2.05) *	←	1.06 (.66)	←	1.07 (.77)	←	1.06 (.73)	←	1.10 (1.15)	←
Negative events (z)	1.53 (4.25) ***	1.21 (2.02) *	1.31 (2.53) *	1.10 (.93)	1.39 (2.94) **	1.10 (.86)	1.19 (1.44)	1.11 (.93)	.96 (.29)	1.02 (.14)
Perceived discrimination (z)	1.57 (4.73) ***	1.27 (2.52) *	1.44 (3.51) *	1.24 (2.11) *	1.48 (3.75) ***	1.24 (2.09) *	1.38 (2.89) *	1.26 (2.13) *	1.30 (2.32) *	1.21 (1.79) †
Parent alcohol abuse (ever)	1.68 (3.09) **	←	1.38 (1.78) †	←	1.31 (1.49)	←	1.25 (1.22)	←	←	←
Parent major depression (ever)	1.99 (3.57) ***	←	1.72 (2.56) *	←	1.70 (2.51) *	←	1.67 (2.36) *	←	1.56 (2.04) *	←
<i>Support^d</i>										
Child-parent warmth/approval (z)	.82 (2.59) *	←	.80 (2.27) *	←	.80 (2.27) *	←	.90 (.94)	←	.92 (.69)	←
Positive experiences (z)	.81 (2.03) *	1.00 (.01)	.75 (2.47) *	.99 (.06)	.75 (2.47) *	.99 (.06)	.90 (.86)	1.00 (.02)	.90 (.80)	.99 (.10)
<i>Psychosocial</i>										
Positive school attitudes (z)	.56 (5.87) ***	.94 (.59)					.68 (3.39) **	.98 (.15)	.77 (2.19) *	1.03 (.26)
Feelings of anger (z)	1.63 (4.94) ***	.96 (.34)					1.26 (2.07) *	.86 (1.27)	1.28 (2.08) *	.86 (1.20)
Depressive symptoms (z)	1.80 (5.07) ***	1.19 (1.73) †					1.21 (1.42)	1.09 (.73)	1.06 (.46)	1.03 (.27)
Friend delinquency (z)	2.62 (8.84) ***	1.50 (3.94) ***							2.15 (6.26) ***	1.43 (2.91) **
<i>Intercepts</i>										
			-1.38 (13.15) ***	-1.28 (12.70) ***	-1.43 (13.11) ***	-1.28 (12.67) ***	-1.51 (12.97) ***	-1.28 (12.57) ***	-1.60 (12.87) ***	-1.27 (12.37) ***

† $p < .1$,* $p < .05$,** $p < .01$,*** $p < .001$ (two-tailed). t-ratios are presented in parentheses.

Notes: Variables are entered in groups in models M1-M4. Coefficients for the intercepts are multinomial logit coefficients; other coefficients are relative risk ratios (e^b).

^aArrows ("←") indicate that the parameter has been fixed across the early- and adolescent-onset equations.

^bThe other background variables were not statistically significant so they have been excluded.

^cParent perceived discrimination was not statistically significant.

^dThe other support variables were not statistically significant.

Table 3

Odds-ratios from logistic regression equations predicting DSM-IV alcohol and/or abuse diagnosis (N=727 for the classification GMM, and N=657 for the diagnostic DSM-IV outcome). Results are based on a multiple imputation analysis using 10 replicate data sets

Variables	Bivariate		N1		N2		N3		N4			
	<i>e^b</i>	(t)	<i>e^b</i>	(t)	<i>e^b</i>	(t)	<i>e^b</i>	(t)	<i>e^b</i>	(t)		
<i>Classification</i>												
Early-onset alcohol use	8.86	(9.51)	***						6.75	(7.48)	***	
Adolescent-onset alcohol use	5.09	(7.14)	***						4.97	(6.63)	***	
<i>Fundamental Causes^a</i>												
Age at wave 6	1.45	(3.88)	***	1.44	(3.67)	***	1.38	(3.24)	**	1.27	(2.30)	*
Female respondent	1.32	(1.65)	+	1.36	(1.75)	+	1.36	(1.73)	+	1.37	(1.75)	+
<i>Stressors^b</i>												
Negative events (z)	1.36	(3.67)	***	1.20	(2.04)	*	1.14	(1.37)		1.03	(.32)	
Perceived discrimination (z)	1.46	(4.48)	***	1.35	(3.33)	**	1.33	(3.11)	**	1.29	(2.79)	**
Parent alcohol abuse (ever)	1.92	(3.51)	***	1.61	(2.39)	*	1.54	(2.12)	*	1.52	(2.06)	*
Parent major depression (ever)	1.59	(2.25)	*	1.40	(1.51)		1.34	(1.27)		1.26	(1.02)	
<i>Support^c</i>												
<i>Psychosocial</i>												
Positive school attitudes (z)	.75	(3.41)	**			.85	(1.73)	+	.89	(1.29)		
Feelings of anger (z)	1.33	(3.24)	**			1.13	(1.24)		1.14	(1.31)		
Depressive symptoms (z)	1.38	(3.44)	**			1.05	(.46)		.98	(.18)		
Friend delinquency (z)	1.72	(5.97)	***						1.41	(3.23)	**	
<i>Intercept</i>				-86	(9.53)	***	-87	(9.49)	***	-88	(9.52)	***
									-7.40	(11.72)	***	

+ p<.1,

* p<.05,

** p<.01,

*** p<.001 (two-tailed). t-ratios are presented in parentheses.

Notes: Variables are entered in groups in models N1-N4. Coefficients for the intercept are logit coefficients; other coefficients are odds-ratios.

- ^aThe other background characteristics were significant so they have been omitted.
- ^bThe other stressor variables were not statistically significant.
- ^cNone of social support measures were statistically significant.