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Factors Associated with Growth in Daily Smoking among Indigenous Adolescents

Les B. Whitbeck, **Kelley J. Sittner Hartshorn**, **Julia McQuillan**, and **Devan M. Crawford**
Department of Sociology University of Nebraska-Lincoln

Abstract

North American Indigenous adolescents smoke earlier, smoke more, and are more likely to become regular smokers as adults than youth from any other ethnic group yet we know very little about their early smoking trajectories. We use multilevel growth modeling across five waves of data from Indigenous adolescents (aged 10 to 13 years at Wave 1) to investigate factors associated with becoming a daily smoker. Several factors, including number of peers who smoked at Wave 1 and meeting diagnostic criteria for major depressive episode and conduct disorder were associated with early daily smoking. Only age and increases in the number of smoking peers were associated with increased odds of becoming a daily smoker.

Even though the health risks associated with cigarette use have been well known for decades, each day about 1,000 teenagers become regular smokers (Centers for Disease Control and Prevention (CDC), 2008). About 26.2% of American Indian/Alaska Native (AIAN) adolescents aged 15 to 17 years, nearly 10% more than the national average, are current cigarette smokers (Substance Abuse and Mental Health Services Administration (SAMSHA), 2011). The rates of smoking among Indigenous adolescents in the northern Midwest are even higher. According to one report, 37% of American Indian adolescents living in Minneapolis-St. Paul used cigarettes in the previous 30 days (Forster, Brokenleg, Rhodes, Lamont, & Poupart, 2008). These early ethnic differences in smoking persist into adulthood. National reports show that in general, AIAN adults smoke at higher rates than any other ethnic group: 36.4% vs. 21.4% for White non-Hispanics, 19.8% of African Americans, 13.3% of Hispanics, and 9.6% of Asians (CDC, 2008). Generalizing about AIAN smoking behaviors should be done with caution, however, because there is no single AIAN "culture." For example, a recent large epidemiological study of multiple cultures reported that 14% of adults in a Southwestern culture were current smokers compared to 50% of adults in a Northern Plains culture (Henderson, Jacobsen, & Beals, 2005).

Indigenous cultures and communities with high rates of smoking are subject to discrepant smoking-related health burdens such as cardiovascular disease (Department of Health & Human Services (DHHS), 2003), lung cancer (CDC, 2003), and type 2 diabetes (Rimm, Chan, Stampfer, Colditz, & Willett, 1995; for a review see Willi, Bodenmann, Ghali, Faris, & Cornuz, 2007). Moreover, tobacco use is a known precursor of other substance abuse (Chen, Unger, Palmer, Weiner et al., 2002; Grucza & Bierut, 2006), and the rates of substance dependence and abuse among AIAN people aged 12 years and older are higher than any other ethnic group (SAMSHA, 2011).

Nicotine is highly addictive. About 32% of those who initiate tobacco use become dependent, compared to 23% of those who experiment with heroin, 16.7% with cocaine, and

Address correspondence to the first author, University of Nebraska-Lincoln, Department of Sociology, 739 Oldfather Hall, Lincoln, NE 68588-0324.Phone: 402 472-5562. Fax: 402 472-4983. lwhitbeck2@unlnotes.unl.edu. .

15.4% with alcohol (Anthony, Warner, & Kessler, 1994). Once addicted to nicotine, it is very hard to quit. More than 85% of people who attempt to quit smoking on their own relapse in the first week (National Institute on Drug Abuse (NIDA), 2009), and even with professional interventions, the relapse rate is 60% to 80% for 6 to 12 months abstinence (Juliano, Donny, Houtsmiller, & Stitzer, 2006). This health risk behavior is difficult to modify because the onset of smoking typically occurs during adolescence, a phase of life when information about health risks are less persuasive than the behaviors of peers and other role models (Collins, Sussman, Rauch, Dent et al., 1987). Early onset of smoking is strongly associated with nicotine dependence (for a review see Hoffman, Sussman, Unger, & Valente, 2006). The process of becoming nicotine dependent among adolescents often progresses from first experimentation in middle school through regular use in high school (Eichner , Cravatt, Beebe, Blevins et al., 2005).

Although AIAN adolescents smoke earlier, smoke more, and are more likely to become regular smokers as adults than those in other ethnic groups, we know very little about their early smoking trajectories. In this study, we use multilevel growth modeling across five waves of data from Indigenous (American Indian and Canadian First Nation) adolescents aged 10 to 13 years at first interview to investigate factors associated with becoming a daily smoker. Early onset daily smokers are an important group to understand because they are most at risk for adult smoking and long-term health consequences.

Early Cigarette Smoking

Adolescent cigarette smoking patterns are very diverse. Classification systems typically range from abstainers to experimental users, and then build according to frequency of use (e.g., weekly or daily use; for a review of stages of adolescent smoking see Mayhew, Flay, & Mott, 2000) or by the number of cigarettes smoked in a given period (Wills, Sandy, Yaeger, Cleary, & Shinar, 2001). As longitudinal studies have become more sophisticated, various smoking trajectories have been identified. Among these trajectories, categories such as “early rapid escalators” (Colder, Mehta, Balanda, Campbell et al., 2001) or “early stable smokers” (Chassin, Presson, Pitts, & Sherman, 2000) show the most rapid progression from experimentation to regular smoking. These are the adolescents most at risk for early nicotine dependence, and, because they are regular smokers at an early age, they will have more years in jeopardy for health problems. Moreover, these rapid escalators to regular smoking may be those who, by heredity and social context, are most resistant to prevention efforts (Lerman & Berrettini, 2003).

Daily smokers or early stable smokers make up the smallest group in the spectrum of adolescent cigarette smokers. According to the CDC, in 2007 only 8.1% of high school students were frequent smokers (smoked cigarettes on 20 or more of the past 30 days). Monitoring the Future findings indicate daily use by 3.1% of 8^{th} graders, 5.9% of 10^{th} graders, and 11.4% of 12^{th} graders (Johnston, O’Malley, Bachman, & Schulenberg, 2009). In their most recent multisite school survey, Beauvais and colleagues (Beauvais, Thurman, Burnside, & Plested, 2007) reported daily smoking rates for American Indian (AI) 12^{th} graders of 16% with no significant differences between AI and non-AI students.

Correlates of Cigarette Smoking among Adolescents

Correlates of daily or frequent smoking differ from those of other categories of smokers. For example, Chassin and colleagues (2000) found that early onset, persistent smokers had the most friends who smoked and were more likely to be from households where parents smoked than were other groups of smokers. Peer pressure may be especially pertinent to AI young people because active refusal or rejection of offers of cigarettes by friends who smoke may violate cultural norms of affiliation, respect, and polite behaviors (Beauvais,

1980). Siblings affect smoking behaviors similarly to non-related peers (Rose, Chassin, Pressen, & Sherman, 1999).

Cigarette smoking is so ubiquitous in some AIAN communities that there are many adult role models who smoke and often little community disapproval or concern. For example, more than three-fourths of AIAN adolescents in the Minneapolis-St. Paul study lived with an adult who smoked and 44% had a sibling who smoked (Forster et al., 2008). AIAN adolescents also are less likely to report parental or peer disapproval of smoking than their non-AIAN counterparts (SAMHSA, 2002). Possibly because of community and family norms, AIAN adolescents are less likely than non-AIAN adolescents to perceive cigarette use as harmful (Beauvais et al., 2007).

Along with modeling of smoking behaviors, early onset smoking is part of a cluster of problem behaviors associated with lack of parental warmth and support, low parental approval, and lack of parental monitoring of children's activities and behaviors. These clusters of problem behaviors emerge through a process of non-optimal parenting often associated with parents who are depressed, substance abusing, and who engage in coercive/aggressive interactions with their offspring. These interaction styles fail to socialize conventional social skills that may result in academic problems and rejection by conventional peers at school. The children drift into affiliations with non-conventional friends who reinforce and enhance antisocial behaviors and substance use (Dodge, Malone, Lansford, Miller, Pettit et al., 2009; Reid, Patterson, & Snyder, 2002). The influence of peers on smoking onset and the transition to regular use is well-established, as evidenced by dozens of studies over three decades in the U.S. and other countries (see review by Kassel, Stroud, & Paronis, 2003). In a recent review, Simons-Morton & Farhat (2010) examined 40 prospective studies of adolescent smoking, of which all but one reported an association between peer smoking and adolescent smoking.

Smoking commonly co-occurs with behavioral problems and psychiatric disorders during adolescence (Lawrence, Mitrou, Sawyer, & Zubrick, 2010), particularly externalizing disorders among boys (Breslau, 1995) and internalizing disorders among girls (Acierno, Kilpatrick, Resnick, Saunders et al., 2000; Needham, 2007; Munafò, Hitsman, Rende, Metcalfe, & Niaura, 2007). Early onset cigarette smoking also foreshadows future psychopathology and substance abuse (for a review see Upadhyaya, Deas, Brady, & Kruesi, 2002). The most consistent evidence links depressive symptoms and nicotine dependence among adolescents and young adults (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Fergusson, Lynskey, & Horwood, 1996). The relationship between smoking onset and depressive symptoms is stronger for adolescent girls than for boys (Acierno et al., 2000; Borrelli, Niaura, Keuthan, Goldstein, et al., 1996; Borrelli, Marcus, Clark, Bock, et al., 1999), a finding recently replicated with Indigenous adolescents (Whitbeck, Yu, McChargue, & Crawford, 2009). Oppositional defiant disorder (ODD), and conduct disorder (CD) have been consistently linked to early cigarette smoking (Breslau, 1995). Symptoms of CD represent a pattern of defiant behaviors such as breaking social norms, disregard for rules, and rebellion against authority that may lead to early initiation of smoking and rapid progression to daily smoking among early adolescents. Smoking cigarettes may symbolize rejection of adult authority and imitate adult behaviors. There also is evidence that adolescents with comorbid CD and ADHD initiate smoking earlier and make an earlier transition to regular use (Riggs, Mikulich, Whitmore, & Crowley, 1999).

Theory and Hypotheses

From a life course and problem behavior perspective, cigarette smoking is part of a cluster of maladaptive behaviors that emerge during adolescence (Donavan & Jessor, 1985; Jessor

& Jessor, 1977). The strong associations between cigarette use, externalization, and substance use suggest that this may be part of a progression of negative behaviors. As use advances to dependence and experimentation with and regular use of other substances emerges, behavioral change may become more and more difficult. Co-occurring externalizing and internalizing disorders further complicate this process. The life course concept of "cumulative continuity" suggests delinquent adolescents may become ensnared in the consequences of their own behaviors, resulting in life-long effects that include psychiatric disorders and substance abuse (Caspi, Elder, & Bem, 1987; Caspi, Bem, & Elder, 1989). At minimum, early cigarette smoking may result in nicotine dependence, which portends serious health problems in later life. At worst, it is linked to internalizing and externalizing symptoms that may have life-long consequences.

Although it is obvious that not all smokers have other substance abuse or mental health problems, there is extensive research linking smoking to psychiatric disorders (see Upadhyaya et al., 2002 for a review). As we have pointed out, this linkage may be particularly important in regards to early onset smoking. Other problem behaviors and meeting criteria for internalizing and externalizing disorders typically precede substance abuse disorders (Kessler, Berglund, Demler, Jin & Walters, 2005). We therefore hypothesize that existing internalizing disorders (e.g., major depressive episode (MDE) and generalized anxiety disorder (GAD)), externalizing disorders (ODD, CD), and ADHD will be associated with cigarette smoking at baseline and that these disorders also will be associated with growth in cigarette smoking across time. Moreover, comorbid disorders will be associated with greater likelihood of smoking at baseline and increases in smoking across time.

The importance of peers in the origination and perpetuation of problem behaviors is universally acknowledged. Friends offer cigarettes, model smoking behaviors, and create and maintain smoking norms such as places and times to smoke. Entire theories of substance abuse have been based on peer associations (e.g., Oetting & Beauvais, 1986; 1987). We have three basic research questions regarding nuances of peer influence. First, does the initial number of deviant peers (Wave 1) modify the trajectory of daily smoking? Second, does change in the number of deviant peers influence daily smoking? Third, do the presence of psychiatric disorders and/or adolescent and family characteristics modify the influence of deviant peers on daily smoking?

Method

Participants and Data

These data were collected as part of a longitudinal study designed in partnership with eight reservations and reserves and a university-based research team. The study focused on risk and protective factors for adolescent mental health and substance abuse problems from early adolescence to young adulthood. The reservations/reserves share a common cultural tradition and language with minor regional variations in dialects. The sample represents one of the most populous Indigenous cultures in the United States and Canada. As part of this partnership's confidentiality agreements, the names of the cultural group and participating reservations and reserves will be not be identified. At each site, Tribal Council appointed advisory boards are responsible for handling personnel difficulties, advising the research team on questionnaire development, and reviewing/approving reports and presentation proposals. All participating staff on the reservations and reserves (i.e., interviewers, site coordinators) were approved by advisory boards and were either enrolled tribal members or spouses of enrollees. Interviewers for this project were trained concerning methodological guidelines of personal interviewing and all were certified for work with human subjects.

Each participating tribe provided us a list of families of tribally-enrolled children aged 10-12 years who lived on or proximate to (within 50 miles) the reservation or reserve. We attempted to contact all tribally enrolled families with a target child within the specified age range in order to achieve a population sample. Families were recruited through personal interviewer visits during which they were presented a traditional gift, an overview of the project, and an invitation to participate. For those families who agreed to participate, both the study adolescent and at least one adult caretaker (and in some cases, two adults) were interviewed annually and each $40 upon completion of the interviews. Recruitment and incentive procedures were approved both by community-based advisory boards and the university IRB. The overall baseline response rate for the study was 79.4%. Subsequent annual retention rates remained quite high (94.6% in Wave 2, 93% in Wave 3, 90% in Wave 5, and 84.5% in Wave 7).

This paper includes youth self-report and parent/caregiver report data from Waves 1, 2, 3, 5, and 7, collected in yearly intervals between 2002 and 2010 (only diagnostic information was collected at Waves 4 and 6). At Wave 1, target adolescents were between 10 and 13 years of age (13-year-olds are those youth who experienced birthdays between recruitment and interview dates). The mean age in the sample was 11 years. There were 746 children interviewed at Wave 1. Only those adolescents for whom we had at least three observations were included in the analysis. Thirty-one adolescents had two or fewer observations and thus were excluded. An additional 41 adolescents were excluded because of missing values on variables in the between-person level of analysis (Level 2), resulting in 674 adolescents and 3,102 total person-observations (within persons at Level 1). The adolescents excluded due to missing values were more likely to live in remote areas, but did not differ significantly from those included in the analyses on any other variables.

Measures

Dependent variable—*Daily smoking* was measured at Waves 1, 2, 3, 5, and 7 by asking adolescents a series of questions to identify whether they smoke cigarettes every day or nearly every day (the questions were not asked in Waves 4 or 6). Respondents were first asked whether they had ever smoked cigarettes. Those who responded “yes” were then asked if they had smoked cigarettes in the past

12 months, and if they had, how often in the past 12 months they smoked. Responses were coded so that 1=daily or almost daily, 0=weekly or less often. Distributions of daily smokers are displayed for males and females in Figure 1. Means and standard deviations of study variables are presented in Table 1.

Time-varying covariates (Level 1)—*Smoking peers* was assessed with one item at each of the five waves of the study. Adolescents were asked how many of their three best friends smoked cigarettes. Response categories range from 0 = none to 3 = all three friends. The mean numbers of best friends who smoke are presented in Figure 2.

Time-invariant covariates (Level 2)—The Diagnostic Interview Schedule for Children-Revised Version II (DISC-R) was used to assess attention deficit hyperactivity disorder (ADHD), two internalizing disorders (*generalized anxiety disorder (GAD)*, *major depressive episode (MDE)*) and two externalizing disorders (, *conduct disorder (CD)*, and *oppositional defiant disorder (ODD)*) at Wave 1. Computer algorithms were used to determine whether each behavior was present at clinically significant frequency/severity levels. We use the combined parent-youth reports, which are more reliable than child reports alone (Schwab-Stone, Shaffer, Dulcan, Jensen, et al., 1996; Shaffer, Schwab-Stone, Fisher, Cohen, et al., 1993). Adolescents meeting criteria for a disorder were coded as 1, and coded as 0 if they

did not meet criteria. Prevalence rates of individual and cooccurring disorders are presented in Table 2.

Initial smoking peers was assessed at Wave 1 with a two item mean scale of peer delinquency. Adolescents were asked how many of their three best friends smoked cigarettes or drank alcohol. Response categories from both questions range from 0 = none to 3 = all three friends. Cronbach's alpha coefficient was 0.83.

Family monitoring was measured as a sum of the number of family members (mother, father, sibling, grandparent, or aunt/uncle) who monitor adolescents' behavior at Wave 1. Adolescents were first asked to identify the person in their family most likely to know where the adolescents were and what they were doing. They were then asked to identify who else would monitor their behavior. Responses range from 0 = none to 5 = five family monitors.

Two parent variables are included. Parent self-reported *Parental depression* and *Parental SUD* were assessed with the University of Michigan Composite International Diagnostic Interview (UM-CIDI). Computer algorithms were used to determine whether each behavior was present at a clinically significant frequency or severity level. Parents meeting clinical definitions for depressive disorder were coded as 1 and those not meeting clinical definitions were coded as 0. A single indicator of substance use disorder was constructed from individual substance-related diagnoses (alcohol abuse, alcohol dependence, and drug abuse). If a caretaker met criteria for one or more of the three substance abuse or dependence diagnoses, he or she was coded as 1; caretakers not meeting criteria were coded as 0. The UM-CIDI is based on DSM-III-R criteria and represents the University of Michigan's revision to the Composite International Diagnostic Interview (CIDI) (World Health Organization, 1990) that was used in the National Comorbidity Study (Wittchen and Kessler, 1994) and the National Comorbidity Study Replication (Kessler, & Merikangas, 2006). As evidenced by its use in these major national epidemiological studies, the UM-CIDI is a well-established diagnostic instrument that has shown excellent interrater reliability, test-retest reliability, and validity for the diagnoses that were used in this study (Kessler, Wittchen, Abelson, McGonagle et al., 1998; Wittchen, Kessler, & Ustun, 2001).

We did not have a measure of parental smoking until the fifth wave (when the adolescents were at least fifteen years old). The measure in the fifth wave showed that over eighty percent of the parents had smoked at some point in their life. Given the difficulty of quitting smoking and the high failure rates, we suspect that the values would have been similar in the initial year (CDC, Nov. 11, 2011) . Despite how ubiquitous parental smoking is, because prior research shows the importance of this variable (e.g., Jackson & Henriksen, 1997), we explored adding this variable to the analysis but found that it was not associated with teen smoking nor did it alter the findings. We therefore show the more parsimonious model without parental smoking status.

Control variables used in the study, all from the first wave of data collection, were gender, per capita family income, and remote location. *Gender* was coded so that 1=female, 0=male. *Per capita family income* was measured by asking parents/caretakers to indicate their overall household incomes as above or below $25,000 in the past year. Two other questions narrow that response to $10,000-$15,000 ranges. Mean imputation was used for missing values on income. Midpoints of these ranges were divided by the number of people living within the household, which was then divided by 1,000 to set the metric of this measure in thousands of dollars. *Remote location* refers to the proximity to towns or other communities, either rural or remote. A rural location refers to those reservations/reserves within somewhat close proximity to other towns, and remote refers to those far removed from other communities. The variable is coded so that 1=remote, 0=rural.

Analysis Plan

To address our hypotheses and research questions, we used growth models in a multilevel framework to estimate the initial and the change in the odds of daily smoking over time. The first level measures involve repeated observations within individuals over time and the second level measures involve stable between individual characteristics measured either in the first wave or as an aggregated average. Multilevel analysis is particularly useful for longitudinal studies because it allows for missing observations at some time points as well as for participants to have different numbers of observations (Luke, 2004). We employ hierarchical generalized linear models (HGLM) with penalized-quasi likelihood (PQL; also known as Generalized Estimating Equations) estimation to appropriately accommodate the lack of independence among the observations clustered within individuals over time, and the dichotomous dependent variable (see Raudenbush & Bryk, 2002 for more details). Unlike models with continuous outcomes, the level 1 variance (R_{ij}) = P (1-P_j) is a function of the mean and is not a free parameter for estimation. The dependent variable is transformed with a logit link function for the estimation; therefore the outcome is the linearized log odds of daily smoking (Lee & Nelder, 1996). Following Diaz (2007), we evaluate the population average coefficients with robust standard errors.

The within person (level 1) model provides estimates of how the odds of daily smoking change over time (the trajectory) and how changes in the number of smoking peers are associated with changes in the odds of daily smoking. The between person (level two) model includes stable individual characteristics measured in the first wave of the study (e.g., parental substance abuse). The HGLM model estimates how individual characteristics are associated with the initial odds of smoking (the intercept model) and the change in the odds of smoking over time (cross-level interactions with the daily smoking growth trajectory) (Snijders & Bosker, 1999).

All variables are grand mean centered at level two, and there is one random effect (the intercept). Centering facilitates interpretation of direct and interaction effects and reduces multicollinearity (Bickel, 2007; Nezlek 2001). We follow the practice described in Raudenbush and Bryk (2002) called “parallel models”, and thus have the same cross level interactions for the growth curve (by age-10) and for the measure of the change in number of smoking peers. This approach has the benefit of facilitating interpretation by ensuring the same comparison group for all of the coefficients. Time-varying smoking peers is group-mean centered at Level 1, within adolescents over time because it is appropriate for cross-level interactions (Enders & Tofighi, 2007). Group mean centering, however, measures deviations from each adolescent’s own mean, therefore preventing estimation of overall level of time-varying characteristics. Thus we include grand mean centered initial smoking peers at level two to estimate the overall level of smoking peers. Separating this one variable into two parts and including them both in the analyses simultaneously allows us to estimate the effects of changes in and overall level of smoking peers on daily smoking (Kreft, De Leeuw, & Aiken, 1995). The baseline model is summarized in the following equation with only an intercept and error term:

$$\text{Prob}(\text{Daily smoking}=1|\pi) = p$$
$$\text{Log}[P/(1-P)] = \pi_{00} + r_0$$
$$\text{Level 1 variance} = 1/[P(1-P)]$$

First, we evaluated an unconditional growth model (not shown in Table 2) using the following equations: Level 1: Within Person Changes

$$\text{Log}\,[P/(1-P)] = \pi_{0j} + \pi_{1j}^{*}\,(\text{Age} - 10)$$

Level 2: Between Person Differences

$$\pi_0\,(\text{Initial log odds}) = \beta_{00} + r_{0j}$$
$$\pi_1\,(\text{Growth trajectory}) = \beta_{10}$$

Age is included as the growth term, centered at age 10 (the age of the youngest adolescents in the sample) for individual *j*. In the level two equations, β_{00} and β_{10} indicate the odds of daily smoking at age 10 and the rate of growth in daily smoking for each year past age 10, respectively. The error term for the intercepts, r_0, gives an estimate of the variance in the odds of daily smoking between adolescents (Snijders & Bosker, 1999; Luke, 2004; Raudenbush & Bryk, 2002).

In Model 1 we include the invariant family characteristics and control variables at the person level (level two). To calculate odds ratios for the cross-level interactions, the log odds of a level 2 predictor must be added to the slope, the sum of which is transformed into the odds through exponentiation. For example, to calculate the trajectory of smoking for females, the coefficient for female is added to the coefficient for daily smoking trajectory and then exponentiated into the females' odds of daily smoking for each year past age 10. In this and all subsequent models, the estimated odds of daily smoking are the expected odds when the predictor values are all 0.

In Model 2, we add individual and comorbid mental disorders as person characteristics at level two. These measures assess the association between mental disorders and the initial odds of daily smoking, as well as whether the disorders modify the daily smoking trajectory. Based on the comorbidity and smoking literature, we also include co-occurring ADHD and any other disorder.

In Model 3 we add a measure smoking peers to the within person change model at level one (π_{2j}) to assess if changes in the number of smoking peers is associated with changes in the odds of daily smoking. We include initial smoking peers in the between person level two equations to estimate the direct association with initial odds of smoking, as well as the modifying effects for the daily smoking trajectory or the change in smoking peers over time. In addition, we assess whether mental disorders modify the association between smoking peers and daily smoking. Below we provide the full equation for the third and final model; the prior models are nested within this one: Level 1: Within Person Changes

$$\text{Log}\,[P/(1-P)] = \pi_{0j} + \pi_{1j}^{*}\,(\text{Age} - 10) + \pi_{2j}^{*}\,(\text{smoking peers})$$

Level 2: Between Person Differences

$$\begin{aligned}\pi_0\,(\text{Initial log odds}) = {} & \beta_{00} + \beta_{01}(\text{female})_j \\ & + \beta_{02}(\text{per capita income})_j \\ & + \beta_{03}(\text{remote location})_j \ldots \\ & + \beta_{16}(\text{initial smoking peers})_j + r_{0j}\end{aligned}$$

$$\pi_1\,(\text{Growth trajectory}) = \beta_{10} + \beta_{11}(\text{female})_j(\text{Age} - 10)_j + \beta_{12}(\text{per capita income})_j(\text{Age} - 10)_j + \beta_{13}(\text{remote location})_j(\text{Age} - 10)_j \ldots + \beta_{116}(\text{initial smoking peers})_j(Age - 10)_j$$

$$\pi_2\,(\text{Smoking peers slope}) = \beta_{20} + \beta_{21}(\text{female})_j(\text{smoking peers})_j + \beta_{22}(\text{per capita income})_j(\text{smoking peers})_j + \beta_{23}(\text{remote location})_j(\text{smoking peers})_j \ldots + \beta_{26}(\text{initial smoking peers})_j(\text{smoking peers})_j$$

Results

Trajectories of Daily Smoking

We began the analyses by estimating an unconditional growth model. As shown in Figure 3, with only age (the growth factor) included in the model, the predicted probability of daily smoking increased steadily with adolescent age. We then added family characteristics and control variables (Model 1, Table 3). Although girls started out with odds of daily smoking that were 73% higher than boys' odds, there was no significant gender difference in the trajectory of daily smoking. In addition, adolescents who had a parent with depression were two and one half times more likely to smoke daily at Wave 1 (OR =2.64). Controlling for gender and family characteristics, the odds of becoming a daily smoker increased by 67% for each year past age 10. Neither gender nor any of the family characteristics modified the growth trajectory.

Adolescent individual and comorbid mental disorders were included in Model 2. We found mixed support for the hypotheses that existing disorders and comorbidity would be associated with cigarette smoking at Wave 1, and that these disorders would be associated with growth in cigarette smoking across time. Because the sample size is modest and odds ratios can be artificially inflated with smaller samples, we focus on the direction and significance but not the magnitude of the coefficients for the psychiatric diagnoses (Nemes, Jonasson, Genell, & Steineck 2009). Compared to adolescents without any disorder, those with MDE and CD had significantly greater odds of daily smoking at Wave 1. Contrary to our expectations, meeting criteria for ADHD and any other disorder was associated with decreased odds of daily smoking at Wave 1, although meeting criteria for ADHD alone was not associated with daily smoking.

In addition to modifying initial smoking status, individual and comorbid disorders also modified the trajectory of daily smoking (Model 2, Table 3). For adolescents with no disorder, the odds of daily smoking increased by 70% for each year past age 10 years (OR =1.70). Contrary to our hypotheses, however, trajectories were flatter for adolescents with MDE and CD. That is, the odds of becoming a daily smoker increased for adolescents with MDE and CD but at a slower rate than for adolescents with no disorders.

To answer our research questions regarding the influence of smoking peers on daily smoking, time-varying smoking peers and initial number of smoking peers were included in Model 3. Adolescents with an above-average number of peers who smoked at Wave 1 were 68% more likely to smoke daily at Wave 1 (OR =1.68). Initial smoking peers did not,

however, influence the trajectory of smoking. It should be noted that once smoking peers were included in the model, none of the psychiatric disorders remained significant. That is, the influence of smoking peers trumped the effects of psychiatric disorders on growth in daily smoking even though having above average numbers of smoking peers at Wave 1 did not influence the growth rate.

Additionally, an increase in the number of peers who smoked cigarettes also was associated with increased odds of daily smoking. For each additional smoking peer above an adolescent's own average, the odds of daily smoking increased more than twofold (OR =2.44). The effect of change in smoking peers was weaker for adolescents who had above average numbers of family monitors (OR =.90), for adolescents who had a parent with SUD (OR =.51), for adolescents who met criteria for MDE (OR =.58), and for adolescents who had above average number of smoking peers at Wave 1 (OR =.89).

Discussion

In this AI culture in the northern Midwest and Canada, beginning at age 10 years, girls were more much more likely than boys to be regular smokers, even though the rate of growth across time did not differ by gender. Gender interactions with the additional Wave 1 variables in the model did not account for this gender difference but more specific models that focus on depressive symptoms and smoking indicate that depressive symptoms have a greater effect on growth for girls' smoking than for boys (Whitbeck et al., 2009). Most of the effects of parents are likely indirect through their effects on adolescent internalizing and externalizing disorders (Dodge et al., 2009; Patterson, 1986; Patterson, Dishion, & Bank, 1984; Reid et al., 2002), which typically precede the onset of regular smoking. Adolescents with a parent who met criteria for MDE were more likely to be regular smokers at Wave 1, but this appears to be limited to early onset of regular smoking and does not affect growth in the odds of an adolescent becoming a regular smoker over time.

For adolescents with no psychiatric disorders, the odds of becoming a regular smoker increased by more than 50% each year after age 10 years. Although those who met criteria for MDE and CD were much more likely to be regular smokers initially, the presence of these disorders did not amplify smoking risk across time above that of those with no disorders. The magnitude of the odds ratios may be attributable to small cell sizes, but the effects of early psychiatric disorders on early regular smoking were very robust. Adolescents who met criteria for MDE or CD at Wave 1 were substantially more likely to be regular smokers than those who did not meet diagnostic criteria. ADHD comorbidity, however, did not inflate the odds of the onset of regular smoking at Wave 1.

As anticipated, peer influence was strongly associated with both the early onset of regular smoking and with smoking trajectories. At Wave 1, adolescents with more friends who smoked were more likely to be regular smokers. Each additional smoking friend above an adolescent's own average number of smoking friends more than doubled the odds of becoming a regular smoker. Moreover, when smoking peers were added to the models, the across time effects of psychiatric disorders were lost. The effects of meeting criteria for MDE and CD were subsumed by increasing associations with smoking friends. These findings may be attributable to peer processes among some AIAN cultures. AIAN teens may be more vulnerable to peer influence than those in other cultures due to the presence of kin, small communities, and norms of affiliation, respect, and courtesy (Beauvais, 1980).

Limitations

Although these findings are from a population sample of one of the largest Indigenous cultures in North America, they are from a single culture. As noted, the prevalence of

smoking varies widely across Indigenous cultures and communities so that in some cultures adolescents may be more exposed to cigarette smoking than in others. Moreover, all of this information comes from rural and remote reservations and reserves and may not represent urban Indigenous adolescents even from the same cultural heritage. There is always the important caution that diagnostic criteria are not normed for Indigenous adolescents. With respect to our omission of parental smoking from the analytic models, we determined that there was so little variation in parent/caretaker smoking that the effects were nonsignficant. Because almost all (80%) of the study adults smoked, both smoking and non-smoking adolescents were likely to have grown up in families where one or both parent/caretakers were smokers. We suspect that this variable is less relevant in this particular Indigenous population than in populations in which parent/caretaker smoking is less common. The adolescent smoking data were based on self-reports of adolescents. They may understate whether they smoke or not and/or how often they smoke. Our peer measures were limited to adolescent reports of their three best friends and did not assess the adolescents' entire social networks. Regardless of these limitations, the findings are an important step forward in understanding the comorbidity of cigarette smoking and psychiatric disorders among Indigenous young people.

Conclusions

Health researchers have fallen behind in addressing the factors that cause Indigenous people to have the highest smoking rates of any ethnic group in the United States and Canada. At the first wave of the study, only two percent of the adolescents were daily smokers. The rates of regular smoking increased rapidly after age 10 years until, by age 19, sixty percent of girls and half of boys were daily smokers. About 90% of smokers begin smoking tobacco by age 18 years (NIDA, 2011), so it reasonable to predict that most of these young people will be adult smokers. The presence of comorbid psychiatric disorders makes quitting even more difficult (Upadhyaya et al., 2002). This portends risk for all the smoking-related health problems including heart disease, cancer, and type 2 diabetes (Rimm et al., 1995; for a review see Willi et al., 2007).

Our findings have several implications for smoking prevention in AIAN communities. First, regular smoking occurs early among AIAN adolescents and progresses very rapidly. Because of the addictive properties of tobacco, early (pre-adolescent) universal interventions are a must. Second, the key to prevention may be in identifying malleable risk factors, such as cooccurring early oppositional behaviors and depressive symptoms. For example, there may be separate trajectories to regular smoking among AIAN adolescents, one an internalizing path (predominantly girls, see Whitbeck, et al., 2009) and another, a rebellious, oppositional path (predominantly boys, see Breslau, 1995). Moreover, given the increasing agreement across studies of co-occurring psychiatric disorders and early smoking (Upadhyaya et al., 2002), it may make sense to clinically target those most at risk for early smoking and the progression to nicotine addiction. Adolescents who manifest early symptoms of conduct problems and depression are highly likely to smoke. If they present for treatment or come under the purview of the social services or criminal justice systems, smoking prevention could be part of the intervention.

Third, early smoking is a social behavior, which is also a malleable risk factor. Even though early onset smoking was associated with early internalizing and externalizing problems, teen social networks overrode these factors in their influence on smoking trajectories over time. Smoking in social networks increases the likelihood and regularity of smoking. These smoking networks may be self-reinforcing and serve to support each others' opposition to universal anti-smoking campaigns and warnings. Future research should focus on understanding AIAN adolescent social network patterns including gender variations in

network configurations and influence, and potential cultural differences in network influence. This information could guide innovative culturally based smoking interventions. Addressing the smoking networks as a group may be an effective approach that would fit with AIAN cultural norms of inclusiveness and community.

Although the long-term health consequences are enormous and may account for a significant proportion of the health disparities experienced by Indigenous communities there is little sense of concern or urgency about adolescent smoking. Because cigarette smoking begins and progresses to nicotine addiction so early in life it has been called a “pediatric disease” (Upadhyaya et al., 2002). It is preventable, but we need to raise awareness and concern in AIAN communities.

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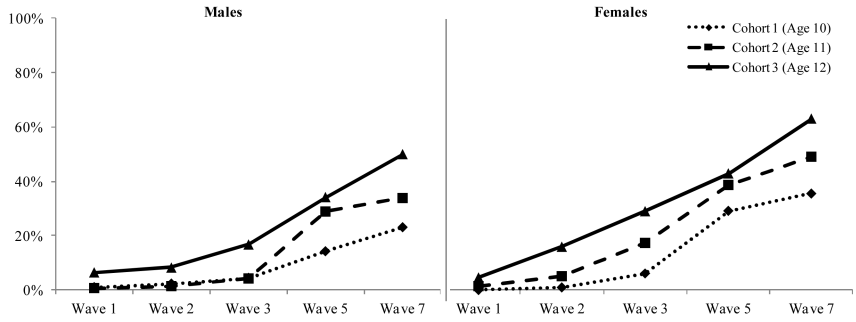

Figure 1.
Percent of male and female daily smokers – by cohort and wave.

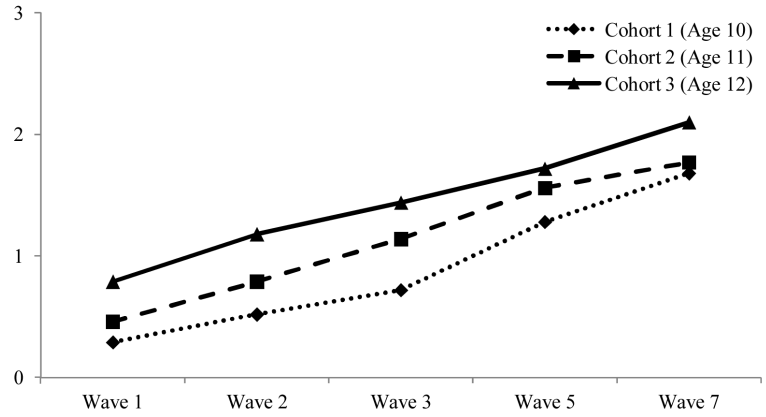

Figure 2.
Mean number of best friends who smoke – by cohort and wave.

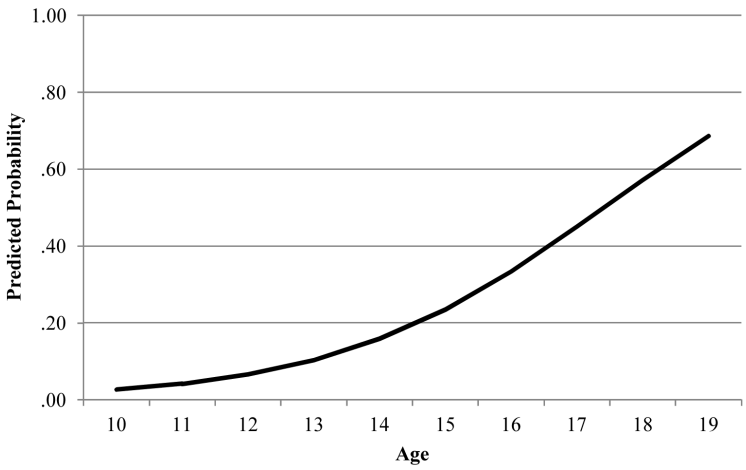

Figure 3.
Unconditional growth in the probability of daily smoking.

Table 1

Descriptive Statistics for All Study Variables at Wave 1 (N=674)

Variable	*M*	*SD*	%
Initial Age	11.06	0.77	
Female			49.40
Per Capita Income	$5,669	$4,860	
Remote Location			8.5
Family Monitoring	2.72	1.15	
Parental SUD[a]			73.6
Parental MDE[b]			20.3
GAD[c]			3.4
MDE[b]			3.7
ODD[d]			8.0
CD[e]			9.2
ADHD[f]			9.8
Initial Smoking Peers	0.52	0.92	

[a] *Note*. Substance use disorder;

[b] Major depressive episode;

[c] Generalized anxiety disorder;

[d] Oppositional defiant disorder;

[e] Conduct disorder;

[f] Attention deficit/hyperactivity disorder.

Table 2

Distribution of Mental Disorders at Wave 1 (N=674)

	N	%
None	518	76.9
GAD[a] only	11	1.6
MDE[b] only	13	1.9
ODD[c] only	32	4.7
CD[d] only	34	5.0
ADHD[e] only	30	4.5
ADHD[e] with any disorder	36	5.3

[a] *Note*. Generalized anxiety disorder;

[b] Major depressive episode;

[c] Oppositional defiant disorder;

[d] Conduct disorder;

[e] Attention deficit/hyperactivity disorder.

Table 3

Growth Curve Model of Daily Smoking (n=674)

	Model 1: Growth Model			Model 2: Addition of Mental Disorders			Model 3: Addition of Smoking Peers		
Predictor	***B***	***SE***	***OR***	***B***	***SE***	***OR***	***B***	***SE***	***OR***
Fixed Effects									
Initial Status	−3.83***	0.11	0.02	−3.98***	0.10	0.02	−3.94***	0.11	0.02
Female	0.55*	0.22	1.73	0.57**	0.21	1.77	0.44†	0.23	1.55
Family Monitoring	0.02	0.08	1.02	0.00	0.09	1.00	0.00	0.10	1.00
Parental SUD[a]	0.20	0.24	1.22	0.17	0.24	1.19	0.28	0.28	1.32
Parental MDE[b]	0.97***	0.26	2.64	0.68**	0.25	1.97	0.62*	0.26	1.86
GAD[c] only				0.24	0.46	1.27	0.14	0.42	1.15
MDE[b] only				1.77***	0.46	5.87	1.31**	0.42	3.71
ODD[d] only				0.60	0.45	1.82	0.44	0.49	1.55
CD[e] only				1.20**	0.36	3.32	0.86*	0.39	2.36
ADHD[f] only				0.38	0.68	1.46	0.10	0.65	1.11
ADHD[f] with Any Disorder				−1.35†	0.82	0.26	−0.55	0.84	0.58
Initial Smoking Peers							0.52***	0.11	1.68
Daily Smoking Trajectory	0.51***	0.02	1.67	0.53***	0.02	1.70	0.43***	0.02	1.54
Female	0.02	0.04	1.02	0.02	0.04	1.02	0.02	0.05	1.02
Family Monitoring	−0.01	0.02	0.99	−0.01	0.02	0.99	0.00	0.02	1.00
Parental SUD[a]	0.00	0.05	1.00	0.00	0.05	1.00	0.05	0.05	1.05
Parental MDE[b]	−0.05	0.05	0.95	−0.01	0.05	0.99	−0.02	0.05	0.98
GAD[c] only				0.00	0.09	1.00	−0.05	0.10	0.95
MDE[b] only				−0.14†	0.08	0.87	−0.03	0.08	0.97
ODD[d] only				0.04	0.09	1.04	0.04	0.10	1.04
CD[e] only				−0.12†	0.07	0.89	−0.08	0.08	0.92
ADHD[f] only				−0.13	0.11	0.88	−0.06	0.11	0.94

Predictor	Model 1: Growth Model			Model 2: Addition of Mental Disorders			Model 3: Addition of Smoking Peers		
	B	***SE***	***OR***	***B***	***SE***	***OR***	***B***	***SE***	***OR***
ADHD[f] with Any Disorder				0.24	0.15	1.27	0.10	0.19	1.11
Initial Smoking Peers							0.02	0.02	1.02
Change in Smoking Peers							0.89***	0.07	2.44
Female							0.07	0.12	1.07
Family Monitoring							−0.10†	0.05	0.90
Parental SUD[a]							−0.67***	0.14	0.51
Parental MDE[b]							0.00	0.15	1.00
GAD[c] only							0.18	0.31	1.20
MDE[b] only							−0.55*	0.22	0.58
ODD[d] only							0.22	0.21	1.25
CD[e] only							0.21	0.19	1.23
ADHD[f] only							−0.15	0.23	0.86
ADHD[f] with Any Disorder							−0.05	0.42	0.95
Initial Smoking Peers							−0.12*	0.05	0.89
Random Effects									
Initial Status		**1.99** ***			**1.88** ***			**1.73** *	

Unconditional Growth Model: Intercept - B=−3.63, SE=0.10, Exp(B)=0.03; Slope - B=0.49, SE=0.03, Exp(B)=1.62

Note. Per capita family income and Remote location are controlled for in all models.

[a]Substance use disorder;

[b]Major depressive episode;

[c]Generalized anxiety disorder;

[d]Oppositional defiant disorder;

[e]Conduct disorder;

[f]Attention deficit/hyperactivity disorder.

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*** p<.001,

** p<.01,

* p<.05,

† p<.10