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Marijuana use development over the course of adolescence among North American Indigenous youth

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Abstract

This study investigated the links between marijuana use trajectories and marijuana abuse/dependence (DSM-IV) using five waves of data from 718 North American Indigenous adolescents between 10 and 17 years from eight reservations sharing a common language and culture. Growth mixture models indicated that 15% of youth began using by 11–12 years of age and that another 20% began shortly thereafter. These early users had odds of abuse/dependence 6.5 times larger than abstainers. Girls were also unexpectedly found to be particularly at risk of early use, and this did not reflect other background and psychosocial factors, including friend use. While the timing, patterns, and consequences of use were similar to those reported for alcohol use previously, the social influences on use differed in important ways.

Keywords

Adolescent; Indigenous; Native American; Marijuana use

1. Introduction

Although alcohol use has garnered the majority of the attention dedicated to risk behaviors in North American Indigenous (NAI) communities (May, 1994), marijuana is the most commonly used illicit substance (Grob and Dobkin de Rios, 1992). One recent developmental study shows that negative live events and discrimination influence feelings of anger and associations with delinquent friends to increase alcohol use early in life, dramatically elevating risks for alcohol abuse and dependence (Cheadle and Whitbeck, 2011). Moreover, the risk of drinking prior to age 13 is higher for girls than boys. Yet comparable studies distinguishing marijuana use development have yet to be undertaken, even though delineating the progression of risk behaviors across many domains beyond alcohol use is needed for substance use prevention and intervention strategies for at-risk NAI youth. This paper builds from and extends prior work on NAI substance use by examining developmental typologies of early marijuana use progression and later abuse and dependence using growth mixture models (Muthén, 2001, 2004), combined with an etiological analysis informed by several sociological perspectives.

NAI adolescent marijuana use fluctuations correspond to those in the general population (Beauvais et al., 1989), but prior year usage rates (23.5% vs. 13.9% for whites) and disorder prevalences (ages 12–17 population estimate is 6.2% vs. 3.7% for whites) are higher than for

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other groups (Wu et al., 2011). Though marijuana use is not related to some of the social consequences that alcohol use precipitates (e.g., violence; Greenfeld, 1998), heavy marijuana users exhibit decreased cognitive performance even after periods of abstinence (Bolla et al., 2005), which may indicate neuromaturational deficits for those who begin using it early in life (Medina et al., 2007). Moreover, marijuana use can have lifelong consequences by influencing academic performance, antisocial behaviors, and other drug use (Novins and Baron, 2004; Novins et al., 2001; Novins and Mitchell, 1998). Given the risks associated with early marijuana use, understanding the developmental course of its use over adolescence is the principal focus of this investigation.

To this end, we orient the first set of research goals around describing the timing, shape, and consequences of 'early use' patterns from ages 10 to 14 in one of the most populous Indigenous cultures in the United States and Canada. After describing early use typologies, we then use these developmental trajectories to predict whether early marijuana use leads to 'problem use,' measured using lifetime DSM-IV marijuana abuse and dependence, in late adolescence (15–17 years).[1] The second set of research goals situates the timing and patterns of use into an etiological analysis guided by a model incorporating 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 proximate intermediate psychosocial developmental pathways (e.g., Agnew and White, 1992). The second set of research goals thus builds a mediational developmental model of marijuana use over the course of adolescence that illuminates how social factors propagate through NAI adolescents' early marijuana use to promote risky pre-adult use.

2. Literature review

Though NAI communities are highly heterogeneous across the more than 560 federally recognized tribal governments in the US (Bureau of Indian Affairs, 2007) and 615 First Nations communities in Canada (Indian and Northern Affairs Canada, 2003), NAI high school seniors are about three times more likely to have used marijuana within the last 30-days than other youth (Mitchell and Plunkett, 2000). Despite wide variation in substance use patterns between cultures and among communities within cultures (Whitbeck et al., 2006a,b; Mitchell et al., 1999), reservation youth appear to use more than non-reservation NAI youth, who in turn use more than European American adolescents (Beauvais, 1992). NAI adolescents in some communities thus appear to begin using marijuana at younger ages (Kosterman et al., 2000), which may result in earlier onset of substance use problems than in the general population (Whitbeck et al., 2008).

Longitudinal studies of NAI and non-NAI populations suggest that early marijuana use is associated with persistence, abuse, polysubstance use, and other harmful social consequences (Ehlers et al., 2007; Kandel et al., 1992; Hix-Small et al., 2004; Schulenberg et al., 2005). Thus, the first part of the analysis describes developmental trajectories over early adolescence (ages 10–14) using semi-parametric growth modeling to capture the timing and patterns of early use. We subsequently consider whether early marijuana smoking leads to elevated and problematic use reflecting broader impacts on youths' lives by linking early use trajectories to lifetime marijuana abuse and dependence measured in later adolescence (ages 15–17). Given the evidence that youth in some NAI populations are

[1]As one Reviewer pointed out, the term 'problem use' suggests that some level of use is not problematic. This reflects our use of the DSM-IV diagnostic criteria as an indicator of whether marijuana is causing problems in adolescents lives (e.g., spending a great deal of time obtaining it, greater tolerance, failure to fulfill major role obligations, continued use despite problems caused or exacerbated by its use, etc.), and thus whether adolescents are not having problems. Though marijuana use is illegal, it is undoubtedly true that many people who use it, just as with other substances (e.g., alcohol) and behaviors (e.g., gambling), turn out to do just fine and that using it does not cause substantial problems in life for them.

early marijuana users, and that the parents and caretakers of the youth in this study have high rates of substance use problems themselves (Whitbeck et al., 2006a,b), using marijuana early may lead to problematic use and associated consequences in later adolescence, just as they begin life stage and role transitions to early adulthood.

3. Theoretical frameworks

The first framework we use to guide the etiological analysis, the *fundamental causes of disease*, is predicated on the observation that social inequality puts individuals at risk of risks (Link and Phelan, 1995). NAI peoples have experienced generations of oppression (e.g., Americanization "civilizing" policies), including loss of land and culture, economic marginalization, and political disenfranchisement. These historical processes have generated inequality within and across NAI communities, and relative to the general public (Duran and Duran, 1995). Economic opportunities in many NAI communities are poor (Gregory et al., 1996), educational attainment is low, crime and risk of victimization rates are high (Greenfeld and Smith, 1999), alcohol, drug, and mental health problems are widespread (Beals et al., 2005; Beauvais, 1998), and social disorganization is endemic (Indian Health Services, 1997; Sandefur et al., 1996).

Although many of these factors are present in the communities in this study (Whitbeck et al. 2006a,b), we have only a limited ability to examine variations across communities due to the data collection design. Therefore, fundamental causes are operationalized as family stratification (i.e., parent education, income, and employment) and ascribed characteristics (i.e., gender and age; i.e., Walls, 2008) following Pescosolido et al. (2008) and McLeod and Nonnemaker (2000). These stratification characteristics can directly increase negative coping behaviors like marijuana use (i.e., Mirowsky and Ross, 2003) as a response to the associated negative stressors (Pearlin et al., 1981), lowered social support (Turner and Marino, 1994), and resulting distress (e.g., Kessler and Neighbors, 1986).

According to the next framework we employ, the *stress process*, negative stressors arising from fundamental social causes and other sources induce stress that people respond to internally (e.g., HPA-axis cascades; Booth et al., 2008) and via expressed behaviors (see Pearlin, 1999). As Pescosolido et al. (2008, p. S176) write, "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." From this perspective, the stress process is critical to NAI adolescent marijuana use because proximal (e.g., financial strain, discrimination, recent negative events) and parenting stressors (e.g., parent history of drug abuse and poor mental health) promote negative coping behaviors, such as increased marijuana use at early ages, in order to reduce the stress, pain, and frustration of life circumstances (e.g., Lindenberg et al., 1994; Agnew and White, 1992).

Specifically, reservation NAI adolescents experience chronic financial strain, violence, negative life events, and discrimination (Trosper, 1996; Bachman, 1992; Kessler et al., 1999; Williams et al., 1997). Evidence from this NAI culture shows that perceived discrimination is an important contributor to both early alcohol use and alcohol disorders in later adolescence (Cheadle and Whitbeck, 2011), as well as to depressive symptoms among adolescents and adults (Whitbeck et al., 2002). Parent substance abuse may be a particularly potent source of stress because it leads to ineffective parenting and decreased social support (Conger, 1997). The youth in this study have parents with high rates of alcohol abuse, dependence, and mental health problems, each of which can be important sources of stress for their children (Whitbeck et al., 2006a,b), and thus contributors to risk behaviors such as marijuana use.

However, relationships with significant others such as family members, friends, and school personnel can provide resources that protect youth from the negative consequences of stress (see Thoits, 1995), and so we also consider the *social support* framework. Adolescents view two-parent families to be warmer and more supportive than single-parent families (McLanahan and Sandefur, 1994) and also report higher levels of school adjustment (Astone and McLanahan, 1991). This suggests that youth in some alternative family formations receive less family-based social support. At the same time, families may be stronger deterrents to substance abuse among NAI than European American children (Swaim et al., 1993). These social supports can mediate fundamental cause and social stress processes (see Ensel and Lin, 1991) and may delay marijuana use initiation and also prevent the escalation of use to more harmful levels. Therefore, we include family structure, parental warmth and approval, and positive school-based experiences as indicators of social support.

The argument so far is that NAI marijuana use is a response to aversive social conditions arising from fundamental social causes, social stressors, and low levels of social support (e.g., Stockdale et al., 2007). Together, these factors comprise a socioenvironmental model that influences marijuana at least in part through *psychosocial intermediate pathways* reflecting psychological states housed within the individual but responsive to these broader social forces. These intermediate pathways are manifestations of distress and strain that some adolescents address by using marijuana as a negative coping mechanism (Powers, 1987; Agnew, 1992). It is through these pathways that contemporaneous stressors like negative life events and cultural stressors such as discrimination and cultural losses are associated with NAI substance abuse (Walters et al., 2002).

For example, discrimination is an external stressor that can be experienced internally as emotional distress (Whitbeck et al., 2002), and it can have consequences for orientations to the outside world by decreasing school attachment (e.g., DuBois et al., 1992; Crawford et al., 2010). Because the school can provide social support, facilitating school attachment may be a protective intermediate mediating pathway (e.g., Bryant et al., 2003), while depression and feelings of anger indicate manifestations of distress that individuals attempt to mediate through negative health behaviors (e.g., Deas and Thomas, 2002). Delinquent peer affiliations are an additional risky psychosocial factor situated at the intersection of individual agency and friendship opportunities. Friends' delinquency is generally considered a major factor for early alcohol and drug use (Conger, 1997; Dinges and Oetting, 1993). In some NAI cultures substance use is considered indicative of adulthood (Topper, 1980) while active refusal or rejection of drug-using friends can be viewed as rude and confrontational (Beauvais, 1980; Weibel-Orlando, 1984).

2.1. This study

By addressing the first set of goals, this study contributes to the existing literature on health risk behaviors among minority youth by showing the extent to which early marijuana use trajectories are precursors to later health and substance use problems among at-risk NAI youth. This part of the analysis echoes the life course view that the developmental impact of life events depends upon their timing (Elder, 1998).

The second set of goals contributes to understanding the etiological backdrop of this process and considers (a) the *fundamental causes of disease* arising from structural constraints and circumstances affecting local environments and the people within them (Link and Phelan, 1995); (b) more proximate *stress processes* linking these external factors to individuals' inner lives (e.g., Pearlin, 1989); (c) the *social supports* mediating these processes (Turner, 1999); and (d) the *intermediate psychosocial pathways* capturing how multiple overlapping behavioral and psychological trajectories influence adolescent health behaviors. The

etiological analysis is thus concerned with how these factors promote problem use in adolescence through early use patterns.

Taken together, the analysis we present builds an integrated socio-environmental model of adolescent marijuana use development among reservation NAI youth, who are among the most disadvantaged groups in the US and one of the least studied sociologically. Moreover, this work builds from prior work on alcohol use (Cheadle and Whitbeck, 2011), and so allows the contributions and developmental processes across outcomes to be compared, thereby providing a richer understanding of the sociological backdrop of NAI adolescent substance use and facilitating the design of culturally appropriate intervention strategies (i.e., Hawkins et al., 2004).

4. Methods

4.1. Sample

The data for this project were collected in partnership with the participating communities under an agreement specifying confidentiality of culture, reservation, and reserve. The design is a multi-year lagged sequential study on four American Indian reservations in the Northern Midwest and four Canadian First Nation reserves. Three of the Canadian Reserves are classified as “remote” because they are considerable distances from even small towns and are accessed by non-paved roads. The communities are from one the most populous Indigenous cultures in the United States and Canada and 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. Retention rates for Waves 2 through 6 ranged from 94.6% at Wave 2 to 87.7% at Wave 6. There were 722 adolescents (96.7%) with at least three observations, and 702 (94%) with at least 4.

Measures of marijuana use frequency were collected in Waves 1, 2, 3 and 5 and the DSM criteria come from the Wave 6 diagnostic interview. The baseline sample was 747 cases, but was reduced to 718 after dropping cases with fewer than two observations for the repeated marijuana use measures. Youth with fewer than two repeat observations were removed to ensure that each case was able to provide trajectory information. By Wave 6 the available sample with DSM-IV marijuana abuse and dependence measures was 652 cases.[2] Because there was also missingness on the independent variables, which were all collected at Wave 1, multiple imputation was used to maintain the covariance structure of the data[3] (Little and Rubin, 2002). The proportion of nonmissing cases for the full 718-case sample is reported along with the descriptive statistics in Table 1.[4] These descriptive results indicate that only small amounts of missingness generally needed to be imputed for the independent variables.

[2]The over-time average marijuana use is 2.5 for the youth with observed DSM scores and 2.55 for the 89 youth with missing DSM diagnoses.

[3]The multiple imputation model included the independent variables used in the analysis, the probability of trajectory classification from a baseline growth mixture model, the repeated marijuana use measures, and indicators for marijuana dependence and abuse diagnosis. A chained equations approach was implemented in ICE for Stata so that the imputations reflect the appropriate measurement level (e.g., the abuse/dependence measure was imputed using logistic regression; see Royston, 2004). The analyses presented used the observed unimputed dependent variables, however, and so the models utilize multiple imputation for the covariates and full information maximum likelihood for early marijuana use development and DSM diagnosis. Thus, all 718 children contribute to the estimation of the growth mixture models, but only the 652 cases with diagnostic information contribute to the estimation of parameters for problem use.

[4]The $N = 470$ for marijuana use at age 11 in Table 1 is because some youth were older than 11 at Wave 1 and thus are missing by definition, so the%Ncolumn overstates missingness.

4.2. Measures

The analysis uses two dependent variables. The first is frequency of marijuana 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 10–13[5] years of age at the first assessment and contributed observations at subsequent waves up until they reached 14-years of age. The second dependent variable, problem marijuana use, is a combined measure of abuse[6] and/or dependence[7] based upon DSM-IV (APA, 2000) criteria from a diagnostic inventory administered at Wave 6 when the youth were aged 15–17 years.[8] As a diagnostic measure, this variable gauges whether marijuana use is causing "significant substance- related problems" (APA, 2000, p. 192), such as an inability to control its use, and/or "recurrent and significant adverse consequences" (APA, 2000, p. 198), such as trouble at work, school, or home, in late adolescence. At Wave 6 over 27% of the sample met the joint abuse/dependence criteria (178 of 652), of whom 23% met criteria for dependence (and, by definition, abuse), and 10% met criteria for abuse without dependence.

Given the way that these variables are constructed, younger children thus contribute more observations to the early use trajectories since it is their observations that provide the most information for understanding trajectories early in life.[9] We have adopted this age range, 10–14 for early use and 15–17 for problem use, to clearly delineate the causal ordering from early growth trajectories to later use.[10] In this way, the representation of these measures within the models we estimate addresses the research goals to describe the timing, shape, and consequences of early marijuana use for later problem use.

4.3. Parent and child variables

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

4.3.1. Fundamental causes—We include status characteristics for *age* in the problem use model series to account for additional heterogeneity not captured in the growth mixture model structure and an indicator for *female*. Additional fundamental cause indicators reflect class-based structural inequalities. We include two measures of family SES: family *Income*

[5]There were eight 9-year olds in the sample, but we coded them to 10 years of age because the sample size was too small and caused estimation problems in the growth mixture models. In addition, there were 18 13-year olds. We did not code them into a lower age category because there was no issue with overlap in the mixture models by age.

[6]Regarding the abuse classification, 6.3% met criteria for past-year marijuana abuse (without dependence). Adolescents were asked questions regarding difficulties with school, employment, police, family, and friends. The majority classified as marijuana abusing met only one of the criteria (70.7%), 26.8% met two, and 2.4% met three. Of the other components, the most common was failure to fulfill major role obligations (65.9%), followed by use despite problems caused or exacerbated by marijuana use (31.7%), use in situations where it is hazardous (24.4%), and substance-related legal problems (9.8%).

[7]9.6% met criteria for past-year marijuana dependence. To meet criteria for marijuana dependence, adolescents first met criteria for abuse, as well as at least three additional criteria: tolerance, withdrawal, unintentional increases in dosage, persistent desire or unsuccessful efforts to reduce use, spending a great deal of time trying to obtain it, giving up important activities because of it, and/or continuing use despite its having caused a physical or psychological problem. Youth were not asked questions about withdrawal. Of those classified as dependent, 54% met three criteria, 30.2% met four, 9.5% met five, and 6.3% met all six. Three criteria were most common: increases in dosage (96.8%), great deal of time spent obtaining it (87.3%), and increased tolerance (85.7%). The remaining three were less common: persistent desire or unsuccessful efforts to cut down (42.9%), activities given up or reduced (36.5%), and using marijuana despite physical or psychological problem (19%).

[8]There were eight 14-year olds and 41 18-year olds.

[9]A 10-year old at Wave 1 contributes observations at the following ages: age 10 (Wave 1), age 11 (Wave 2), age 12 (Wave 3), and age 14 (Wave 5). A 13 year old, the maximum age at Wave 1, contributes to age 13 (Wave 1) and age 14 (Wave 2). There are 2040 observations in the 10–14 year age range with sample sizes of 191 (age 10), 473 (age 11), 660 (age 12), 484 (age 13), and 612 (age 14).

[10]In addition, the number of marijuana use classes increases with the age-range, leading to a more complicated model structure as more youth transition to using regularly. That is, a new class is added for each additional year of age as more youth transition into smoking marijuana.

below $25,000 and parent education, entered into the 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.

4.3.2. Stress processes—Proximal stress process variables include *Financial strain* (α = .73), both parent and child experiences of *Discrimination* (α = .93, α = .92, respectively), and the child's *Negative life experiences* (α = .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 of parenting stressors used DSM-III lifetime criteria for *Parent alcohol abuse (ever)* and *Parent major depression (ever)*.

4.3.3. Social support—*Family structure* is included as a sociodemographic proxy for social support. The variable is included as dummy variables for *Partnered* but unmarried, *Never married*, and *Separated/divorced/widowed* parent/guardians, with *Married* omitted. Direct social support measures include both parent and child perceptions of *Parental warmth and support* (α = .73, α = .82. respectively; correlation = .17) and *Positive school experiences* (α = .58). The parent-reported parental warmth and supportiveness measure was created from six 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-reported 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 primarily school-based positive life events scale employed six dichotomous items (e.g., won an award or recognition for school work in the last 6 months, etc.).

4.3.4. Intermediate psychosocial characteristics—Because we view marijuana use as a developmental process, we also adjust for intermediate risky/protective developmental outcomes. The measures included in the analysis are *Depressive symptoms* (α = .83), *Positive school attitudes* (α = .75), *Feelings of anger* (α = .84), and *Friend delinquency* (α = .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 seven ordinal items (e.g., how many of your three best friends drink alcohol, etc.).

4.4. Analytic plan

Marijuana use development patterns are modeled using growth mixture models (GMM). 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 uncovers sub-populations with discrete 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. 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.

Following Bollen and Curran (2006, pp. 180–181; see also Cheadle et al., 2010), we modeled marijuana use at level 1 (within-adolescent) using multilevel notation as

$$f(y_{it})=\ln(y_{it})=\sum_{g=1}^{G}\pi_i^{(g)}\left[\beta_{0i}^{(g)}+\beta_1^{(g)}age_{it}+\beta_2^{(g)}age_{it}^2\right] \quad (1)$$

and at level 2 (between-adolescents) as

$$\beta_j^{(g)}=\gamma_j^{(g)}, \text{ for } j=0,1,2 \quad (2)$$

In this application, the density of y_{it}, marijuana use for adolescent i at age t, is modeled as a Poisson process for count outcomes so that $f(y_{it})=\sum_{g=1}^{G}\pi_i^{(g)}f^{(g)}(y_{it})$ where $f^{(g)}(y_{it}) \sim Poisson(\lambda_{it}^{(g)})$. In these equations $\pi_i^{(g)}$, the mixing proportion, is the probability that child i belongs to the gth group with $0\leqslant\pi_i^{(g)}\leqslant 1$ and $\sum_{g=1}^{G}\pi_i^{(g)}=1$ (all probabilities sum to 1). According to Eqs. (1) and (2), change in marijuana use follows a quadratic growth curve where patterns of use are allowed to vary across latent groups (g).

The second component of the model incorporates the covariates into a multinomial logit equation predicting class/group membership in which the covariates are incorporated as the x-variables:

$$Pr(C_g=j|\mathbf{X})=\pi_i^{(j)}=\frac{\exp(\mathbf{X}\theta^j)}{\sum_{g=1}^{G}\exp(\mathbf{X}\theta^{(g)})} \quad (3)$$

In Eq. (3), C_g is as a latent variable capturing adolescent trajectory classifications with specific classes denoted j, $\mathbf{X}$ is a matrix of predictor variables (i.e., gender), and $\Theta^{(j)}$ is a vector of regression weights for group j of g. The final component of the model then employs C_g and $\mathbf{X}$ as predictors of probability of DSM-IV abuse/dependence diagnosis using logistic regression. Diagnoses is treated as a latent class, C_d, within the model, but where the classes are known. The full model is depicted graphically in Fig. 1.

Because group membership C_g is probabilistic – it is latent and must be inferred – all results are based on simultaneous estimation of the regression parameters, the GMM, and diagnosis (C_d, for the final models). In order to determine the optimal number of classes (g), 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,[11] the third incorporated a quadratic term for time, and the fourth added a random intercept (as with 2). The BIC was used to compare models by class and type (Raftery, 1995) and we selected a 3-class quadratic growth curve without random intercept variation (from model set 3) based on the fact that the BIC for this model compared favorably to more complicated baseline LGCMs

[11]This amounts to $\beta_{0i}^{(g)}=\gamma_{00}^{(g)}+\zeta_{0i}$ with the assumption that $\zeta \sim N(0, \sigma_{00})$ so that the model allows for within group (g) heterogeneity between adolescents.

and GMMs and those with more classes (Nylund et al., 2007). In addition, the model we present maintains parsimony and adequate sample sizes for the etiological analysis of class membership (Nagin, 2005), which could not be maintained with additional classes for this sample because of small cell sizes when additional latent groups are included in the analysis.

5. Results

5.1. 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. These youth also live in a diverse range of family structures. Only 32% reside in two-parent homes, 26% live with a partnered but unmarried guardian, 22% with a never married parent, and 19% with a separated or divorced (or widowed) parent. Nearly 65% 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 that may lead to early use trajectories and problematic use in later in adolescence (i.e., Schulenberg et al., 2005).

5.2. Classification of marijuana use trajectories

Average levels of marijuana use over the previous year for the three trajectory classes are plotted in Fig. 2 for ages 10–14 years. The legend contains (a) the *N* and % classified, (b) the odds-ratio for later problem use diagnosis versus abstainers, (c) and the percentage meeting the problem use abuse/dependence DSM-IV criteria for each latent class. The majority of youth, about 66%, largely abstained from marijuana through age 14. About 13% of these *abstainer* youth, however, met the abuse/dependence criteria by late adolescence when they were between 15 and 17 years of age. The first group is labeled *early-onset,* about 15% of this NAI population, because they have increasing use from age 11. A majority (60%) of early-onset youth met criteria for abuse/dependence when they were older and the odds were 10 times larger than for the youth who abstained. A similar percentage, 19%, began transitioning to fairly regular marijuana smoking in early adolescence and half of them later met the diagnostic criteria. The odds for this group of *adolescent-onset* smokers was nearly seven times larger than for those who abstained. Fig. 2 shows that nearly 40% of the youth in this population begin using marijuana, at least occasionally, by age 14 and that risks for problem use by late adolescence are greatly elevated for them.[12]

5.3. The etiology of early marijuana use trajectories

Exponentiated multinomial logistic regression coefficients (relative risk [RR] ratios[13] [RRR]) and absolute *t*-values for the etiological models predicting membership in the early-onset and adolescent-onset classes relative to abstaining are presented in Table 2 (paths to C_g in Fig. 1). There are a few important things to note. First, the full covariate list reported in Table 1 does not appear. Covariates not related to early use are omitted in order to (a) conserve space and (b) to simplify the model specification. Thus, an omitted variable indicates that it was not associated with classification. Second, before moving to the multinomial logistic regressions, we tested whether or not classification could be treated as

[12]In terms of model fit, the average latent class posterior assignment probabilities are all above .89 for the baseline GMM and the entropy measure is .83. Entropy increases to .978 in the final model in Table 3, indicating that the models overall do a good job of classifying individuals.

[13]The relative risk is the ratio of the probability for one group to the probability for another and so is conceptually similar, though not identical, to the odds.

an ordinal outcome such that the latent classes (abstainer → adolescent-onset → early-onset) can be considered ordered by intensity of use with respect to each predictor. This proportionality assumption[14] 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), 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 marijuana use (1.25 vs. 1.28, males versus females, at age 14), are more likely to be early-onset users than boys. The RR (relative risk) is over two times larger across specifications when all measures are included in the equation (M4). This result suggests that gender differences in early use do not result from differential exposure to stressors, support, depressive symptoms, or from having more delinquent friends. The stressors, as indicated in the Bivariate column, all increase risk for early-onset use, although perceived discrimination does not predict adolescent-onset. Both negative life events and perceived discrimination apparently operate through friend delinquency (M4) while the effect of parent depression is not significant in M1 when the other stressors are included, indicating that this association reflects how it is intertwined with the broader package of stressors. Parent alcohol abuse, which raises the RR of being early- or adolescent-onset by nearly 50% (M4), is marginally significant ($p < .1$) across equations.

Positive school-based experiences was the only social support variable statistically related to marijuana use development ($p < .1$, M4). Each standard deviation increase in positive school experiences reduced the RR of both early-use classifications by 15%. All of the psychosocial variables had bivariate associations with use classification and only the effect of positive school attitudes was held constant across categories. Positive school attitudes is protective (M3, $p < .1$) until friend delinquency is controlled (M4), suggesting that school attachment protects use by promoting friendships with less delinquent peers. At the same time, depression influences early-onset use primarily by fostering friendships with more delinquent peers. The friend delinquency correlation indicates that each standard deviation increase raises the RR of early-onset by nearly 2.5 times and those for adolescent-onset by 44%.

5.4. The etiology of marijuana dependence and/or abuse in late adolescence

Odds ratios and *t*-ratios for coefficients predicting DSM-IV marijuana abuse/dependence are reported in Table 3 (paths to C_d in Fig. 1). As with the results in Table 2, the coefficients are restricted to those statistically associated with diagnosis in at least one model. In other words, variables from Table 1 that have been omitted indicate that the variable is not associated with problem use. The "Bivariate" column presents findings for covariates singly, model N1 includes the stressor and adolescent background variables (the fundamental cause measures were not statistically related to abuse/dependence), 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. N4 incorporates the early trajectory classifications, treating them as mediators of the fundamental causes, stressors, and psychosocial influence pathways.

[14]The regression parameter is fixed to be equal for the early- and adolescent-onset equations as with ordinal logit regression.

The age coefficient is significant across equations, leading to the following pessimistic conclusion: even after controlling for early use trajectories and other factors, more adolescents in this population will elevate their marijuana use to the point that it causes problems in their lives. In fact, each year of age is associated with a 45% increase in the odds of meeting the diagnostic criteria in the final model (N4) even after early use trajectories are accounted for.

Although each of the stressor variables predicted problem use in the bivariate models, only negative events and major depression were associated in model N1. These factors appear to operate by decreasing school attachment and promoting friendships with delinquent peers. Indeed, school attachment is protective because it leads to friendships with others who are more prosocial. As with trajectory classification, delinquent friendships (N4) play a key role in the model, raising the odds of abuse/dependence by 43% for each standard deviation when early- and adolescent-onset trajectory classifications are included.

The largest influences on problem use arise from prior histories of marijuana use. Results for both early- and adolescent-onset are in fact relatively similar in magnitude. Early-onset use multiplies the odds of abuse/dependence by 6.9 times and adolescent-onset by approximately 6.5. These findings clearly indicate that early marijuana use greatly increases the risk for problematic use in later adolescence. The model-based odds ratios are smaller than those reported in Fig. 2, which may suggest that use trajectories feed back to influence friendships with delinquent peers in a negative cycle of homophilous friend selection and social influence (i.e., Kandel, 1978). In this way the social lives and risk behaviors of adolescents become intertwined into patterns of relationships reinforcing marijuana use.

6. Discussion

While marijuana use has been studied for decades in NAI communities (Beauvais et al., 1989, 2004), there remains much to learn about the etiology and progression of use (Ehlers et al., 2007), and how use patterns are similar and different to those for other substance use outcomes such as alcohol use. Developmental trajectory studies from early experimentation to regular use are ongoing in the general population (e.g., Schulenberg et al., 2005), but this is the first study to do so among a sample of NAI youth. NAI adolescent marijuana usage rates are in general higher than for other groups (Mitchell and Plunkett, 2000; Beauvais, 1992), so with the first set of research goals we described the timing and patterns of marijuana use over the early life course and illustrated the consequences for problem use in later adolescence.

From ages 10 to 14 years, three groups characterize adolescent marijuana use in this particular NAI population: early-onset, adolescent-onset, and abstainer. The early-onset group (14%) showed increasing use after age 11 and the adolescent-onset group (19%) had sharply increasing consumption at age 13. The early-onset users were far more likely to meet DSM-IV criteria for marijuana abuse and/or dependence. These diagnostic criteria were used to indicate problematic marijuana use because they suggest negative impacts on important areas of life that may have long-term consequences (i.e., Rohde et al., 2001). In fact, 60% of the early-onset and 50% of the adolescent-onset groups met the abuse and/or dependence problem use criteria, while a much smaller 13% of the abstainers did. This shows that any use, particularly early use, carries substantially elevated risk for later problems, possibly setting the stage for genetic factors to contribute to more substantial marijuana use problems (Kendler et al., 2008). Considered with the age effect in Table 3, more of the youth who abstained in early adolescence are also likely to show signs of problem use by late adolescence. In fact, nearly 30% of all youth in this study did so.

Overall, less than 10% of adolescents age 17 in the general population independently meet either of the abuse or dependence criteria (Young et al., 2002).

In addition, we conducted an etiological analysis of marijuana over the early life course. This analysis was guided by a developmental model integrating research into fundamental causes of disease reflecting structural characteristics (Link and Phelan, 1995), stress processes (e.g., Pearlin, 1989), social support (Turner, 1999), and intermediate psychosocial developmental pathways (i.e., Cheadle and Whitbeck, 2011). The statistical models were oriented around whether these factors distinguish early and later problem users, and whether early trajectories explain the relationships between these factors and later problem marijuana use in an analysis spanning from approximately 10 to 17 years of age.

The global picture portrayed in the analysis is of a developmental process whereby negative events, perceived discrimination, and parent alcohol abuse combine with few positive school experiences to decrease school attachment, increase depressive symptoms, and promote friendships with delinquent peers. That is, these factors suggest a mediational model whereby environmental stressors filter through adolescents' lives to influence their psychosocial characteristics and then early marijuana use trajectories. Early marijuana use subsequently plays a key role in problem use so that by approximately age 16 the odds of marijuana abuse/dependence criteria are nearly seven times larger for early-onset users, and nearly 6.5 times larger adolescent onset users. Taken together, these results paint a dramatic picture of the role of stress in promoting negative affects leading to behaviors that substantially increase the risk of harmful patterns of marijuana consumption over the early life course (Labouvie, 1986). Future studies will be able to address how these processes unfold during the transition to adulthood.

The etiological analysis of early marijuana use trajectories and problem use indicated that features of fundamental causes of disease were not the primary factors differentiating early marijuana users in these NAI communities. None of the parent education, income, or employment measures were related to these outcomes. The NAI culture comprising this study is one of the most populous in the US and Canada and disadvantage is common in these communities, which reduces variation and our ability to detect these effects (see Whitbeck et al., 2006a,b). Moreover disadvantage is widespread and so contextual effects are likely to have widespread influences (see, e.g., Boardman et al., 2001), but these require a more heterogeneous and larger collection of communities in order to be studied directly. These unmeasured community effects may also "swamp" other measures of disadvantage, which could be why indicators of financial strain and social support variables were also not associated with the marijuana use outcomes.

Two indicators of fundamental causes were associated with marijuana use, however: females were more likely to be early-onset users, and older adolescents were more likely to have problem use in late adolescence. In both cases, these factors appeared to structure the risk of early marijuana use largely independently of the other characteristics. 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 use as they age into later adolescence and early adulthood. To qualify the importance of this age effect, 50% of these adolescents' parents meet lifetime criteria for alcohol abuse and 22% for drug abuse (Whitbeck et al., 2006a,b). Indeed, parent alcohol abuse was a consistent predictor of early- and adolescent-onset use, though it was unrelated to problem use. This latter finding points to the complicated intergenerational cycle of substance use that persists in many NAI communities and may reflect the complex interplay between genetic risk, risky environments, and early substance use (e.g., Pescosolido et al., 2008).

The finding that girls in this NAI population have odds of early-onset marijuana use two times higher than boys is striking. The analysis indicated that the gender 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, or romantic partners. 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 marijuana and other substances. Having an older romantic partner is associated with many risk behaviors for girls, including early sexual activity, drinking problems, and illegal drug use (Mezzich et al., 1997; Young and d'Arcy, 2005). At the same time, females who engage in more risky behaviors are more likely to date older boys and to be further encouraged to engage in risk behaviors by their partners (Cauffman et al., 2008).

So how does the sequence identified in this analysis compare to Cheadle and Whitbeck's (2011) findings for alcohol use? There are important similarities, but also important differences. For example, both marijuana and alcohol use show similar early- and adolescent-onset classifications, and early use is highly predictive of abuse/dependence for both. Thus, the finding that early use trajectories are predictive of later problem substance use in this population is not novel. The findings here are important, however, because they corroborate the findings for alcohol with another outcome, marijuana use, which reinforces the suggestion that intervention efforts should target both risk behaviors when NAI youth are quite young.

Etiologically, however, there are important differences. For example, perceived discrimination is a significant predictor of early and problem drinking, but is not for marijuana use. Thus, whereas factors such as positive school attitudes, anger, and parent depression are important for alcohol use development, adolescents do not use marijuana for all of the same reasons that they drink. In fact, the only thing that seems to matter to both onset of use and problem use is delinquent peers, suggesting a joint role for social learning through associations, reinforcement, and definitions favorable to use (Akers and Lee, 1999). However, the results are consistent that gender is an important risk factor for both alcohol and marijuana outcomes, which strengthens the suggestion that gender-responsive intervention programming may be required in this population.

6.1. 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, the age-range of this study covers only a single cohort of youth over a few years. Implications for the life course, including the transition to adulthood, have yet to be assessed. Fourth, though we considered a range of factors promoting early use and later problem use, the measures we employed were fixed in time and relatively early in life. Circumstances change and future work should more fully consider and operationalize how changes in these factors contribute to marijuana use. Finally, peer delinquency was found to be an important component of early use. The measure used, however, was perceptual so the coefficients may be biased upwards, though the finding of friend effects is consistent with numerous social network studies on risky behavior (e.g., Haynie and Osgood, 2005; Ennett et al., 2006).

7. Conclusions

Nearly 35% of youth from this Northern Midwest and Canada NAI culture begin using marijuana at a young age with sharply increasing rates of consumption over early

adolescence. The early use patterns reflect a process whereby social stressors such as negative life events, perceived discrimination, and parent alcohol abuse, along with few positive school experiences, influence school attitudes, depressive symptoms, and delinquent friendships. This pattern of social influence is consistent with the view that social environments are critical determinants of substance use, particularly early on, and that they set the stage for subsequent genetic effects promoting increased use and addiction (e.g., Kendler et al., 2008). The substance use literature, however, also tends to focus on males as being particularly at-risk for substance use problems (Schulte et al., 2009), yet females were two times more likely to be early-onset users in this population. Because early use is strongly predictive of problem use by approximately age 16, these findings indicate that substance use intervention efforts should begin prior to age 11 and incorporate gender-specific programming. Later interventions may be too late to stem the transition to more dangerous patterns of illicit substance use if early histories of marijuana and alcohol use are not deflected.

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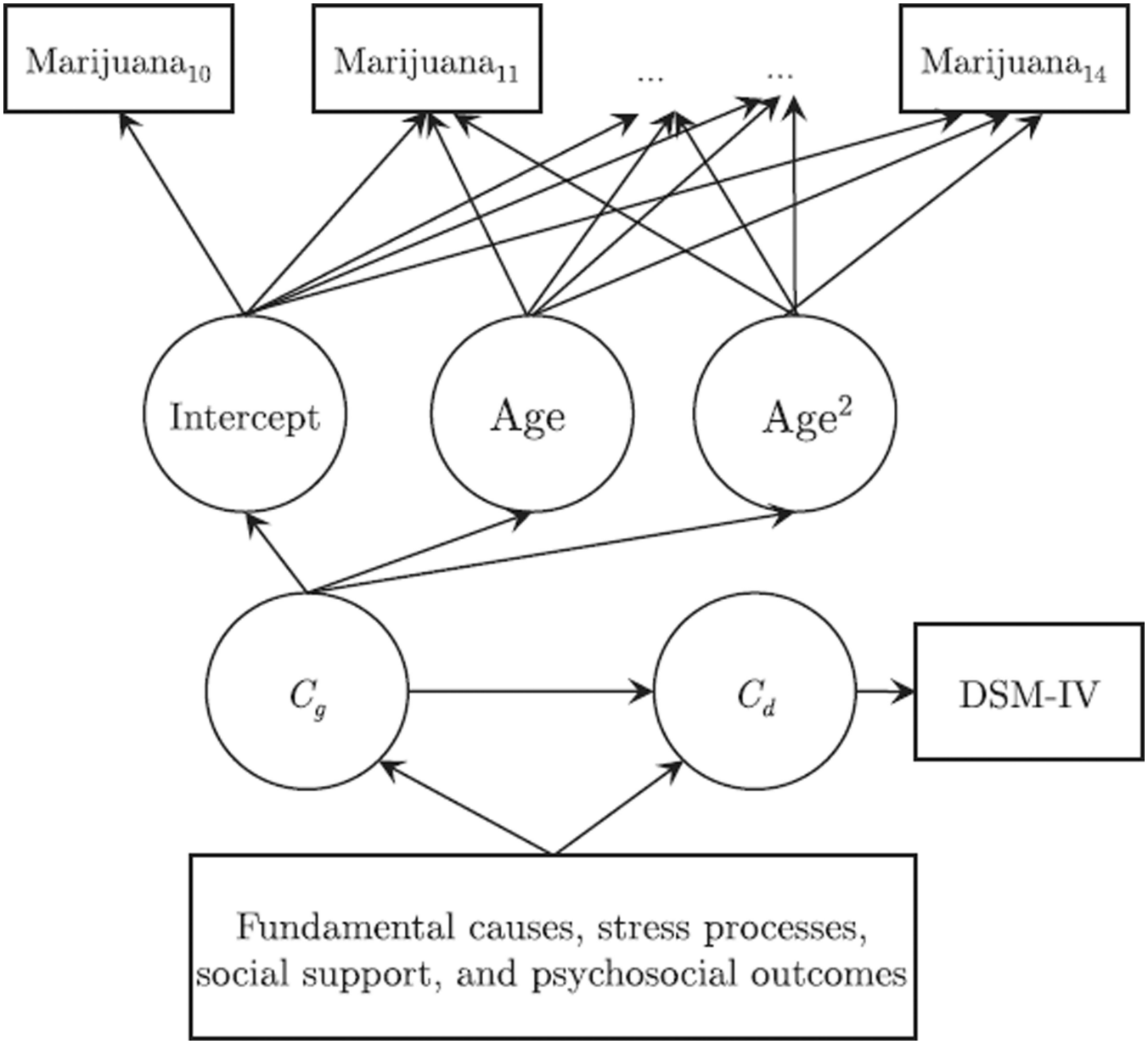

Fig. 1.
Graphical depiction of the full analysis model.

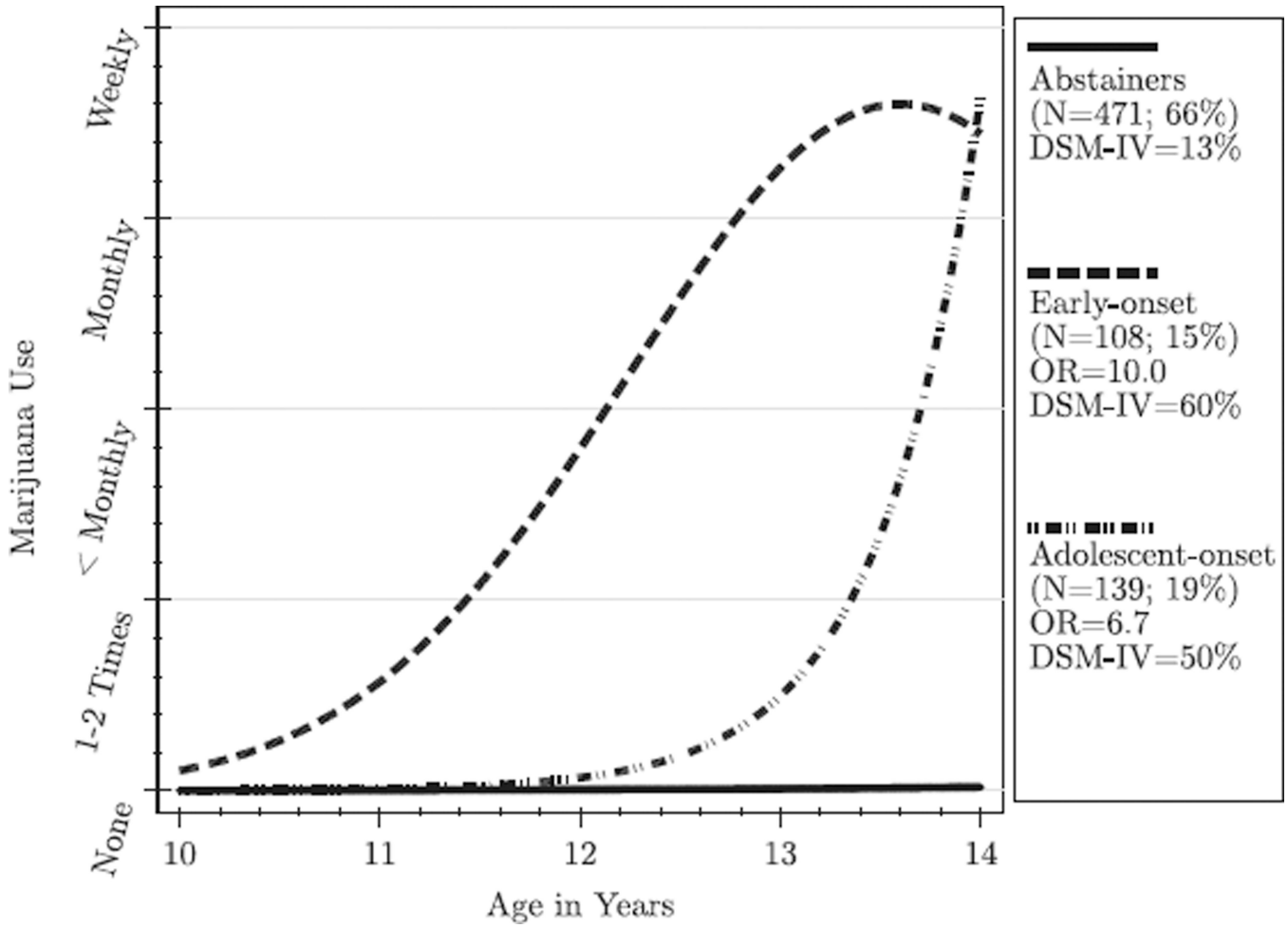

Fig. 2.
Predicted marijuana use frequency development patterns for the 3-class quadratic growth mixture model without intercept heterogeneity ($N = 718$).

Table 1

Descriptive statistics for the complete covariate list ($N \approx 718$).

Variables	*N*	% *N*	Mean	Sd	Min	Max	Male	Female
Fundamental causes/background								
Age at Wave 1	715	100	11.09	[0.82]	10	13	11.13	11.05
Age at Wave 6	643	90	16.21	[0.88]	14	18	16.25	16.18
Female respondent	718	100	0.50	[0.50]	0	1	0.00	1.00
Income below $25,000	702	98	0.44	[0.50]	0	1	0.44	0.45
Education:<high school	715	100	0.17	[0.38]	0	1	0.16	0.19
Education: high school	715	100	0.36	[0.48]	0	1	0.38	0.35
Education: some college/degree	715	100	0.46	[0.50]	0	1	0.46	0.47
Employment: full time	716	100	0.58	[0.49]	0	1	0.57	0.58
Employment: part-time	716	100	0.10	[0.30]	0	1	0.10	0.10
Employment: unemployed	716	100	0.18	[0.39]	0	1	0.17	0.20
Employment: other	716	100	0.14	[0.35]	0	1	0.16	0.12
Stressors								
Financial strain (*z*)	659	92	0.00	[1.00]	−1	4	0.02	−0.02
Negative events (*z*)	709	99	0.00	[1.00]	−2	4	−0.03	0.02
Perceived discrimination (*z*)	706	98	−0.01	[1.00]	−1	4	0.03	−0.05
Parent discrimination (*z*)	626	87	0.00	[0.99]	−2	3	−0.06	0.06
Parent alcohol abuse (ever)	711	99	0.64	[0.48]	0	1	0.65	0.62
Parent major depression (ever)	711	99	0.19	[0.39]	0	1	0.17	0.21
Support								
Family structure: married	714	99	0.32	[0.47]	0	1	0.34	0.31
Family structure: partnered	714	99	0.26	[0.44]	0	1	0.25	0.28
Family structure: never married	714	99	0.22	[0.41]	0	1	0.18	0.26
Family structure: sep/div/widowed	714	99	0.19	[0.40]	0	1	0.23	0.15
Child-parent warmth/approval (*z*)	715	100	0.00	[1.00]	−4	2	−0.04	0.05
Parent–parent warmth/support (*z*)	716	100	0.00	[1.00]	−4	2	−0.12	0.12
Positive school experiences (*z*)	710	99	−0.01	[1.00]	−2	2	0.07	−0.09
Psychosocial								
Positive school attitudes (*z*)	713	99	−0.01	[1.00]	−3	1	−0.06	0.04

Variables	*N*	% *N*	Mean	Sd	Min	Max	Male	Female
Feelings of anger (*z*)	716	100	−0.01	[0.99]	−2	3	−0.01	0.00
Depressive symptoms (*z*)	699	97	−0.01	[0.99]	−2	3	−0.08	0.06
Friend delinquency (*z*)	663	92	−0.01	[1.00]	−1	3	−0.01	0.00
Outcomes								
Marijuana use at age 11	470	65	0.07	[0.42]	0	5	0.04	0.09
Marijuana use at age 14	611	85	1.26	[1.93]	0	6	1.25	1.28
Marijuana abuse/dependence	652	91	0.27	[0.45]	0	1	0.28	0.27

Table 2

Relative risk ratios from multinomial logistic regression equations predicting early and adolescent-onset marijuana use trajectory classification versus abstaining (N = 718). 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.
Fundamental causes/background[b]										
Female respondent	2.16**	1.26	2.23***	1.27	2.14**	1.22	2.06	1.27	2.11	1.29
	(3.47)	(1.19)	(3.53)	(1.22)	(3.33)	(1.00)	(3.16)	(1.17)	(3.14)	(1.26)
Stressors[c]										
Negative events (*z*)	1.47**	←[a]	1.36***	←	1.46***	←	1.30**	←	1.14	←
	(4.81)		(3.59)		(4.24)		(2.78)		(1.22)	
Perceived discrimination (*z*)	1.50**	1.12	1.35**	1.01	1.38**	1.03	1.26*	1.01	1.15	.97
	(4.00)	(1.27)	(2.85)	(.06)	(3.05)	(.31)	(2.06)	(.07)	(1.25)	(.27)
Parent alcohol abuse (ever)	1.77**		1.57*		1.51*		1.40+	←	1.39+	←
	(3.34)	←	(2.47)	←	(2.24)	←	(1.81)		(1.73)	
Parent major depression (ever)	1.60*	←	1.35	←	1.29		1.26	←	1.20	←
	(2.42)		(1.40)		(1.21)	←	(1.07)		(.83)	
Support [d]										
Positive school experiences (*z*)	.84*	←			.76**	←	.85+	←	.85+	←
	(2.16)				(3.05)		(1.70)		(1.66)	
Psychosocial										
Positive school attitudes (*z*)	.70***	←					.84+	←	.92	←
	(.00)						(1.84)		(.84)	
Feelings of anger (*z*)	1.48***	1.30**					1.11	1.13	1.10	1.15
	(3.62)	(2.77)					(.85)	(1.22)	(.78)	(1.39)
Depressive symptoms (*z*)	2.01***	1.36**					1.49**	1.11	1.24	1.07
	(5.47)	(2.91)					(2.79)	(.88)	(1.41)	(.59)
Friend delinquency (*z*)	2.80***	1.61***							2.30***	1.44**
	(8.41)	(4.69)							(6.11)	(3.23)

Variables	Bivariate		M1		M2		M3		M4	
	Early	Ad.	Early	Ad.	Early	Ad.	Early	Ad.	Early	Ad.
Intercepts										
Early-onset			−1.59***	−1.24***	−1.60***	−1.25***	−1.66***	−1.24***	−1.80***	−1.22***
			(13.89)	(12.48)	(14.00)	(12.43)	(13.60)	(12.25)	(13.35)	(12.02)

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

t-Ratios are presented in parentheses.

[a] Arrows ("←") indicate that the parameter has been fixed across the early- and adolescent-onset equations.

[b] The other background variables were not statistically significant so they have been excluded.

[c] Financial strain and parent perceived discrimination were not statistically significant.

[d] The other support variables were not statistically significant.

+ $p < .1$.

* $p < .05$.

** $p < .01$.

*** $p < .001$ (two-tailed).

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Table 3

Odds-ratios from logistic regression equations predicting DSM-IV marijuana dependence and/or abuse diagnosis (N = 718 for the classification GMM, and N = 652 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	
	e^b	(t)	e^b	(t)	e^b	(t)	e^b	(t)	e^b	(t)
Classification										
Early-onset marijuana use									6.89	(6.87)***
Adolescent-onset marijuana use									6.48	(7.80)***
Fundamental causes/background[a]										
Age at Wave 6	1.63	(4.79)***	1.64	(4.76)***	1.56	(4.23)***	1.37	(2.88)**	1.45	(2.92)**
Stressors[b]										
Negative events (z)	1.39	(3.75)***	1.33	(2.93)**	1.25	(2.25)*	1.07	(.66)	1.08	(.65)
Perceived discrimination (z)	1.27	(2.79)**	1.12	(1.22)	1.10	(1.00)	1.04	(.41)	1.04	(.37)
Parent alcohol abuse (ever)	1.52	(2.17)*	1.19	(.84)	1.11	(.51)	1.10	(.46)	1.00	(.01)
Parent major depression (ever)	1.72	(2.57)*	1.68	(2.17)*	1.59	(1.92)+	1.47	(1.59)	1.52	(1.57)
Support[c]										
Psychosocial										
Positive school attitudes (z)	.70	(4.21)***			.81	(2.21)*	.86	(1.55)	.91	(.91)
Feelings of anger (z)	1.28	(2.69)**			1.07	(.62)	1.07	(.66)	1.01	(.09)
Depressive symptoms (z)	1.39	(3.38)**			1.11	(.90)	.99	(.06)	.93	(.59)
Friend delinquency (z)	2.04	(7.40)***					1.73	(4.63)***	1.43	(2.69)**
Intercept			−1.069	(11.43)***	−1.083	(11.42)***	−1.122	(11.38)***	−1.940	(13.19)***

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

[a]The other background characteristics were not significant so they have been omitted.

[b]The other stressor variables were not statistically significant.

[c]The social support measures were not statistically significant.

+ $p < .1$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.