

HHS Public Access

Author manuscript

Health Psychol. Author manuscript; available in PMC 2023 December 01.

Published in final edited form as:

Health Psychol. 2022 December; 41(12): 912–922. doi:10.1037/hea0001227.

Smoking across adolescence and adulthood with cardiovascular risk among American Indian Peoples

Ashley H. Clawson, Ph.D.^{1,2}, Ashley B. Cole, Ph.D.², Nicole M. Ruppe, M.S.^{1,2}, Cara N. Nwankwo, B.A.^{1,2}, Alexandra L. Blair, B.S.^{1,2}, Kristoffer S. Berlin, Ph.D.^{3,4}, Monique M. Naifeh, M.D.⁵

¹Center for Pediatric Psychology, Department of Psychology, Oklahoma State University, 116 Psychology Building, Stillwater, Oklahoma, 74078.

²Department of Psychology, Oklahoma State University, 116 Psychology Building, Stillwater, Oklahoma, 74078.

³The University of Memphis, Psychology, 202 Psychology, Memphis, TN 38152.

⁴University of Tennessee Health Science Center, Memphis, Pediatrics, Memphis, TN.

⁵Department of Pediatrics, The University of Oklahoma Health Sciences Center, Oklahoma City, OK

Abstract

Objective: American Indian peoples (AIs) have high smoking rates and cardiovascular risk factor burden. This study aimed to: 1) investigate latent smoking classes across adolescence and adulthood, 2) investigate adolescent predictors of smoking classes, and 3) assess how smoking class is related to adult cardiovascular risk in a sample of AIs.

Methods: A sample of AIs (*N*=338) from the National Adolescent to Adult Health Study self-reported on smoking across four assessment waves (W1: 7–12th grade; W2: 8–12th grade; W3: ages 18–26; W4: ages 24–32). The socioecological framework for addressing tobaccorelated disparities was used to identify potential adolescent (W1) risk and resource factors. C-reactive Protein, blood pressure, and lipids were collected at W4. Growth mixture modeling and regressions were used.

Results: Six smoking classes were identified: *light smoking* (36%), *nonsmoking* (23%), *escalating, adult daily smoking* (13%), *chronic heavy smoking* (12%), *escalating, young adult daily smoking* (9%), and *reducing smoking* (7%). Risk factors for being in the *chronic, heavy smoking class* included peer smoking and older age at W1. Compared to the *chronic heavy smoking* class, AIs in the *reducing smoking* class lived in in more impoverished neighborhoods during adolescence. Relative to several classes with less smoking, being in the *chronic heavy smoking* class was associated with higher C-reactive Protein and less favorable lipid levels. W1 social support was a resource factor for adult diastolic blood pressure and some lipids.

Conclusions: Socio-ecologically-informed tobacco interventions have the potential to reduce smoking and cardiovascular risk among AIs, and bolstering social support may be important.

Keywords

American Indian; smoking; cardiovascular risk; social support; development

American Indian (AI) peoples exhibit some of the highest rates of commercial cigarette smoking (henceforth, *smoking*) compared to all ethnic/racial groups in the U.S.; specifically, 20.9% of AI adults smoke compared to 14.0% of adults overall (Centers for Disease Control and Prevention, 2021; Jamal et al., 2016). AI individuals are also more likely to initiate smoking at earlier ages during adolescence and continue smoking across time relative to other ethnic/racial groups (Lawrence et al., 2014). Yet, there is limited information about the longitudinal smoking trajectories of AI individuals across adolescence and into adulthood and how smoking impacts adult health. AI peoples also experience high rates of cardiovascular disease, which is influenced by smoking and social determinants of health (Breathett et al., 2020; Fabsitz et al., 1999). Research on how developmental smoking patterns relate to cardiovascular health among AI populations is needed, particularly given that impacts of smoking on cardiovascular health begin in childhood (McGill et al., 2008) and because smoking-attributable mortality from cardiovascular disease is higher among Als relative to non-Hispanic White individuals (Mowery et al., 2015; Zhang et al., 2015). Prior research has established relations between smoking and cardiovascular risk factors, including higher cholesterol, triglycerides, low-density lipoprotein (LDL-C), blood pressure, C-reactive protein (CRP), and lower high-density lipoprotein (HDL-C) (Campbell et al., 2008; Craig et al., 1989; Howard et al., 1999; U.S. Department of Health and Human Services, 2014). The current longitudinal study examined how smoking patterns across adolescence and into adulthood (i.e., latent smoking classes) related to risk factors for cardiovascular disease (lipids, blood pressure, CRP) among AI peoples.

Consistent with a socioecological approach to understanding tobacco-related inequities, this study aimed to expand our understanding of how multilevel influences from interlocking, systemic structures of racism (e.g., deleterious social determinants of health) and resource factors influence tobacco-related disparities among AI populations across adolescence and into adulthood (Henson et al., 2017; National Cancer Institute, 2017). This study focused on AI individuals and used person-centered data analytic techniques (i.e., a within-group approach) to identify heterogeneity in developmental smoking patterns, including subgroups of individuals with similar smoking patterns (i.e., latent smoking classes), rather than assuming all AI individuals had the same smoking pattern. Furthermore, the within-group approach was used over the commonly used between-group approach (which identifies average smoking patterns across a population and looks at race/ethnicity as a predictor) because 1) tobacco-related disparities among AI populations are well-documented, 2) there is a need to understand drivers of these disparities, 3) using ethnicity/race (i.e., a sociopolitical construct corresponding to privilege and oppression) as a predictor of smoking may mask within-group differences (Burlew et al., 2009; National Cancer Institute, 2017; Pearson et al., 2021), and 4) identifying predictors and health correlates of developmental

smoking patterns specific to AIs may support more nuanced tobacco and cardiovascular risk prevention efforts for AI individuals.

Only two studies have explored heterogenous smoking patterns exclusively among AI/ Indigenous youth across development. First, Whitesell and colleagues (2014) examined smoking trajectories among AI middle-school students (N=381; mean age at baseline was 13; baseline data collected in 2006) from a Northern Plains reservation across four waves spanning two years (Whitesell et al., 2014). Three smoking classes emerged: non-users (62%), occasional smokers (30%) who had a consistent intermittent use (once in a while, not daily), and experimental smokers (8%) who reported daily smoking at Wave 1 followed by a decline to intermittent/non-daily use. Female gender, greater life stress, and poorer parent-child relationship quality significantly increased the odds of classification in the experimental smokers versus the non-users class (Whitesell et al., 2014). Further, female gender, greater life stress, and higher deviant peer influences predicted membership in the occasional smokers class (Whitesell et al., 2014). Second, Hautala and colleagues (2020) examined smoking across ten waves spanning from ages 10–12 through ages 24– 27 years-old among an Indigenous sample (N=708) living on/near four reservations in the upper-Midwest US and Canada. Four smoking classes emerged, with the first two representing low levels of smoking: the low/non-smokers class (35%) who largely reported no smoking; and the occasional smokers class (17%) who had increased smoking across adolescence followed by a reduction in adulthood. The remaining classes initiated smoking during adolescence and increased use over time: the early-onset smokers class (26%; mean age of onset=11) and the *mid-adolescent onset smokers* class (22%; mean age of onset=13). Youth in the low/non-smoking class had higher family income, higher caregiver education, and less household smoking. Females were more likely to be in the early-onset smoker class, and males were more likely to be in the mid-adolescent onset smokes and low/non-smoking classes (Hautala et al., 2020).

The aforementioned longitudinal studies identified significant heterogeneity in smoking patterns among AI/Indigenous populations, with results indicating between three and five smoking classes (Hautala et al., 2020; Whitesell et al., 2014). These studies also identified important multilevel risk and health promotive factors related to smoking patterns among AI populations. Risk factors were female sex, life stress, deviant peer influences, socioeconomic status, and living in the home with people who smoked (Hautala et al., 2020; Whitesell et al., 2014). An identified resource factor (i.e., a variable that reduces risk) against smoking trajectories was greater positive parent-child relationships, which may be conceptualized as an aspect of social support (Hautala et al., 2020; Whitesell et al., 2014). Social support literature has identified connectedness as important resource factor against smoking among AI and Alaska Native (AN) youth (Henson et al., 2017). Social support has also been linked to improved cardiovascular health in the general population (Uchino, 2006) and perceptions about health among AI individuals (Bassett et al., 2014). The longitudinal associations between social support, smoking, and cardiovascular risk among AI populations is largely unknown. Further, few longitudinal studies have identified smoking trajectories specifically among AIs, and no study to date has investigated the association between longitudinal smoking patterns and adult cardiovascular risk.

The current study aimed to identify: 1) longitudinal smoking patterns (i.e., smoking classes) across four waves from adolescence (grades 7–12) into adulthood (ages 26–34), 2) risk and resource factors associated with smoking class membership, and 3) how smoking class membership and risk and resource factors in adolescence were related to adult cardiovascular risk (i.e., CRP, blood pressure, and lipid levels) among a sample of AI individuals (*N*=338). Regarding Aim 1, based on the existing studies on longitudinal smoking classes among AI individuals (Hautala et al., 2020; Whitesell et al., 2014), it was hypothesized that at least four longitudinal smoking classes would emerge, including a nonsmoking smoking class and adolescent onset smoking classes with escalating and intermittent smoking patterns. Regarding Aims 2 and 3, the socioecological framework for addressing tobacco-related disparities (National Cancer Institute, 2017) and prior literature on smoking and cardiovascular health correlates, as well as prior literature on smoking classes among AI populations (Breathett et al., 2020; Fuemmeler et al., 2013; Henson et al., 2017; LeMaster et al., 2002; Raghuveer et al., 2016; Uchino, 2006; Whitesell et al., 2014), were used to identify potential risk and resource factors: parental smoking, personal mental health symptoms, peer factors (i.e., peer smoking, peer relationships), supportive adult relationships, and contextual variables (i.e., poverty, healthcare access difficulties, neighborhood connectedness). It was hypothesized that 1) parental smoking, peer smoking, mental health symptoms, neighborhood poverty, and healthcare access difficulties would be risk factors associated with being in classes with greater smoking and greater cardiovascular risk; 2) classes with greater smoking across time would be associated with greater adult cardiovascular risk; and 3) adolescent social support (i.e., supportive adult relationships, feeling that friends care about you, neighborhood connectedness) would be associated with decreased smoking and cardiovascular risk.

Methods

This article includes a description of the sample, data exclusions and manipulations, and all measures that were included in the study; the APA Reporting Standards for quantitative research were followed. Analysis code is available by emailing the corresponding author; all data and research materials are on the Add Health website (https://addhealth.cpc.unc.edu/). This study was not pre-registered. Mplus 8.5 was used for analyses (Muthen & Muthen, 2017); the subpopulation command was used to select AI individuals (*N*=338) and to incorporate accurate weighting (Chantala & Tabor, 1999).

Participants and Study Design

This study included four waves of data from the National Longitudinal Study of Adolescent Health (Add Health), a nationally representative longitudinal study of health (Harris, 2013; Harris et al., 2019; UNC Carolina Population Center, 2020): Waves I (1994–1995; grades 7–12; M=15.65 years [SD = 1.56]), II (collected one year later; grades 8–12; M=16.06 years [SD = 1.60]), III (2001–2002; age range 18–26 years; M=21.54 years [SD = 1.58]), and IV (2008; age range 24–32 years; M=28.55 years [SD = 1.59]). Detailed information about Add Health are provided elsewhere (Harris, 2013; Harris et al., 2019; UNC Carolina Population Center, 2020); information about sampling methods, response and retention rates, and the absence of non-response bias are provided in the Online Supplement 1. This

study included 338 youth who self-identified as AI; no data were available on where youth resided (e.g., reservations/reserves or other settings). The Add Health study was approved by the University of North Carolina Institutional Review Board (IRB) and informed consent was obtained (Harris, 2013). This secondary analysis was not considered human subjects research by the Oklahoma State University IRB.

Measures

Participants self-reported age and sex at baseline.

Baseline Measures

Neighborhood Characteristics.: Parents reported on neighborhood drug exposure ("In this neighborhood, how big a problem are drug dealers and drug users?"); higher scores reflected greater drug exposure (range: 1–3). Perceived neighborhood connectedness was assessed by asking the following: "People in this neighborhood look out for each other;" responses were "true" (0)/ "false" (1). Participants reported difficulties with accessing medical care (1 [very easy] to 4 [very hard]). Census data identified the block level proportion of individuals <18 years living below the poverty line; higher scores reflected greater poverty (range: 0 to .84). Prior research has identified these contextual factors as smoking correlates (Hautala et al., 2020; Henson et al., 2017; National Cancer Institute, 2017).

Adult Relationships.: Participants answered five questions about how much they believed parents cared about them, how supported they felt by adults, and about family relationships. Response options were identical for all questions, with scores ranging from 0 (not at all) to 5 (very much). A latent variable was created using these five indicators: Supportive Adult Relationships. Prior research has used similar questions when assessing social support among AI youth (Henson et al., 2017).

<u>Caregiver Smoking.</u>: Caregivers indicated if adolescents lived with caregivers who smoked (no[0]/yes[1]), a risk factor for smoking in prior research (Vuolo & Staff, 2013), including among AI individuals (Hautala et al., 2020).

<u>Peer Factors.</u>: Peer relationship perceptions were assessed by asking, "How much do you feel that your friends care about you?" with responses ranging from 1 (not at all) to 5 (very much). Participants reported how many of their three best friends smoked daily; higher scores reflected more peer smoking. Peer smoking has been identified as risk factor for smoking (Fuemmeler et al., 2013); peer support has been linked to improved mental health among AI youth (Henson et al., 2017).

<u>Delinquency</u>: Participants reported on the frequency of engagement in delinquent behaviors across fifteen items (e.g., fighting, stealing); an overall score was calculated by averaging across items (range: 0–33), and higher scores reflected greater delinquency (Kelley & Lee, 2018). This measure demonstrated good internal consistency in prior research using Add Health data ($\alpha = .84$)(Kelley & Lee, 2018) and in the current sample ($\alpha = .85$).

<u>Depression.:</u> Depression symptoms were assessed using a nine-item version of the Center for Epidemiological Study-Depression (CES-D). Total scores ranged from 0 to 24. The modified CES-D has demonstrated good psychometric properties in the general population (Grzywacz et al., 2006; Primack et al., 2009), including studies that used Wave I Add Health data ($\alpha = .81$) (Primack et al., 2009) and in the current sample ($\alpha = .80$).

<u>Life Expectancy.</u>: Participants were asked if they believed that they would live to be age 35 years; responses ranged from 0 (almost no chance) to 5 (almost certain). Low perceived life expectancy, as measured here, has been linked to future risk (Nguyen et al., 2012).

Waves I-IV Smoking—Multiple questions that assessed history and current cigarette use frequency were used to create overall ordinal smoking variables that were consistent across waves and used as indicator variables in the growth mixture models. Response options included: not smoking (0); smoked, but not in the past month (1); smoked in the past month, but not daily (2); and smoked daily in the past month (3). This ordinal smoking measure was used over a measure of recent smoking intensity to enhance our ability to capture light or intermittent smoking patterns, a common pattern among younger populations (Chassin et al., 2009). A similar ordinal smoking variable was used in another study on smoking trajectories among AI individuals (Hautala et al., 2020).

Wave IV Health Markers—The following biospecimens were collected in Wave IV using standardized and recommended procedures; detailed procedures can be found in prior studies (Harris, 2013). High sensitivity CRP was measured as described previously (Whitsel et al., 2012). Systolic (SBP) and diastolic (DBP) blood pressure were measured three times and scores were averaged; the average SBP and DBP were used in analyses (Entzel et al., 2009). Triglycerides, HDL-C, and LDL-C cholesterol levels were measured via blood samples collected using finger-pricks; levels were described using deciles (Whitsel et al., 2013).

Wave IV Health Marker Control Variables—All Aim 3 analyses controlled for Wave IV body mass index (BMI) and diabetes (Entzel et al., 2009; Howard et al., 1999; Wang et al., 2006; Whitsel et al., 2012, 2013; Zhang et al., 2015). Other potential confounding variables included in the analyses investigating CRP were (Whitsel et al., 2012): count of potential subclinical sources of infection/inflammation, count of infectious/inflammatory diseases, and recent use of anti-inflammatory medications. Self-reported hyperlipidemia history or use of antihyperlipidemic medication were included in the analyses with lipids (Whitsel et al., 2013).

Data Analysis

Mplus 8.5 was used for all analyses, and the subpopulation command was used to select AI youth for this study (N=338) and to incorporate accurate weighting procedures (Chantala & Tabor, 1999). Missing data indicators were created for the smoking variable at each wave (i.e., a variable indicating whether smoking data was present or missing); missingness rates for smoking ranged from 0.3% (n=1) to 1.5% (n=5). No covariates were significantly related to missingness or smoking (Enders, 2006). Missingness ranged from 9.2% (n=31) to 14.2%

(*n*=48) across Wave IV health variables. Missing data were addressed using the robust full information maximum likelihood approach (Enders & Bandalos, 2001); this approach uses a casewise likelihood function to maximize use of complete and partially available data to estimate parameters (Enders, 2006; Enders & Bandalos, 2001). Robust full information maximum likelihood yields superior performance relative to listwise and pairwise deletion (Enders & Bandalos, 2001) and performs similarly to multiple imputation (Enders, 2006).

For Aim 1, growth mixture modeling (GMM) was used to identify latent smoking classes (Berlin et al., 2013). Several unconditional, latent growth curve models were conducted (intercept only, linear, quadratic, and piecewise; i.e., linear slope from baseline to Wave III and linear slope from Wave III to IV) to determine the best fitting growth model. Based on information criteria and visualization of the data, the quadratic latent growth curve model best fit the data (Online Supplement 2). Next, mixture models identifying one to seven classes were estimated, and an iterative approach to freeing the growth factor variances was used to identify the best fitting model (Feldman et al., 2009). Additionally, a set of models with a forced nonsmoking class were examined (i.e., a user-specified nonsmoking class). Overall model interpretation, information criteria, the Vuong-Lo-Mendell-Rubin (VLMR) test, and the adjusted VLMR tests were used to identify the best fitting GMM. The entropy summary statistics provided information on the accuracy of classification; values closer to 1 indicated greater accuracy (Muthén, 2004). Because the preferred Lo, Mendell, and Rubin likelihood ratio test and the bootstrap likelihood ratio test (Nylund et al., 2007) are not available when using TYPE=COMPLEX in MPlus (the command used to account for the complex survey design), the VLMR and adjusted VLMR were also considered. However, because of documented limitations of VLMR tests (Guerra-Peña & Steinley, 2016; van der Nest et al., 2020) and data supporting the use of the Bayesian Information Criteria (BIC) (Nylund et al., 2007), the BIC was more strongly considered. A recommended consensus-based method was used whereby three co-authors independently identified their proposed best fitting model using the available data, prior research, and theory, and then subsequently met to discuss and reach consensus (Berlin et al., 2018). Regarding Aims 2–3, the Bolk, Croon, and Hagenaars (BCH) method (Asparouhov & Muthén, 2021) for examining covariates associated with class membership is not available when using ALGORITHM=INTEGRATION (which was used in these models); therefore, the most likely smoking class membership was exported and used in Aims 2–3 as an observed variable. This approach can be effectively used when BCH is not available and entropy is high (e.g., >0.90) (Clark & Muthén, 2009). See Online Supplement 3 for diagrams of the models tested in all aims.

For Aim 2, latent variable modeling was conducted to identify predictors, including a latent predictor, of smoking class membership (treated as an observed nominal variable); because the dependent variable was nominal, the model used multinomial logistic regression. A latent variable, *Supportive Adult Relationships*, was modeled using five indicators (standardized loadings ranged from 0.57–0.80; CFI: 0.98, TLI: 0.96, RMSEA: 0.05) and was used as a predictor of class membership. The following predictors were also examined: demographic variables (age, sex), caregiver smoking, peer variables (perception of friendships, peer smoking), contextual variables (perceptions about neighborhood drug exposure, perception of neighbors looking out for one another, difficulties accessing

healthcare, and proportion of individuals <18 years in neighborhood below the poverty line), child perception about living to age 35, and adolescent mental health symptoms (baseline depression symptoms and delinquency). The *chronic heavy smoking* class served as the referent group as it was hypothesized that this class would have the highest health risk. Continuous predictors were centered.

For Aim 3, latent variable modeling was used to simultaneously identify the associations between the observed smoking class membership (derived from Aim 1), all Aim 2 predictors, *Supportive Adult Relationships* (a latent variable), and Wave IV health markers; because the dependent variables were continuous, the model used linear regressions. Wave IV health markers included CRP, triglycerides, HDL-C, LDL-C, and SBP and DBP. Relevant control variables were also included (as described in the Wave IV Health Marker Control Variables subsection of the measures section). The *chronic heavy smoking* class served as the referent group as it was hypothesized that this class would have the highest health risk. Continuous predictor variables were centered. All presented results are standardized; binary predictors retained their original coding and predicted a standardized dependent variable.

Results

Descriptive statistics on participant demographics and study variables are presented in the Online Supplement 4.

Aim 1: Identification of Latent Smoking Classes

The best fitting solution was a six class quadratic GMM where all growth factor variances were estimated (Figure 1; Online Supplement 2). This model was chosen for several reasons. First, it had one of the lowest BIC values, the lowest AIC, and the second lowest adjusted BIC values; further, the entropy value suggested a high degree of separation between classes (.88). Second, the classes that emerged were consistent with prior research on smoking trajectories among general adolescent samples and samples of other racially minoritized youth (Chassin et al., 2009). Further, a nonsmoking class naturally emerged (in other models a nonsmoking class only emerged when user-specified), and a chronic heavy smoking class emerged. See Online Supplement 5 for visual depictions of competing models (i.e., 3- and 4-class models and a 5-class model with a forced nonsmoking class).

The largest class was characterized by light levels of smoking from adolescence into adulthood (n = 121, 35.80%) and was named the *light smoking* class (See Online Supplement 6 for smoking probabilities across classes). The second largest class (n = 79, 23.29%) was named the *nonsmoking* class; this class had high probabilities of abstaining across time. Individuals in the *escalating, adult daily smoking* class (n = 44, 13.06%) had escalating smoking probabilities across adolescence and young adulthood (Waves I-III), with most individuals reporting daily smoking by adulthood (Wave IV). The next largest class, the *chronic heavy smoking* class (n = 40, 11.80%) demonstrated the highest level of smoking across time. Individuals in the *escalating, young adult daily smoking* class (n = 32, 9.49%) differed from individuals in the *escalating, adult daily smoking* class because these youth reached daily smoking by young adulthood (Wave III). The smallest class, the

reducing smoking class (n = 22, 6.55%), escalated to daily smoking by Wave III but reported decreased smoking in Wave IV.

Aim 2: Identification of Predictors of Smoking Class Membership

Multiple individual and contextual predictors of smoking classes were investigated (Table 1). Compared to the *chronic heavy smoking* class, AI peoples in both the *nonsmoking* and *light smoking* classes had fewer friends who smoked (this second finding only approached significance: OR: 0.67, CI: 0.45–0.999). Compared to the *chronic heavy smoking* class, AI individuals in the *light smoking* and *escalating, young adult daily smoking* classes were younger; AI individuals in the *reducing smoking* class lived in more impoverished neighborhoods during adolescence.

Aim 3: Associations between Smoking, Aim 2 Predictors, and Wave IV Health Markers

The *chronic heavy smoking* class served as the referent group because this class was hypothesized as having the highest health risk due to the highest levels of smoking (Table 2).

CRP—Being female and having greater *Supportive Adult Relationships* during adolescence were associated with higher CRP in adulthood; greater adolescent beliefs about living to age 35 were related to lower CRP. Individuals in the *escalating, young adult daily smoking* class had lower CRP compared to individuals in the *chronic heavy smoking* class (Table 2).

Blood Pressure—Female sex was associated with lower adult SBP and DBP. Having more *Supportive Adult Relationships* was related to lower adult DBP. Though not statistically significant, individuals in the *escalating, adult daily smoking* and the *light smoking* classes had lower DBP relative to those in the *chronic heavy smoking* class (p = .07; Table 2).

Lipids—Predictors of HDL-C, LDL-C, and triglyceride levels in adulthood were also examined. Higher levels of HDL-C are considered healthy while lower levels of LDL-C and triglycerides are considered healthy (Arnett et al., 2019). Individuals in the *light smoking* class had higher HDL-C relative to those in the *chronic heavy smoking* class (Table 2). Having the perception that neighbors <u>do not</u> look out for one another during adolescence was related to lower <u>HDL-C</u> in adulthood; female sex was associated with higher HDL-C. Regarding <u>LDL-C</u>, AIs in the *escalating, young adult daily smoking* class had lower LDL-C in adulthood compared to those in the *chronic heavy smoking* class (Table 2). Being older and perceiving that friends cared about you in adolescence were associated with lower LDL-C. Lastly, greater delinquency, difficulties accessing medical care, and neighborhood drug exposure during adolescence were associated with higher <u>triglycerides</u>. Being in the *escalating, adult daily smoking* class was related to lower <u>triglycerides</u> relative to being in the *chronic heavy smoking* class (Table 2).

Discussion

This study revealed significant heterogeneity in smoking patterns among AI individuals from adolescence through adulthood, identifying six latent smoking classes: *light smoking*

(36%), nonsmoking (23%), escalating, adult daily smoking (13%), chronic heavy smoking (12%), escalating, young adult daily smoking (9%), and reducing smoking (7%). Consistent with the socioecological framework for addressing tobacco-related disparities, multilevel risk and resource factors for smoking and adult cardiovascular health were also identified (National Cancer Institute, 2017). Social support during adolescence was an important resource factor against several cardiovascular risk outcomes.

The present results were somewhat distinct from the two existing studies that investigated longitudinal smoking classes among AI/Indigenous populations (Hautala et al., 2020; Whitesell et al., 2014). Methodological and geographical differences may partially account for differential findings (e.g., Whitesell and colleagues (2014) assessed middle schoolers over a two-year period). The developmental smoking patterns in this study appear more similar to Hautala and colleagues' (2020) findings except for the number of classes identified (i.e., six versus four). For example, the present study's nonsmoking class was similar to their low/non-smokers class, and the present light smoking class was similar to their occasional smokers class; yet, there were notable differences between classes. Hautala and colleagues' (2020) occasional smokers class had higher probabilities of daily/ near daily smoking across time relative to the present *light smoking* class. Further, their class demonstrated daily/near daily smoking reductions by age 26 and was the only class with declining smoking; therefore, the present study's reducing smoking class also overlaps with this class, though with larger reductions in daily smoking in adulthood. This finding highlights a subgroup of AI youth who progressed to daily smoking by young adulthood and yet reduced their smoking in adulthood. Given that youth in the current study were 16-years-old on average at baseline, the present *chronic heavy smoking* class most closely overlaps with their early onset smoker class, and the present escalating, young adult daily smoking class most closely overlaps with their mid-onset smokers class (Hautala et al., 2020). Current findings highlighted two unique classes: the reducing smoking and the escalating, adult daily smoking classes.

Consistent with the socioecological framework for tobacco-related health disparities, the second aim identified individual and contextual factors that were associated with longitudinal smoking patterns (National Cancer Institute, 2017). Being older and having more friends who smoked were risk factors for being in the *chronic*, *heavy smoking* class, which echoes prior research that identified peer deviance as a risk factor for smoking among AI youth (Whitesell et al., 2014). Having friends who smoke poses multifaceted risk due to environmental tobacco smoke exposure and increased risk for personal smoking (Rajkumar et al., 2017; U.S. Department of Health and Human Services, 2014). Lastly, exposure to neighborhood poverty during adolescence was related to an increased likelihood of being in the *reducing smoking* class (versus the *chronic*, *heavy smoking* class). Experiences with poverty during adolescence may have influenced smoking *reductions* across time because of responsivity to tobacco costs (Hyland et al., 2004), including increases in cigarette excise taxes from 1995 to 2009 (Jamison et al., 2009). Cigarette tax increases are one of the most effective and cost-effective methods for reducing smoking, with the most

¹Hautala et al. (2020) *occasional smokers* class' probabilities of daily smoking across time ranged from .01-.38; the present study's *light smoking* class' probabilities of daily/near daily smoking across time ranged from .07-.11.

pronounced benefits for youth and lower income populations (U.S. Department of Health and Human Services, 2020). Overall, these results highlight potential influences on tobaccorelated disparities for AI individuals; importantly, exposure to these, and other, risk factors is influenced by historical and current systemic racism (National Cancer Institute, 2017; Pearson et al., 2021). Findings support the need for increased tobacco interventions among AI populations and illuminate the importance of interventions across individual, family, school, neighborhood, and public health levels.

The third aim of this study examined how longitudinal smoking patterns, and resource and risk factors, were associated with adult cardiovascular risk. After controlling for covariates, including BMI and diabetes, a pattern of results emerged that documented greater cardiovascular risk for individuals in the *chronic heavy smoking* class. Those in the *chronic heavy smoking* class had higher CRP and LDL-C relative to the *escalating, young adult daily smoking* class, and higher triglycerides and DBP (though only a trend) relative to those in the *escalating, adult daily smoking* class. Finally, though these results only trended towards significance, individuals in the *chronic heavy smoking* class also had lower HDL-C and higher DBP compared to those in the *light smoking* class.

Overall, the results demonstrate that being in the *chronic heavy smoking* class was related to a higher CRP, a less favorable lipid profile (i.e., lower HDL-C and higher LDL-C and triglycerides), and higher blood pressure compared to being in a class with less smoking; however, being in the *chronic heavy smoking* class was differentially related to these individual biomarkers across comparisons. For example, those in the *chronic heavy smoking* class had higher LDL-C relative to those in the escalating, young adult daily smoking class; however, there were no differences in HDL-C or triglycerides between these classes. Prior research, largely with non-AI populations, has documented the associations between smoking and these lipids/lipoproteins (Campbell et al., 2008; Craig et al., 1989; U.S. Department of Health and Human Services, 2014); however, data on how developmental smoking patterns relate to these biomarkers among younger adults, and especially with AI younger adults, remains limited. The same gaps in the literature are present for blood pressure, and similar observation can be made about how our findings reflect how smoking is related to higher DBP, but unrelated to SBP. Yet, interpretation of these blood pressure findings are additionally complicated by prior research documenting transient, but potentially not sustained, changes to blood pressure among individuals who smoke (Liu & Byrd, 2017; U.S. Department of Health and Human Services, 2014), including a study documenting lower SBP and DBP among older AI/AN individuals who smoke (Wang et al., 2006). Future research is needed to investigate these differential associations more closely. Furthermore, while important differences in cardiovascular risk emerged across the smoking classes, numerous expected differences between classes were nonsignificant, e.g., differences between the nonsmoking class and the chronic heavy smoking class. This may have due to insufficient power, omission of important variables (e.g., discrimination; cultural identity), or other factors. Nevertheless, these results enhance our understanding of developmental smoking patterns related to adult cardiovascular risk among AI peoples, pointing to potential intervention opportunities for reducing smoking and other risk factors for cardiovascular disease.

This study identified numerous multilevel risk and resource factors associated with AI adult cardiovascular health, and exposure to these risk factors is influenced by systemic racism (Churchwell et al., 2020). Greater delinquency, difficulties accessing medical care, and neighborhood drug exposure during adolescence were associated with higher triglycerides. These findings are consistent with prior research that demonstrated early risk environments were associated with poorer adult metabolic functioning, blood pressure, and CRP levels through multiple pathways, including psychological functioning (Taylor, 2010). Current findings revealed the prospective importance of multi-level social support: Social support during adolescence was protective against several adult cardiovascular risk markers (i.e., DBP and some lipids). This finding is consistent with prior work documenting associations between social support and physiological regulation markers (e.g., lower blood pressure reactivity under stress and lower daily blood pressure) that are important for cardiovascular health among the general population (Uchino, 2006; Yang et al., 2016); yet, there are limited prospective studies (Yang et al., 2016) and scant data on AI health. AI youth with more supportive relationships also had higher CRP, which may reflect the tendency for youth with distress to seek additional social support.

Several limitations should be considered. Add Health is primarily a school-based cohort, which may limit generalizability. Second, smoking rates have significantly declined since the baseline data were collected, which suggests the age of the data may be a limitation. However, AI smoking rates and smoking-related morbidity and mortality have remained high, which highlights that examining longitudinal smoking patterns among AI peoples was warranted (Centers for Disease Control and Prevention (CDC), 2017; Jamal et al., 2016; Zhang et al., 2015). Further, the smoking classes found in this study are similar to those identified in a recent study of AI youth (Hautala et al., 2020) and with patterns commonly identified across studies, populations, and time (Chassin et al., 2009; Park et al., 2018). Future research should investigate other nicotine products, adversity measures (e.g., education; family income), and psychological variables; differentiate between commercial and ceremonial tobacco use; and identify the dose-response relationship between smoking intensity and health among AI peoples. The secondary data analysis precluded an examination of important cultural factors (e.g., trauma, discrimination, cultural identity/ connectedness, and place of residence [e.g., urban, tribal reservation/reserve]) and statistical control of involvement in tobacco programs, limiting the generalizability of findings. Additionally, the lack of baseline cardiovascular health measures limits conclusions that can be drawn.

This study advances our knowledge about developmental smoking patterns for AI peoples and how these patterns, and risk and resource factors, are linked to cardiovascular health. Prior qualitative research (Myhra & Wieling, 2014) and community-based participatory research (CBPR) methods with AI populations (Anderson et al., 2019) highlight that cultural and strengths-based approaches have the potential to advance efforts in reducing tobacco exposure among AI populations. While there is a paucity of empirically-based tobacco prevention and cessation programs among Indigenous populations (Carson, Brinn, Labiszewski, et al., 2012; Carson, Brinn, Peters, et al., 2012; Soto et al., 2018), a promising intervention is the All Nations Breath of Life Smoking Cessation Program, which is a culturally-tailored cessation program that includes social support, motivational interviewing,

culturally-tailored psychoeducation, and nicotine replacement therapy (Choi et al., 2016). Collectively, this prior work highlights the importance of amplifying AI voices in tobacco research and, taken with current findings, incorporating multi-level interventions to reduce tobacco-related inequities among AI peoples.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Funding:

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Information on how to obtain the Add Health data files and about prior publications that uses this data is available on the Add Health website (https://addhealth.cpc.unc.edu/). No direct support was received from grant P01-HD31921 for this analysis. This study was funded by funds from the Oklahoma Tobacco Research Center. Dr. Clawson and Cole's time were also supported by P20 CA253255.

References

- Anderson KM, Kegler MC, Bundy LT, Henderson P, Halfacre J, & Escoffery C (2019). Adaptation of a brief smoke-free homes intervention for American Indian and Alaska Native families. BMC Public Health, 19(1), 1–9. 10.1186/s12889-019-7301-4 [PubMed: 30606151]
- Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, Himmelfarb CD, Khera A, Lloyd-Jones D, McEvoy JW, Michos ED, Miedema MD, Muñoz D, Smith SC, Virani SS, Williams KA, Yeboah J, & Ziaeian B (2019). 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation, 140(11), e596–e646. 10.1161/CIR.000000000000000678 [PubMed: 30879355]
- Asparouhov T, & Muthén B (2021). Auxiliary variables in mixture modeling: Using the BCH method in Mplus to estimate a distal outcome model and an arbitrary second model. Mplus Web Notes No. 21. https://www.statmodel.com/examples/webnotes/webnote21.pdf
- Bassett DR, Nelson L, Rhoades DA, Krantz EM, & Omidpanah A (2014). A national study of social networks and perceptions of health among urban American Indian/Alaska Natives and non-Hispanic whites. Journal of Biosocial Science, 46(4), 556–559.10.1017/S0021932013000679 [PubMed: 24999507]
- Berlin KS, Ankney RL, & Rybak TM (2018). Methods commentary: Uncovering unobserved data patterns with latent variable mixture modeling. Journal of Pediatric Psychology, 43(7), 733–736. 10.1093/jpepsy/jsy042 [PubMed: 29893895]
- Berlin KS, Parra GR, & Williams NA (2013). An introduction to latent variable mixture modeling (part 2): longitudinal latent class growth analysis and growth mixture models. Journal of Pediatric Psychology, 39(2), 188–203. 10.1093/jpepsy/jst085 [PubMed: 24277770]
- Breathett K, Sims M, Gross M, Jackson EA, Jones EJ, Navas-Acien A, Taylor H, Thomas KL, & Howard BV (2020). Cardiovascular health in American Indians and Alaska Natives: A scientific statement from the American Heart Association. Circulation, 141(25), E948–E959. 10.1161/CIR.0000000000000773 [PubMed: 32460555]
- Burlew AK, Feaster D, Brecht M-L, & Hubbard R (2009). Measurement and data analysis in research addressing health disparities in substance abuse. Journal of Substance Abuse Treatment, 36(1), 25–43. 10.1016/j.jsat.2008.04.003 [PubMed: 18550320]
- Campbell SC, Moffatt RJ, & Stamford BA (2008). Smoking and smoking cessation-The relationship between cardiovascular disease and lipoprotein metabolism: A review. Atherosclerosis, 201(2), 225–235. 10.1016/j.atherosclerosis.2008.04.046 [PubMed: 18565528]

Carson KV, Brinn MP, Labiszewski NA, Peters M, Chang AB, Veale A, Esterman AJ, & Smith BJ (2012). Interventions for tobacco use prevention in Indigenous youth. Cochrane Database of Systematic Reviews, 8, Article CD009325. 10.1002/14651858.CD009325.pub2

- Carson KV, Brinn MP, Peters M, Veale A, Esterman AJ, & Smith BJ (2012). Interventions for smoking cessation in Indigenous populations. Cochrane Database of Systematic Reviews, 1, Article CD009046. 10.1002/14651858.CD009046.pub2 [PubMed: 22258998]
- Centers for Disease Control and Prevention. (2021). Burden of cigarette use in the U.S.: Current cigarette smoking among U.S. adults aged 18 years and older. https://www.cdc.gov/tobacco/campaign/tips/resources/data/cigarette-smoking-in-united-states.html
- Centers for Disease Control and Prevention. (2017). 1991–2019 High School Youth Risk Behavior Survey Data. http://nccd.cdc.gov/youthonline/
- Chantala K, & Tabor J (1999). National Longitudinal Study of Adolescent Health: Strategies to perform a design-based analysis using the Add Health data. https://addhealth.cpc.unc.edu/wp-content/uploads/docs/user_guides/weights1.pdf
- Chassin L, Curran P, Presson C, Sherman S, & Wirth R (2009). Developmental trajectories of cigarette smoking from adolescence to adulthood. In Phenotypes and endophenotypes: Foundations for genetic studies of nicotine use and dependence. (pp. 189–244).
- Choi WS, Beebe LA, Nazir N, Kaur B, Hopkins M, Talawyma M, Shireman TI, Yeh H-W, Greiner KA, & Daley CM (2016). All Nations Breath of Life. A randomized trial of smoking cessation for American Indians. American Journal of Preventive Medicine, 51(5), 743–751. 10.1016/j.amepre.2016.05.021 [PubMed: 27436332]
- Churchwell K, Elkind MSV, Benjamin RM, Carson AP, Chang EK, Lawrence W, Mills A, Odom TM, Rodriguez CJ, Rodriguez F, Sanchez E, Sharrief AZ, Sims M, & Williams O (2020). Call to action: Structural racism as a fundamental driver of health disparities: A presidential advisory from the American Heart Association. Circulation, 142, E454–E468. 10.1161/CIR.00000000000000936 [PubMed: 33170755]
- Clark S, & Muthén BO (2009). Relating latent class analysis results to variables not Included in the analysis. https://www.statmodel.com/download/relatinglca.pdf
- Craig WY, Palomaki GE, & Haddow JE (1989). Cigarette smoking and serum lipid and lipoprotein concentrations: an analysis of published data. BMJ, 298(6676), 784–788. [PubMed: 2496857]
- Enders CK (2006). A primer on the use of modern missing-data methods in psychosomatic medicine research. Psychosomatic Medicine, 68(3), 427–436. 10.1097/01.psy.0000221275.75056.d8 [PubMed: 16738075]
- Enders CK, & Bandalos DL (2001). The Relative Performance of Full Information Maximum Likelihood Estimation for Missing Data in Structural Equation Models. Structural Equation Modeling: A Multidisciplinary Journal, 8(3), 430–457. 10.1207/S15328007SEM0803_5
- Entzel P, Whitsel EA, Hallquist S, & Halpern CT (2009). Add Health Wave IV documentation: Cardiovascular and anthropometric measures. https://addhealth.cpc.unc.edu/wp-content/uploads/docs/user_guides/Wave_IV_Cardiovascular_and_anthropometric_documentation.pdf
- Fabsitz RR, Sidawy AN, Go O, Lee ET, Welty TK, Devereux RB, & Howard BV (1999). Prevalence of peripheral arterial disease and associated risk factors in American Indians: The Strong Heart Study. American Journal of Epidemiology, 149(4), 330–338. [PubMed: 10025475]
- Feldman BJ, Masyn KE, & Conger RD (2009). New approaches to studying problem behaviors: A comparison of methods for modeling longitudinal, categorical adolescent drinking data. Developmental Psychology, 45(3), 652–676. [PubMed: 19413423]
- Fuemmeler B, Lee CT, Ranby KW, Clark T, McClernon FJ, Yang C, & Kollins SH (2013). Individual- and community-level correlates of cigarette-smoking trajectories from age 13 to 32 in a U.S. population-based sample. Drug and Alcohol Dependence, 132(1–2), 301–308. 10.1016/j.drugalcdep.2013.02.021 [PubMed: 23499056]
- Grzywacz JG, Hovey JD, Seligman LD, Arcury TA, & Quandt SA (2006). Evaluating short-form versions of the CES-D for measuring depressive symptoms among immigrants from Mexico. Hispanic Journal of Behavioral Sciences, 28(3), 404–424. 10.1177/0739986306290645 [PubMed: 31768090]

Guerra-Peña K, & Steinley D (2016). Extracting spurious latent classes in growth mixture modeling With nonnormal errors. Educational and Psychological Measurement, 76(6), 933–953. 10.1177/0013164416633735 [PubMed: 29795894]

- Harris KM (2013). The Add Health Study: Design and accomplishments. https://addhealth.cpc.unc.edu/wp-content/uploads/docs/user_guides/DesignPaperWave_I-IV.pdf
- Harris KM, Halpern CT, Whitsel EA, Hussey JM, Killeya-Jones LA, Tabor J, & Dean SC (2019). Cohort profile: The National Longitudinal Study of Adolescent to Adult Health (Add Health). International Journal of Epidemiology, 48(5), 1415–1415k. 10.1093/ije/dyz115 [PubMed: 31257425]
- Hautala D, Sittner K, & Walls M (2020). Latent trajectories and profiles of commercial cigarette smoking frequency from adolescence to young adulthood among North American Indigenous People. Nicotine & Tobacco Research, 22(11), 2066–2074. 10.1093/ntr/ntaa063 [PubMed: 32270190]
- Henson M, Sabo S, Trujillo A, & Teufel-Shone N (2017). Identifying protective factors to promote health in American Indian and Alaska Native adolescents: A literature review. The Journal of Primary Prevention, 38(1–2), 5–26. 10.1007/s10935-016-0455-2 [PubMed: 27826690]
- Howard BV, Lee ET, Cowan LD, Devereux RB, Galloway JM, Go OT, Howard WJ, Rhoades ER,
 Robbins DC, Sievers ML, & Welty TK (1999). Rising tide of cardiovascular disease in American
 Indians: The Strong Heart Study. Circulation, 99(18), 2389–2395. 10.1161/01.CIR.99.18.2389
 [PubMed: 10318659]
- Hyland A, Li Q, Bauer J, Giovino G, Steger C, & Cummings KM (2004). Predictors of cessation in a cohort of current and former smokers followed over 13 years. Nicotine & Tobacco Research, 6(6), 363–369. 10.1080/14622200412331320761
- Jamal A, King BA, Neff LJ, Whitmill J, Babb SD, & Graffunder CM (2016). Current cigarette smoking among adults—United States, 2005–2015. MMWR. Morbidity and Mortality Weekly Report, 65(44), 1205–1211. 10.15585/mmwr.mm6544a2 [PubMed: 27832052]
- Jamison J, Tynan M, MacNeil A, & Merritt R (2009). Federal and state cigarette excise taxes United States, 1995--2009. Morbidity and Mortality Weekly Report (MMWR), 58(19), 524–527. [PubMed: 19478719]
- Kelley MS, & Lee MJ (2018). When natural mentors matter: Unraveling the relationship with delinquency. Children and Youth Services Review, 91, 319–328. 10.1016/j.childyouth.2018.06.002
- Lawrence EM, Pampel FC, & Mollborn S (2014). Life course transitions and racial and ethnic differences in smoking prevalence. Advances in Life Course Research, 22, 27–40. 10.1016/ j.alcr.2014.03.002 [PubMed: 26047689]
- LeMaster PL, Connell CM, Mitchell CM, & Manson SM (2002). Tobacco use among American Indian adolescents: Protective and risk factors. Journal of Adolescent Health, 30(6), 426–432.
- Liu X, & Byrd JB (2017). Cigarette smoking and subtypes of uncontrolled blood pressure among diagnosed hypertensive patients: Paradoxical associations and implications. American Journal of Hypertension, 30(6), 602–609. 10.1093/ajh/hpx014 [PubMed: 28203691]
- McGill HC, McMahan CA, & Gidding SS (2008). Preventing heart disease in the 21st century: Implications of the pathobiological determinants of atherosclerosis in youth (PDAY) study. Circulation, 117(9), 1216–1227. 10.1161/CIRCULATIONAHA.107.717033 [PubMed: 18316498]
- Mowery PD, Dube SR, Thorne SL, Garrett BE, Homa DM, & Nez Henderson P (2015). Disparities in smoking-related mortality among American Indians/Alaska Natives. American Journal of Preventive Medicine, 49(5), 738–744. 10.1016/j.amepre.2015.05.002 [PubMed: 26163166]
- Muthén BO (2004). Mplus. Statistical analysis with latent variables. Technical appendices. Muthen & Muthen. https://www.statmodel.com/download/techappen.pdf
- Muthen LK, & Muthen BO (2017). Mplus User's Guide. Eighth Edition. 10.1111/j.1600-0447.2011.01711.x
- Myhra LL, & Wieling E (2014). Intergenerational patterns of substance abuse among urban american indian families. Journal of Ethnicity in Substance Abuse, 13(1), 1–22. 10.1080/15332640.2013.847391 [PubMed: 24564557]
- National Cancer Institute. (2017). National Cancer Institute Tobacco Control Monograph 22. A socioecological approach to addressing tobacco-related health disparities (NIH Publication No.

- 17-CA-8035A). U.S. Department of Health and Human Services, National Institutes of Health. https://cancercontrol.cancer.gov/brp/tcrb/monographs/monograph-22
- Nguyen QC, Hussey JM, Halpern CT, Villaveces A, Marshall SW, Siddiqi A, & Poole C (2012). Adolescent expectations of early death predict young adult socioeconomic status. Social Science and Medicine, 74(9), 1452–1460. 10.1016/j.socscimed.2012.01.006 [PubMed: 22405687]
- Nylund KL, Asparouhov T, & Muthén BO (2007). Deciding on the number of classes in latent class analysis and growth mixture modeling: A Monte Carlo simulation study. Structural Equation Modeling: A Multidisciplinary Journal, 14(4), 535–569. 10.1080/10705510701575396
- Park E, McCoy TP, Erausquin JT, & Bartlett R (2018). Trajectories of risk behaviors across adolescence and young adulthood: The role of race and ethnicity. Addictive Behaviors, 76, 1–7. 10.1016/j.addbeh.2017.07.014 [PubMed: 28734192]
- Pearson JL, Waa A, Siddiqi K, Edwards R, Nez Henderson P, & Webb Hooper M (2021). Naming racism, not race, as a determinant of tobacco-related health disparities. Nicotine & Tobacco Research, 23(6), 885–887. 10.1093/ntr/ntab059 [PubMed: 33822185]
- Primack BA, Swanier B, Georgiopoulos AM, Land SR, & Fine MJ (2009). Association between media use in adolescence and depression in young adulthood: A longitudinal study. Archives of General Psychiatry, 66(2), 181–188. 10.1001/archgenpsychiatry.2008.532 [PubMed: 19188540]
- Raghuveer G, White DA, Hayman LL, Woo JG, Villafane J, Celermajer D, Ward KD, de Ferranti SD, & Zachariah J (2016). Cardiovascular consequences of childhood secondhand tobacco smoke exposure: Prevailing evidence, burden, and racial and socioeconomic disparities: A scientific statement from the American Heart Association. Circulation, 134(16), e336–e359. 10.1161/CIR.0000000000000443 [PubMed: 27619923]
- Rajkumar S, Fretts AM, Howard BV, Yeh F, & Clark ML (2017). The relationship between environmental tobacco smoke exposure and cardiovascular disease and the potential modifying effect of diet in a prospective cohort among american indians: The Strong Heart Study. International Journal of Environmental Research and Public Health, 14(5), 1–11. 10.3390/ijerph14050504
- Soto C, Unger JB, Sussman S, & Zeledon I (2018). International approaches to tobacco use cessation programming and prevention interventions among Indigenous adolescents and young adults. Current Addiction Reports, 5(1), 35–41. 10.1007/s40429-018-0186-y
- Taylor SE (2010). Mechanisms linking early life stress to adult health outcomes. Proceedings of the National Academy of Sciences of the United States of America, 107(19), 8507–8512. 10.1073/pnas.1003890107 [PubMed: 20442329]
- U.S. Department of Health and Human Services. (2014). The health consequences of smoking: 50 years of progress. A report of the Surgeon General. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. https://www.ncbi.nlm.nih.gov/books/NBK179276/pdf/Bookshelf_NBK179276.pdf
- U.S. Department of Health and Human Services. (2020). Smoking cessation: A report of the Surgeon General. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. https://www.hhs.gov/sites/default/files/2020-cessation-sgr-full-report.pdf
- Uchino BN (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. Journal of Behavioral Medicine, 29(4), 377–387. 10.1007/s10865-006-9056-5 [PubMed: 16758315]
- UNC Carolina Population Center. (2020). National Longitudinal Study of Adolescent to Adult Health: Study design. https://addhealth.cpc.unc.edu/wp-content/uploads/docs/documentations/2020_Study_Design.pdf
- van der Nest G, Lima Passos V, Candel MJJM, & van Breukelen GJP (2020). An overview of mixture modelling for latent evolutions in longitudinal data: Modelling approaches, fit statistics and software. Advances in Life Course Research, 43(September 2019), 100323. 10.1016/j.alcr.2019.100323
- Vuolo M, & Staff J (2013). Parent and child cigarette use: a longitudinal, multigenerational study. Pediatrics, 132(3), e568–77. 10.1542/peds.2013-0067 [PubMed: 23918887]

Wang W, Lee ET, Fabsitz RR, Devereux R, Best L, Welty TK, & Howard BV (2006). A longitudinal study of hypertension risk factors and their relation to cardiovascular disease: The Strong Heart Study. Hypertension, 47(3), 403–409. 10.1161/01.HYP.0000200710.29498.80 [PubMed: 16432042]

- Whitesell NR, Asdigian NL, Kaufman CE, Crow CB, Shangreau C, Keane EM, Mousseau AC, Mitchell CM, Big Crow C, Shangreau C, Keane EM, Mousseau AC, & Mitchell CM (2014). Trajectories of substance use among young American Indian adolescents: Patterns and predictors. Journal of Youth and Adolescence, 43(3), 437–453. 10.1007/s10964-013-0026-2 [PubMed: 24136376]
- Whitsel EA, Cuthbertson CC, Tabor J, Potter AJ, Wener MH, Clapshaw PA, Killeya-Jones L, Halpern CT, & Mullan Harris K (2013). Add Health Wave IV documentation report: Lipids. https://addhealth.cpc.unc.edu/wp-content/uploads/docs/user_guides/Wave_IV_Lipids_documentation.pdf
- Whitsel EA, Cuthbertson CC, Tabor JW, Potter AJ, Wener MH, Killeya-Jones L, & Harris KM (2012). Add Health Wave IV documentation: Measures of inflammation and immune function. 10.17615/C60M2R
- Yang YC, Boen C, Gerken K, Li T, Schorpp K, & Harris KM (2016). Social relationships and physiological determinants of longevity across the human life span. Proceedings of the National Academy of Sciences of the United States of America, 113(3), 578–583. 10.1073/ pnas.1511085112 [PubMed: 26729882]
- Zhang M, An Q, Yeh F, Zhang Y, Howard BV, Lee ET, & Zhao J (2015). Smoking-attributable mortality in American Indians: findings from the Strong Heart Study. European Journal of Epidemiology, 30(7), 553–561. 10.1007/s10654-015-0031-8 [PubMed: 25968176]

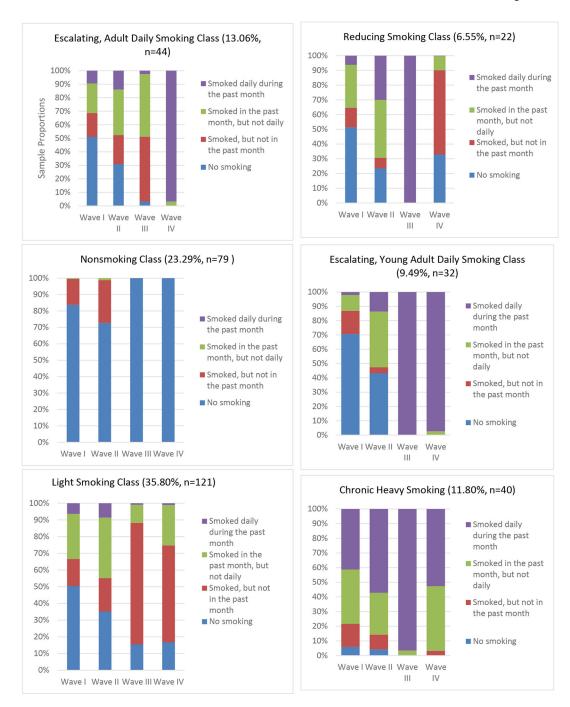


Figure 1.

Longitudinal Latent Smoking Classes from Adolescence to Adulthood: Six Class Model.

Table 1.

Predictors of Smoking Class Membership

		Chr	Chronic Heavy Smoking Class versus $^{\it a}$	ss versus ^a	
	Escalating, Adult Daily Smoking Class	Nonsmoking Class	Light Smoking Class	Reducing Smoking Class	Escalating, Young Adult Daily Smoking Class
	OR (CI)	OR (CI)	OR (CI)	OR (CI)	OR (CI)
Baseline Predictors					
Age	.68 (.43–1.08)	.76 (.55–1.06)	.71 (.51–.98)	1.08 (.65–1.78)	.43 (.28–.66)
Female	.43 (.12–1.57)	1.47 (.42–5.19)	1.07 (.29–3.91)	.69 (.15–3.26)	.76 (.20–2.90)
Perception that will live to be 35	1.22 (.67–2.20)	1.19 (.66–2.15)	1.31 (.81–2.11)	.94 (.42–2.09)	3.67 (.89–15.24)
Delinquency	1.02 (.92–1.13)	.93 (.86–1.01)	.99 (.91–1.08)	.97 (.87–1.08)	.99 (.89–1.09)
Depression	.94 (.77–1.15)	1.01 (.92–1.12)	.98 (.88–1.08)	1.00 (.87–1.15)	.90 (.74–1.10)
Feel that friends care about you	.98 (.46–2.10)	.79 (.45–1.41)	.91 (.49–1.72)	1.18 (.57–2.43)	.88 (.41–1.90)
# of friends who smoke daily	.80 (.49–1.31)	.41 (.21–.81)	.67 (.45–.999)	.79 (.44–1.43)	.58 (.32–1.07)
Live in home with people who smoke	.69 (.14–3.32)	.84 (.26–2.71)	.63 (.23–1.73)	1.79 (.47–6.83)	2.89 (.59–14.23)
Latent adult supportive relationships variable	1.39 (.21–9.05)	1.15 (.28–4.75)	.98 (.22–4.31)	.89 (.12–6.54)	.21 (.03–1.29)
Proportion of those <18 who live in poverty	3.63 (.23–57.34)	4.67 (.25–88.39)	9.86 (.92–106.30)	50.13 (6.18-406.43)	2.52 (.08–83.18)
Difficulties accessing healthcare	2.81 (.96–8.22)	2.16 (.74–6.30)	1.85 (.71–4.87)	1.68 (.54–5.23)	2.50 (.81–7.70)
Drug problems in neighborhood	1.39 (.48–4.04)	1.48 (.70–3.14)	.97 (.42–2.25)	.75 (.30–1.86)	1.57 (.56-4.39)
Perception that neighbors in neighborhood do not watch out for each other	1.49 (.35–6.33)	2.56 (.71–9.20)	1.58 (.47–5.37)	2.17 (.59–8.03)	1.57 (.56-4.39)

Notes:

OR = Odds Ratio; CI = 95% Confidence Interval. Continuous predictor variables were centered (except the latent supportive relationships variable).

 $^{^{4}\}mathrm{The}$ "Chronic, heavy smoking class" is the reference category for latent classes.

Author Manuscript

Table 2.

Predictors of Adult (Wave IV) Health Markers when Smoking Referent Class is the Chronic Heavy Smoking Class

	C-Reactiv	ve Protein	Systolic Blo	Systolic Blood Pressure	Diastolic Blood Pressure	od Pressure	High-density	High-density Lipoprotein	Low-d Lipopi	Low-density Lipoprotein	Triglycerides	erides
Predictors	В	d	В	d	В	d	В	d	В	d	В	р
Escalating, Adult Daily Smoking Class ^a	.01	96.	17	49.	43	.07	25	.26	.26	.42	99	.04
Light Smoking Class ^a	07	.74	09	59.	31	.07	.39	.05	03	68°	23	.30
Reducing Smoking Class ^a	09	77.	.001	266	01	76.	31	.33	52	.14	57	.11
Escalating, Young Adult Daily Smoking Class ^a	40	.049	12	.72	30	.37	90.	98.	61	.04	38	.25
Nonsmoking Class ^a	.12	.61	05	LL:	12	.52	03	68°	02	56.	43	.10
Age	80.	.22	.04	.48	.00	.72	04	.58	25	100'	.07	.29
Female	.30	<.001	30	<.001	23	.001	.31	<.001	.001	86.	03	99.
Perception that will live to be 35	13	.04	01	.93	.07	.45	.03	69.	.07	.23	.07	.42
Delinquency	90.	.38	.01	.91	.01	.90	.13	.05	.11	.14	.18	.047
Depression	10	.12	13	.21	15	.16	05	.36	11	.16	05	.61
Feel that friends care about you	07	.26	.09	.21	.13	.08	.07	.30	16	.01	09	.26
# of friends who smoke daily	.06	.23	05	.40	04	.52	01	.87	02	.81	01	.82
Live in home with people who smoke	.002	86.	02	.83	.01	.90	.01	.94	.004	96.	10	.13
Latent supportive relationships variable	.21	.004	15	.12	16	.04	15	.13	.04	69.	.04	.65
Proportion of those <18 who live in poverty	.09	.21	.001	66.	.02	.81	01	.90	04	.55	08	.32
Difficulties accessing healthcare	.00	.73	.03	69°	.004	.95	.01	.80	12	.16	.13	.047
Drug exposure in neighborhood	08	.26	.07	.45	.07	.47	01	.90	.04	.61	.16	900.
Perception that neighbors do not watch out for each other	02	62.	08	.24	05	.47	18	.005	02	.81	08	.13

Notes:

index and diabetes. The following additional covariates were included based on the distal outcome: CRP: Wave IV including a count of potential subclinical sources of infection or inflammation, a count of common infectious or inflammatory diseases, and recent use of anti-inflammatory medications; TG, HDL, LDL: Wave IV self-reported history of hyperlipidemia or use of antihyperlipidemic medication. ^aThe Chronic Heavy Smoking Class is the reference category for latent classes. Analyses also controlled for relevant covariates for the distal outcomes: All analyses controlled for Wave IV Body mass STDY standardization was used for binary independent variables and STDYX standardization was used for continuous independent variables.