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Adolescent tolerance for deviance, cigarette smoking trajectories, and premature mortality: A longitudinal study

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ABSTRACT

Cigarette smoking is a well-established cause of excess morbidity and mortality in the United States and globally. The current study builds on the existing literature by examining how smoking trajectories might be a mechanism through which adolescent tolerance for deviance predicts premature all-cause and tobacco-specific mortality. Participants were from a cohort-sequential study conducted in the Midwestern United States of the natural history of cigarette smoking from adolescence through midlife that collected nine waves of data from 1980 to 2011. For the current study, we selected participants who were measured at least once at age 18 or older and who did not die before age 24 (n = 7575). Participants' tolerance for deviance was assessed in adolescence, smoking trajectory group was based on self-reported smoking status during the first six waves of data collection, and cause of death for deceased participants (n = 222) was obtained from the National Death Index. Mediation analyses using the joint significance test were conducted separately for all-cause mortality and tobacco-specific mortality. Adolescent tolerance for deviance significantly predicted smoking trajectory group over and above the influence of covariates. Adolescents with higher tolerance for deviance were more likely to belong to any smoking trajectory group compared to abstainers, and membership in a smoking trajectory group characterized by early onset and heavy, persistent smoking was related to premature all-cause and tobacco-specific mortality. Finally, smoking trajectory group was a significant mediator of the relation between adolescent tolerance for deviance and all-cause mortality.

1. Introduction

Cigarette smoking is a well-established cause of excess morbidity and mortality and is responsible for about 480,000 deaths annually in the United States (U.S. Department of Health and Human Services, 2014) and 5 million deaths each year globally (World Health Organization, 2012). Previous research linking cigarette smoking to mortality reported that 21% of deaths among men and 17% of deaths among women in the United States in a year were attributable to smoking (Fenelon and Preston, 2012), and even individuals who smoked at low levels were found to be at elevated risk for all-cause mortality and some smoking-related causes of death (Inoue-Choi et al., 2017).

Most prior studies examining the link between cigarette smoking and mortality have tested death at relatively old ages. For example, Doll et al. (2004) analyzed the effects of smoking on mortality at age 60 or older, and Inoue-Choi et al. (2017) followed a sample of participants whose ages ranged from 59 to 82 at baseline to prospectively predict mortality. One exception found a significant positive relationship between smoking and heart disease mortality among individuals age 18 to 44 (Khan et al., 2015).

To our knowledge, this is the first study that has compared the effect of membership in different smoking trajectory groups on subsequent mortality. In contrast, previous work has relied on comparisons among current smokers of different quantities, former smokers, and non-smokers (Doll et al., 2004; Inoue-Choi et al., 2017; Khan et al., 2015). Using empirically derived trajectory groups provides a better picture of smoking behavior than static measures by taking into account how individuals' smoking behavior evolves over time. Specifically, developmental trajectories consider multiple features such as age of onset, speed of acceleration in smoking rate, and variability versus persistence in smoking over time (Chassin et al., 2009). Moreover, because smoking trajectory groups have been shown to vary in their antecedents and correlated risk factors, we expected them to similarly vary in terms of

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premature mortality risk.

In addition to predicting premature mortality, cigarette smoking may serve as a mechanism by which other factors raise risk for early death. For example, low socioeconomic status, family problems, familial substance use and anti-sociality (Conrad et al., 1992), and child psychopathology (Voorhees et al., 2002), especially attention deficit hyperactivity disorder (Milberger et al., 1997), have all been associated with cigarette smoking and may be linked to early death because of cigarette smoking. Personality may also be linked to early death because certain personality characteristics predict cigarette smoking. For example, studies have found that higher levels of extraversion and neuroticism at age 16 predicted subsequent cigarette smoking (Munafó and Black, 2007) as did lower levels of childhood conscientiousness (Hampson et al., 2006). Adolescents who are more tolerant of deviant behavior are more likely to engage in problem behavior (Jessor and Jessor, 1977) including substance use (Wills et al., 1996) and risky sexual behavior (Costa et al., 1995). Their risky behavior may make it more likely for them to die prematurely and, if they engage in early, persistent, and heavy smoking, their exposure may partially explain their risk.

The current longitudinal study builds on the existing literature by examining deaths that occurred at an earlier age than most prior studies, using cigarette smoking trajectory groups as the measure of smoking behavior, and testing whether tolerance for deviance measured in adolescence predicts mortality indirectly through smoking trajectory group. Specifically, the study tested mortality due to all causes and tobacco-specific causes at a relatively young age, where age of death ranged from 24 to 51. We hypothesized that participants in a smoking trajectory group characterized by early onset of smoking and heavy persistent smoking over time would be at the highest risk for premature mortality. Furthermore, we tested tolerance for deviance as an adolescent personality characteristic that might be related to premature death because of its relation to cigarette smoking. We tested adolescent tolerance for deviance as a prospective predictor of mortality outcomes, and examined whether this relationship was mediated by smoking trajectory group. Finally, our models controlled for other factors available in the longitudinal data set that might impact the risk for premature mortality. These included educational attainment, other tobacco use, physical activity, healthy eating, and seat belt use. Previous studies testing predictors of elevated mortality risk have similarly included demographic factors and engagement in other healthy and risky behaviors as covariates (Batty et al., 2016; Inoue-Choi et al., 2017; Khan et al., 2015).

2. Methods

2.1. Participants

Participants were from the Indiana University Smoking Survey, an ongoing cohort-sequential study of the natural history of cigarette smoking (Chassin et al., 2000; Chassin et al., 2018). Between 1980 and 1983, all consenting 6th to 12th graders in a Midwestern county school system completed annual surveys. A total of 8543 participants were enrolled in the study. Follow-up surveys were conducted in 1987, 1993, 1999, 2005, and 2011. At each wave, 70% or more of the original sample was retained. In 1987, survey data were collected with group-administered questionnaires in school for cohorts who were still in high school. For older cohorts and for all participants in 1993, 1999, 2005, and 2011, a survey was sent by mail followed by telephone interviews, and an online survey in 2011, if surveys were not returned. Participants were paid $15 to $35 over the waves, and in 1999, 2005, and 2011 they were also entered into lottery drawings for cash prizes up to $500. The research protocol was approved by the Institutional Review Board at Indiana University, and informed consent was obtained for participation in the research.

For the current study, we selected all participants who were measured at least once at age 18 or older (n = 7586). After excluding 11 participants who died before the age of 24 to be able to prospectively predict death during adulthood, the final sample eligible for analyses was 7575. The sample was 49% female.

2.2. Measures

2.2.1. Educational attainment

At each wave starting in 1987, participants reported the highest level of education completed. For analyses, we created a binary variable (no college education (35%) versus some college education or higher (65%)) based on the educational attainment reported at the last wave of measurement. If the last reported educational attainment was prior to age 25, we used the participant's parents' education as a proxy.

2.2.2. Tolerance for deviance

As adolescents, participants' tolerance for deviance was assessed with 10 items measured on a five-point scale from not wrong to very wrong (e.g., “To damage property that does not belong to you just for fun is…) during the first five measurement waves (1980 to 1987). The items were taken from Schlegel and DiTecco's (1978) empirically shortened version of the Jessor and Jessor (1977) problem behavior theory questionnaire. A mean score was computed at each wave, and for the current analyses, we computed the mean of the available scores from the five waves. A higher value indicated less tolerance for deviance.

2.2.3. Smoking trajectory

At each wave of data collection, participants self-reported their smoking status (as “never smoked, not even a single puff”; “smoked once or twice ‘just to try’ but not in the last month”; do not smoke, but in the past I was a regular smoker”; “smoke regularly, but not more than once a month”; “smoke regularly, but not more than once a week”; “smoke regularly, but not more than once a day”; and “smoke more than once a day”), and they reported the number of cigarettes that they usually smoked each day (from 0 to 20 or more).

To obtain the participant's smoking trajectory group, we used latent growth analysis on data collected from the first six waves. Groups that were defined a priori were stable abstainers (those who had never progressed past trying a cigarette; 52.7%), stable quitters (who were never measured as smokers, but only as ex-smokers; 7.8%); and relapsing/remitting smokers (who reported periods of smoking, quitting, and then smoking again over the waves; 6.9%). All other participants were clustered empirically using Proc Traj (Jones et al., 2001; see Chassin et al., 2009 for details). In addition to the three a priori groups, the latent class growth analysis produced the following groups: developmentally limited smokers (smoked less than half a pack at their peak and gave up smoking by age 30; 3.8%), experimenters (who never smoked more than occasionally and generally quit by age 22; 4.8%), early-onset persistent smokers (started smoking around age 11, escalated quickly, and smoked more than half a pack a day; 6.4%), high school-onset persistent smokers (started to smoke around age 16 and smoked almost as heavily as the early-onset group; 10.5%), and adult-onset smokers (started to smoke around age 21 and smoked at low levels; 7.2%).

2.2.4. Lifetime other tobacco product use

In the 1987 through 2005 waves, participants reported their lifetime and current use of smokeless tobacco, pipes, and cigars. In 2011, participants were also asked about lifetime and current use of snus, hookah, and bidis. For analyses, participants were divided into those who reported lifetime use of any other tobacco product (21.9%) versus those who reported no lifetime use (78.1%).

2.2.5. Other health behaviors

In all waves from 1987 to 2011, participants reported how often

they exercised vigorously or participated in sports or other similar activities, how often they made choices about what to eat based on the health value of foods, and how often they used a seat belt while driving or riding in a car. For the current study, participants were divided into those who exercised 2–3 times per week or more (40.1%) versus those who exercised less than 2–3 times per week (59.9%); those who made choices based on the health value of foods often or always (45.4%) versus those choices never, rarely, or sometimes (54.6%); and those who always used a seat belt (76.1%) versus those who did not (23.9%) at the last measurement available.

2.2.6. All-cause mortality and tobacco-specific mortality

Cause of death for deceased participants (n = 222) was obtained from the National Death Index (National Center for Health Statistics, Centers for Disease Control). The U.S. Surgeon General's 2014 Report, "The Health Consequences of Smoking – 50 Years of Progress" (U.S. Department of Health and Human Services, 2014) was used to confirm causal relationships between cause of death and tobacco use. Four cause of death categories were created: tobacco-related (n = 53), non-tobacco related (n = 66), external cause (e.g., accident, suicide; n = 99), and unknown cause (n = 4). For analyses, binary all-cause mortality (tobacco related, non-tobacco related, external cause, and unknown cause; n = 222) and tobacco-specific mortality (n = 53) variables were created.

2.3. Data analytic strategy

The current study examined how adolescent tolerance for deviance predicted mortality outcomes indirectly through smoking trajectories, for both all-cause mortality and tobacco-specific mortality. Penalized likelihood logistic regression, multinomial logistic regression, and mediation analysis were used to investigate these questions. SAS software version 9.4 for Windows was used for all analyses. A multinomial logistic regression analysis was used to assess the influence of adolescent tolerance for deviance on smoking trajectories, to accommodate the nominal smoking trajectories variable. For each of the mortality outcomes, logistic regression with Firth's penalized maximum likelihood (ML) estimation was used to assess the influence of smoking trajectories and adolescent tolerance for deviance on mortality. Penalized ML estimation was used to account for rare events in the mortality outcome (Firth, 1993), in this case relative rarity of deaths in the data. Missing data was assumed to be missing at random, an assumption made when using ML estimation (Enders, 2010), and ML estimation allowed for the inclusion of participants with incomplete data.

Mediation analyses were conducted separately for the all-cause mortality and tobacco-specific mortality outcomes. As recent research has shown that there is no need to test for an overall effect before testing for mediation (O'Rourke and MacKinnon, 2015, 2018), we did not conduct initial analyses examining the sole influence of adolescent tolerance for deviance on mortality outcomes. In mediation models this effect of the independent variable on the dependent variable is often referred to as the c path, the influence of the independent variable on the mediator is often referred to as the *a* path, and the influence of the mediator on the dependent variable is often referred to as the *b* path (MacKinnon, 2008). The joint significance test was used to detect the presence of mediation by assessing the significance of the *a* and *b* paths separately (MacKinnon, 2008). This test has been shown to have an optimal balance of statistical power and Type I error as compared to other tests of mediation (MacKinnon et al., 2002). As the mediator was nominal and the outcomes were dichotomous, no single value of the mediated effect was calculated. The mediation models depicting these hypothesized effects are shown in Fig. 1a and b.

3. Results

3.1. Correlations among variables

We began by examining Pearson (for continuous variables), point biserial (for continuous and categorical variables), and tetrachoric (for categorical variables) correlations among all study variables in the models. The correlations among the hypothesized covariates, sex, education, other tobacco product use, seat belt use, and healthy eating, were all significant, based on the criteria that confidence intervals calculated for the correlations did not include one. These correlations were used to determine the inclusion of covariates for each outcome. Sex ($r = -0.036$), education ($r = -0.069$), other tobacco product use ($r = 0.033$), seat belt use ($r = -0.093$), and healthy eating ($r = -0.038$) were all significantly correlated with all-cause mortality, so they were included as covariates in the analysis for all-cause mortality. Sex ($r = -0.028$), education ($r = -0.051$), and seat belt use ($r = -0.058$) were significantly correlated with tobacco-specific mortality, so they were included as covariates in the analysis for tobacco-specific mortality. Exercise was not significantly associated with either mortality outcome, so it was excluded from subsequent analyses.

3.2. Covariate effects on smoking trajectories

Our models tested the unique effects of the covariates on membership in the smoking trajectory groups with abstainers as the reference trajectory group in each case. Men were more likely than women to belong to the stable quitter group (OR = 1.59; 95% CI 1.28, 1.98), the experimenter group (OR = 2.40; 95% CI 1.82, 3.17), the early onset group (OR = 2.43; 95% CI 1.88, 3.13), the high school onset group (OR = 1.88; 95% CI 1.54, 2.29), and the relapsing/remitting group (OR = 2.14; 95% CI 1.68, 2.73). Participants with at least some college education were less likely than those with no college education to belong to stable quitter group (OR = 0.71; 95% CI 0.58, 0.87), the experimenter group (OR = 0.67; 95% CI 0.52, 0.85), the early onset group (OR = 0.21; 95% CI 0.17, 0.27), the high school onset group (OR = 0.39; 95% CI 0.33, 0.47), and the relapsing/remitting group (OR = 0.34; 95% CI 0.28, 0.42). Participants who reported using other tobacco products in their lifetime were more likely than participants who did not report using other tobacco products to belong to stable quitter group (OR = 2.34; 95% CI 1.84, 2.99), the developmentally limited group (OR = 1.52; 95% CI 1.09, 2.12), the experimenter group (OR = 1.91; 95% CI 1.40, 2.61), the early onset group (OR = 1.45; 95% CI 1.10, 1.92), the high school onset group (OR = 1.95; 95% CI 1.56, 2.43), the adult onset group (OR = 1.93; 95% CI 1.53, 2.45), and the relapsing/remitting group (OR = 2.58; 95% CI 2.00, 3.32). Participants who reported always wearing a seat belt were less likely than those who reported not always wearing a seat belt to belong to the early onset group (OR = 0.73; 95% CI 0.58, 0.92), the high school onset group (OR = 0.71; 95% CI 0.59, 0.85), the adult onset group (OR = 0.69; 95% CI 0.55, 0.85), and the relapsing/remitting group (OR = 0.75; 95% CI 0.60, 0.94). Finally, participants who reported making choices based on the health value of foods more often were less likely than those who reported making choices based on the health values of foods less often to belong to the early onset group (OR = 0.53; 95% CI 0.42, 0.68), the high school onset group (OR = 0.70; 95% CI 0.59, 0.84), and the relapsing/remitting group (OR = 0.69; 95% CI 0.55, 0.86).

3.3. Adolescent tolerance for deviance predicting smoking trajectories

Results from the multinomial logistic regression with adolescent tolerance for deviance predicting smoking trajectories are shown in Table 1. Profile-Likelihood confidence intervals are reported for the parameter estimates, and Wald confidence intervals are reported for the odds ratios (ORs). Covariate effects are excluded from the table in order to focus on the effects of adolescent tolerance for deviance, the variable

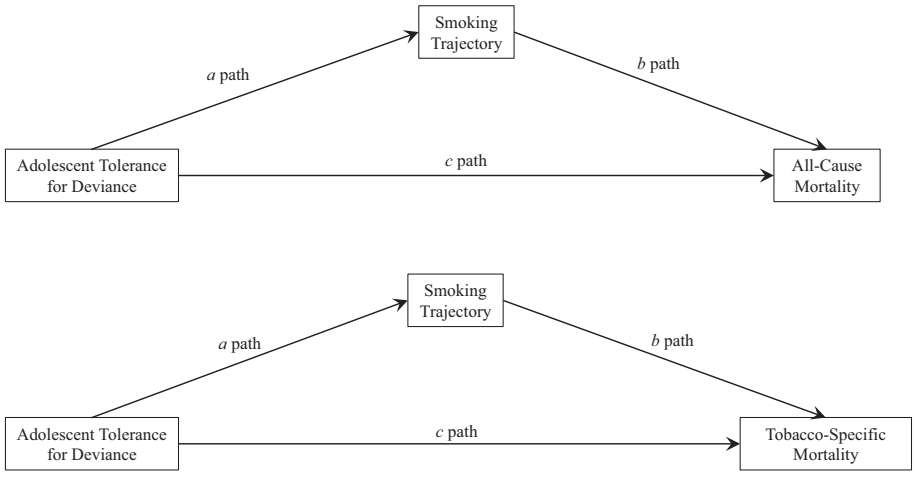

Fig. 1. a. Mediation model for the effect of adolescent tolerance for deviance on all-cause mortality through smoking trajectory, excluding covariates. b. Mediation model for the effect of adolescent tolerance for deviance on tobacco-specific mortality through smoking trajectory, excluding covariates.

of primary interest for the current study. Adolescent tolerance for deviance was a significant predictor of smoking trajectories over and above the influence of the covariates. The estimates in Table 1 are multinomial logit estimates holding all other variables in the model constant, and can be interpreted for each group with abstainers as the reference group, coded as 1. For instance, $\beta = -1.251$ for the effect of tolerance for deviance on the early onset smoking trajectory compared to the abstainer trajectory group. This can be interpreted such that for a one-unit increase in tolerance for deviance, the multinomial log-odds of being in the early onset smoking group would be expected to increase by 1.25, compared to the abstainers group, holding all other model variables constant.

3.4. Adolescent tolerance for deviance and smoking trajectories predicting all-cause mortality

Table 2 shows results from the logistic regression analyses with adjusted ML estimation for the models assessing adolescent tolerance for deviance and smoking trajectories predicting the two mortality outcomes. Both adolescent tolerance for deviance and smoking trajectories were significant predictors of all-cause mortality, with sex and seat belt use as significant covariates in the model. Two of the eight estimates comparing smoking trajectory groups predicting mortality were significant, holding all other variables in the model constant. First, early onset smokers were significantly more likely than abstainers, coded as 1, to have died from any cause (OR = 2.80, 95% CI 1.79, 4.37). Second, high school onset smokers were significantly more likely than abstainers to have died from any cause (OR = 1.61, 95% CI 1.05, 2.45). Finally, adolescents who were less tolerant of deviance were significantly more likely to have died from any cause (OR = 1.37, 95% CI 1.11, 1.69), holding all other variables constant.

3.5. Adolescent tolerance for deviance and smoking trajectories predicting tobacco-specific mortality

Results for tobacco-specific mortality are also shown in Table 2. Education and seat belt use were significant covariates in this model. Adolescent tolerance for deviance and the overall smoking trajectory variable were not significant predictors of tobacco-specific mortality. As hypothesized, however, membership in the early onset smoking trajectory was a significant predictor of tobacco-specific mortality (OR = 2.68, 95% CI 1.13, 6.33).

3.6. Mediation analysis

We used the joint significance test to assess presence of significant mediation for each of the mortality outcomes. For both mediation models, the *a* path was the effect of adolescent tolerance for deviance on smoking trajectories, and this path was significant ($p < .001$). Smoking trajectory group was a significant predictor of all-cause mortality holding adolescent tolerance for deviance and covariates constant (*b* path; $p < .001$). However, smoking trajectory group was not a significant predictor of tobacco-specific mortality holding adolescent tolerance for deviance and covariates constant (*b* path; $p = .257$). In summary, wave 1–6 smoking trajectory group was a significant mediator of the relation between adolescent tolerance for deviance and all-cause mortality but was not a significant mediator of the relation between adolescent tolerance for deviance and tobacco-specific mortality.

4. Discussion

This study adds to our understanding of the link between cigarette smoking and early mortality by examining the causal pathway from tolerance for deviance in adolescence to cigarette smoking trajectory

Table 1
Results from multinomial regression of adolescent tolerance for deviance predicting smoking trajectories, 1980 to 2011, Midwestern U.S.

Outcome	β	SE	Wald χ^2	95% CI β	OR	95% CI OR
Smoking trajectories^						
Stable quitters	−0.442	0.072	37.69	[−0.582, −0.300]	0.643§	[0.558, 0.740]
Developmentally limited	−0.381	0.098	15.15	[−0.571. −0.188]	0.683§	[0.564, 0.828]
Experimenters	−1.077	0.085	161.20	[−1.244, −0.911]	0.340§	[0.288, 0.402]
Early onset	−1.251	0.077	266.01	[−1.402, −1.101]	0.286§	[0.246, 0.333]
High school onset	−0.425	0.064	43.73	[−0.551, −0.299]	0.654§	[0.576, 0.741]
Adult onset	−0.386	0.073	27.62	[−0.529, −0.241]	0.680§	[0.589, 0.785]
Relapsing/remitting	−0.907	0.073	152.98	[−1.051, −0.763]	0.404§	[0.350, 0.466]

Note: ^For all effects, the abstainer trajectory group is the reference group, coded as 1. § indicates 95% Wald confidence limits did not include zero.

Table 2
Results from logistic regressions of adolescent tolerance for deviance and smoking trajectories predicting mortality outcomes, 1980 to 2011, Midwestern U.S.

Predictor	β	SE	Wald χ^2	95% CI β	OR	95% CI OR
	All-cause mortality					
Smoking trajectories (*b* path)						
Stable quitters	−0.079	0.072	0.066	[−0.732, 0.489]	0.924	[0.506, 1.687]
Developmentally limited	−0.977	0.643	2.308	[−2.552, 0.089]	0.376	[0.107, 1.328]
Experimenters	−0.051	0.388	0.017	[−0.902, 0.649]	0.95	[0.444, 2.034]
Early onset	1.028	0.228	20.245	[0.568, 1.470]	2.795§	[1.786, 4.373]
High school onset	0.474	0.216	4.826	[0.036, 0.890]	1.607§	[1.052, 2.452]
Adult onset	0.153	0.282	0.296	[−0.438, 0.679]	1.166	[0.671, 2.024]
Relapsing/remitting	0.418	0.260	2.583	[−0.118, 0.911]	1.518	[0.912, 2.527]
Adolescent tolerance for deviance (*c′* path)	0.314	0.108	8.370	[0.101, 0.531]	1.368§	[1.106, 1.692]
	Tobacco-specific mortality					
Smoking trajectories (*b* path)						
Stable quitters	0.112	0.576	0.038	[−1.225, 1.144]	1.118	[0.362, 3.455]
Developmentally limited	−1.097	1.413	0.603	[−5.945, 0.897]	0.334	[0.021, 5.324]
Experimenters	−1.293	1.413	0.838	[−6.144, 0.711]	0.274	[0.017, 4.378]
Early onset	0.985	0.439	5.030	[0.061, 1.838]	2.677§	[1.132, 6.329]
High school onset	0.384	0.435	0.780	[−0.548, 1.215]	1.468	[0.626, 3.444]
Adult onset	0.748	0.453	2.726	[−0.235, 1.602]	2.113	[0.869, 5.135]
Relapsing/remitting	0.486	0.493	0.972	[−0.596, 1.410]	1.625	[0.619, 4.267]
Adolescent tolerance for deviance (*c′* path)	0.394	0.212	3.463	[−0.036, 0.840]	1.482	[0.979, 2.244]

Note: § indicates 95% Wald confidence limits did not include zero.

into adulthood to premature mortality. The unique contributions include the prospective prediction of mortality among younger people, the use of smoking trajectory groups, and capitalizing on longitudinal data to test the influence of tolerance for deviance measured in adolescence. The first finding of note was the association between smoking behavior and death at a young age, thus demonstrating the high risk of cigarette smoking. Specifically, compared to participants who abstained from cigarette smoking from adolescence into adulthood, those in the smoking trajectory group characterized by early onset and persistent, heavy smoking were significantly more likely to die prematurely from any cause and from a tobacco-specific cause. In addition, study participants in the trajectory group characterized by onset in high school and persistent, heavy smoking were significantly more likely to die from any cause. Importantly, these effects were significant after controlling for other covariates, including education, other lifetime tobacco product use, and other health behaviors. This finding highlights the need to invest in efforts to prevent adolescent initiation of smoking.

A second key finding of the current study was the significant prospective association between tolerance for deviance measured in adolescence and smoking trajectory in adulthood. This effect was significant after controlling for covariates related to smoking trajectory group. For example, men, those with less educational attainment, those who used tobacco products other than cigarettes, and those who reported less engagement in healthy behaviors were more likely to belong to the trajectory groups characterized by higher levels of smoking. In terms of the variable of primary interest for the current study, adolescents who were more tolerant of deviance were more likely to be members of any trajectory group characterized by smoking compared to the abstainer trajectory group. This is consistent with prior work demonstrating a link between adolescent personality traits and subsequent smoking behavior (Hampson et al., 2006; Hayatbaksh et al., 2013; Milberger et al., 1997; Munafó and Black, 2007; Voorhees et al., 2002) and further demonstrates the continued need for adolescent tobacco use prevention campaigns, especially if they can be tailored to reach teens who are more tolerant of deviance. Smoking is one mechanism by which adolescents who are more tolerant of deviant behavior are put at risk for early mortality. In our models that controlled for other factors that elevate the risk for premature death, there was no association between adolescent tolerance for deviance and tobacco-specific mortality, but adolescents who were less tolerant of deviance were more likely to die prematurely of any cause in our sample. Although this finding was in the unexpected direction, previous studies, including meta-analyses, have found no direct association between personality and mortality (Batty et al., 2016; Jokela et al., 2013; Jokela et al., 2014). Indeed, the zero-order correlations from the current data indicated no prospective relation between tolerance for deviance in adolescence and either mortality outcome.

Rather than a direct relationship between personality traits in adolescence and premature mortality, a more plausible mechanism is the one demonstrated in our mediational models. That is, tolerance for deviance in adolescence increases the likelihood of initiating cigarette smoking, and cigarette smoking behavior characterized by early onset that is heavy and persists into adulthood, in turn, increases the risk for premature mortality. In our mediational models, smoking trajectory group was a significant mediator of the relation between adolescent tolerance for deviance and all-cause mortality. When controlling for adolescent tolerance for deviance and covariates, there was a significant association between the overall smoking trajectory variable and all-cause mortality but not tobacco-specific mortality. However, as hypothesized, there was a significant effect of early onset smoking (compared to abstainers) on tobacco-specific mortality. This demonstrates the serious health risk of initiating smoking at early age and escalating quickly to high levels of smoking that persists into adulthood. This smoking trajectory group, characterized by early onset, heavy smoking, and persistence into adulthood likely experiences the highest 'dose' of smoking, putting them at risk for smoking-related death even at a relatively young age. Indeed, a study of a large national sample reported a strong association between smoking and mortality due to heart disease by the age of 44 (Khan et al., 2015).

There are limitations of the current study that should be considered. First, participants' exposure to secondhand smoke was not measured and therefore not included as a covariate in the models. Secondhand smoke exposure has been associated with premature mortality in non-smokers (U.S. Department of Health and Human Services, 2006). Second, the community from which this sample was drawn is predominantly white, non-Hispanic, and located in the Midwestern U.S. Thus, these findings may not generalize to other racial and ethnic groups and other geographic regions or countries. Third, as noted previously, because our models predicted mortality at a young age, there was a relatively small number of deaths from smoking-related cause in the sample. This lopsided distribution of the tobacco-specific mortality outcome variable likely elevated the Type II error rate. Therefore, whereas harm caused by smoking (reflected in the overall smoking trajectory variable) may not be strong enough to induce

tobacco-specific mortality, the fact that early onset stable smoking significantly predicted tobacco-specific mortality is noteworthy given the likely very high Type II error.

5. Conclusions

Despite these limitations, the findings of this study are important in addressing the relationship between cigarette smoking and death at an early age from all causes, identifying the role that an adolescent personality trait plays in prospectively predicting cigarette smoking behavior, and further refining the link between cigarette smoking and mortality by using smoking trajectories to better characterize an individual's smoking behavior over time. These findings reinforce the need to prevent adolescent initiation of cigarette smoking and offer cessation services to those who smoked regularly into adulthood.

Conflicts of interest

None.

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