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Differences in time to onset of smoking and nicotine dependence by race/ethnicity in a Midwestern sample of adolescents and young adults from a high risk family study

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Abstract

Objective—The objective of this study was to determine whether race/ethnicity was associated with time to smoking initiation and time from first cigarette to onset of DSM-IV nicotine dependence (ND) after adjusting for familial and individual psychosocial risk factors.

Methods—Cox proportional hazards models with time-dependent covariates were used to analyze data from 1376 offspring aged 12–33 years from 532 families at high risk for substance use problems due to paternal alcohol problems and 235 low risk families. Fifty-six percent of the sample self-identified as African-American (AA) and 44% were mainly of European descent.

Results—Controlling for covariates, AAs began smoking at older ages (HR=0.58; 95% CI: 0.48–0.70) and had longer times between smoking initiation and onset of ND compared to non-AAs (HR=0.25, 95% CI: 0.16–0.39 for ND onset occurring <18 years and HR=0.49, 95% CI: 0.30–0.80 for ND onsets ≥ age 18). After additionally controlling for number of cigarettes smoked daily, the racial/ethnic effects for onset of ND were attenuated, but remained statistically significant for ND onset <18 (HR=0.34, 95% CI: 0.19–0.61); however, the estimate was no longer significant for later ND onset (HR=0.84, 95% CI: 0.50–1.41).

Conclusions—AA adolescents and young adults initiate smoking at older ages and have longer transition periods between initiation and onset of ND compared to non-AAs, even after controlling for many relevant psychiatric and psychosocial covariates; however, racial/ethnic differences in time to onset of nicotine dependence in late adolescence and young adulthood may be explained by differences in daily quantity smoked.

Keywords

smoking initiation; nicotine dependence; racial/ethnic differences

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1. Introduction

Convergent results from published literature show that African American (AA) adolescents differ in their tobacco use behaviors from their non-AA counterparts. AA adolescents are less likely to initiate cigarette smoking (Bohnert et al., 2009; Dierker et al., 2008; Escobedo et al., 1990; Griesler et al., 2002; Gritz et al., 1998; Voorhees et al., 2011) and begin smoking at older ages (Gutman et al., 2011; Kandel et al., 2005). They are also less likely to become regular smokers and do so at older ages than non-AA youth (Choi et al., 2002; Griesler et al., 2002; Robinson et al., 2004; Voorhees et al., 2011). Among smokers, AA adolescents smoke fewer cigarettes per day (Gutman et al., 2011; Kandel et al., 2005) and increase the quantity they smoke more slowly after initiation (Gutman et al., 2011). In addition, AA adolescent smokers are less likely than non-AA smokers of the same age to meet criteria for DSM-IV nicotine dependence (Dierker et al., 2008; Kandel et al., 2005; Robinson et al., 2006; Schroeder and Moolchan, 2007), even after controlling for daily quantity smoked (Kandel et al., 2005). While racial/ethnic differences remain into young adulthood, with lower proportions of smokers among AAs compared to their EA counterparts, a “crossover effect” has been observed whereby the magnitude of between-group differences declines with age, and by middle age, smoking rates in AAs typically exceed those in EAs (e.g., (Geronimus et al., 1993; Kandel et al., 2011)).

Although racial/ethnic differences in smoking behaviors among adolescents and young adults are consistently observed, the reason for these differences has yet to be elucidated. Given that many sociodemographic risk factors for smoking are also positively associated with AA race/ethnicity, it would be expected that taking these other variables into account would explain the association, yet racial/ethnic differences in smoking behaviors persist despite controlling for variables such as friends’ smoking and indicators of socio-economic status (Bohnert et al., 2009; Escobedo et al., 1990; Gritz et al., 1998; Gutman et al., 2011; Voorhees et al., 2011). There are, however, racial/ethnic differences in other smoking-associated domains, such as individual psychopathology and parental substance use behaviors, that have not yet or rarely been examined in the context of the relationship between race/ethnicity and smoking. In addition, although the literature consistently shows that AA adolescents have slower rates of transition to regular or daily smoking than non-AA adolescents (e.g., (Flint et al., 1998) it is unknown whether these same group differences exist for transitioning to DSM-IV nicotine dependence.

The aims of the present study were to determine (a) whether AA race/ethnicity was associated with lower likelihood of transitioning from smoking initiation to DSM-IV nicotine dependence and (b) whether racial/ethnic differences in individual and parental alcohol use disorder and regular smoking explain the relationships between AA race/ethnicity and time smoking initiation and time from smoking initiation to onset of DSM-IV nicotine dependence. This investigation was conducted in a racially/ethnically diverse sample of adolescent and young adult participants in the Missouri Family Study (MOFAM), which is enriched for families at high risk for alcohol and other substance use problems. The MOFAM study features birth record ascertainment of families, over-sample of AA families, telephone screening interviews to determine family risk status, and detailed substance use and psychiatric histories from structured interviews administered to offspring and their parents.

2. Methods

2.1 Sample ascertainment

The Missouri Family Study (MOFAM) is a longitudinal, high risk family study designed to investigate the impact of paternal alcoholism on offspring outcomes over time in an

ethnically diverse sample of youth identified from the general population. State birth records were used to randomly select families with children who would be aged 13, 15, 17 or 19 at the time of baseline interview, and with at least one additional child aged 13 or older born to the same parents, with oversampling of AA families (identified using race of the mother on the birth record) in order to increase the statistical power to detect differences in outcomes by race/ethnicity. Mothers were screened over the telephone in 2003–2009 to confirm that the children were full siblings, and to determine risk status of the family. Families were identified as "high risk" (HRSK) based on maternal report that the father had a history of excessive drinking; otherwise, the families were considered "low risk" (LRSK). In addition, a second high risk sample ("very high risk"; VHRSK) was created that consisted of families of men with two or more driving while intoxicated convictions based on state driving records. A total of 767 families with 1378 offspring (768 AA, 610 non-AA; median age 16) enrolled in the study: Three hundred seventeen families of non-AA, primarily European-American (EA) descent (84 LRSK, 79 HRSK and 154 VHRSK), and 450 AA families (151 LRSK, 150 HRSK and 149 VHRSK). A detailed description of the method of family ascertainment is provided in a previous publication (Calvert et al., 2010).

2.2 Assessments

Mothers, fathers and offspring were each interviewed by telephone by different raters, who were blind to family risk status, using a structured psychiatric interview developed by the Midwest Alcoholism Research Center that was based on the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; (Bucholz et al., 1994)), the Diagnostic Interview Schedule (DIS; (Robins et al., 2000)) and the Childhood Semi-Structured Assessment for the Genetics of Alcoholism (CSSAGA), and the nicotine dependence module adapted from the Composite International Diagnostic Interview (CIDI; (Cottler et al., 1991)).

Although offspring were re-interviewed at 2 year intervals, the present study uses only information from offspring and their mothers that was collected at the offspring's initial interview. The study protocol was approved by the Washington University School of Medicine Human Research Protections Office and by the Ethics Board of the State Department of Health and Senior Services in accordance with regulations governing the use of vital records in research. All subjects provided informed consent.

Mothers were interviewed with a comprehensive assessment that covered her own diagnostic history of psychiatric disorders and substance use, abuse and dependence. Additionally, mothers provided reports of the children's biological father's history of substance use, abuse and dependence, along with brief reports of regarding his other psychopathology and educational attainment.

The offspring assessment was a comprehensive psychiatric interview that covered a wide range of behaviors and psychiatric disorders, including smoking and nicotine dependence, and non-diagnostic sections probing other topics associated with substance use, abuse and dependence, such as traumatic experiences, suicidality, perceived peer substance use behaviors, and school grades.

2.3 Smoking outcomes

Age at which the respondents smoked their first cigarette, symptoms of and age at onset of nicotine dependence and number of cigarettes smoked per day during period of heaviest smoking were obtained from the offspring report of their smoking behaviors at interview. Respondents were considered to have met the tolerance criterion of nicotine dependence if they had smoked more than 20 cigarettes per day during their period of heaviest smoking or if they endorsed at least one of the following questions: "After you had been smoking for a

while, did you start to smoke more cigarettes on those days when you smoked in order to feel satisfied?" and "After you had been smoking cigarettes for a while, did you switch to a stronger type of cigarettes to feel satisfied?" Consistent with the syndromal nature of ND in the DSM-IV classification, ND was defined as at least 3 of 7 dependence criteria occurring within the same 12-month period. Offspring who affirmed the clustering of ND criteria were asked their age at the first occurrence of the syndrome, which is the onset age for ND that was used in the current analyses. The current DSM-IV ND assessment, as well that of the CIDI on which it was based, has been used widely in epidemiologic studies of ND (e.g., (Dierker et al., 2008; Sartor et al., 2008; Scherrer et al., 2012; Strong et al., 2009; Xian et al., 2010)), as well as other DSM-IV substance use disorders (e.g., (Duncan et al., 2008; Sartor et al., 2009)) in adolescents and young adults.

2.4 Covariates

Psychiatric, social and familial variables available from the dataset that had been identified in the literature as associated with race/ethnicity and smoking outcomes were included in the statistical models as covariates. These variables included respondent gender, alcohol and cannabis use (ever), alcohol intoxication, lifetime major depression, three or more conduct disorder symptoms lifetime, poor grades in high school (the lowest quartile of responses in the sample), and report of friends' smoking behavior (most, some or no friends smoke). In addition, from the mother's interview, household income (< or ≥$ 45,000, the median household income in Missouri), parental lifetime regular smoking (≥100 cigarettes), nicotine dependence, and education (less than high school, high school diploma or GED, and more than high school) were included, with separate variables constructed for mothers and fathers (based on mother's report). Maternal report of parental separation and her own lifetime alcohol abuse or dependence status were also examined. Family risk type (HRSK, VHRSK, and LRSK [referent]) was included in all models as a set of dummy variables, which served to control for the study design and for paternal alcohol problems.

2.5 Data Analysis

Statistical analyses were conducted with STATA, version 8.2 (StataCorp, 2003). Since a portion of our sample was not through the age of risk for onset of smoking initiation and onset of nicotine dependence, a Cox proportional hazards model with time-dependent covariates was used to model time to these events (Cox, 1972). This statistical technique, a form of survival analysis, allows for censoring of individuals who have not yet developed the outcome (or may never develop the outcome), rather than assuming that those who have not experienced the outcome at the time of the analysis will never do so, as in logistic regression. Consequently, estimates of risk derived using survival analysis are considered to be more accurate than those obtained from logistic regression. For the current analyses, data were transformed into a "person-years" format such that each individual had a row of data for each year of his or her life. For a given time-dependent variable (e.g. ever use of alcohol, alcohol intoxication, cannabis use, major depression, three or more conduct disorder symptoms, parental separation/divorce/death), an individual was coded zero for each line of data until he or she reached the age at onset, after which the variable was coded one. Fixed covariates or covariates for which age at onset were not available were coded as time invariant (i.e., the same value on all rows of data for a given participant). These were gender, proportion of current friends who smoke cigarettes, low high school grades (lowest quartile), maternal and paternal educational attainment, maternal and paternal smoking, maternal alcohol use disorder and family risk status. The proportional hazards assumption that risk remains constant over time was assessed using the Grambsch and Therneau test of the Schoenfeld residuals (Grambsch and Therneau, 1994). Upon identification of a proportional hazards violation, separate dummy variables were defined for different age periods for that variable based on which division(s) eliminated the proportional hazards

violation (e.g., cannabis use before age 14, between ages 14 and 16, and after age 16); these were retained in the model with separate hazards ratios estimated for each period. Confidence intervals for Cox regression models were adjusted for clustering within family using Huber-White robust standard errors and the Efron approximation was used to handle ties (Hosmer and Lemeshow, 1999).

Given the suggestion of interactions between race and sex for smoking outcomes in results from a previous study (Robinson et al., 2006), interactions between race/ethnicity and gender were also assessed by adding the interaction term along with the main effects for each term into the models.

3. Results

Fifty-six percent participants were from AA families and 44% were from non-AA families. Thirty-seven percent (37.45%) of the sample came from LRSK families, 28.01% came from HRSK families and 34.54% came from VHRSK families. As can be seen in Table 1, compared to AAs, non-AAs had higher rates of ever having smoked a cigarette (52.46% vs. 44.13%; p=.01) and nicotine dependence (32.81% vs. 18.51% p<.001) and earlier ages of onset for smoking initiation and for nicotine dependence (p=0.017 and p<0.001, respectively). More non-AA compared to AA participants had ever used alcohol or been intoxicated, had parents with more than a high school education, mothers with alcohol use disorder and fathers who were ever regular smokers or nicotine dependent. AA participants were in turn more likely to have poor high school grades, parents who were not together, and to have mothers with less than a high school education and fathers who were reported to have a high school diploma or GED.

In a model for time to first cigarette that did not include covariates, AA race/ethnicity was associated with decreased likelihood of ever smoking a cigarette (HR=0.72; 95% CI: 0.60–0.86). After adjusting for covariates, the magnitude of the association between being AA and smoking initiation increased (i.e., was further from the null value [1.00] HR=0.63; 95% CI: 0.51–0.78). The interaction between race and gender was not statistically significant.

Results from the model of time from first cigarette to onset of nicotine dependence are presented in Table 2. A violation of the proportional hazards assumption was observed for AA race/ethnicity, which was fixed by dividing it into two time periods -- before age 18 and age 18 and older – allowing the effect of race on the development of nicotine dependence to be estimated separately for each time period. In the base model that also included family risk status (Model 1 in Table 2), AAs who had ever smoked a cigarette were significantly less likely to become nicotine dependent than non-AAs before age 18 (HR=0.31; 95% CI: 0.20–0.49); however, at age 18 or older the association was not statistically significant (HR= 0.68; 95% CI: 0.43–1.07). After adjusting for covariates (Model 2), AA race/ethnicity was significantly associated with lower likelihood of becoming nicotine dependent during both risk periods, although the protective effect was of greater magnitude for transition before age 18 (HR=.28) than at age 18 or older (HR=.56). The interaction between race/ethnicity and gender was not statistically significant.

Given that previous studies have found that AA individuals become nicotine dependent at lower levels of cigarette smoking compared to non-AA individuals (Luo et al., 2008; Edens et al., 2010), we added terms to the model reflecting number of cigarettes per day (CPD) during the heaviest smoking period (dummy variables for 11–19, 20–25 and >25 CPD, with ≤10 CPD as the referent group). Results from this model are displayed in the column labeled Model 3 of Table 2. After the adjustment for CPD, AAs were still significantly less likely to transition to nicotine dependence than non-AAs before age 18, although the effect was somewhat attenuated (RR=0.34; 95% CI: 0.19–0.61). The association between race/ethnicity

and transition to nicotine dependence at age 18 or older was also attenuated and was no longer statistically significant (HR=0.84; 95% CI: 0.50–1.41).

4. Discussion

In this sample of adolescents and young adults, African-Americans were less likely to initiate smoking and to transition to nicotine dependence, particularly before age 18, even after controlling for familial and individual psychiatric and socio-demographic risk factors. The relationship between AA race/ethnicity and transition to nicotine dependence after age 18 was explained by racial/ethnic differences in quantity smoked. The effects of race/ethnicity on these outcomes did not differ by gender.

The results regarding time to smoking initiation are largely consistent with those from previous studies using adolescent samples (e.g., (Johnston et al., 2010)) and extend the literature by controlling for the effect of many well-characterized familial and individual psychiatric variables as potential confounders of the relationship between race/ethnicity and smoking initiation. The fact that the protective effect of AA race/ethnicity is observed even after controlling for these variables suggests that there are additional factors that are important to smoking initiation that have not been examined here.

Our results regarding time from smoking initiation to onset of DSM-IV nicotine dependence are similar to those of other studies that have reported that AAs have longer time to onset of regular or daily smoking (Choi et al., 2002; Griesler et al., 2002; Robinson et al., 2004; Voorhees et al., 2011), which is highly associated with nicotine dependence (e.g., (Pergadia et al., 2006). Although we are unaware of previous studies examining time to DSM-IV nicotine dependence onset, there have been several studies that have found that AAs are less likely to meet criteria for nicotine dependence (Kandel et al., 2005; Robinson et al., 2004; Schroeder and Moolchan, 2007).

We observed that among those who ever smoked 100 cigarettes or more, AA youth were significantly less likely than their non-AA counterparts to endorse the tolerance and withdrawal symptoms, which are correlated with CPD. After controlling for average number of cigarettes smoked per day the protective effect of AA race/ethnicity diminished for adolescent onsets and was no longer significant for onsets after age 18. Indeed, after eliminating tolerance and withdrawal from the count of ND symptoms, there were no significant differences in mean number of symptoms (2.38 for AA and 2.39 for non-AA, p=0.99). This finding is consistent with findings from an early interim analysis of mothers in MOFAM who smoked during pregnancy (Edens et al., 2010) that found that AA women reported more nicotine dependence symptoms than non-AA women at lower levels of quantity smoked.

A plausible biological explanation for these differences is that AAs have slower average clearance of nicotine and of its proximal metabolite cotinine compared to European Americans (Perez-Stable et al., 1998). Consistent with this finding, AA smokers have higher plasma or urine cotinine levels compared to EA smokers (Ahijevych and Gillespie, 1997; Caraballo et al., 1998; Kandel et al., 2007; Moolchan et al., 2007). The longer availability of nicotine and cotinine in the blood could contribute to the need to smoke fewer cigarettes per day, which could explain the lighter smoking seen among AA smokers. To the extent that daily smoking quantity reflects level of nicotine dependence, differences in nicotine metabolism may help explain, in part, differences in DSM-IV nicotine dependence, but a large amount of the variance still remains unexplained. In addition, metabolic differences still cannot explain racial/ethnic differences in smoking initiation.

It is possible that these findings, as well as our own, could be explained, in part, by a lack of validity due to racial variance in the ND criteria (i.e., that the DSM-IV ND criteria do not measure the same trait in AAs vs. non-AAs). The validity of the DSM-IV ND diagnosis has been questioned with regard to the lack of empirical evidence supporting the 3-symptom threshold and appropriateness for use in younger and non-white samples (DiFranza et al., 2010). Our findings that the association between AA race/ethnicity and DSM-IV ND is not consistent over different developmental periods and is at least partially explained by quantity of cigarettes smoked could be taken as evidence for lack of validity. Evidence from studies using item response modeling to assess differential item functioning (an indication of measurement variance) has been equivocal, with some observing no differential item functioning by race-ethnicity (Saha et al., 2010), and others demonstrating limited evidence (Strong et al., 2003), although studies in adolescent/young adult-aged samples are somewhat uncommon. It is important to note that measurement variance comes about because of actual differences between groups either in the way that members of a particular group tend to interpret items (an indication of lack of validity) or due to biological differences with regard to the construct being measured. Therefore, differences in DSM-IV ND incidence, prevalence, and age at onset by demographic factors may provide valuable insights into the etiology of nicotine dependence and/or the validity of the DSM-IV criteria, the latter being of particular importance in light of revisions to the criteria that have been proposed for DSM-V, currently underway.

The current study is not without limitations. First, the smoking assessment used in this study was retrospective and therefore our findings may be affected by recall bias, although the relatively young age of the sample makes this possibility less likely. Due to the fact that some of the members of the sample were as young as 13 years old at the time of data collection, however, there likely are individuals in the sample who have yet to initiate smoking or develop nicotine dependence. The use of Cox proportional hazards models is appropriate in this situation, as it makes no assumptions about the future outcome status of individuals in the sample; however, given that the proportional hazards violations identified in the nicotine dependence model, it is possible that different associations between race/ethnicity and the outcomes may be seen for individuals at older ages. Additional waves of data are being collected that will enable us to examine changes over time in these relationships with prospective data. Finally, study participants were born in a single Midwestern state. Thus results may not be generalizable to persons born in other parts of the U.S. By design, families with full siblings were included, thus results may not generalize to families with other configurations. It is possible that offspring who chose not to participate in the interview might be more or less likely to be smokers than those who did. If this were the case and smokers of a particular race/ethnicity were under- or over-represented, our findings could be biased.

4.1 Conclusions

African-American adolescents and young adults were less likely to initiate smoking and are less likely to transition to nicotine dependence before age 18 after adjusting for covariates, indicating that there are additional unmeasured factors that explain these relationships. At age 18 and older, however, differences in nicotine dependence were explained by lower quantity of cigarettes smoked among African-Americans. Elucidation of the factors that account for the observed protective effect of African-American ethnicity on adolescent smoking behavior may provide valuable information to guide prevention and intervention efforts to reduce smoking initiation and progression to nicotine dependence in non-African-Americans as well as decrease higher levels smoking persistence in African American adults.

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Table 1

Sample characteristics by race/ethnicity.

	Non-African-American n=610	African-American n=768	p-value[a]
Ever smoked a cigarette	52.46	44.13	0.01
Mean age onset (SE)	13.26(.18)	13.86 (.18)	0.017
DSM-IV nicotine dependence[b]	32.81	19.82	<0.001
Mean age onset (SE)	16.91(.24)	18.51 (.39)	<0.001
Cigarettes per day when smoked most[b]			0.009
≤10	54.26	72.93	
11–19	18.09	10.53	
19–25	18.09	10.53	
≥26	9.57	6.02	
Male gender	49.34	49.35	0.999
Mean age (SE)	17.33 (.14)	17.69 (.13)	0.065
Parents do not live together	46.23	71.48	<0.001
Low grades in high school	19.34	33.12	<0.001
Friends' smoking status			0.349
No Friends Smoke	23.36	19.92	
Some Friends Smoke	42.76	43.93	
Most Friends Smoke	33.88	36.15	
Ever Used Alcohol	59.18	49.87	0.004
Ever Intoxicated	43.35	24.58	<0.001
Ever Used Cannabis	33.33	33.86	0.859
Major Depressive Disorder	10.0	10.94	0.593
≥ 3 Conduct Disorder Symptoms	9.72	11.29	0.39
Maternal variables[c]			
Household income <$45,000	40.03	68.13	<0.001
Alcohol use disorder	24.43	13.99	0.001
Ever smoked ≥100 cigarettes	52.30	42.19	0.014
Education level			0.024
Less than high school	5.42	11.49	
High school diploma or GED	29.39	29.63	
More than high school	65.19	58.88	
Paternal variables[c]			
Ever smoked ≥100 cigarettes	66.39	57.55	0.028
Education level			0.004
Less than high school	18.2	17.06	
High school diploma or GED	37.9	51.04	
More than high school	43.91	31.9	

[a] from chi square (categorical variables) or t-test (continuous variables)

[b] Among ever smokers

[c] Maternal report

Table 2

Cox proportional hazards model for time from first cigarette to onset of nicotine dependence by race/ethnicity in adolescent and young adult participants in the Missouri Family Study.

	Hazard Ratio (95% Confidence Interval)		
	Model 1*	**Model 2****	**Model 3*****
African-American, onset before age 18	0.31 (0.20–0.49)	0.28 (0.17–0.47)	0.34 (0.19–0.61)
African-American, onset ≥ age 18 years	0.68 (0.43–1.07)	0.56 (0.34–0.92)	0.84 (0.50–1.41)

* adjusted for family risk group only

** additionally adjusted for: Offspring report: ever intoxicated, friends' smoking, ≥3 conduct disorder symptoms, major depressive disorder, ever used cannabis, poor high school grades, parents divorced/never married; Mother report: maternal household income, alcohol abuse and dependence, education and regular smoking, and paternal education and regular smoking

*** adjusted for all variables in Model 2 and for cigarettes smoked per day during period of heaviest smoking