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Childhood Sexual Abuse and Two Stages of Cigarette Smoking in African-American and European-American Young Women

Carolyn E. Sartor, Ph.D.^{a,b,†}, Julia D. Grant, Ph.D.^b, Alexis E. Duncan, Ph.D.^{b,c}, Vivian V. McCutcheon, Ph.D.^b, Elliot C. Nelson, MD^b, Wilma J. Calvert, R.N., Ph.D.^d, Pamela A.F. Madden, Ph.D.^b, Andrew C. Heath, D.Phil.^b, and Kathleen K. Bucholz, Ph.D.^b

^aDepartment of Psychiatry, Yale University School of Medicine, New Haven, CT, USA

^bMidwest Alcoholism Research Center, Department of Psychiatry, Washington University School of Medicine, St. Louis, MO, USA

^cGeorge Warren Brown School of Social Work, Washington University, St. Louis, MO, USA

^dUniversity of Missouri-St. Louis College of Nursing, St. Louis, MO, USA

Abstract

Objective—The aim of the current study was to determine whether the higher rates of childhood sexual abuse (CSA) but lower rates of cigarette smoking in African-American vs. European-American women can be explained in part by a lower magnitude of association between CSA and smoking in African-American women.

Methods—Data were drawn from a same-sex female twin study of substance use (n=3,521; 14.3% African-American). Cox proportional hazards regression analyses using CSA to predict smoking initiation and progression to regular smoking were conducted separately by race/ethnicity. Co-twin status on the smoking outcome was used to adjust for familial influences on smoking (which may overlap with family-level influences on CSA exposure).

Results—After adjusting for co-twin status, CSA was associated with smoking initiation in European Americans (hazards ratio (HR)=1.43, 95% confidence intervals (CI): 1.26–1.62) and with smoking initiation 16 in African Americans (HR=1.70, CI: 1.26–2.29). CSA was associated

[†]Please address correspondence to Carolyn E. Sartor, Ph.D., Department of Psychiatry, Yale University School of Medicine, VA CT Healthcare System, 950 Campbell Drive, West Haven, CT 06516; phone: 203-937-3894; fax: 203-785-6196; ; Email: carolyn.sartor@yale.edu

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Contributors

Dr. Sartor conducted the literature searches and statistical analyses and wrote the first draft of the manuscript. Drs. Bucholz, Heath, and Madden designed the studies from which data were drawn and oversaw data collection. All authors provided input on the study design (e.g., operationalization of childhood sexual abuse and stratification of analyses by race/ethnicity) and edited the manuscript. All authors have approved the final manuscript.

Conflicts of Interest

We have no conflicts of interest to declare.

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with regular smoking onset 15 in European Americans (HR=1.63, CI:1.21–2.18), with no change in HR after adjusting for co-twin status. In the African-American subsample, the HR for CSA was reduced to non-significance after adjusting for co-twin status (from HR=3.30, CI:1.23–8.89 to HR=1.16, CI:0.71–1.92 for regular smoking 15).

Conclusions—CSA is associated with moderate elevation in risk for initiating smoking among African-American and European-American women. By contrast, CSA is associated with elevated risk for (adolescent onset) regular smoking only in European-American women. Furthermore, there is significant overlap between risk conferred by CSA and familial influences on regular smoking in African-American but not European-American women.

Keywords

smoking; sexual abuse; women; African Americans

1. Introduction

1.1. Childhood sexual abuse and cigarette smoking in African Americans vs. European Americans

An estimated one in five women has experienced childhood sexual abuse (CSA) (Bensley, Van Eenwyk, & Simmons, 2000; Dube, Felitti, Dong, Giles, & Anda, 2003; Molnar, Buka, & Kessler, 2001) and although findings are mixed (Chiu et al., 2013; Scher, Forde, McQuaid, & Stein, 2004), higher rates of exposure to CSA in African-American compared to European-American girls have been reported in several studies (Hussey, Chang, & Kotch, 2006; McCutcheon et al., 2010; Ullman & Filipas, 2005). Differences between African Americans and European Americans in smoking outcomes, specifically, lower rates of smoking (Garrett, Dube, Winder, Caraballo, & Centers for Disease Control and Prevention, 2013; Johnson & Hoffmann, 2000; Scarinci, Robinson, Alfano, Zbikowski, & Klesges, 2002) and later initiation of smoking (Andreski & Breslau, 1993; Duncan, Lessov-Schlaggar, Sartor, & Bucholz, 2012; Mickens, Ameringer, Brightman, & Leventhal, 2010) among African Americans have consistently been found, which is at odds with the well-documented association of CSA with smoking.

Elevated rates of regular smoking and nicotine dependence among individuals exposed to CSA have been observed in numerous population-based studies (Al Mamun et al., 2007; Chartier, Walker, & Naimark, 2009; Nelson et al., 2002; Roberts, Fuemmeler, McClernon, & Beckham, 2008). For example, using data from the National Epidemiologic Survey on Alcohol and Related Conditions, Afifi and colleagues (Afifi, Henriksen, Asmundson, & Sareen, 2012) found that nicotine dependence criteria were endorsed by 33.2% of women who reported experiencing CSA but only 18.0% of those who did not. CSA has also been linked to elevated risk for onset of regular smoking in early adolescence (Anda et al., 1999).

To make sense of the seemingly contradictory findings of higher exposure to CSA but lower risk for smoking in African Americans vs. European Americans, we need to consider the progression of smoking behaviors. The few studies that have examined the association of CSA with more than one stage of substance use suggest that it varies, with a higher

magnitude of association in early stages, i.e., initiation (Benjet, Borges, Medina-Mora, & Méndez, 2013; Sartor et al., 2007), which typically occurs during adolescence.

Differences in rates of smoking between African Americans and European Americans also appear to vary by developmental stage (Ellickson, Orlando, Tucker, & Klein, 2004; Hu et al., 2006). Large-scale epidemiological studies have documented lower rates during adolescence but equivalent or even higher rates of smoking during adulthood in African Americans compared to European Americans (Garrett et al., 2013; Kandel, Schaffran, Hu, & Thomas, 2011). Consistent with these findings, Moon-Howard's study based on National Health Interview Survey data (2003) revealed that the prevalence of smoking increases later into adulthood among African-American than European-American women (Moon-Howard, 2003).

1.2. Familial influences common to cigarette smoking and risk for experiencing CSA

CSA frequently occurs in the context of additional familial risk factors such as parental psychopathology and other forms of child maltreatment, which are also linked to smoking (Afifi et al., 2012; Chartier, Hesselbrock, & Hesselbrock, 2009; Fuller-Thomson, Filippelli, & Lue-Crisostomo, 2013; Perez-Fuentes et al., 2013; Shorey et al., 2013). Determining if the elevated risk for early initiation and progression to regular smoking in individuals who have experienced CSA is attributable to sexual abuse specifically or to the constellation of risk factors that cluster with it is therefore extremely challenging. An alternative approach that can be applied in twin samples is to use information on smoking in co-twins – who are exposed to nearly all the same familial influences – to adjust for familial influences on the smoking outcome; the resulting estimated effects of CSA are independent of familial influences common to CSA and the smoking outcome.

1.3. Aims of the current study

Building on the evidence for CSA associated risk for smoking, distinctions in the course of smoking behaviors between African Americans and European Americans and higher rates of CSA in African Americans, the current study aimed to identify possible racial/ethnic differences in the independent association of CSA with the timing of smoking initiation and progression to regular smoking in women. We hypothesized that the independent association of CSA with smoking outcomes would be lower, i.e., a greater degree of CSA associated risk would be accounted for by shared familial influences, in African-American than European-American women.

2. Methods

2.1. Participants

The sample was composed of female twins from the Missouri Adolescent Female Twin Study (MOAFTS), a longitudinal study begun in 1995 that targeted all female twins identified through birth records as being born in Missouri between July 1, 1975, and June 30, 1985. (See Waldron, Bucholz, Lynskey, Madden, & Heath, 2013 for details on sample ascertainment). All twins in the targeted birth cohort (excluding those who had withdrawn from the study or whose parents requested that the family not be re-contacted) were

contacted for wave 4 interviews between 2002 and 2005, when they were 18 to 29 years of age, so parental consent was not needed. As a result, a greater number of twins - 80% of those identified from birth records - completed wave 4 than wave 1 interviews (3,787 vs. 3,258). We therefore limited the sample to wave 4 participants, but data from other waves (1, 3, and/or 5), which were available for over 95% of participants, were integrated as well. (See 2.3.). A total of 3,521 participants were included in the current study: all twins who answered CSA questions and whose co-twins also participated in wave 4 of MOAFTS. (CSA status was missing for 23 of the 3,544 twins with participating co-twins). Mean ages at first and last interview were 16.8 (SD=3.4) and 24.5 (SD=2.7) years, respectively; 14.3% self-identified as African-American (249 monozygotic and 254 dizygotic twins), the remainder as European-American (1,815 monozygotic and 1,203 dizygotic twins).

2.2. Procedures and assessment

Data were collected using an interview modified for telephone administration from the Semi-structured Assessment for the Genetics of Alcoholism (Bucholz, Helzer, Shayka, & Lewis, 1994; Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999), which assesses DSM-IV psychiatric disorders and related psychosocial constructs. Consent was obtained verbally prior to starting the telephone interview. The study was approved by the Human Research Protections Office at Washington University.

2.3. Predictors and outcomes

In cases where age at first CSA experience, age at first cigarette, or age at onset of regular smoking was reported in more than one interview, we used the age at first occurrence reported the first time the participant endorsed it.

2.3.1. CSA history—CSA was assessed in Waves 1, 3, and 4 using the questions in Table 1. CSA criteria were met if any sexual abuse item was endorsed and reported as occurring at age 15 or younger in any wave of data collection. The prevalence of CSA was 20.5% and 11.0% in the African-American and European-American subsamples, respectively. Mean age at first CSA experience was 8.4 (SD=3.9) in African Americans and 8.6 (SD=4.1) in European Americans. The concordance rates for CSA were 54.9% and 50.2% in the African-American and European-American subsamples, respectively.

2.3.2. Age at first cigarette and onset of regular smoking—Initiation of smoking was queried by asking, ‘Have you ever tried a cigarette?’ Participants who responded ‘yes’ were asked their age at the time. As there is no standard cut off for early initiation, we chose the lowest quartile of the age at first cigarette distribution (12) to represent early initiation relative to peers. Participants who reported trying a cigarette were asked the total number they had ever smoked and lifetime maximum frequency of smoking. If they reported smoking at least weekly for two consecutive months, age at onset of weekly smoking was also queried. As there is no universal definition of regular smoking (see White, Pandina, & Chen, 2002), we integrated a common quantity based definition, 100 or more cigarettes over the lifetime, with a common frequency based definition, at least weekly for two or more consecutive months. Age at regular smoking onset was defined as the age weekly smoking began.

2.3.3. Familial influences—A dichotomous variable representing co-twin early initiation status and a dichotomous variable representing co-twin regular smoking status were used to adjust for familial influences on smoking initiation and regular smoking, respectively. Zygosity as well as co-twin status by zygosity interaction terms, which indicate the degree to which familial influences are attributable to genetics, were also included in adjusted models.

2.4. Data analysis

2.4.1. Initiation of smoking, regular smoking, and timing of transitions by race/ethnicity and CSA history—Logistic regression analyses were conducted to test for differences in rates of smoking initiation and regular smoking by race/ethnicity and by CSA status within subsamples. Independent t-tests were conducted to test for differences in age at first cigarette, age at onset of regular smoking, and rate of progression from first cigarette to regular smoking by race/ethnicity and by CSA status within subsamples.

2.4.2. Predicting initiation of smoking and onset of regular smoking with CSA history—Analyses were stratified by racial/ethnic group, as a pooled analysis would be statistically compromised, given the substantial sociocultural and socioeconomic separation between African Americans and European Americans in our sample and its impact on relevant risk factors (see Imbens and Rubin, 2015). Cox proportional hazards (PH) regression analyses were conducted to predict initiation of smoking and onset of regular smoking as a function of CSA status. Age at first cigarette was the point of origin in the regular smoking models; thus, regular smoking analyses were limited to participants who endorsed having tried a cigarette (331 African-American and 2,234 European-American participants). This survival analysis approach accounts for the possibility that participants who have not yet reached the outcome may do so in the future. It also involves testing the PH assumption that risk remains constant over time, thus revealing the extent to which risk associated with CSA varies over the period of risk (i.e., when an individual can develop the outcome). CSA was modeled as a time-varying covariate by creating a data set with each line of data representing a single year in the participant's life. CSA was coded as absent in the years prior to CSA exposure and present from the year of first occurrence onward, so that only CSA preceding the smoking outcome was treated as a predictor. Analyses were conducted in Stata (StataCorp, 2007) using the clustered sandwich estimator to adjust for the non-independence of observations in twins. All analyses were adjusted for age at the time of the interview when the smoking outcome was first reported. (For participants who did not endorse the outcome, age at report was coded as age at last interview, i.e., age their status as a never smoker/non regular smoker was established.) The regular smoking models also included age at first cigarette.

The Grambsch and Therneau test of the Schoenfeld residuals (Grambsch & Therneau, 1994) was used to test the PH assumption in each model. Violations of the assumption were resolved by breaking up the risk period and estimating hazard ratios (HRs) for each age range. Cut offs for risk periods were determined individually for each variable, starting with a single split, then increasing splits and shifting cut offs as needed. Adjustments to correct for PH violations are described in supplementary materials.

3. Results

3.1. Initiation of smoking, regular smoking, and timing of transitions by race/ethnicity

The lifetime prevalence of smoking (i.e., ever trying a cigarette) was 75.5% in the European-American subsample and 67.2% in the African-American subsample (odds ratio (OR)=1.49, 95% confidence intervals (CI):1.21–1.82). Mean age at first cigarette did not differ between European-American and African-American participants: 14.2 (SD=3.2) vs. 14.4 (SD=3.7) ($t_{(404.89)}=0.98$, $p=0.325$). However, a higher proportion of European-American than African-American participants who had ever smoked became regular smokers (53.1% vs. 30.2%; OR=2.62, CI:2.05–3.35) and mean age at onset of regular smoking was younger in the European-American than African-American subsample: 15.9 (SD=2.8) vs. 17.5 (SD=3.2) ($t_{(110.54)}=4.79$, $p<0.001$). Mean transition time from first cigarette to onset of regular smoking did not vary by race/ethnicity.

3.2. Smoking outcomes by race/ethnicity and CSA history

As seen in Table 2, across racial/ethnic groups, women with a history of CSA were more likely to have ever smoked a cigarette and began smoking at an earlier age than women without a history of CSA. European-American women with a history of CSA were more likely to progress to regular smoking and to become regular smokers at a younger age than those without a history of CSA. No differences by CSA status in risk for progressing to regular smoking or age at onset of regular smoking were observed in African-American women. The timing of transitions from first cigarette to onset of regular smoking did not differ by CSA status in African-American or European-American women.

3.3 CSA and smoking outcomes

Results of Cox PH regression analyses using CSA history to predict initiation of smoking and regular smoking are shown with 95% confidence intervals (CIs) by race/ethnicity in Tables 3 and 4, respectively (without zygosity and co-twin by zygosity interactions, which were nonsignificant). Model 1 is unadjusted for co-twin early initiation status, zygosity, and co-twin early initiation status by zygosity; Model 2 is adjusted.

3.3.1. Initiation of smoking—The change in HR for CSA from Model 1 to Model 2 was non-significant for the African-American subsample. In the European-American subsample, the CSA estimate shifted from Model 1, where CSA associated risk was higher for the 17 risk period, to Model 2, where the HR was consistent over time and nearly identical to the 17 estimate in Model 1. In the adjusted models, risk for initiation of smoking was elevated in European Americans with a history of CSA (HR=1.43, CI: 1.26–1.62) and in African Americans with a history of CSA, specifically in the 16 risk period (HR=1.70, CI: 1.26–2.29). The contribution of familial influences was modest and consistent across the period of risk in the African-American subsample (HR=2.45, CI:1.26–4.76). By contrast, in the European-American subsample, the contribution of familial influences was significant across the period of risk but much greater in the pre-adolescent to early adolescent years, with HRs ranging from 10.38 (CI:7.06–15.24) for 11 to 1.62 (CI:1.18–2.21) for 16.

3.3.2. Progression from first cigarette to regular smoking—In Model 1 for the African-American subsample, CSA associated risk was significant in the 16 risk period (HR=3.30, CI:1.23–8.89) but not thereafter. The addition in Model 2 of co-twin regular smoking status, which was strongly associated with regular smoking risk (HR=13.03, CI: 3.76–45.18), reduced the estimate to non-significance across the period of risk. In the European-American subsample, the association between CSA and progression to regular smoking in the 15 risk period did not change (HR=1.63, CI:1.21–2.18) after adjusting for co-twin regular smoking status, which was associated with elevated risk for progressing to regular smoking (HR=3.52, CI:2.50–4.98).

4. Discussion

The current investigation extends the literature on CSA and smoking outcomes and racial/ethnic differences in the progression of smoking behaviors among women in several ways. First, after adjusting for confounding effects of familial risk factors for smoking, we found evidence for independent associations of CSA with initiation of cigarette smoking across racial/ethnic groups and progression to regular smoking in European Americans. Second, we uncovered differences between African-American and European-American women in risk for regular smoking conferred by CSA, thus expanding our relatively limited understanding of the etiology of cigarette smoking in African-American women. Third, we found partial support for our hypothesis that a greater degree of CSA associated risk would be accounted for by shared familial influences in African-American vs. European-American women, with findings specific to regular smoking.

4.1. CSA and smoking outcomes in African-American vs. European-American women

Our study provides further support for lower rates of lifetime smoking (Garrett et al., 2013; Johnson & Hoffmann, 2000; Scarinci et al., 2002) and lower likelihood of progressing to regular smoking in African-American compared to European-American women (Griesler et al., 2002; Kandel et al., 2004; Vega et al., 2007). The higher observed prevalence of CSA in African-American vs. European-American women in our sample is also consistent with the majority of studies that have examined differences by race/ethnicity in exposure to CSA (Hussey et al., 2006; McCutcheon et al., 2010; Ullman & Filipas, 2005). However, our finding that risk for initiating smoking was elevated in both African-American and European-American women with a history of CSA has not been previously reported. It suggests that risk conferred by CSA has even broader effects on smoking than has been documented in the literature, which has focused primarily on regular smoking and nicotine dependence.

We were somewhat surprised that the association of smoking initiation with CSA was not limited to early initiation in European Americans. Whereas early initiation is associated with later alcohol and drug problems, trying a cigarette at some point over the lifetime is relatively normative (i.e., not presumed to be a possible outcome of exposure to adverse conditions). Interestingly, we did observe an early onset specific effect of CSA on regular smoking in European-American women. The majority of investigations linking CSA to regular smoking have not distinguished early vs. later onset (Agrawal et al., 2005; Chartier

et al., 2009; Nelson et al., 2006; Roberts et al., 2008), but results of the one known study to do so are highly consistent with ours. Anda et al. (1999) found that the odds of smoking regularly by age 14 was twice as high in participants who had experienced CSA than in those who had not.

Our findings raise the question of why CSA is associated with elevated risk for initiation of smoking and progression to regular smoking in European-American women but only initiation in African-American women. Although there were fewer African-American than European-American women in the sample and their overall rate of regular smoking was lower, over 20% of African-American women reported CSA, so it is unlikely that we simply lacked sufficient power to detect the association. One possible explanation is related to variability in the association of CSA with substance use across developmental stages. African Americans progressed to regular smoking at an older age than European Americans (17.5 vs. 15.9 years). CSA may simply be a more salient risk factor for smoking behaviors in middle than late adolescence. The specificity of the association between CSA and onset of regular smoking by age 15 in European Americans further suggests that CSA plays less of a role in late adolescence, when other more proximal risk factors (e.g., peer smoking) may impact smoking to a greater degree.

Another possible source of this distinction can be inferred from the 13-fold increase in risk associated with co-twin regular smoking and reduction of the estimated association of CSA with regular smoking from a greater than 3-fold elevation in risk to non-significance in the adjusted model for the African-American subsample. This reduction indicates significant overlap between CSA and familial influences on regular smoking, i.e., that CSA is a marker of family-level risk for regular smoking rather than an independent risk factor. Although almost twice as many African-American as European-American women reported experiencing CSA, the twin concordance rates for CSA were highly comparable across racial/ethnic groups, so this finding cannot be explained by greater within family similarity in risk for CSA among African Americans. Follow-up analyses investigating racial/ethnic differences in the clustering of psychosocial risk factors are planned to further explore differences between African-American and European-American women in the independence of the contribution of CSA to risk for regular smoking.

4.2. Limitations

Certain limitations of the study should be noted. First, the rate of endorsement of CSA among European-American women in our study is somewhat lower than prevalence estimates in the larger literature (Bensley et al., 2000; Dube et al., 2003; Molnar et al., 2001). Second, co-twin status on early initiation of smoking and regular smoking only approximates familial risk; it does not capture all variance attributable to the full spectrum of risk factors common to CSA and these smoking outcomes. Third, although the concordance rates for CSA did not differ across racial/ethnic groups, it is possible that the greater overlap of CSA with familial influences in the African-American subsample reflects a higher rate of abuse by the same perpetrator among African-American twins, and we are unable to determine that from our data. Fourth, due to the smaller number of African-American than

European-American participants, statistical power was lower for analyses conducted with the African-American than European-American subsample.

4.3. Conclusions and future directions

In addition to demonstrating an independent association of CSA with initiation of smoking in women, our findings indicate that one etiological model of cigarette smoking in women does not fit all. African-American and European-American women appear to differ with respect to age at onset of regular smoking, the influence of CSA on regular smoking risk, and the degree of overlap between familial influences on regular smoking and risk conferred by CSA. Extending this line of research to identify factors that buffer against risk stemming from exposure to CSA in African-American and European-American girls can inform the development of tailored smoking prevention and intervention efforts.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Sexual abuse was associated with smoking initiation in both racial/ethnic groups.
- Sexual abuse predicted adolescent onset regular smoking in European Americans.
- These associations were independent of familial influences on smoking.
- Sexual abuse did not independently elevate African Americans' regular smoking risk.

Table 1

Items used to define childhood sexual abuse

Traumatic Events (Waves 1, 3, and 4)
<i>Endorsement of event 1 or 2, with reported age at first experience 15 years or younger</i>
1. Raped
2. Sexually molested
Health Problems and Health Habits (Waves 1, 3, and 4)
<i>Endorsement of item, with reported age at first experience 15 years or younger</i>
Has anyone ever forced you to have sexual intercourse?
Parental Discipline and Early Childhood Experiences (Wave 4)
<i>Endorsement of item 1 or 2</i>
1. Before you turned 16, was there any forced sexual contact between you and any family member like a parent or step-parent, grandfather, etc? By sexual contact I mean their touching your sexual parts, your touching their sexual parts or intercourse.
2. Before you turned 16, was there any forced sexual contact between you and anyone who was 5 or more years older than you (other than a family member)?

Prevalence of lifetime cigarette use and regular smoking and timing of transitions to first cigarette and regular smoking by race/ethnicity and childhood sexual abuse history

Table 2

	African Americans		European Americans	
	CSA n=103	No CSA n=400	CSA n=333	No CSA n=2,685
Ever smoked a cigarette ^{a,b}	81.6%	63.5%	88.6%	73.9%
		OR=2.54 (CI: 1.48–4.35)		OR=2.75 (1.94–3.89)
Proportion of ever smokers who smoked regularly ^b	32.1%	29.5%	73.6%	50.1%
		OR=1.13 (CI: 0.67–1.92)		OR=2.77 (2.11–3.65)
	Mean (SD)		Mean (SD)	
Age at first cigarette ^{a,b}	13.2 (4.0)	14.8 (3.5)	13.0 (3.3)	14.3 (3.1)
		$t_{(329)}=3.45$, $p<0.001$		$t_{(2232)}=6.80$, $p<0.001$
Age at onset of regular smoking ^b	16.5 (3.5)	17.9 (3.1)	15.1 (3.0)	16.1 (2.7)
		$t_{(97)}=1.92$, $p=0.058$		$t_{(286.5)}=4.54$, $p<0.001$
Years from first use to onset of regular smoking	3.4 (3.2)	3.1 (3.2)	2.6 (2.8)	2.6 (2.6)
		$t_{(92)}=0.42$, $p=0.673$		$t_{(1152)}=0.08$, $p=0.934$

CSA = childhood sexual abuse; SD = standard deviation; OR = odds ratio; CI = 95% confidence intervals;

^a $p < 0.05$ in African-American subsample;

^b $p < 0.05$ in European-American subsample.

Table 3

Results of Cox proportional hazards regression analyses using childhood sexual abuse history to predict initiation of cigarette smoking^a

	Hazard Ratios (95% Confidence Intervals)					
	African Americans (n=496)		European Americans (n=2,974)			
	Model 1		Model 2 ^b		Model 1	
CSA	I6	I7	I6	I7	I6	I7
	1.83 (1.36–2.49) *	1.18 (0.81–1.71)	1.70 (1.26–2.29) *	1.07 (0.73–1.56)	1.71 (1.47–1.99) *	1.42 (1.13–1.81) *
Co-twin 1 st cigarette 12	-----		2.45 (1.26–4.76) *		-----	
					I1	I2–I3
					I4–I5	I6
					1.43 (1.26–1.62) *	
					10.38 (7.06–15.24) *	
					3.71 (2.67–5.16)	
					2.50 (1.89–3.31)	
					1.62 (1.18–2.21)	

CSA = childhood sexual abuse; Model 1 = unadjusted for co-twin early initiation status, zygosity, or co-twin early initiation status by zygosity; Model 2 = adjusted for co-twin early initiation status, zygosity, and co-twin early initiation status by zygosity;

^a adjusted for proportional hazards violations and age at the time of interview when reported age at first cigarette;

^b sample size for Model 2 = 489 due to missingness on co-twin early initiation status;

^c sample size for Model 2 = 2,939 due to missingness on co-twin early initiation status;

* $p < 0.05$.

Age ranges refer to risk for initiation of cigarette smoking during that period.

Results of Cox proportional hazards regression analyses using childhood sexual abuse history to predict transition from first cigarette to regular smoking^a

Table 4

	Hazard Ratios (95% Confidence Intervals)						
	African Americans (n=331)			European Americans (n=2,234)			
CSA	Model 1		Model 2	Model 1		Model 2	
	I5	3.30 (1.23–8.89) [*]	1.16 (0.71–1.92)	I5	1.64 (1.22–2.21) [*]	I5	1.63 (1.21–2.18)
	I6	0.95 (0.52–1.74)		I6	1.14 (0.86–1.52)	I6	1.11 (0.84–1.47)
	Co-twin regular	-----		13.03 (3.76–45.18) [*]	-----		3.52 (2.50–4.98) [*]

CSA = childhood sexual abuse; Model 1 = unadjusted for co-twin regular smoking status, zygosity, or co-twin regular smoking status by zygosity; Model 2 = adjusted for co-twin regular smoking status, zygosity, and co-twin regular smoking status by zygosity;

^a adjusted for proportional hazards violations and age at the time of interview when reported age at onset of regular smoking;

* $p < 0.05$.

Age ranges refer to risk for onset of regular smoking during that period.