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Racial differences in heritability of cigarette smoking in adolescents and young adults

Cristina B. Bares^a, Kenneth S. Kendler^b, and Hermine H. M. Maes^c

^a1080 S. University, School of Social Work, The University of Michigan, Ann Arbor, MI 48109

^b800 E. Leigh Street, Department of Psychiatry and Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University, Richmond, VA 23298-0126;

^c800 E. Leigh Street, Department of Human and Molecular Genetics and Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University, Richmond, VA 23298-0126

Abstract

Introduction—Although epidemiologic studies suggest low levels of cigarette use among African American adolescents relative to White U.S. adolescents, it is not known whether this may be due to racial differences in the relative contribution of genes and environment to cigarette use initiation and progression to regular use.

Methods—Using data from White (n=2,665) and African American (n=809) twins and full siblings sampled in the National Longitudinal Study of Adolescent, we fitted age-, sex- and race-specific variance decomposition models to estimate the magnitude of genetic and environmental effects on cigarette use initiation and cigarette use quantity in Whites and African Americans across adolescence and adulthood. We employ a causal-contingent-common pathway model to estimate the amount of variance explained in quantity of cigarettes smoked contingent on cigarette use initiation.

Results—African Americans had lower cigarette use prevalence from adolescence through adulthood, and used cigarettes less heavily than Whites. Race-specific causal-contingent-common pathway models indicate that racial differences in genetic and environmental contributions to cigarette use initiation and cigarette use quantities are not present in adolescence but appear in young adulthood. Additive genetic factors were an important risk factor for cigarette use initiation for White but less so for African American young adults and adults.

Corresponding Author. cbb@umich.edu.

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[†]For example, a_{WFI}^2 refers to the additive genetic influence for White Females on cigarette use initiation and a_{AAFI}^2 refers to the additive genetic influence for African American Females.

[‡]The total variance explained is calculated by multiplying the percent of variance explained by the specific influence of each component on the first phenotype (cigarette use initiation, a_i) with the square of the common pathway, and adding the variance explained by the specific influence of each component on the second phenotype (cigarette use quantity, a_q).

Conclusions—Genetic and environmental contributions for cigarette use are similar by race in adolescence. In adulthood, genes have a stronger influence for cigarette use among White adolescents while the influence of the environment is minimal. For African Americans, both genetic and environmental influences are important in young adulthood and adulthood.

Keywords

behavioral genetics; heterogeneity; cigarette use initiation; cigarette use quantity; African American; adolescents

1. Introduction

Many epidemiologic and community-based studies have consistently reported relatively low levels of cigarette use among African American U.S. adolescents compared to White U.S. adolescents (Bachman et al., 1991; CDC, 2004; Griesler and Kandel, 1998). Between the ages of 12 and 17, White males and females initiate cigarette use at higher rates than African American males and females (Anderson and Burns, 2000). In fact, twice as many Whites are regular smokers compared to African Americans. National estimates suggest that in adolescence 4.2% of African American 10th graders (and 9.0% of 12th graders) report being a regular smoker compared to 9.5% of White 10th grade adolescents (17.5% of 12th graders) (Bachman et al., 2011; Bachman et al., 1991).

During adolescence, African Americans initiate smoking during young adulthood (Taylor et al., 1997; Trinidad et al., 2004) rather than during adolescence, as has been found for White adults (White et al., 2004). When African American adolescents do initiate cigarette use fewer youth transition into regular cigarette use than White or Hispanic youth (Griesler and Kandel, 1998; Kandel et al., 2004). Similar risk factors predispose to cigarette use during adolescence and influence the cigarette use trajectories that African American and White adolescents follow (Fergus et al., 2005). Racial/ethnic differences in smoking prevalence exist even after controlling for the influence of socio-economic status (Blum et al., 2000). Despite lower levels of cigarette use among African Americans in adolescence, racial differences in cigarette use behaviors and smoking-related diseases emerge during adulthood and contribute to health disparities in lung cancer.

When African American adults smoke, they tend to be light smokers (smoke less than 10 cigarettes per day) (Kandel and Chen, 2000) and show signs of nicotine dependence when smoking 6 to 10 cigarettes per day (Okuyemi et al., 2007). In a smoking cessation trial African Americans were found to smoke on average three fewer cigarettes per day than Whites (Branstetter et al., 2015). Previous studies also suggest that when smoking two packs per day, a greater proportion of Whites meet criteria for nicotine dependence than African Americans (Kandel and Chen, 2000). Although African American adults report smoking fewer cigarettes per day (Benowitz et al., 1999; Perez-Stable et al., 1998; Wagenknecht et al., 1990) the metabolism of nicotine into its metabolite cotinine is slower for African Americans than White adults and, as a result, African Americans have higher concentrations of cotinine (Benowitz et al., 2009). Lastly, rates of smoking-related cancers, such as lung (Haiman et al., 2006) and head and neck cancers (Molina et al., 2008; Sankaranarayanan et

al., 1998) are higher among African Americans adults (Haiman et al., 2006). Lastly, data from the National Cancer Institute's SEER program indicates that African American men have the highest incidence of lung and bronchial cancer than other racial/ethnic groups and have lower survival rates when lung cancer is diagnosed (Ward et al., 2004).

Given the health disparities in lung cancer, studies have examined mechanisms that may give rise to racial disparities in cigarette use. Together these studies indicate that biological predispositions as well as social determinants are associated with cigarette use among African Americans in adulthood. The preference among African Americans to smoke mentholated cigarettes may contribute to the racial disparities in smoking-related cancers (Apelberg et al., 2012; Clark et al., 1996; Jones et al., 2013). To date, few studies have found support for the hypothesis that individuals who use mentholated cigarettes have higher rates of lung cancer (Mustonen et al., 2005). Other studies examining the link between the use of mentholated cigarettes and lung cancer have found no such support (Brooks et al., 2003; Carpenter et al., 1999; Stellman et al., 2003). Additional mechanisms to explain the health disparities in lung cancer among African Americans despite later onset and lower levels of use might be the experience of financial strain and stress (Advani et al., 2014), emotional distress (Bares and Andrade, 2012), and the exposure to everyday discrimination (Kendzor et al., 2014; Landrine and Klonoff, 2000). In their sample, Kendzor and colleagues found that White participants had been smoking for more years and smoked more cigarettes per day than African Americans, but greater proportions of African Americans smoked within 5 minutes of waking up and reported the highest scores on the Heaviness of Smoking Index. African Americans who report high rates of discrimination have 34% to 51% higher odds of being current smokers (Borrell et al., 2010). In addition, inequalities in health care access may be an additional contributing factor to higher rates of lung cancer and poorer prognosis among African Americans (Ward et al., 2004).

Previous studies examining racial differences in the influence of social determinants of cigarette use have found that African American adolescents may experience a different set of protective factors for cigarette use than Whites. African Americans adolescents who attend majority African American schools have one of the lowest rates of substance use compared to schools with a more mixed racial composition (O'Malley et al., 2006). In addition, having a positive relationship with one's mother (Mahabee-Gittens et al., 2011) is a stronger protective effect against cigarette use initiation in African Americans than in Whites.

Family, twin and adoption studies are used to estimate the proportion of variance in a trait or behavior that is accounted for by genetic factors, or heritability. Ample evidence exists for the contribution of both genetic and shared environmental factors to cigarette initiation, with shared environmental factors being more important in adolescence with an increase in the role of genetic factors towards young adulthood (Hopfer et al., 2003; Maes and Neale, 2009; Maes et al., submitted). Furthermore, studies examining the heritability of cigarette use among adolescents and adults have uncovered that the genetic factors that influence cigarette initiation are shared with those influencing regular tobacco use as well as nicotine dependence (Bares et al., 2015; Do et al., 2015; Maes et al., 2004; Morley et al., 2007; Oncel et al., 2014). These results, however, are based on samples of twins who are predominantly White. Twin studies focused on differences in heritability by race have examined body mass

index or hypertension (Jacobson and Rowe, 1998; Snieder et al., 2003). Using the first wave of the National Longitudinal Study of Adolescent Health (Harris et al., 2009), Jacobson and Rowe found that the genetic and environmental influences on BMI differ by race (1998). A recent study of female twins in Missouri has found no racial differences in the additive genetic contribution to the liability of cigarette use initiation but significant racial differences in the shared environment (Sartor et al., 2015).

Few adolescent twin studies have a sizeable number of non-White participants to determine whether heterogeneity exists in the heritability of cigarette use initiation and progression. For example, the Virginia Twin Study of Adolescent Behavioral Development contains primarily White twins (Simonoff et al., 1997), the Minnesota Twin and Family Study (Iacono et al., 2006), and the Colorado Longitudinal and Community Twin Studies (Rhea et al., 2006) each contain less than 2% African American twin pairs in each of their sample. To date, the study that includes the largest proportion of African American twin pairs is the National Longitudinal Study of Adolescent to Adult Health (Harris et al., 2013).

The question of whether racial differences exist in the degree of influence of genes and environment on adolescent cigarette use is best addressed by a classic twin study that includes both racial groups from a nationally representative U.S. sample. The goal of the present study was to examine the presence of racial differences in the contribution of genes, or environment, or both, to the initiation of cigarette use and quantity of cigarettes smoked in White and African American U.S. adolescents. Given the evidence on how aspects of the environment influence the cigarette use of African American and White adolescents differently we expected that there would be heterogeneity in the genetic and familial influences on the initiation and continued use of cigarettes by race.

2. Method

2.1 Sample

Data from twin and sibling pairs available in the National Longitudinal Study of Adolescent to Adult Health (Add Health) (Harris, Halpern, Smolen, & Haberstick, 2006; Harris & Udry, 2013) was used for the present analyses. Adolescent participants were recruited¹ into the nationally representative study when they were enrolled in high school and subsequently assessed at various points as they transitioned into adulthood. At the time of the initial assessment (Wave 1) participants were asked to list individuals who lived in their household. From this list, participants were identified who belonged to the same family and were either twins, full siblings, half siblings or unrelated individuals (Harris et al., 2013).

For the present analyses, participants were selected who self-identified as White or African American and who were either twin pairs or full-sibling pairs. Zygosity of the twin pairs was determined by matching on 12 molecular genetic markers and through self-report questions regarding the twins' similarity (Harris et al., 2006). For this study, participants were divided into five groups based on sex and zygosity (monozygotic males (MZM), monozygotic females (MZF), dizygotic males (DZM), dizygotic females (DZF) and opposite sex dizygotic twins (DZO)) so that we could test for qualitative and quantitative sex differences in the sources of genetic and environmental influences on the phenotypes. Due to small

numbers of African American twins in this sample, we further selected into the analytic sample White and African American individuals who indicated having a full sibling 4 years, or less, apart in each of three age groups. Full siblings were considered in these analyses as dizygotic male-male twins, female-female twins or opposite-sex dizygotic pairs, as they have the same genetic relatedness (50%) as dizygotic twins. In families that had both a twin pair and sibling pairs, the twin pair was preferentially selected over the sibling pairs into the analytic sample. In families where there were multiple sibling pairs, the pair of oldest siblings was retained for the analyses. Demographic characteristics of the sample are presented in Table 1 broken down by race.

2.2 Measures

Cigarette use initiation—At each wave of the 4 wave longitudinal study, participants reported on whether they had ever smoked a cigarette and if they indicated they had smoked, they were subsequently asked to report the age at which they had first used a cigarette and how many cigarettes they had smoked per day in the previous 30 days. For each participant a cigarette use initiation variable was created to indicate whether they had responded “Yes” (1), or “No” (0) to the survey question “Have you ever smoked a cigarette?”.

Cigarette use quantity—To assess quantity of cigarettes smoked, we used data available from those participants who had affirmatively answered the question about having ever initiated cigarette smoking. Participants who answered “Yes” to having initiated the use of cigarettes were asked how many cigarettes they had smoked in the previous 30 days. The quantity of cigarettes smoked variable used in the present analyses was an ordered categorical variable such that “0” referred to not having smoked any cigarettes in the previous 30 days, “1” referred to smoking between 1 and 11 cigarettes per day, and “2” indicated smoking more than 11 cigarettes per day. Further, those participants who reported not having ever smoked a cigarette were coded as having missing information for the cigarette use quantity variable (Neale et al., 2006).

Age groups—Across the four waves of the Add Health study, participant ages ranged from 12 to 33 years. At the younger ages (age 12 and 13), the prevalence of smoking was very low and no participants smoked more than 11 cigarettes per day, so data for this age group was not used. To examine developmental changes in genetic and environmental contributions, age groups that corresponded to adolescence (14- to 17-year-olds), young adulthood (18- to 25-year-olds), and adulthood (26- to 33-year-olds) were constructed. Using these three age groups, we ran separate cross-sectional analyses for multiple years spanning adolescence to adulthood. A longitudinal model would have allowed us to ask questions about the presence of heritability differences by race in initial cigarette use as well as in the rate of change in cigarette use across time. Given the scarcity of research in this area, our aim in this study was to test for racial differences in heritability. In addition, due to the low number of twins available to us we would not be adequately powered to run longitudinal and heterogeneity analyses by race in the same model.

Each pair of participants and their co-sibling had to both fall within the age range of the groups above to be included. Beginning with age 14 we collapsed across ages resulting in a

group of adolescents who were between 14 and 17 years of age, a second group of 18- to 25-year-olds, and a third group of participants who were 26-33 years of age. Participants and their co-sibling had to both fall within the age range to be included in these age groups. To include both siblings in a pair in any of the three age groups, we created an indicator variable that was coded 1 when both siblings fell in the 14- to 17-year-old group, for example, or coded 0 if one sibling's age was in the range but their co-sibling was not.

The sample included 3,474 twin and sibling pairs with complete data. When broken down by race 2,665 pairs were White and 809 pairs were African American. Data were available for 1,546 monozygotic (414 White MZ pairs; 156 African American MZ pairs) and dizygotic twin pairs (418 White DZ pairs; 159 African American DZ pairs) and 1,928 full-sibling pairs (1,565 White FS pairs; 363 African American FS pairs). About half of each racial group was female (percent female in White group: 48.7%, percent female in African American group: 52.8%). There were no significant differences in average age between the White and African American groups (see Table 1) in any of three age groups. There were no racial differences in rates of attrition across the first three waves of the longitudinal study, but between the first and fourth wave, a greater proportion of African American (17.7%) participants were lost to follow up than Whites (14.6%, $\chi^2=4.09$, $p=0.04$).

2.3 Analysis Plan

2.3.1 Twin Model Background—The classical twin model decomposes the variance in a phenotype into components that represent the contribution of additive genetic (a^2), shared environmental (c^2) and unique environmental (e^2) sources (Neale and Cardon, 1992).

Additive genetic sources refer to latent genetic effects on a phenotype. The shared environment refers to aspects of the environment that twins share with one another that increase their similarity on a particular phenotype. The shared environment can be broken down further when extensions of the classical twin model are employed. One such extension involves including full sibling pairs along with twins. It is important to note that in the present analyses we supplement our sample with full sibling pairs but do not model their influence, nor model the special twin environment that the inclusion of full siblings permits, as we are not fully powered to test for both racial differences and the influence of the special twin environment simultaneously. In other analyses that are under preparation we specifically model and test for the influence of age and the special twin environment on cigarette use initiation.

When examining the genetic and environmental contributions to the liability of quantity of substance use one has to also take into account the liability to initiate the substance in the first place. The causal-contingent-common pathway model is a specific type of bivariate twin model (Kendler et al., 1999; Maes et al., 2004) developed to appropriately account for the dependency involved in these two phenotypes and provides estimates of the proportion of variance explained for drug use initiation (a_i^2 , c_i^2 , e_i^2) and for drug use progression (a_q^2 , c_q^2 , e_q^2). Specifically, the causal-contingent-common pathway model (illustrated in Kendler et al., 1999 and Bares et al., 2015) assumes that there is a direct common path linking the liability for the initiation of drug use to the liability for drug use progression. Therefore, the causal-contingent-common pathway model estimates specific sources of variance for the

initiation of a drug (a_i^2 , c_i^2 , e_i^2), the common pathway (beta), and novel sources of variance associated with progressing into drug use past initiation (a_q^2 , c_q^2 , e_q^2).

2.3.2 Data Analysis Steps—Each twin model run allowed thresholds to be estimated separately by age group, sex and race. The present analysis began by running a full causal-contingent-common pathway model for cigarette use initiation and quantity with sex- and race-specific estimates for each age group (Model 1). Next, qualitative and quantitative sex differences in the genetic and environmental sources of variance for cigarette initiation (a_i^2 , c_i^2 , e_i^2), for cigarette use quantity (a_q^2 , c_q^2 , e_q^2) and the common pathway (b^2), were tested by equating parameters across males and females, and fixing the correlation between the genetic liabilities (r_g : a_i^2 and a_q^2) to 1 (Model 2). Racial differences in the relative contribution of genetic and environmental sources of variance were then tested by constraining parameters to be equal for the White and African American groups (Model 3). Next, race differences in the common pathway, as well as in the variance components, between initiation and quantity of cigarettes were tested (Model 4). The significance of variance components was tested in subsequent models by constraining them to zero and examining subsequent changes to model fit. The fit of the full model (Model 1) was used to compare more restricted models (Models 2-18) using the chi-square statistic. Analyses were done in R (RCore, 2012) using the OpenMx package (Boker et al., 2011).

3. Results

Table 1 presents demographic information, prevalence of ever smoking, and percentage of individuals in each of the three categories of cigarette use quantity for each age group by race. Although the average age of cigarette use onset was similar for White ($\bar{x}=13.8$, $SD=2.1$) and African American adolescents ($\bar{x}=13.8$, $SD=4.4$, $t=-0.11$, $p=ns$), the prevalence of lifetime cigarette use differed significantly by race in each of the three age groups. In the youngest adolescents 27.1% of African Americans reported having ever smoked a cigarette compared to 48.2% of Whites in that same age group ($\chi^2=10.3$, $p<0.0001$). Similarly, in the older two age groups, African Americans reported having ever smoked a cigarette at rates lower than Whites (see Table 1). There were further significant differences in each age group in the number of cigarettes used between White and African American smokers. In each age group, African Americans were more likely to not have smoked in the previous month and, among those who were current smokers, African Americans were less likely to smoke 11 or more cigarettes per day than Whites (see Table 1).

3.1 Testing twin model assumptions

Prior to estimating variance components contributing to the liability to cigarette use, tetrachoric correlations for cigarette use initiation within each age, sibling type and racial group were computed (presented in Table 2). In each age group for Whites, the pattern of correlations suggest the influence of the shared environment for female pairs but additive genetic influences for male pairs. The pattern of correlations in the African American group differs by sex as well. Among 14- to 17-year-old females, the correlations suggest shared environmental influences, but additive genetic and shared environmental influences when one examines the correlations in the older two groups. For the African American males in

the 14- to 17-year-old group, the correlations suggest additive genetic influences and dominance while the 18- to 25-year-old male correlations suggest shared environmental influences. Lastly, for 26- to 33-year-old males the correlations indicate additive genetic and shared environmental influences. The supplementary Table includes the correlations for the MZs and DZs with the full siblings included as DZs which form the basis of the following tests and models.

Next, twin model assumptions were tested separately in the three age groups. In all age groups, we were able to equate the thresholds for the binary variable of cigarette use initiation by twin order and zygosity without observing a significant deterioration in model fit. In the 14-17 and 26-33 age groups, thresholds could also be equated between same-sex and opposite-sex pairs and across males and females. However, thresholds could not be equated across White and African American groups in any age group. Subsequent twin models allowed thresholds to be estimated separately for Whites and African Americans and within each race group, males and females had separate thresholds.

3.2 Biometric Model Fitting

We first examined the fit of a full causal-contingent-common pathway model (Model 1) which included race-specific as well as sex-specific additive genetic, shared and unique environmental parameters estimates (i.e. $a_{WFI}^{2\ddagger}$, $c_{WFI}^{2\ddagger}$, and $e_{WFI}^{2\ddagger}$; $a_{AAFI}^{2\ddagger}$, $c_{AAFI}^{2\ddagger}$, and $e_{AAFI}^{2\ddagger}$) for both cigarette smoking initiation, on cigarette use quantity, as well as the causal path between the two phenotypes. In the next model, parameter estimates were constrained to be the same for males and females within each race group (Model 2). Comparing the fit of Model 2 to Model 1 tests sex differences in parameter estimates in the three age groups (see Table 3). Similarly, comparing Model 3 to Model 2 tests racial differences in parameter estimates for each of the three sources of variance. Subsequent models tested the significance of specific parameters and are described in greater detail in the Supplementary Material.

3.2.1 Model Fitting Results for 14- to 17-Year-Old Group—In the 14- to 17-year-old group parameter estimates (a^2 , c^2 , and e^2) could be equated for Whites and African Americans without a significant deterioration of model fit. When the common pathway was equated across race in addition to variance components (Model 4), there was no significant worsening of model fit. Removing the common pathway between the liabilities to initiate cigarette use and cigarette quantity significantly reduced the fit of the model (Model 5).

Variance component estimates[‡] are presented in Table 4 for the specific influence of each variance component on cigarette use initiation (a_i), cigarette use quantity (a_q), the common pathway (β), and the total variance explained. The variance components were estimated from a model in which only racial differences were allowed for, and for the 14- to 17-year-old group, indicate that additive genetic influences accounted for a greater proportion of variance in cigarette use among Whites than in African Americans. The shared environment accounted for 8% of the variance in cigarette use initiation for the White 14-to 17-year-old group and 15% in the African American. Recall, however, that the model testing racial differences in this age group did not fit significantly worse than a model that allowed for sex

differences as well as racial differences. The inability to find racial differences in this age group may be due to low statistical power and caution is recommended when interpreting the differences that appear in the variance components.

3.2.2 Model Fitting Results for 18- to 25-Year-Old Group—Parameter estimates for additive genetic (a^2), shared environmental (c^2) and unique environmental (e^2) influences could be equated by sex (Model 2) for the 18- to 25-year-old group as well as by race (Model 3) without reductions in the fit of these models. However, when the next model constrained the overlap in genetic and environmental influences to be the equal by racial group, a significant deterioration in the model (Model 4) was observed. In the 18- to 25-year-old group we could not detect racial differences in the amount of variance explained by genetic and environmental influences, but there were racial differences in the overlap between genetic influences on the initiation of cigarettes and the quantity of cigarettes smoked.

Examining the estimate of the common pathway among Whites suggests that about 46% of the co-variance between additive genetic influences in cigarette use initiation is shared with those that influence cigarette use quantity. In African Americans, however, the estimate of the common pathway is negative suggesting that the overlap between additive genetic influences on cigarette use initiation are inversely related to genetic influences on quantity smoked.

The variance components of the 18- to 25-year-old group are presented in Table 4 and indicate that for both Whites and African Americans additive genetic influences (53%, 31%, respectively) are larger than shared environmental influences (1%, 10%).

3.2.3 Model Fitting Results for 26- to 33-Year-Old Group—Racial differences in all three sources of variance (additive genetic, shared and unique environmental; Model 3) and in the common pathway (Model 4) between cigarette use initiation and cigarette quantity were detected for the 26- to 33-year-old group (Table 3). The variance components for this age group suggest that additive genetic influences are greater for Whites (65%) than for African Americans (34%) and that shared environmental influences are greater among African Americans (5%) than in Whites (0%). Estimates of the common pathway in White 26- to 33-year-olds suggest that 62% of the variance in cigarette use initiation is shared with cigarette use quantity. Similar to what was found for the 18- to 25-year-old African American group, in this age group, we find that the estimate of the common pathway (−86%) suggests an inverse relationship in the percent of variance that is shared between initiating cigarette use and in the number of cigarettes smoked for the 26- to 33-year-old African American group.

4. Discussion

The aim of this study was to use a genetically informative, population-based sample to examine racial differences in the heritability of cigarette use initiation and quantity of cigarettes smoked between White and African American participants using the largest and most diverse sample of twins and full sibling pairs. Consistent with previous research we

found a lower prevalence of lifetime cigarette use across adolescence and adulthood for African American participants compared to Whites (Bachman et al., 2011; Blustein et al., 2015). There was, however, no difference in the average age of cigarette use initiation between White and African American adolescents among those who initiate cigarette use. Previous studies have reported that African Americans initiate cigarette use in adulthood rather than in adolescence (Trinidad et al., 2004) and the results of the present analyses indicate that among the African American adolescents who had initiated cigarette use the age of onset did not differ from the sample of Whites, and in fact was 5 years earlier than in previous reports (Taylor et al 1997; White et al., 2004; Trinidad et al., 2004). It should be noted that the age of onset of the African American sample in this study has greater variation than in Whites, suggesting that there are greater individual differences in age of onset in the African American group. The discrepancy between the present finding and existing research might arise from the fact that previous studies have used adult reports of when cigarette use onset occurred rather than reports from adolescents. For instance, although Duncan and colleagues reported an earlier age of cigarette use onset for Whites compared to African American adolescents, the average difference in age of onset between these groups was 5 months. In addition, the average age of onset reported for African American adolescents in Duncan's sample (13.8) is nearly identical to what the present analyses show (13.8). Lastly, among those who had initiated cigarette use, there was a significant difference, consistent with previous reports, in the amount of cigarette smoked such that African American individuals at all ages reported smoking less heavily than Whites.

In adolescence we did not detect racial differences in the contribution of additive genetic and shared environmental influences on the initiation of cigarette use and cigarette use quantity or in the degree to which the liability of cigarette use quantity was shared with the liability to initiate cigarette use. In addition, the significance and magnitude of the common pathway indicated a shared liability between the factors that influence cigarette initiation and quantity for adolescents, regardless of race. This suggests that the factors that contribute to the initiation of cigarette use among adolescents are related to the factors that contribute to the quantity of cigarette smoked for both racial groups.

In young adulthood, however, racial differences were detected in the degree to which genetic and environmental influences contribute to initiating cigarette use and to the quantity of cigarettes smoked. Genetic factors were found to have a greater influence in White adolescents, consistent with previous twin studies of White young adults (Hopfer et al., 2003; Maes and Neale, 2009), than African Americans in this sample. The influence of the shared environment was quite small for young adult Whites (1%), but 10% of the total variance in cigarette use was accounted for by shared environmental influences in African Americans. In young adulthood we found racial differences in the shared liability between using cigarettes and quantity smoked. In fact, the causal pathway was estimated to be positive in Whites, and similar in magnitude to previous reports (Bares et al., 2015; Maes et al., 2004), but negative in African Americans. Among Whites, the overlap in liability indicates that the genetic factors that influence initiating cigarette use are the same that contribute to how many cigarettes are smoked. For African Americans, the negative common pathway between the phenotypes would suggest that the factors that influence the initiation of cigarette use make individuals use fewer cigarettes. This finding is consistent with the

differences by race observed in the descriptive analyses. A greater proportion of African American young adults are non-smokers and among those who do use cigarettes, they use smoke fewer cigarettes per day. Previous studies have indicated that there is a greater time to nicotine dependence among African Americans that is primarily due to smoking fewer cigarettes per day (Duncan et al., 2012), consistent with the findings of the present study.

Finally, in adulthood racial differences were detected in the contribution of additive genetic and shared environmental factors to cigarette use initiation and quantity, as well as the degree of overlap between the liabilities. Additive genetic influences were found to be significant for Whites and African Americans, but they are not identical. Previous studies have reported that the heritability for cigarette initiation in predominantly White adult samples is between 37% to 50% (Li et al., 2003). In the present study, the magnitude of the total influence of genes in White adults (65%) is nearly twice as large than the total genetic influence in the African American adults (34%).

The present findings for the White group across all three age groups are consistent with previous analyses indicating that additive genetic factors play a role in cigarette use initiation that get stronger with age (Do et al., 2015; Maes et al., 2004). To date, only one other study exists that has examined racial differences in heritability of cigarette use initiation (Sartor et al., 2015) but that sample included only female-female pairs. Sartor and colleagues did not detect racial differences in heritability of cigarette use initiation perhaps because their analyses were not sensitive to developmental or sex differences. As can be seen from our table of correlations, the pattern among the African American females is much more consistent with what has been previously reported for White adolescent samples than is observed among African American male-male pairs or opposite-sex pairs.

4.1 Limitation

The findings of the present study have to be taken into account with some caution. To be adequately powered to test for racial differences in the role of genes and environment on cigarette use we made use of full siblings available in the data and modeled them as dizygotic twins. Doing so may have resulted in overestimating the influence of the shared environment because full siblings do not experience exactly the same shared environment as MZ or DZ twins. Because full sibling pairs are not the same age as each other, estimates of the influence of the shared environment in this study may be inflated.

In spite of this limitation, this study is among the first to examine the heritability of cigarette use behaviors by appropriately modeling the conditionality between initiation and quantity used and to compare White and African American individuals as they develop from adolescence into adulthood. Further, the results of this study contribute to our understanding of sources of health disparities in health behaviors by race through obtaining group-specific sources of genetic and environmental influences. Although genetic and environmental influences on the cigarette use behaviors of African American and White adolescents are similar, genes become a stronger risk factor for the use of cigarettes among White young adults while the influence of the familial environment is minimal. For African Americans, both additive genetic and familial influences are important in young adulthood and decrease in adulthood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Sample Differences by Race

	White (n=2,665*)	African American (n=809*)	p
	n(%) / Mean(SD)	n(%) / Mean(SD)	
Demographic characteristics			
Sex (% Female)	1,298 (48.7%)	427 (52.8%)	ns
Sibling type:			
Monozygotic twins	414 (15.5%)	156 (19.3%)	
Dizygotic twins	686 (25.7%)	290 (35.8%)	
Full sibling pairs	1,565 (58.7%)	363 (44.8%)	0.0001
14- to 17-year-olds	15.74 (1.06)	15.72 (1.07)	ns
18- to 25-year-olds	21.75 (1.8)	21.62 (1.8)	ns
26- to 33-year-olds	29.00 (1.6)	28.96 (1.6)	ns
Cigarette smoking behavior			
Age at first cigarette use	13.8 (2.1)	13.8 (4.4)	ns
Ever smoked a cigarette:			
14-17 year old group	48.2%	27.1%	0.0001
18-25 year old group	83.3%	77.7%	0.001
26-33 year old group	83.3%	77.6%	0.002
Cigarette use quantity:			
<u>14- to 17-year-olds</u>			
Not in past 30 days	48.7%	58.0%	
1 to 10 cigarettes per day	37.2%	37.0%	
11 or more cigarettes per day	14.1%	5.0%	0.016
<u>18- to 25-year-olds</u>			
Not in past 30 days	70.0%	81.8%	
1 to 10 cigarettes per day	8.5%	8.6%	
11 or more cigarettes per day	21.5%	18.8%	0.0001
<u>26- to 33-year-olds</u>			
Not in past 30 days	64.7%	80.0%	
1 to 10 cigarettes per day	11.2%	10.9%	
11 or more cigarettes per day	24.1%	21.1%	0.0001

*
Note: number of twin/sibling pairs

Table 2
Tetrachoric Correlations (95% Confidence Intervals) of Cigarette Use Initiation by Age, Sex, Race and Sibling Type

White															
MZ			DZ			DZO			FS			FSO			
n=414			n=418			n=268			n=937			n=628			
Corr.*	95% CI	n	Corr.*	95% CI	n	Corr.*	95% CI	n	Corr.*	95% CI	n	Corr.*	95% CI	n	
14-17 group															
Males	0.75	(0.40, 0.93)	50	0.60	(0.26, 0.83)	63	0.30	(-0.07, 0.61)	70	0.17	(-0.21, 0.51)	122	0.02	(-0.30, 0.34)	168
Females	0.56	(0.18, 0.81)	55	0.65	(0.30, 0.86)	56				0.29	(-0.10, 0.61)	127			
18-25 group															
Males	0.96	(0.82, 1.00)	73	0.57	(0.07, 0.87)	87	0.14	(-0.37, 0.60)	97	0.55	(0.26, 0.76)	182	0.50	(0.22, 0.72)	236
Females	0.79	(0.49, 0.94)	82	0.37	(-0.12, 0.74)	66				0.52	(0.25, 0.72)	174			
26-33 group															
Males	0.94	(0.77, 0.99)	73	0.77	(0.33, 0.95)	81	0.33	(-0.25, 0.75)	101	0.40	(0.04, 0.69)	166	0.52	(0.23, 0.74)	224
Females	0.77	(0.47, 0.93)	81	0.35	(-0.14, 0.73)	65				0.55	(0.29, 0.74)	166			
Black															
MZ			DZ			DZO			FS			FSO			
n=156			n=159			n=131			n=195			n=168			
Corr.*	95% CI	n	Corr.*	95% CI	n	Corr.*	95% CI	n	Corr.*	95% CI	n	Corr.*	95% CI	n	
14-17 group															
Males	0.49	(-0.24, 0.90)	20	0.63	(-0.28, 0.97)	12	-0.95	(-1.00, 0.12)	40	-0.98	(-1.00, 0.44)	25	0.29	(-0.40, 0.80)	42
Females	0.27	(-0.45, 0.81)	22	0.39	(-0.31, 0.85)	30				0.28	(-0.53, 0.85)	28			
18-25 group															
Males	0.26	(-0.52, 0.84)	28	0.07	(-0.63, 0.74)	26	0.21	(-0.28, 0.62)	47	0.52	(-0.22, 0.91)	32	0.00	(-0.45, 0.47)	67
Females	0.68	(0.12, 0.94)	32	0.55	(-0.08, 0.90)	34				0.06	(-0.51, 0.61)	43			
26-33 group															
Males	0.14	(-0.65, 0.81)	24	0.29	(-0.51, 0.86)	23	0.05	(-0.49, 0.57)	44	0.46	(-0.30, 0.90)	28	-0.20	(-0.71, 0.43)	59
Females	0.73	(0.08, 0.97)	30	0.51	(-0.04, 0.86)	34				0.31	(-0.37, 0.81)	39			

Table 3**Causal-Contingent-Common Pathway Models by Age Group**

Age Group	Model	Pars.	-2LL	df	AIC	LL	df	P
<u>14 to 17</u>	1. Full CCC Model	44	3951.44	2623	-1294.56			
	2. Test sex differences	26	3959.15	2641	-1322.85	7.710	18	0.983
	3. Test A ¹ C ² E ³ race differences *	20	3963.661	2647	-1330.339	4.511	6	0.608
	4. Test ACE race differences + beta ⁴	19	3962.942	2648	-1333.058	3.792	7	0.803
	5. Test beta	18	3978.886	2649	-1319.114	15.944	1	0.000
	6. Test overall A	17	3975.623	2650	-1324.377	12.681	2	0.002
	7. Test A on CI ⁵	18	3974.248	2649	-1323.752	11.306	1	0.001
	8. Test A on CQ ⁶	18	3962.953	2649	-1335.047	0.011	1	0.916
	9. Test overall C	17	3963.949	2650	-1336.051	1.007	2	0.604
	10. Test Con CI	18	3962.948	2649	-1335.052	0.006	1	0.938
	11. TestC on CQ	18	3963.905	2649	-1334.095	0.963	1	0.326
<u>18 to 25</u>	1. Full CCC Model	44	5455.55	4707	-3958.45			
	2. Test sex differences	26	5470.182	4725	-3979.818	14.632	18	0.687
	3. Test ACE race differences *	20	5481.185	4731	-3980.815	11.003	6	0.088
	4. Test ACE race differences + beta	19	5487.232	4732	-3976.768	17.050	7	0.017
	5. Test beta in Whites	19	5497.873	4732	-3966.127	16.688	1	0.000
	6. Test beta in African Americans	19	5497.862	4732	-3966.138	16.677	1	0.000
	7. Test overall A in Whites	24	5508.724	4727	-3945.276	38.542	2	0.000
	8. Test overall A in African Americans	24	5471.023	4727	-3982.977	0.841	2	0.657
	9. Test A on CI in Whites	25	5488.445	4726	-3963.555	18.263	1	0.000
	10. Test A on CI in African Americans	25	5470.355	4726	-3981.645	0.173	1	0.677
	11. Test Aon CQ in Whites	25	5470.903	4726	-3981.097	0.721	1	0.396
	12. Test Aon CQ in African Americans	25	5470.735	4726	-3981.265	0.553	1	0.457
	13. Test overall C in Whites	24	5470.218	4727	-3983.782	0.036	2	0.982
	14. Test overall C in African American	24	5470.358	4727	-3983.642	0.176	2	0.916
	15. Test Con CI in Whites	25	5470.218	4726	-3981.782	0.036	1	0.850
	16. Test C on CI in African Americans	25	5470.356	4726	-3981.644	0.174	1	0.677
	17. Test C on CQ in Whites	25	5470.182	4726	-3981.818	0.000	1	1.000
	18. Test C on CQ in African American:	25	5470.182	4726	-3981.818	0.000	1	1.000
<u>26 to 33</u>	1. Full CCC Model	44	5372.720	4406	-3439.28			
	2. Test sex differences	26	5389.420	4424	-3458.58	16.700	18	0.544
	3. Test ACE race differences *	20	5405.048	4430	-3454.952	15.628	6	0.016
	4. Test ACE race differences + beta	19	5408.110	4431	-3453.89	18.690	7	0.009
	5. Test beta in Whites	25	5410.329	4425	-3439.671	20.909	1	0.000
	6. Test beta in African Americans	25	5392.761	4425	-3457.239	3.341	1	0.068
	7. Test overall A in Whites	24	5412.588	4426	-3439.412	23.168	2	0.000
	8. Test overall A in African Americans	24	5389.597	4426	-3462.403	0.177	2	0.915
	9. Test A on CI in Whites	25	5406.782	4425	-3443.218	17.362	1	0.000

Age Group	Model	Pars.	-2LL	df	AIC	LL	df	P
	10. Test A on CI in African Americans	25	5391.446	4425	-3458.554	2.026	1	0.155
	11. Test A on CQ in Whites	25	5396.020	4425	-3453.98	4.634	1	0.031
	12. Test A on CQ in African Americans	25	5394.557	4425	-3455.443	5.137	1	0.023
	13. Test overall C in Whites	24	5391.958	4426	-3460.042	2.538	2	0.281
	14. Test overall C in African American	24	5391.139	4426	-3460.861	1.719	2	0.423
	15. Test Con CI in Whites	25	5391.137	4425	-3458.863	1.717	1	0.190
	16. Test C on CI in African Americans	25	5389.901	4425	-3460.099	0.481	1	0.488
	17. Test C on CQ in Whites	25	5391.382	4425	-3458.618	1.962	1	0.161
	18. Test C on CQ in African American:	25	5391.365	4425	-3458.635	1.945	1	0.163

* Notes: This, and subsequent models, are compared to Model 2; A¹ refers to additive genetic effects, C² refers to shared environmental effects, E³ refers to unique environmental effects; beta⁴ refers to the common pathway; CI⁵ indicates cigarette initiation; CQ⁶ indicates cigarette quantity.

Table 4

Variance Components (95% CIs) from No-Sex Differences Model by Race and Age Group

White										
Cigarette use initiation			Common pathway			Cigarette use quantity			Total	
a_i	c_i	e_i	β	a_q	c_q	e_q	a^2_T	c^2_T	e^2_T	
14-17 year olds	0.70 (0.68, 0.81)	0.00 (0.00, 0.01)	0.30 (0.22, 0.31)	0.96 (0.95, 0.96)	0.00 (0.00, 0.01)	0.07 (0.07, 0.07)	0.01 (0.01, 0.12)	64% (0.01, 0.12)	8% (0.01, 0.12)	28% (0.01, 0.12)
18-25 year olds	0.86 (0.68, 0.96)	0.02 (0.00, 0.29)	0.11 (0.06, 0.17)	0.68 (0.48, 0.72)	0.13 (0.07, 0.30)	0.00 (0.00, 0.00)	0.40 (0.29, 0.62)	53% (0.00, 0.62)	1% (0.00, 0.62)	46% (0.00, 0.62)
26-33 year olds	0.89 (0.86, 0.89)	0.00 (0.00, 0.32)	0.12 (0.12, 0.22)	0.79 (0.41, 0.82)	0.10 (0.06, 0.16)	0.00 (0.00, 0.13)	0.28 (0.20, 0.28)	65% (0.00, 0.13)	0% (0.00, 0.13)	35% (0.00, 0.13)
African American										
a_i	c_i	e_i	β	a_q	c_q	e_q	a^2_T	c^2_T	e^2_T	
14-17 year olds	0.31 (0.02, 0.64)	0.01 (0.00, 0.03)	0.68 (0.50, 0.73)	0.14 (0.10, 0.98)	0.00 (0.00, 0.00)	0.16 (0.12, 0.72)	0.83 (0.29, 0.90)	1% (0.00, 0.00)	15% (0.12, 0.72)	84% (0.29, 0.90)
18-25 year olds	0.19 (0.01, 0.42)	0.12 (0.00, 0.27)	0.69 (0.56, 0.87)	-0.92 (-0.97, -0.79)	0.15 (0.00, 0.19)	0.00 (0.00, 0.00)	0.01 (0.01, 0.70)	31% (0.00, 0.00)	10% (0.00, 0.00)	59% (0.01, 0.70)
26-33 year olds	0.19 (0.00, 0.23)	0.00 (0.00, 0.42)	0.81 (0.77, 0.98)	-0.86 (-0.98, 0.71)	0.21 (0.00, 0.91)	0.04 (0.00, 0.62)	0.01 (0.00, 0.87)	34% (0.00, 0.62)	5% (0.00, 0.87)	61% (0.00, 0.87)

Note: a_i refers to additive genetic influences on cigarette initiation; c_i and e_i refer to shared environmental and unique environmental influences, respectively, on cigarette initiation; a_q refers to additive genetic influences on cigarette quantity, c_q and e_q refer to shared environmental and unique environmental influences on cigarette quantity, respectively.

*The total variance explained by each component is calculated by squaring the common pathway, multiplying by the variance component for cigarette initiation, and adding the quantity to the variance component for cigarette quantity.