Published in final edited form as:

Drug Alcohol Depend. 2015 December 1; 157: 54–59. doi:10.1016/j.drugalcdep.2015.10.002.

Genetic and Environmental Contributions to Initiation of Cigarette Smoking in Young African-American and European-American Women

Carolyn E. Sartor^{1,2}, Julia D. Grant², Arpana Agrawal², Brooke Sadler, Pamela A. F. Madden², Andrew C. Heath², and Kathleen K. Bucholz²

¹Department of Psychiatry, Yale University School of Medicine, New Haven, CT, USA

²Department of Psychiatry, Washington University School of Medicine, St. Louis, MO, USA

Abstract

Background—Distinctions in the relative contributions of genetic and environmental factors to initiation of cigarette smoking may explain, in part, the differences between African Americans and European Americans in the prevalence of smoking. The current investigation is the first to compare heritable and environmental influences on smoking initiation between African-American and European-American women.

Methods—Data were drawn from Missouri Adolescent Female Twin Study participants and female Missouri Family Study participants (n=4,498; 21% African-American, the remainder European-American). Mean age at first and last assessments were 17.0 (SD=3.5) and 24.0 (SD=3.2), respectively. Twin-sibling modeling was conducted to estimate the proportion of variance in smoking initiation (i.e., ever trying a cigarette) attributable to additive genetic, shared environmental, special twin environmental, and unique environmental factors.

Results—Additive genetic influences accounted for approximately half of the variance in smoking initiation in both African-American and European-American women. In the African-American subsample, the remaining variance was attributable primarily to unique environmental factors (46%; 95% CI:28–71%). In the European-American subsample, only 12% (95% CI:8–16%) of the variance was attributable to unique environmental factors, with the remainder

Address correspondence to: Carolyn E. Sartor, Department of Psychiatry, Yale University School of Medicine, VA Connecticut Healthcare System, 950 Campbell Avenue (151D), West Haven, CT 06516; carolyn.sartor@yale.edu; phone: (203) 932-5711 extension 3894.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Contributors

Dr. Sartor conducted the literature searches and wrote the first draft of the manuscript, Dr. Grant conducted the statistical analyses, and Dr. Agrawal provided consultation on analyses. 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 and edited the manuscript. All authors have approved the final manuscript.

Author Disclosures

Conflicts of Interest

We have no conflicts of interest to declare.

accounted for by shared environmental (13%; 95% CI:0–41%) and special twin environmental (24%; 95% CI:0–52%) factors.

Conclusions—The estimated heritability of smoking initiation is substantial and nearly identical for African-American and European-American women, but the type of environmental factors that contribute to risk differ by race/ethnicity. Whereas the primary environmental influences on European-American women's smoking initiation are at the family level, those that impact African-American women's smoking initiation are primarily individual-specific.

Keywords

smoking; African Americans; women; twins

1. INTRODUCTION

More than one-third of 12th graders (17- to 18-year olds) in the U.S. report smoking at least one cigarette during the last year (Johnston et al., 2014) and approximately one in three adolescents who try cigarettes will progress from experimentation to regular smoking (Choi et al., 1997; Flint et al., 1998). The rate of cigarette use among adolescents is currently as high for girls as boys (Johnston et al., 2014) and according to the 2014 Report of the Surgeon General (U.S Department of Health and Human Services, 2014), smoking related disease risks for women now equal those for men, highlighting the importance of identifying mechanisms underlying early smoking behaviors in adolescent girls and young women.

Genetic factors play a major role in the development and persistence of smoking behaviors. The heritability of regular smoking (generally defined as smoking 100 or more cigarettes over the lifetime) has been estimated from twin studies to be between 50 and 80% (Agrawal et al., 2005; Kendler et al., 1999; True et al., 1997; Vink et al., 2005), although studies that have found sex differences have reported somewhat lower estimates for women: 32–46% (Broms et al., 2006; Hamilton et al., 2006; Madden et al., 2004). Genetic contributions to the earliest stage of smoking, experimentation with cigarettes or initiation of smoking (i.e., ever smoking a cigarette), have not been as extensively investigated, but heritability estimates of smoking initiation are in the same range as those for regular smoking (Hardie et al., 2006; Maes et al., 2004, 2006; Seglem et al., 2015). The estimated contribution of shared environment - environmental influences that make members of a twin pair alike, such as common peers and parenting - to initiation of smoking is less consistent across studies but appears to vary systematically by age. For example, in Seglem et al.'s study of 12–18 year-old twins (2015) and Boomsma et al.'s study of 13–22 year-old twins (1994), shared environment accounted for 56% and 59%, respectively, of variance in smoking initiation, whereas neither Maes et al. (2004) nor Hardie et al. (2006) found any evidence for shared environmental influences in their studies based on middle aged twins.

The twin studies to date have been instrumental in developing etiological models of smoking initiation in individuals of European ancestry but have only rarely examined smoking behaviors in African Americans.. Differences in smoking behaviors between African Americans and European Americans are well documented. There is consistent evidence that the lifetime prevalence of smoking (Hu et al., 2006; Lawrence et al., 2007) and risk for

progression to regular smoking (Griesler et al., 2002; Kandel et al., 2004; Vega et al., 2007) are lower in African Americans than European Americans. African Americans also initiate smoking at an older age (Andreski and Breslau, 1993; Duncan et al., 2012; Mickens et al., 2010) and continue to smoke later into adulthood (Moon-Howard et al., 2003).

We are aware of only one large-scale twin study to examine smoking behaviors in African Americans, an earlier investigation by our group (Sartor et al., 2009) that was based exclusively on African-American (AA) young women. Consistent with prior studies based on adults of European ancestry, 62% of variance in initiation of smoking (ever smoking a cigarette) was attributable to genetics and the remainder to unique - also known as individual-specific - environmental influences (i.e., environmental influences not shared by family members); shared environment was not implicated. Although an important step toward improving our understanding of liability to smoking initiation among African Americans, in the absence of European-American (EA) participants, a direct comparison between racial/ethnic groups in the relative influence of genetic vs. environmental factors could not be made. The current study, which integrated data from female participants in a high-risk family study (52% AA) with data from female twin study participants (14% AA), was designed to test for differences between AA and EA young women in the relative contribution of genetic, shared environmental, and individual-specific environmental influences on initiation of cigarette smoking.

2. METHODS

Data were drawn from the Missouri Adolescent Female Twin Study (MOAFTS) and the Missouri Family Study (MOFAM). Both studies were approved by Washington University's Human Research Protections Office. MOFAM was also approved by the Ethics Board of the State Department of Health and Senior Services (which was not required at the time that MOAFTS started).

2.1 Participants

The sample was composed of female twins who completed the fourth wave of data collection for MOAFTS and female participants from MOFAM.

2.1.1 MOAFTS—MOAFTS is a longitudinal study of substance use and related psychopathology in adolescent girls and young women. Female twins born in Missouri to Missouri-resident parents between 1975 and 1985 were identified through birth records and recruited from 1995 to 1999. Cohorts of 13-, 15-, 17-, and 19-year-old twin pairs and their families were ascertained in the first 2 years. New cohorts of 13-year-old twins and their families were added in the subsequent 2 years. Interviews were completed by at least one parent in 78% of eligible families. (Details of sample ascertainment are reported in Waldron et al., 2013.) Baseline (wave 1) interviews were conducted with 3,258 twins. Retest (wave 3) interviews were conducted with a subset of wave 1 participants (n=1,370) 2 years after wave 1 assessments. Between 2002 and 2005, all twins from the target cohort (excluding those who had withdrawn from the study or whose parents asked that the family not be re-contacted) were contacted for wave 4 interviews. As all twins were 18 years of age or older at the time of recruitment for wave 4, parental consent was not needed and as a result, an

even greater number of twins completed wave 4 than wave 1 interviews: 3,787 (80% of those identified from birth records). Wave 5 interviews were conducted between 2005 and 2008 with 3,428 twins.

Because wave 4 interviews yielded the largest sample size and covered lifetime psychiatric and psychosocial information in young adulthood, we limited the sample to MOAFTS participants who completed wave 4 interviews, but data from other waves, which were available for over 95% of wave 4 participants, were integrated as well. The wave 4 sample was composed of 1,038 monozygotic (MZ) twin pairs, 735 dizygotic (DZ) twin pairs and 241 twins whose co-twins did not participate.

2.1.2 MOFAM—MOFAM is a high-risk family study of alcoholism and related conditions oversampled for AA families. From 2003 to 2009, Missouri state birth records were used to identify families with at least one child aged 13, 15, 17 or 19 years (the same age range targeted in MOAFTS) and 1 or 2 additional full siblings. Biological mothers completed brief telephone screening interviews to determine level of familial risk for alcoholism. Families in which biological fathers had a history of excessive drinking were classified as “high risk.” All others were classified as “low risk.” Another group of families was selected from men identified through driving records as having 2 or more drunk driving convictions and classified as “very high risk.”

In all participating families, mothers were interviewed first. Biological fathers were then solicited for interview and permission was sought from mothers to contact offspring. (Sample ascertainment is described in detail in Calvert et al., 2010). Ninety-two percent of targeted families: 450 AA families (41.5% low risk, 30.1% high risk, 28.4% very high risk) and 317 of non-AA (primarily EA) descent (30.1% low risk, 21.5% high risk, 48.4% very high risk) were enrolled in the study. Sample enrollment occurred over 6 years. Three of the intake years had sufficient time to collect 4 waves of data; the remainder had 1 to 2 waves. A total of 1,461 (735 female) offspring completed at least one interview. Data were drawn from all available waves of data collection.

The current sample included the 4,498 individuals of known zygosity and race/ethnicity: 3,767 MOAFTS participants (both members of 1038 MZ twin pairs and 735 DZ twin pairs, as well 221 individuals with no co-twin data) and 731 female MOFAM participants (163 full sibling pairs, 30 full sibling trios, and 315 individuals with no sibling interview data). The mean ages at first and last assessment of the combined sample were 17.0 (SD=3.5) and 24.0 (SD=3.2), respectively (16.8 (SD=3.4) and 24.5 (SD=2.7) for MOAFTS and 17.6 (SD=3.6) and 21.6 (SD=4.2) for MOFAM). Twenty-one percent of the sample self-identified as AA (14%, n=547 of MOAFTS; 55%, n=407 of MOFAM), the remainder as EA.

2.2. Procedure and Assessment Battery for MOAFTS and MOFAM

MOAFTS and MOFAM assessments were nearly identical by design to facilitate integration of data across studies (with adjustments for differences in ascertainment strategies). In both studies, data were collected via telephone interview by trained interviewers using a modified version of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) (Bucholz et al., 1995; Hesselbrock et al., 1999). The SSAGA was designed to assess

substance use history, psychiatric disorders, and other related psychosocial domains. Other than the wave 5 MOAFTS interview, which covered the 2 years between wave 4 and 5 assessments, interviews queried lifetime psychiatric and psychosocial history. Consent was obtained verbally prior to the start of the interview.

Initiation of cigarette smoking was defined as ever having smoked a cigarette (thus capturing experimenters as well as regular smokers). Participants who responded "Yes" to the question "Have you ever tried a cigarette?" at any wave of data collection were classified as positive for initiation. A total of 64.1% of AA participants (67.6% in MOAFTS and 59.5% in MOFAM) and 75.2% of EA participants (76.1% in MOAFTS and 66.6% in MOFAM) reported having tried smoking cigarettes. The mean age at initiation of smoking was 14.6 (SD=3.6) for AA participants (14.4, SD=3.6 in MOAFTS; 14.9, SD=3.4 in MOFAM) and 14.1 (SD=3.2) for EA participants (14.2, SD=3.4 in MOAFTS; 14.1, SD=3.2 in MOFAM).

2.3 Data Analysis: Twin-Sibling Modeling

Data from twins reared together can be used to decompose the variance of a phenotype into additive genetic sources (A), shared - also known as common - environmental sources (C), and unique environmental sources (E; which also includes error variance). Because MZ twin pairs share 100% of their genes and DZ twin pairs share on average 50% of their segregating genes, genetic influence is suggested by higher MZ than DZ twin correlations. Shared environmental influence is indicated by DZ twin correlations exceeding half of the MZ twin correlations.

Since ordinary full siblings share the same amount of genetic material as DZ twin pairs, they can contribute to estimates of genetic and environmental variance. When non-twin sibling pairs are included in twin models, DZ twin and non-twin sibling correlations are compared to determine if the former is significantly greater than the latter. A higher DZ than non-twin sibling pair correlation indicates that environmental influences are more similar for members of DZ twin pairs than non-twin sibling pairs. In this case an additional special twin environment parameter (T; environmental influences that make members of a twin pair but not members of an ordinary sibling pair alike, e.g., being treated more similarly than non-twin siblings by parents or teachers) needs to be modeled.

Polychoric correlations were conducted for MZ, DZ, and MOFAM (non-twin) female same sex sibling pairs, separately for the AA and EA subsamples, to estimate concordance of smoking initiation by sibling pair type and race/ethnicity. Twin-sibling modeling was then conducted with the statistical software package Mx (Neale, 2004), which uses maximum likelihood estimation to evaluate the extent to which phenotypic variance can be attributed to additive genetic, shared environmental, special twin environmental, and unique environmental influences. Twin-sibling models were fitted to raw categorical data. Dummy variables representing low risk, high risk, and very high risk MOFAM groups were included as covariates to adjust the thresholds. A series of sub-models was tested to determine which variance components contribute significantly to risk for smoking initiation in the AA and EA subsamples. To test for differences between African Americans and European Americans in the relative contribution of A, C, T, and E, the pathways were equated across

AA and EA subsamples, with a significant reduction in model fit indicating significant differences between the groups in the contribution of the variance component(s). The sub-models were tested by calculating the difference between the -2 log likelihood fit of the full model and the nested sub-model, which is distributed as chi-square for the given degrees of freedom.

3. RESULTS

3.1 Sibling Pair Correlations

Sibling pair correlations, calculated separately for AA and EA participants, are shown in Table 1. For MZ and DZ twin pairs (but not non-twin sibling pairs), correlations were lower in AA than EA participants, suggestive of an overall lesser degree of familial influence on initiation among AA than EA young women.

However, the correlations suggested the presence of genetic factors in both AA and EA subsamples. For African Americans, the DZ and non-twin sibling correlations were about half the size of the MZ correlations and similar in magnitude, suggesting that there were no shared environmental or special twin environmental effects in African Americans. In contrast, among European Americans, the DZ twin correlations were more than half the MZ correlations and the non-twin sibling correlations were lower than DZ twin correlations, indicating that both shared environmental and special twin environmental contributions may be present.

3.2 Twin-Sibling Models

The full twin-sibling model had 16 estimated parameters. Model fitting, summarized here, is reported in detail in Supplementary Table 1¹. The first series of sub-models (2–6 in Supplementary Table 1²) tested for the significance of A, C, T, and E pathways in the AA subsample. It was possible to drop either C and T or A but not both simultaneously, suggesting insufficient power to differentiate between genetic and family environmental influences. A, C, T, and E were therefore retained in the final AA model. The second series of sub-models (7–10 in Supplementary Table 1³) tested for the significance of A, C, T, and E pathways in the EA subsample. It was possible to drop either C or T (not both simultaneously) but not A, indicating significant genetic influence and significant family environmental influences but insufficient power to differentiate between the sources of family environmental influence. Thus A, C, T, and E were also retained in the EA model. The third series of sub-models (11–16) tested for differences between the AA and EA groups in the contribution of genetic, family environmental, and unique influences. It was possible to equate either the proportion of variance attributable to A or to total family environmental influences (C+T) across African Americans and European Americans, but not the total variance attributable to all familial influences (A+C+T). This indicates significant differences in total familiarity across groups but insufficient power to determine which familial influences differ. The best fitting model, shown with 95% confidence intervals (CI)

*Supplementary material can be found by accessing the online version of this paper at <http://dx.doi.org> and by entering doi:...

in Table 2, was the full model, with A, C, T, and E parameters estimated separately for African Americans and European Americans.

Across subsamples, additive genetic influences accounted for about half of variance in smoking initiation (50% in African Americans (95% CI:0–72%) and 51% in European Americans (95% CI:30–74%). For African Americans, shared environmental and twin-specific environmental influences accounted for an additional 4% (95% CI:0–46%) and <1% (95% CI:0–30%) of the variance, respectively. For European Americans, shared environmental influences explained an additional 13% (95% CI:0–41%) and special twin environmental influences accounted for an additional 24% (95% CI:0–52%) of the variance in smoking initiation.

4. DISCUSSION

The current investigation applied a twin-sibling modeling approach to identifying possible sources of distinctions in liability of AA vs. EA adolescent girls and young women to initiation of cigarette smoking. By expanding on our previous study based exclusively on AA twins (Sartor et al., 2009) through the incorporation of EA participants and integration of non-twin sibling pairs, we were able to detect racial/ethnic differences in the total contribution of familial influences to smoking initiation. Differences were evident from the overall lower cross-twin consistency in smoking status among AA vs. EA pairs: $r_{AA-MZ}=0.55$ vs. $r_{EA-MZ}=0.88$ and $r_{AA-DZ}=0.23$ vs. $r_{EA-DZ}=0.62$. Although the low power in our AA subsample meant that we were unable to differentiate the sources of familial influence (A, C, and/or T) in African Americans, twin-sibling models generated estimates of the total proportion of variance attributable to familial influences in each group. Total familial influences were estimated at 88% in European Americans compared to only 54% in African Americans, indicating that family-level influences play a larger role in smoking initiation in EA than AA young women.

There are no prior twin studies of smoking phenotypes that include both African Americans and European Americans against which to compare our findings, but there are several large-scale studies of twins of European descent and a study conducted with a small sample of AA twins that can provide a context for our findings. Both heritability estimates - 50% for African Americans and 51% for European Americans - fall in the middle of the 30–75% range reported in studies conducted with twins of European ancestry (Hamilton et al., 2006; Hardie et al., 2006; Madden et al., 2004; Maes et al., 2006; Seglem et al., 2015). Moreover, they are nearly identical to the estimate of 56% derived in Whitfield and colleagues' study of 200 AA twins from the Carolina African American Twins Study of Aging (CAATSA; Whitfield et al., 2007), the one known study of smoking initiation based on an AA sample other than MOAFTS. The degree of similarity is striking, especially given the differences in mean age at the time of assessment: 46.9 (SD=13.9) in CAATSA compared to 17.0 (SD=3.5) at first assessment and 24.0 (SD=3.2) at last assessment in our study.

In the twin smoking literature (based on European ancestry twins), significant shared environmental influences - accounting for greater than 50% of variance in initiation of cigarette smoking - have been reported in studies of based on adolescent twins (Boomsma et

al., 1994; Seglem et al., 2015), but those based on middle aged twins have revealed no evidence of shared environmental effects (Hardie et al., 2006; Maes et al., 2004). Although somewhat unusual in its inclusion of special twin environmental influences, our estimate of 37% for family environmental influences in the EA subsample falls within the expected range.

We are aware of one prior study to identify special twin environmental influences on initiation of cigarette smoking (although others that have included both twin and non-twin sibling pairs have documented the influence on non-twin siblings on smoking behavior, e.g., Vink et al., 2003). Maes et al. (2006) used an extended twin kinship design to examine genetic and environmental influences on smoking initiation (i.e., ever smoked) in a sample of over 12,000 twins and over 13,000 additional family members. Special twin environment accounted for 15% of variance in smoking initiation in women and 14% in men. Again, the more modest contribution in this older sample (median age of 50 years), compared to the 24% observed among European Americans in our study, is consistent with the pattern in the literature of decreasing influence of shared environmental effects with increasing age. Interpretations of twin specific environmental influences can be challenging, but findings from Pergadia et al.'s study based on a young adult Australian twin cohort (2006) suggest a potential straightforward explanation. The study revealed that 37.8% of twins tried their first cigarette at the same time as their twin, which is consistent with the higher degree of overlap in twins' vs. non-twin siblings' peer groups (McGuire and Segal, 2013).

Although low power precludes a definitive conclusion regarding the source(s) of familial influences in the AA model, given the substantially higher point estimate for A (50%) vs. C (4%) or T (<0.1%) – and the absence of available smoking data from twin studies that include a comparable number of AA twins – speculation based on this trend level finding is warranted. The weak evidence for shared environmental influences on smoking initiation among AA women in our sample may reflect the greater presence of certain protective factors in AA than EA families. Studies examining attitudes toward substance use across different racial/ethnic groups have found higher rates of parental disapproval of alcohol and other drug use in African Americans compared to European Americans (Beck et al., 1995) and linked ethnic identification to lowered intent to use (Corneille and Belgrave, 2007), more negative attitudes about drug use (Stock et al., 2013) and reduced risk for using substances among AA adolescents (Pugh and Bry, 2007). However, relatively few studies have addressed racial/ethnic differences in parental attitudes specifically toward smoking and evidence for higher disapproval or stricter rules about smoking in AA compared to EA parents has been inconsistent (Ellickson et al., 2003; Mahabee-Gittens et al., 2012). Another known protective factor against adolescent smoking is religious involvement (Nonnemaker et al., 2003; Sinha et al., 2007; Walker et al., 2007; Wills et al., 2003), which is higher in African Americans than European Americans (Taylor et al., 1996; Wallace et al., 2007), but some studies in this area – including one based on a subsample of participants in the current study (Heath et al., 1999) – have not observed this protective effect and others indicate that the effect is weaker in AA adolescents than those of other racial/ethnic backgrounds (Steinman et al., 2008; Wallace et al., 2007). A broader examination of family and community level influences is clearly needed to identify factors that reduce risk for initiation of smoking in African Americans.

4.1 Limitations

Findings should be interpreted with certain limitations in mind. First, the relatively low number of AA sibling pairs limited statistical power, resulting in large confidence intervals that included 0 for estimates of additive genetic, shared environmental, and special twin environmental influences in the AA subsample. Second, it is possible that participants with a very limited smoking history who were first assessed in late adolescence were less likely than those assessed in early adolescence to recall trying a cigarette. However, as we did not observe significant racial/ethnic differences in age at first assessment, such a bias would not have influenced the major study findings. Third, the prevalence of smoking initiation was lower in the MOFAM than MOAFTS (population based) sample and because a larger proportion of AA than EA participants were drawn from MOFAM, the prevalence estimate was affected to a slightly greater degree for the AA than EA subsample. Although an important consideration in terms of generalizability of the prevalence of smoking in AA women, estimates of the proportion of variance accounted for by genetic vs. environmental factors would not be affected substantially by such a shift. Fourth, the absence of racial/ethnic differences in non-twin sibling correlations but the presence in twin pair correlations may be attributable in part to the greater similarity across race/ethnicity in the environmental conditions for MOFAM high risk and very high risk participants than for MOAFTS participants, who were drawn from the general population

4.2 Future Directions

Among the questions raised by our findings is whether these same racial/ethnic distinctions are found in males, for whom estimates of shared environmental influences on initiation of smoking have been less robust (Broms et al., 2006; Hamilton et al., 2006; Madden et al., 2007). The possibility that there are racial/ethnic differences in the contribution of heritable factors to later stages of smoking (e.g., regular smoking and quitting) also merits investigation, particularly given the persistence of smoking later into adulthood in African Americans compared to European Americans (Moon-Howard, 2003). Most important with respect to addressing the larger goals of refining etiological models of smoking and improving smoking prevention efforts for African Americans, it is critical to identify the risk factors subsumed under “unique environmental influences” in our model of smoking initiation. Risk factors that are not shared by family members may be more challenging to address than those occurring in the context of the family, requiring more individually tailored prevention efforts, so this line of research should be a high priority.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Role of Funding Sources

Funding for this study was provided by grants AA090022, AA11998, AA12640, AA17688, AA17915, AA017921, AA021492, and AA023549 from the National Institute on Alcohol Abuse and Alcoholism, grant DA23668 from the National Institute on Drug Abuse, grant HD049024 from the National Institute of Child Health and Human Development, and a grant from the Robert E. Leet and Clara Guthrie Patterson Trust.

The National Institutes of Health and the Robert E. Leet and Clara Guthrie Patterson Trust had no further role in study design; in the collection, analysis and interpretation of data; in the writing of the report; or in the decision to submit the paper for publication.

REFERENCES

- Agrawal A, Madden PA, Heath AC, Lynskey MT, Bucholz KK, Martin NG. Correlates of regular cigarette smoking in a population-based sample of Australian twins. *Addiction*. 2005; 100:1709–1719. [PubMed: 16277631]
- Andreski P, Breslau N. Smoking and nicotine dependence in young adults: differences between blacks and whites. *Drug Alcohol Depend*. 1993; 32:119–125. [PubMed: 8508722]
- Beck KH, Scaffa M, Swift R, Ko M. A survey of parent attitudes and practices regarding underage drinking. *J. Youth Adolesc*. 1995; 24:315–334.
- Boomsma DI, Koopmans JR, van Doornen LJ, Orlebeke JF. Genetic and social influences on starting to smoke: a study of Dutch adolescent twins and their parents. *Addiction*. 1994; 89:219–226. [PubMed: 8173488]
- Broms U, Silventoinen K, Madden PA, Heath AC, Kaprio J. Genetic architecture of smoking behavior: a study of Finnish adult twins. *Twin Res. Hum. Genet*. 2006; 9:64–72. [PubMed: 16611469]
- Bruin JE, Gerstein HC, Holloway AC. Long-term consequences of fetal and neonatal nicotine exposure: a critical review. *Toxicol. Sci*. 2010; 116:364–374. [PubMed: 20363831]
- Bucholz KK, Cadoret R, Cloninger CR, Dinwiddie SH, Hesselbrock VM, Nurnberger JI, Reich T, Schmidt I, Schuckit MA. A new, semi-structured psychiatric interview for use in genetic linkage studies: a report of the reliability of the SSAGA. *J. Stud. Alcohol*. 1994; 55:149–158. [PubMed: 8189735]
- Calvert WJ, Bucholz KK, Steger-May K. Early drinking and its association with adolescents' participation in risky behaviors. *J. Am. Psychiatr. Nurses Assoc*. 2010; 16:239–251. [PubMed: 21659276]
- Choi WS, Pierce JP, Gilpin EA, Farkas AJ, Berry CC. Which adolescent experimenters progress to established smoking in the United States. *Am. J. Prev. Med*. 1997; 13:385–391. [PubMed: 9315272]
- Corneille MA, Belgrave FZ. Ethnic identity, neighborhood risk, and adolescent drug and sex attitudes and refusal efficacy: the urban African American girls' experience. *J. Drug Educ*. 2007; 37:177–190. [PubMed: 17977240]
- Duncan AE, Lessov-Schlaggar CN, Sartor CE, Bucholz KK. 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. *Drug Alcohol Depend*. 2012; 125:140–145. [PubMed: 22564873]
- Ellickson PL, Perlman M, Klein DJ. Explaining racial/ethnic differences in smoking during the transition to adulthood. *Addict. Behav*. 2003; 28:915–931. [PubMed: 12788265]
- Flint AJ, Yamada EG, Novotny TE. Black-white differences in cigarette smoking uptake: progression from adolescent experimentation to regular use. *Prev. Med*. 1998; 27:358–364. [PubMed: 9612826]
- Griesler PC, Kandel DB, Davies M. Ethnic differences in predictors of initiation and persistence of adolescent cigarette smoking in the National Longitudinal Survey of Youth. *Nicotine Tob Res*. 2002; 4:79–93. [PubMed: 11906684]
- Hamilton AS, Lessov-Schlaggar CN, Cockburn MG, Unger JB, Cozen W, Mack TM. Gender differences in determinants of smoking initiation and persistence in California twins. *Cancer Epidemiol. Biomarkers Prev*. 2006; 15:1189–1197. [PubMed: 16775180]
- Hardie TL, Moss HB, Lynch KG. Genetic correlations between smoking initiation and smoking behaviors in a twin sample. *Addict. Behav*. 2006; 31:2030–2037. [PubMed: 16675152]
- Heath AC, Madden PA, Grant JD, McLaughlin TL, Todorov AA, Bucholz KK. Resiliency factors protecting against teenage alcohol use and smoking: influences of religion, religious involvement and values, and ethnicity in the Missouri Adolescent Female Twin Study. *Twin Res*. 1999; 2:145–155. [PubMed: 10480749]

- Hesselbrock M, Easton C, Bucholz KK, Schuckit MA, Hesselbrock VM. A validity study of the SSAGA - a comparison with the SCAN. *Addiction*. 1999; 94:1361–1370. [PubMed: 10615721]
- Hu MC, Davies M, Kandel DB. Epidemiology and correlates of daily smoking and nicotine dependence among young adults in the United States. *Am. J. Public Health*. 2006; 96:299–308. [PubMed: 16380569]
- Johnston, LD.; O'Malley, PM.; Miech, RA.; Bachman, JG.; Schulenberg, JE. Institute for Social Research. Ann Arbor: The University of Michigan; 2014. Monitoring The Future National Survey Results On Drug Use: 1975–2013: Overview, Key Findings On Adolescent Drug Use.
- Kandel DB, Kiros GE, Schaffran C, Hu MC. Racial/ethnic differences in cigarette smoking initiation and progression to daily smoking: a multilevel analysis. *Am. J. Public Health*. 2004; 94:128–135. [PubMed: 14713710]
- Kendler KS, Neale MC, Sullivan P, Corey LA, Gardner CO, Prescott CA. A population-based twin study in women of smoking initiation and nicotine dependence. *Psychol. Med*. 1999; 29:299–308. [PubMed: 10218922]
- Lawrence D, Fagan P, Backinger CL, Gibson JT, Hartman A. Cigarette smoking patterns among young adults aged 18–24 years in the United States. *Nicotine Tob. Res*. 2007; 9:687–697. [PubMed: 17558826]
- Madden PA, Pedersen NL, Kaprio J, Koskenvuo MJ, Martin NG. The epidemiology and genetics of smoking initiation and persistence: crosscultural comparisons of twin study results. *Twin Res*. 2004; 7:82–97. [PubMed: 15053857]
- Maes HH, Neale MC, Kendler KS, Martin NG, Heath AC, Eaves LJ. Genetic and cultural transmission of smoking initiation: an extended twin kinship model. *Behav. Genet*. 2006; 36:795–808. [PubMed: 16810566]
- Maes HH, Sullivan PF, Bulik CM, Neale MC, Prescott CA, Eaves LJ, Kendler KS. A twin study of genetic and environmental influences on tobacco initiation, regular tobacco use and nicotine dependence. *Psychol. Med*. 2004; 34:1251–1261. [PubMed: 15697051]
- Mahabee-Gittens EM, Xiao Y, Gordon JS, Khoury JC. The role of family influences on adolescent smoking in different racial/ethnic groups. *Nicotine Tob. Res*. 2012; 14:264–273. [PubMed: 22180584]
- McGuire S, Segal NL. Peer network overlap in twin, sibling, and friend dyads. *Child Dev*. 2013; 84:500–511. [PubMed: 22994629]
- Mickens L, Ameringer K, Brightman M, Leventhal AM. Epidemiology, determinants, and consequences of cigarette smoking in African American women: an integrative review. *Addict. Behav*. 2010; 35:383–391. [PubMed: 20061090]
- Moon-Howard J. African American women and smoking: starting later. *Am. J. Public Health*. 2003; 93:418–420. [PubMed: 12604485]
- Neale, MC. Statistical Modeling with Mx. Box # 980710, Richmond VA 23298: Dept of Psychiatry; 2004.
- Nonnemaker JM, Mcneely CA, Blum RW. National Longitudinal Study of Adolescent Health. Public and private domains of religiosity and adolescent health risk behaviors: evidence from the National Longitudinal Study of Adolescent Health. *Soc. Sci. Med*. 2003; 57:2049–2054. [PubMed: 14512236]
- Pergadia ML, Heath AC, Agrawal A, Bucholz KK, Martin NG, Madden PA. The implications of simultaneous smoking initiation for inferences about the genetics of smoking behavior from twin data. *Behav. Genet*. 2006; 36:567–576. [PubMed: 16477519]
- Pugh LA, Bry BH. The protective effects of ethnic identity for alcohol and marijuana use among Black young adults. *Cultur. Divers. Ethnic Minor. Psychol*. 2007; 13:187–193. [PubMed: 17500608]
- Sartor CE, Agrawal A, Lynskey MT, Bucholz KK, Madden PA, Heath AC. Common genetic influences on the timing of first use for alcohol, cigarettes, and cannabis in young African-American women. *Drug Alcohol Depend*. 2009; 102:49–55. [PubMed: 19261395]
- Seglem KB, Waaktaar T, Ask H, Torgersen S. Genetic and environmental influences on adolescents' smoking involvement: a multi-informant twin study. *Behav. Genet*. 2015; 45:171–180. [PubMed: 25604452]

- Sinha JW, Cnaan RA, Gelles RJ. Adolescent risk behaviors and religion: findings from a national study. *J. Adolesc.* 2007; 30:231–249. [PubMed: 16677701]
- Steinman KJ, Ferketich AK, Sahr T. The dose-response relationship of adolescent religious activity and substance use: variation across demographic groups. *Health Educ. Behav.* 2008; 35:22–43. [PubMed: 16861596]
- Stock ML, Gibbons FX, Gerrard M, Houlihan AE, Weng CY, Lorenz FO, Simons RL. Racial identification, racial composition, and substance use vulnerability among African American adolescents and young adults. *Health Psychol.* 2013; 32:237–247. [PubMed: 23088177]
- Taylor R, Chatters L, Jayakody R, Levin J. Black and white differences in religious participation: a multisample comparison. *J. Sci. Study Relig.* 1996; 35:403–410.
- True WR, Heath AC, Scherrer JF, Waterman B, Goldberg J, Lin N, Eisen SA, Lyons MJ, Tsuang MT. Genetic and environmental contributions to smoking. *Addiction.* 1997; 92:1277–1287. [PubMed: 9489045]
- U.S. Department of Health and Human Services. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014.
- U.S. Department of Health and Human Services. The Health Consequences of Smoking: A Report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2004.
- U.S. Department of Health and Human Services. How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease: A Report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2010.
- Vega WA, Chen KW, Williams J. Smoking, drugs, and other behavioral health problems among multiethnic adolescents in the NHSDA. *Addict. Behav.* 2007; 32:1949–1956. [PubMed: 17254715]
- Vink JM, Willemsen G, Boomsma DI. Heritability of smoking initiation and nicotine dependence. *Behav. Genet.* 2005; 35:397–406. [PubMed: 15971021]
- Vink JM, Willmsen G, Engels CME, Boomsma DI. Smoking status of parents, siblings, and friends: predictors of regular smoking? Findings from a longitudinal twin-family study. *Twin Res.* 2003; 6:209–217. [PubMed: 12855070]
- Waldron M, Bucholz KK, Lynskey MT, Madden PA, Heath AC. Alcoholism and timing of separation in parents: findings in a midwestern birth cohort. *J. Stud. Alcohol Drugs.* 2013; 74:337–348. [PubMed: 23384382]
- Walker C, Ainette MG, Wills TA, Mendoza D. Religiosity and substance use: test of an indirect-effect model in early and middle adolescence. *Psychol. Addict. Behav.* 2007; 21:84–96. [PubMed: 17385958]
- Wallace JM Jr, Delva J, O'Malley PM, Bachman JG, Schulenberg JE, Johnston LD, Stewart C. Race/ethnicity, religiosity and adolescent alcohol, cigarette and marijuana use. *Soc. Work Public Health.* 2007; 23:193–213. [PubMed: 19306594]
- Whitfield KE, King G, Moller S, Edwards CL, Nelson T, Vandenberg D. Concordance rates for smoking among African-American twins. *J. Natl. Med. Assoc.* 2007; 99:213–217. [PubMed: 17393944]
- Wills TA, Yaeger AM, Sandy JM. Buffering effect of religiosity for adolescent substance use. *Psychol. Addict. Behav.* 2003; 17:24–31. [PubMed: 12665078]

Highlights

- Initiation of smoking in women is influenced to a substantial degree by genetics.
- Familial influences are stronger in European-American than African-American women.
- Shared environmental factors contribute to initiation in European-American women.
- Environmental influences are largely individual-specific in African-American women.

Table 1

Sibling-pair correlations for initiation of cigarette smoking for African Americans and European Americans

	MZ twin (MOAFTS)	DZ twin (MOAFTS)	Non-twin sibling (MOFAM)
African Americans	0.55 (0.33–0.78) n=127 pairs	0.23 (0.05–0.52) n=128 pairs	0.32 (0.03–0.61) n=101 pairs
European Americans	0.88 (0.85–0.92) n=911 pairs	0.62 (0.52–0.73) n=606 pairs	0.49 (0.20–0.78) n=92 pairs

MZ=monozygotic; DZ=dizygotic; MOAFTS=Missouri Adolescent Female Twin Study; MOFAM=Missouri Family Study; 95% confidence intervals shown in parentheses

Table 2

Proportions of variance in initiation of cigarette smoking attributable to genetic and environmental factors in African-American and European-American women*

	Additive Genetic	Shared Environmental	Special Twin Environmental	Unique Environmental
African Americans				
Full model	0.50 (0.00 – 0.72)	0.04 (0.00 – 0.46)	<0.001 (0.00 – 0.30)	0.46 (0.28 – 0.71)
European Americans				
Full model	0.51 (0.30 – 0.74)	0.13 (0.00 – 0.41)	0.24 (0.00 – 0.52)	0.12 (0.08 – 0.16)

* dummy variables representing low risk, high risk, and very high risk MOFAM groups included in all models to adjust for sample ascertainment; 95% confidence intervals shown in parentheses