Original investigation

Comparison of Smoking History Patterns Among African American and White Cohorts in the United States Born 1890 to 1990

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Abstract

Introduction: Characterizing smoking history patterns summarizes life course exposure for birth cohorts, essential for evaluating the impact of tobacco control on health. Limited attention has been given to patterns among African Americans.

Methods: Life course smoking histories of African Americans and whites were estimated beginning with the 1890 birth cohort. Estimates of smoking initiation and cessation probabilities, and intensity can be used as a baseline for studying smoking intervention strategies that target smoking exposure. US National Health Interview Surveys conducted from 1965 to 2012 yielded cross-sectional information on current smoking behavior among African Americans and whites. Additional detail for smokers including age at initiation, age at cessation and smoking intensity were available in some surveys and these were used to construct smoking histories for participants up to the date that they were interviewed. Age-period-cohort models with constrained natural splines provided estimates of current, former and never-smoker prevalence in cohorts beginning in 1890.

Results: This approach yielded yearly estimates of initiation, cessation and smoking intensity by age for each birth cohort. Smoking initiation probabilities tend to be lower among African Americans compared to whites, and cessation probabilities also were generally lower. Higher initiation leads to higher smoking prevalence among whites in younger ages, but lower cessation leads to higher prevalence at older ages in blacks, when adverse health effects of smoking become most apparent.

Conclusions: These estimates provide a summary that can be used to better understand the effects of changes in smoking behavior following publication of the Surgeon General's Report in 1964.

Implications: A novel method of estimating smoking histories was applied to data from the National Health Interview Surveys, which provided an extensive summary of the smoking history in this population following publication of the Surgeon General's Report in 1964. The results suggest that some of the existing disparities in smoking-related disease may be due to the lower cessation rates in African Americans compared to whites. However, the number of cigarettes smoked is also lower among African Americans. Further work is needed to determine mechanisms by which smoking duration and intensity can account for racial disparities in smoking-related diseases.

Introduction

Among preventable causes of death in the United States (US), cigarette smoking has remained firmly at the top of the list for decades.¹ Publication of the first report of the Surgeon General on the health consequences of smoking in 1964² inspired the public health community to launch multipronged efforts to limit those consequences.^{3–5} As a result, an estimated 8 million premature deaths were averted by 2012, amounting to 31 percent fewer than estimated without tobacco control.⁶ Nevertheless, between 2004 and 2012, 3.2 million people died prematurely from cigarette smoking,⁶ keeping smoking a major risk factor affecting health of the US public. Racial/ethnic disparities underlie smoking exposure trends and the health consequences of exposure.

Current smoking prevalence among adults 18 years or over has declined steadily for blacks and whites, and currently stands at about 18 percent for both (see p. 725, Figure 13.13 in the reference¹). However, a more detailed breakdown by age and gender reveals differences that can ultimately result in important dissimilarities as birth cohorts approach ages that have higher risk of tobacco related disease.¹ Black young adults have lower rates of initiation than whites,⁷ which results in correspondingly lower prevalence of current smoking among black high school students, although the prevalence trends among all ages 18 and older from 1991 to 2011 are similar to whites. This apparent contradiction results from other differences in the life course of smoking histories for birth cohorts, that is, blacks quit smoking at rates that are 10% lower than their white counterparts.¹ Because smoking-related diseases tend to occur at older ages, it is most relevant to consider the effects of exposure on disease and mortality rates in older age groups. Our goal is not to estimate effects of smoking on specific diseases, but we will show that differences in initiation, cessation and intensity can ultimately lead to very different cumulative summaries of smoking history in subsets of the US population, in the age groups most affected by exposure to this important risk factor.

A case in point is lung cancer, a disease risk that is strongly related to cigarette smoking, incidence rates which also differs between blacks and whites. Rates for males are considerably higher for blacks than for whites, and for females rates for blacks are slightly lower than whites.^{8,9} Reasons for these disparities are not well understood, but are likely to be due to either different intensities or durations of cigarette exposure, especially at ages with high absolute risk of lung cancer. Levels of carcinogen exposure may not be conveyed by an interview response because of differences in the way individuals smoke, the chosen brand or the individuals metabolic function for removing toxins. For example, Pérez-Stable et al.¹⁰ have shown substantial differences between blacks and whites in nicotine uptake, thus affecting the net exposure from each cigarette smoked. Hence, genomic differences may also play a role in the effect of cigarette smoking exposure on disease risk.

In this study, we focus on differences in the level of cigarette smoking exposure between blacks and whites. We extend our earlier work on smoking trends for all races¹¹ to examine racial differences and the effects this might have on risk of tobacco related diseases. We use data from the National Health Interview Surveys to construct smoking histories for African Americans and whites born after 1889, and compare the differences. Our analysis provides information on current and former smoker prevalence by age and gender as well as average duration of smoking, quantity smoked, and summary measures of overall smoking history.

Methods

Data

Smoking status (current, former, or never) was elicited in 36 National Health Interview Surveys (NHIS) administered from 1965 to 2012 (1965–1966, 1970, 1974, 1976–1980, 1983, 1985, 1987–1988, 1990–1995, 1997–2012), where ever-smokers were defined as those who smoked 100 lifetime cigarettes.¹² While there is some variation in the exact wording in the various surveys, smoking status was determined by first asking whether they had smoked 100 cigarettes over their lifetime (ever-smokers) and if the response was “Yes” they were asked if they still smoked regularly (current or former smokers). These provided cross-sectional estimates of the smoking prevalence by age and survey year. Further detail was provided by a subset of these surveys (1970, 1978–1980, 1987–1988, 1992, 1995, 1997–2001, 2010–2012), which provided retrospective histories of age at smoking initiation and cessation, prior to the interview.

Smoking histories among blacks and whites by gender to age 99 were estimated using the NHIS variable that gives the race classification of the respondent for calendar years 1965–2012. Individuals reporting race as African American or black represent 13% or 131 125 (47 782 males and 83 343 females) out of 1 000 387 subjects in the NHIS data set with smoking histories, whereas whites represent 82% or 817 751 (338 195 males and 479 556 females). Smoking experience over the life course was constructed using yearly initiation and cessation probabilities beginning with those born after 1889. No birth cohort has been followed from birth to age 99. For example, subjects 75 and older are the only subjects available from the 1890 birth-cohort in the first survey conducted in 1965. Similarly, since the minimum age for survey participants is 18, the most recent cohort actually interviewed was born in 1994 and would have reported a very short smoking history. Extrapolation beyond the observed range was necessary to more fully capture the overall history of cigarette smoking in the population.

Statistical Methods

The method of analysis¹¹ employs a compartment model for smoking history, which begins with the transition from never to current smoker, followed by the transition to former smoker. This clearly over-simplifies a more complex reality in which individuals often attempt cessation several times of varying duration. However, the NHIS data are cross-sectional and they do not provide the detail required for a more complex model. Separate analyses were conducted for each gender and race. Interpretation of individual temporal effects attributed to age, period (calendar year) and cohort (year of birth) is complicated by the well-known identifiability problem of age-period-cohort models, but we are only interested in fitted rates that result from the analysis and these are unique and unaffected by this phenomenon.^{13,14} We briefly describe the analytical approach below, but further detail on the manner in which the estimates were obtained is provided in the [Supplementary Material](#).

Each temporal effect was represented as a constrained natural spline,¹⁵ which is a semiparametric function for the additive effects of age, period and cohort. Maximum likelihood estimates were obtained using PROC GENMOD in SAS that uses a generalized inverse to resolve the identifiability problem, which in our case constrained the cohort slope to zero, but fitted values for outcomes are independent of arbitrary constraints used to specify the generalized inverse.

Using the retrospective histories obtained for ever-smokers, we first estimate the probability an individual reports initiating at given age conditional on the individual being a never-smoker at the beginning of that year. The cumulative proportion of ever-smokers was obtained using life table methods,¹⁶ but at older ages this estimate is biased when compared to estimates in the observed population at that age because smokers tend to die earlier than nonsmokers. We thus empirically adjusted the initiation probabilities by determining cross-sectional prevalence trends by age within cohorts. To align actuarial and cross sectional estimates of ever-smoker prevalence at a specified age, we obtained a multiplicative constant that was applied to the initiation probability used in the actuarial estimate that made the two estimates coincide. Cohorts born before 1935 only have NHIS data for ages 30 or over. Individuals in the survey sample represent increasingly older individuals as one evaluates earlier birth cohorts, and the differential mortality in older ages that results from cigarette smoking leads to a biased representation of the size of the smoking cohort when it was younger. Currently, cohorts usually initiate smoking before 30, but it was not uncommon for those born early in the twentieth century to initiate later in life, especially females.¹⁷ Because later smoking initiation would also postpone the effect of differential mortality, we force alignment for these cohorts at the age first surveyed. Otherwise, cohorts are aligned at age 30. This process provided adjusted initiation probabilities, as well as the prevalence of ever-smokers.

Because smoking cessation is often not successful and shows a high rate of relapse in the first 2 years, we defined an individual as a former smoker if they had quit at least 2 years before the interview; otherwise their observation was censored at the given age of quitting. We estimated the conditional cessation probabilities based on the proportion of current smokers at a given age, who quit during that year, beginning at age 15. These were then used to generate the cumulative probability of quitting by age for each cohort. For most surveys, the upper age limit for recruitment was 85, and because cessation probabilities were already high they were held constant at that point. Alternative assumptions were also tried, but by age 85 smoking prevalence is low due to cumulative effects of cessation and high death rates among smokers, and consequently the specific choice made little difference to the results.

The conditional cessation probabilities are used to determine the cumulative proportion of smokers who had not quit by age for a given cohort. We multiply this cumulative proportion by the ever-smoker prevalence at each age to obtain current smoking prevalence. Former smoker prevalence is found by subtracting current from ever prevalence, and never-smoker prevalence is obtained as 1 minus ever-smoker prevalence. A detailed presentation of the formulae used in these calculations are provided in the [Supplementary Material](#).

In the NHIS data, reported numbers of cigarettes smoked per day (CPD) were highly clumped at half pack intervals, so smoking intensity was classified as an ordered categorical response: $CPD \leq 5$; $5 < CPD \leq 15$; $15 < CPD \leq 25$; $25 < CPD \leq 35$; $35 < CPD \leq 45$; and $45 < CPD$. A cumulative logistic age-period-cohort model was fitted, once again using constrained natural splines for temporal effects. Fitted probabilities for each dose category provide an estimated smoking intensity distribution for single years of age by birth cohort. Estimates for cohorts born before 1920 were constrained to be the same as the 1920 birth cohort. Similarly, estimates for cohorts born after 2002 were arbitrarily constrained to be identical to those of the 2005 cohort, who would be seven in 2012, the year before earliest age at initiation. The mean number of cigarettes smokers consumed

per day was then determined by using the estimated distribution of the categories and an approximate mean of the reported values within a category (3: $CPD \leq 5$; 10: $5 < CPD \leq 15$; 20: $15 < CPD \leq 25$; 30: $25 < CPD \leq 35$; 40: $35 < CPD \leq 45$; and 60: $45 < CPD$).

Using details from the smoking histories by birth cohort, we obtained summaries of smoking history when the cohort reached a particular age. Average cumulative years of exposure for current smokers was determined by the average difference between current age and age at initiation. Correspondingly, for former smokers the average difference between age at cessation and age at initiation represented their contribution to years of exposure. Weighting by the distribution of smoking categories, we obtained the overall average exposure per capita for a specific cohort at a given age. Similarly, we obtained average per capita pack-years by cumulating CPD over the duration of cigarette use in each category. This approach yielded estimates by birth cohort, but these were also rearranged by period in order to consider the temporal perspective normally considered for public health planning. Details on the formulae used to obtain these estimates are provided in the [Supplementary Material](#).

Results

Single birth-cohort estimates of yearly initiation and cessation probabilities, and prevalence were generated by the analysis, but Figures only display decennial cohorts. In the [Supplementary Material](#), we display animated graphs that show a comparison of observed and fitted estimates of ever, current, and former smokers by gender and race for all cohorts ([Supplementary Figures A2–A4](#)). [Figure 1](#) shows age-specific initiation probabilities for blacks (solid lines) and whites (broken lines) ([Supplementary Figure A9](#) displays the difference).

For males ([Figure 1a](#)), the initiation probabilities follow similar time trends, that is, each cohort reaches a peak in the late teens and the overall pattern for those born in the 20th century showed an increase until 1920–1930 followed by a decline. However, black males have generally had lower rates of initiation when compared to whites.

Initiation probabilities for females ([Figure 1b](#)) show more complex trends than their male counterparts. In the cohorts born before 1910, when smoking was considered to be less socially acceptable in women, initiation probabilities were low, but slightly higher for black women younger than about 30 compared to whites. For these cohorts, the analysis indicates more of a decline after age 25 in black women compared to their white counterparts, which showed little decline until well after their 30th year, although the small sample size results in considerable uncertainty in these estimates. In cohorts born after about 1930, the age pattern of increasing to a peak in the late teens and then falling to near zero by age 30 was similar for both races and both genders, although overall levels tend to be lower for females and gender-specific rates for blacks are lower than their white counterparts. The initiation probabilities appear to already be quite high for males born at the start of the 20th century, and they continue to increase before beginning to decline for those born around 1950 and later. In comparison, initiation probabilities for females mostly occurred after the start of the 20th century and did not begin to decline until cohorts born after 1960. This decline appears to have been much steeper for black females than for white females, otherwise, the patterns for blacks and whites appear to track for both genders, with somewhat lower initiation probabilities for blacks.

Cessation probabilities are shown in [Figure 2](#) ([Supplementary Figure A10](#) displays the difference). The overall patterns reveal an

Figure 1. Initiation probability by race (black = solid line, white = broken line), gender, calendar year, and birth cohort (insert shows the trend by age for the 1930 cohort).

increasing trend with age and with year of birth. Particularly striking is the fairly consistent upturn in 1950s and early 1960s that occurs for many cohorts. Females have slightly lower cessation probabilities than males, but the cessation probabilities are considerably lower for blacks compared to whites, especially for those under 50 years of age. As already noted for initiation probabilities, there appears to also be a racial difference in cessation for women born early in the century. For these women, the cessation probabilities for blacks under 50 were higher than whites.

Figure 3 shows current smoker prevalence for each cohort by period (Supplementary Figure A11 displays the difference), which is essentially driven by the initiation and cessation probabilities. Tracing each line from left to right follows the life course of a cohort. For male cohorts, the prevalence tends to reach a peak that is higher in whites than blacks and but the estimates fall more rapidly in whites due to higher cessation probabilities, resulting in higher prevalences for blacks older than 50. For females, there is a much steeper increase among those born before 1930 because

Figure 2. Cessation probability by race (black = solid line, white = broken line), gender, calendar year, and birth cohort (insert shows the trend by age for the 1930 cohort).

of the higher initiation probabilities noted above. Because initiation in these cohorts extends over a longer time span, the prevalence for whites eventually overtake those for blacks, but they ultimately return to very similar levels in older ages. For cohorts born after 1930 but before 1950, the pattern resembles that of the males, that is, higher for whites in the younger ages, but higher for blacks later in life due to lower cessation probabilities. In recent cohorts, the differences in initiation probabilities are much greater,

so that the prevalence for white females remains higher than that of blacks over all ages observed and the lower cessation probabilities for black females is not enough for the age-specific prevalence to overtake the prevalence for whites. In [Supplementary Figure A5](#) we show 90% confidence intervals for the corresponding trends in blacks.

Former smoker prevalence is displayed in [Figure 4](#) ([Supplementary Figure A12](#) displays the difference), which is shown to be considerably

Figure 3. Current smoker prevalence by race (black = solid line, white = broken line) gender, calendar year, and birth cohort.

higher for whites than blacks. This follows from the results noted earlier, which indicated that both initiation and cessation tend to be lower for blacks. Some modest exceptions to this phenomenon occur in female cohorts born before 1930, when initiation probabilities among those younger than 30 were higher for blacks, but eventually high cessation probabilities for whites result in higher former smoker prevalence for even these cohorts. [Supplementary Figure A6](#) shows corresponding 90% confidence intervals for the estimates in blacks.

[Figure 5](#) ([Supplementary Figure A13](#) displays the difference) shows the estimated mean cigarettes per day for each calendar year by cohort. In males born before the 1970s, consumption increased with age before declining later in life. In more recent cohorts, the trend with age appears to be much less prominent. The most prominent difference by race is that the increase in intensity is much less for blacks than for whites in the earlier birth cohorts. At the ages of peak consumption, the difference is approximately 10 CPD. Females

Figure 4. Former smoker prevalence by race (black = solid line, white = broken line), gender, calendar year, and birth cohort.

show an overall similar pattern albeit at lower levels than males, with a lower difference in consumption at the peak, that is, approximately 7 CPD.

Figure 6 (Supplementary Figure A14 displays the difference) shows the mean cumulative years of exposure (duration) for all individuals (current, former, and never smokers) at a given age for a specified cohort, which provides one population measure of the smoking history burden experienced by a cohort over its lifetime. For males, per capita duration is slightly higher for whites but at

older ages the levels are greater for blacks and the magnitude of the difference tends to be greater. With a few exceptions, duration burden tends to remain lower for black females, compared to their white counterparts. The crossover with age noted for duration in males, is not apparent for pack-years, as shown in Figure 7 (Supplementary Figure A15 displays the difference). This reflects the considerably lower smoking intensity in blacks of either gender when compared to corresponding whites. This results in considerably lower estimates of smoking exposure among blacks using this alternative measure of

Figure 5. Mean cigarettes per day by race (black = solid line, white = broken line), gender, calendar year, and birth cohort.

exposure. [Supplementary Figure A7](#) shows 90% confidence intervals for the estimated trends in cumulative years smoked in blacks.

[Figure 8](#) presents the duration burden for each single year birth cohort at age 65 by race and gender. After 1980, black males have higher levels of duration burden than whites. For females, on the other hand, levels for blacks are somewhat lower than for whites prior to 1990, with little difference thereafter.

Trends in pack-years for those aged 65 are shown in [Figure 9](#). While the shapes of the curves are similar, the values are considerably lower for blacks, which results from the correspondingly lower estimates of intensity (mean cigarettes per day) already noted. For males, there has been a substantial decline since 1990, but there has been little changes for black females. [Supplementary Figure A8](#) shows 90% confidence intervals for the trends in mean pack-years for blacks.

Figure 6. Mean cumulative years of smoking by race (black = solid line, white = broken line), gender, calendar year, and birth cohort.

Discussion

The Smoking History Generator (SHG) that was developed by the National Cancer Institute and the Cancer Intervention Modeling Network (CISNET)¹⁷ was recently updated to include surveys conducted through 2009.¹¹ Results presented here provide a further update by including NHIS data through 2012 and by investigating race differences. The analytical approach enhanced the initial estimates of smoking history parameters by making use of a more

comprehensive age-period-cohort model that considered all of the data in a single analysis, instead of separate analyses for 5-year birth cohorts that were subsequently smoothed in a secondary analysis of the results for each cohort.^{17,18} Combining these two steps into a single modeling framework provided greater statistical power, which made it feasible to obtain yearly estimates with smaller sample sizes. In this analysis we made use of that power to consider smoking histories in the African American population, which represents only 13% or 131 125 (47 782 male and 83 343 female) out of 1 000 387

Figure 7. Mean pack-years by race (black = solid line, white = broken line), gender, calendar year, and birth cohort.

subjects in the NHIS data set. This allowed us to compare smoking histories in this minority population with the experience in the majority white population. Another strength of this new approach is that it can be modified to include the effects of covariates, for example, ethnicity, education, and socioeconomic status.

Race is one of the variables elicited by survey respondents, but the manner in which this was obtained changed over time, especially when the need for better data on minority groups was appreciated. Initially, race was based on the interviewer's observation,

but even when respondents identified their race in later surveys there was considerable variation in how the question was worded.¹⁹ In this analysis we used the race classification reported in the survey, but this variation over time is an unavoidable limitation in these data. It is difficult to determine how these changes in NHIS methodology would have affected the results presented here. However, it is clear that race issues need to be carefully considered when developing public health policies to control health effects of smoking.

Figure 8. Mean cumulative years of smoking at age 65 by race (BK = black, WH = white), gender, calendar year, and birth cohort.

Figure 9. Mean pack-years of smoking at age 65 by race (BK = black, WH = white), gender, calendar year, and birth cohort.

We have used data from the NHIS surveys that were collected from 1965 to 2012 to estimate initiation and cessation probabilities; current, former, and never smoking prevalences; and, the distribution of smoking intensities by single years of age and single year birth cohorts beginning in 1890. Some detail, for example, age at initiation and cessation, was necessarily obtained retrospectively from subjects' memories of their past history, which is a limitation that results from the lack of longitudinal data. Differential mortality due to adverse effects of cigarette smoking result in a biased sample when cohorts are queried later in life, making it necessary to adjust

the crude estimates of initiation probabilities.²⁰ We employed a method that empirically controls for this bias, and provides a means for making the adjustment by estimating the effect of selection bias using only the data from NHIS.¹¹ In the results presented here, the analyses were carried out separately by race and gender, which provides for any differences in the adjustment that may have arisen.

Figure 1 shows the contrasting ways in which initiation probabilities changed over the course of the 20th century by both race and by gender, which reflects some of the societal influences on cigarette smoking. For males, the shape of the curves was similar and the birth

cohort pattern was also similar, with the peak increasing for cohorts born early in the century and reaching a zenith for those born in the 1920s. This pattern is consistent with the increased popularity of cigarette smoking among males and the very effective strategy of building a market for male smokers by distributing free cigarettes to those enlisted for military service in World War II. The subsequent decline coincides with the growth in understanding of the health consequences of smoking. In general, the initiation probabilities are consistently lower for blacks in comparison to whites.

By contrast, the initiation probability patterns for women differ somewhat by race. For the 1900 birth cohort there is more of a decline after the late teens for black females while the probabilities for whites changed very little well into their 40s. This suggests somewhat less of a rebound for black females as a result of increasing acceptability of smoking by women. In addition, this period is one of the few instances in which a black cohort had somewhat higher initiation probabilities compared to whites. As the century progressed, initiation probabilities for blacks are lower than whites, and the difference in the peak levels are generally greater than the corresponding race comparison for males.

Cessation probabilities are consistently lower for blacks, but the pattern of increase with age is much less for black females than for either white females or males of either race. The differences in initiation and cessation give rise to distinct prevalence trends and they explain why adult smoking prevalence for blacks is very similar to whites even though initiation probabilities are lower for blacks. For males, current smoker prevalence (Figure 3) is consistently high in younger ages for whites, a pattern that is subsequently reversed for older ages as a result of lower cessation probabilities in blacks so that one can only appreciate what is occurring in the population by evaluating the age patterns. In birth cohorts born before 1920, this reversal is not as clear for females, but among the later birth cohorts the pattern is similar to males. The higher prevalence of smoking among blacks for older ages is significant because many of the health conditions associated with smoking, like lung cancer or chronic obstructive pulmonary disease, tend to occur later in life.

The framework in which we have developed these estimates made use of simplifying assumptions, which were necessary due to data that were limited to cross sectional surveys. We only considered single transitions from never- to ever-smoker and current to former smoker. For initiation this is reasonable, in that we define smokers as those who have smoked at least 100 cigarettes. Cessation, on the other hand, often requires more than one attempt, and the nature of the NHIS data does not provide the necessary detail that would allow for a detailed analysis of this process. Our definition of former smokers were those smokers who had not smoked for at least 2 years in order to limit the number of transitions between current and former smokers. The model provides a good fit to the crude estimates of prevalence obtained directly from the NHIS data, except for age 25–34 in which the fitted estimates for former smokers are slightly lower and for current smokers they are slightly higher (see results in Supplementary Material). This would be expected if a subject reported that they had quit but then relapsed after the interview. With no way to determine post-interview relapse from the cross-section NHIS survey, relapses would not ultimately satisfy the definition of cessation used in our analysis.

Two commonly used summary measures of smoking history in the population are mean per capita duration of smoking and mean pack-years. Work by Doll and Peto²¹ and by Flanders et al.²² indicate that duration is more strongly associated lung cancer mortality

risk so this is likely to be a better predictor of risk for this disease. While smoking intensity improves estimates of risk, pack-years is not the optimal way to include this information. More recent work with additional cohort data indicates that an even better estimate of lung cancer mortality risk can be achieved by including life history of exposure.^{23,24} This work implies that age at initiation and cessation provides better estimates of risk that are not captured by duration, alone, and the history of smoking intensity further improves estimates of risk. Hence, both summary measures of smoking history have limitations and this is especially true for pack-years. Nevertheless, pack-years are still widely used, perhaps because it is relatively easy to compute and interpretation makes intuitive sense. The US Preventive Services Task Force²⁵ and the Centers for Medicare & Medicaid Services (CMS)²⁶ use 30 pack-years as one of the criteria for lung cancer screening eligibility. Our summary of these two measures at age 65 (Figures 8 and 9), when risk of tobacco related diseases are increasing, shows that black males tend to have lower mean pack-years exposure but higher mean duration compared to whites. However, lung cancer risk among black males is as great as or greater than whites. The substantially lower mean pack-years for black males that does not result in a correspondingly lower lung cancer risk may result in fewer screens than would be appropriate for this group. When quantifying the impact of smoking on disease control, a single summary is usually not adequate, but it is best accomplished by assessing the manner in which risk for that specific disease is affected by age, duration, time since cessation, and intensity.

While smoking prevalence is slightly lower for younger black males, the lower cessation probabilities result in higher prevalence of current smokers later in the history of the birth cohort. The net effect of this has resulted in declining mean duration of exposure for both whites and blacks, but the estimates for blacks is slightly greater than whites since 2000 (Figure 8). For females, on the other hand, there has been little temporal trend in mean duration and little difference by race. There are considerably larger differences in exposure as measured by pack-years, which are considerably lower for blacks than whites, as a result of substantially lower levels of CPD reported to NHIS. This is puzzling given the higher rates of lung cancer in blacks. However, the reasons for the difference are not well understood, and they may reflect racial/ethnic differences in the metabolism of toxic components of tobacco smoke, the manner in which cigarettes are used or the preference of different brands. For example, Pérez-Stable et al. showed that total and nonrenal clearances of cotinine were significantly lower for blacks compared to whites. In addition, blacks had 30% higher nicotine intake per cigarette. In combination, this may explain higher levels of cotinine per cigarette smoked, which may in turn reduce the number of cigarettes blacks would need to smoke in order to produce a comparable pharmacologic effect.¹⁰ Duration, of course, would not be affected by this and it remains unclear as to how these differences would affect the toxic effects on lung cancer. Further work is needed to understand reasons for these racial/ethnic differences in smoking behavior, but what does seem clear is that there may be important racial/ethnic differences in the effects that need to be considered when developing control strategies for cigarette smoking.

The tobacco industry has historically targeted particular brands in different segments of the cigarette market. Menthol cigarettes have been aggressively advertised to African Americans and this group disproportionately use mentholated cigarettes compared to whites. This is one fundamental racial/ethnic difference that has existed for decades and African Americans continue to be a target of

advertisements for menthol cigarettes.²⁷ We found that blacks initiate smoking at lower rates and smokers smoke fewer cigarettes, but have lower cessation. These opposing tendencies may be due to the greater use of menthol cigarettes among black smokers than white smokers but it is not clear that this is actually the underlying cause for the difference.²⁸ Further investigation is needed in this area.

These results provide an early step in the search for effective ways to control the many diseases caused by cigarette smoking, including lung cancer, heart disease and chronic obstructive pulmonary disease. Lung cancer, for example, has been thought to be more strongly related to duration than intensity, which would suggest that the difference in mean duration was more relevant than the difference in pack-years. However, other work with carcinogenesis models indicates that not only does intensity play a role, but one must also consider both age at initiation and age at cessation, that is, duration by itself is not an adequate measure of exposure. Both of the exposure summary measures presented here have limitations and they are meant to provide easily understood differences in the experience of African Americans. Pack-years in particular has been seen to have serious limitations, even though it is commonly used to control for smoking history in epidemiological studies. A primary reason for including pack-years in these results is the fact that this is used in identifying eligibility for lung cancer screening. The fact that lung cancer risk is higher for African American males than it is for their white counterparts, even though they have lower mean pack-years of exposure then this might point to a need to consider alternative criteria for determining screening eligibility.

A strength of the NHIS data is that it represents a probability sample of the US population, but a limitation arises from the fact that events prior to 1965 rely on recall of initiation and cessation by the respondents. Before 1974, data were either self-reported or elicited from a proxy who maybe another adult member of the household. Proxy data are usually valid for current smoking status, but they may not be valid for age at initiation and cessation, or intensity. In particular, the data from the 1970 NHIS survey may be biased.^{29,30}

These results will be useful in distinguishing differential effects of smoking by race on lung cancer, heart disease and chronic obstructive pulmonary disease. Our results suggest that some of the existing disparities in smoking related-conditions may be due to the lower cessation rates of black compared to white smokers. However, the number of cigarettes smoked is lower among blacks, which may reflect smoking cigarettes closer to the butt or the use of menthol cigarettes. In further work, we plan to investigate the role of smoking duration, intensity and type of cigarettes in explaining racial disparities in the rate of deaths due to the various diseases.

Supplementary Material

Supplementary Material and Figures A1–A15 can be found online at <http://www.ntr.oxfordjournals.org>

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Declaration of Interests

None declared.

Disclaimer

The findings and conclusions are the author's, not necessarily the CDC's.

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