THE LONG PLUME OF CHILDHOOD: CIGARETTE SMOKING THROUGHOUT THE LIFE COURSE AND ADULT MORTALITY*

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ABSTRACT

Individuals are exposed to the harmful substances in cigarette smoke throughout the life course. Yet, demographic studies of health consequences of smoking generally measure only adult smoking status without examining the enduring effect of early-life smoking. Likewise, life course studies of the influence of childhood conditions on adult health generally focus on childhood socioeconomic status and health but ignore cigarette use. I investigate the effects of childhood and adulthood smoking on racial/ethnic/nativity and sex differentials in adult mortality using the 1997-2006 National Health Interview Survey-Linked Mortality Files. Smoking initiation in childhood or adolescence contributes additional mortality risk for current heavy and light smokers relative to never smokers. Foreign-born and U.S-born Hispanics' lower smoking prevalence and later initiation reduce their mortality risk relative to whites. Findings reinforce the benefits of interventions designed to eliminate or delay smoking initiation among children and adolescents and the salience of early-life smoke exposure as a childhood circumstance that deteriorates later-life health.

Keywords: cigarette smoking, mortality, life course, race/ethnicity/nativity

INTRODUCTION

Exposure to cigarette smoke is one of the greatest dangers to population health and longevity (Doll et al. 2004; Mokdad et al., 2004; Preston, Glei and Wilmoth 2010; Rogers et al., 2005). The pernicious effects of cigarette smoke exposure are greatest among the heaviest smokers and those who smoke for extended periods of time. Tobacco consumption or secondhand smoke exposure during childhood and adolescence may inflict indelible damage on the structure and functioning of the cardiovascular and respiratory systems. Given the highly addictive nature of the substances contained in cigarettes, nicotine in particular, initiation[1] to smoking at an early age may set individuals on a life-long trajectory of continued cigarette use and reduced opportunity for a long and healthy life.

While the life course framework has been applied to numerous childhood conditions with far-reaching socioeconomic and health consequences, researchers generally regard cigarette use as an adult health behavior. Although adult smoking prevalence is lower today than among past birth cohorts (Preston and Wang 2006), cigarette use during childhood and adolescence endures (U.S. Department of Health and Human Services 2012). Childhood may represent a sensitive period of the life course, during which exposure to cigarette smoke permanently scars the structure and impairs the functioning of the body (Ben-Shlomo and Kuh 2002).

This paper investigates how cigarette use in early-life affects mortality risk in adulthood as well as how differences in life-long cigarette use influence racial/ethnic/nativity and sex mortality differentials. A life course framework proposes that early-life cigarette smoke will increase the risk of death net of adult smoking status and other covariates. Pooled survey data linked to death records permit examination of early-life health behavior and adult mortality for a

[1] I use the term "initiation" throughout to describe age at initiation to becoming a regular smoker rather than age at first cigarette use.

nationally-representative sample of both sexes born over a 45 year span during historical periods with distinct attitudes, knowledge, and behaviors regarding cigarette use.

Cigarette Smoking and Mortality in the United States

Studies utilizing both direct and indirect methods from over half a century demonstrate the pernicious impact of smoking on population health. Cigarette use is the primary cause of preventable death in the United States; about 18% of deaths occurring in 2000 in the U.S. were attributable to tobacco use (Mokdad et al. 2004). The lower life expectancy of the U.S. population compared to populations of other industrialized nations may be partially due to the high prevalence of cigarette smoking among U.S. adults and adolescents throughout the 20th century. Smoking increases the mortality risk from a number of causes of death other than lung cancer and respiratory diseases, including cardiovascular and cerebrovascular diseases and cancer in sites other than the lungs (Doll et al., 2004; Hummer, Nam and Rogers 1998).

Differences in smoking behavior contribute to racial/ethnic/nativity and sex disparities in health. Rogers and colleagues (2010) showed that about 22% of the female mortality advantage is due to higher prevalence of cigarette smoking among males. While females historically smoke at lower rates than males, increasing female labor force participation is hypothesized to have increased their smoking rates as conflict arises between occupational obligations and traditional household obligations (Waldron 1993). Lower rates of smoking among U.S. minorities relative to non-Hispanic whites present a unique paradox. While some research has reasoned that the socioeconomic adversity and discrimination experienced by racial/ethnic minority populations would motivate them to smoke and engage in other health risk behaviors as short-term coping mechanisms at the expense of long-term health (Geronimus, Neidert and Bound 1993; Lawlor et al., 2003), Hispanics and non-Hispanic blacks are less likely than non-Hispanic whites to smoke

in adolescence (Centers for Disease Control and Prevention 2010). While Hispanics' smoking prevalence remains lower than that of non-Hispanic whites at all ages, smoking prevalence of non-Hispanic blacks converges to that of non-Hispanic whites and a cross-over occurs in the 20's (Geronimus et al. 1993; Pampel 2008). The low prevalence of cigarette use among Hispanics (particularly foreign-born Hispanics) likely contributes to their low mortality risk relative to non-Hispanic whites, particularly for smoking-attributable causes such as cardiovascular diseases, lung cancer, and respiratory diseases. Hummer, Lariscy, and Hayward (2011) show that the risk of death from lung cancer or respiratory disease is lower among both foreign-born and U.S.-born Hispanics relative to U.S.-born non-Hispanic whites. For example, vis-à-vis U.S.-born non-Hispanic white males ages 65-79, foreign-born Hispanic males ages 65-79 are 58% less likely to die from cancer of the lung, trachea, or bronchus during mortality follow-up and 65% less likely to die from respiratory disease during follow-up.

Most demographic studies of cigarette smoking and mortality focus exclusively on adult smoking status without considering the age of smoking initiation and duration. However, life-long cigarette smoke exposure deserves attention given that smoking patterns are shaped by the key components of the life course, including historical shifts in attitudes and behaviors regarding tobacco use, the influence of both agency and structure on individuals' decisions to smoke, and the linked lives of families and peer groups in childhood and adulthood (Elder 1998).

The Life Course Framework: Early-life Circumstances and Adult Mortality

Life course research emphasizes the enduring influence of childhood exposures on adult social, economic, cognitive, behavioral, and health outcomes. Epidemiologists and demographers apply this perspective to the disablement process as it unfolds over time and the persistence (and potential widening) of health disparities at adult ages. Preston, Hill, and Drevenstedt (1998)

developed a typology demonstrating four potential associations between childhood health and adult mortality risk. Early-life health conditions may leave a direct biological imprint on the structures and functions of organ systems through either scarring or immunity. Alternatively, childhood circumstances may indirectly affect adult health, either because individuals who endure disadvantaged early-life health and social status tend to also experience disadvantaged adult health and social status (correlated environments) or infant mortality selects only more robust individuals to survive to advanced ages (selection).

While some instances of negative associations between adverse childhood circumstances and adult mortality (immunity or selection) are documented, the bulk of life course studies find adverse early-life conditions reduce the probability of survival and increase the risk of later-life poor health and disability. The current contentious issue is whether childhood conditions influence adult health directly or indirectly. Hayward and Gorman (2004) found that the effect of childhood socioeconomic and family conditions on adult mortality risk is largely indirect, operating through adult socioeconomic achievement and health behaviors. More recently, Montez and Hayward (2011) found evidence for both pathway and biological imprint processes. Using 1998-2006 Health and Retirement Study (HRS) data, they found that for females, the association between childhood health and adult mortality risk is reduced but remains significant with adjustment for adult height and educational attainment. For males, adjustment for adult conditions reduced the effect of father's education on mortality risk, but men whose fathers attained fewer than eight years of education still had a 15% greater mortality risk than men with higher-educated fathers.

Most research on early-life circumstances as determinants of adult health focus on variables other than health behavior, including nutrition, infectious disease, family disruption,

household socioeconomic status (parental education, father's occupation, home ownership, and moving for financial reasons), and rural/urban residence (Ben-Shlomo and Kuh 2002; Elo and Preston 1992). One exception is Ferraro and Kelley-Moore's (2003) study of life-long obesity and exercise and their impact of lower- and upper-body disability. Additionally, the framework developed by Crimmins and Finch (2006) includes "noninfectious inflammogens" as an additional external factor that operates through inflammation and organ damage to impact morbidity and mortality. However, their analyses examine data recorded prior to the 20th century—before cigarette smoking became prevalent—and therefore focus on sources of inflammation other than smoking.

Throughout the twentieth century, risk of nutritional deprivation and infectious disease burden in early-life diminished in developed countries as living conditions improved. Greater caloric availability ensures that most mothers and infants receive adequate nutrition during critical periods of growth and development (Fogel 2004). Additionally, childhood infectious illnesses have become less common in recent cohorts. As caloric deficiency and infectious diseases become less common, other early life circumstances (i.e., cigarette smoke exposure, obesity, autoimmune conditions, and family disruption) rise in prominence as determinants of later-life health (Ferraro and Kelley-Moore 2003; Okada et al. 2010; Schwartz et al., 1995). This occurrence fits with fundamental cause theory's assertion that reduced or eliminated mechanisms of disease are replaced by new mechanisms that maintain health inequality based on distal social causes, such as race/ethnicity and social class (Link and Phelan 1995).

Previous studies that examined the relationship between early-life smoking and adult health are generally limited by cross-sectional data, non-representative samples, respondents born within a specific time period or geographic location, small numbers of health events, or

health outcomes other than mortality. For instance, McCarron and colleagues (2001), using a sample of males students at Glasgow University in Scotland between 1948 and 1968, found that smoking in early adulthood was statistically associated with all-cause mortality and death from cardiovascular disease, coronary heart disease, and cancers related to smoking but not associated with death due to stroke, cancers unrelated to smoking, respiratory disease, and residual causes. The small number of stroke deaths (d = 66) and deaths from respiratory diseases (d = 46) likely limited the statistical power of their analysis, given that other studies have shown an association between cigarette use and respiratory disease mortality. Additionally, the data lacked any adult variables other than vital status. Hegmann and colleagues (1993) found that men who started smoking before age 19 and women who started smoking before age 25 were more likely to be diagnosed with lung cancer than men and women who began smoking later or never initiated smoking, using a case-control design of Utah residents. And while numerous studies utilizing the National Longitudinal Study of Adolescent health (Add Health), the Tobacco Use Supplements to the Current Population Survey (TUS-CPS), and other data sources demonstrate racial/ethnic/nativity and sex differences in child and adolescent smoking, these sources do not allow study of the effect of early smoking on adult health outcomes permitted by data with retrospective self-reports of age at smoking initiation and prospective mortality follow-up. Recently, Blue and Fenelon used three indirect estimation methods (Peto-Lopez (1992), Preston-Glei-Wilmoth (2010), and a method of their own design) to show that smoking differentials explain more than 75% of the difference in life expectancy at age 50 between Hispanic and non-Hispanic white men and nearly 75% of the life expectancy difference between Hispanic and non-Hispanic white women. However, their indirect method utilized data that do not include direct

observation of individuals' smoking initiation, duration, intensity, or cessation and contain biases due to selective out-migration (which they address) and ethnic misclassification.

The objective of the current study is to address the limitations of this previous research by utilizing current and nationally-representative pooled survey data linked to death records that permit examination of early-life cigarette smoking, adult smoking, and adult mortality risk for females and males born over a 45 year span during historical periods with distinct attitudes, knowledge, and behaviors regarding youth and adult cigarette smoking.

METHODS

Data

I use the 1997-2006 National Health Interview Survey Linked Mortality Files (NHIS-LMF), available through the Integrated Health Interview Series (Minnesota Population Center 2010). This data source is comprised of National Health Interview Survey (NHIS) data from 1986-2004 linked to National Death Index (NDI) death records through 2006. The current study uses Adult Sample File data (roughly one adult from each NHIS household) for years 1997 through 2004, given that these respondents are asked detailed questions regarding past and current cigarette smoking. Prior NHIS years included supplements on cancer risk or health behavior and disease prevention with questions on smoking, but the 1997-2004 data offer the first opportunity to pool multiple consecutive years with identical measures of adult smoking status, smoking intensity, age at smoking initiation, and time since cessation as well as other socio-demographic and behavioral variables associated with both tobacco use and mortality risk. Mortality follow-up is performed through probabilistic matching of surveys to death records maintained by the NDI based on items generally reported in both sources. Respondents deemed ineligible for linkage due to missing information on surveys or death records are excluded from analyses. While the

linkage of NHIS surveys to NDI death records is generally of high quality, matches among Hispanics and the foreign-born are less certain than matches among non-Hispanic whites and U.S.-born adults (Lariscy 2011).

Variables and Measurement

Age at smoking initiation is measured by the question: "How old were you when you first started to smoke fairly regularly?" As with other childhood conditions (Haas 2007), adults are able to accurately recall their age of smoking initiation with limited risk of bias (Huerta et al., 2005; Johnson and Mott 2001; Kenkel, Lillard and Mathios 2003). In the first set of analyses depicting the mean age of smoking initiation by birth cohort, race/ethnicity/nativity, and sex, age at smoking initiation is kept in its continuous form. In the hazard models that follow, age at smoking initiation is dichotomized as initiating smoking at age 16 or younger versus initiating smoking after age 16[2]. Adult smoking status is coded as never smoker, former smoker, current light smoker (fewer than 20 cigarettes per day), and heavy smoker (20 or more cigarettes per day).

I adjust for or stratify by several socio-demographic characteristics associated with adult smoking status and mortality risk in both sets of analyses. I measure race/ethnicity/nativity as foreign-born Hispanic, U.S.-born Hispanic, U.S-born non-Hispanic black (hereafter Black), and U.S.-born non-Hispanic white (hereafter White). I stratify Hispanics by nativity (foreign-born or U.S.-born) since smoking is less prevalent among the foreign-born than the U.S.-born (Blue and Fenelon 2011; Lopez-Gonzalez, Aravena and Hummer 2005). I limit the sample to adults ages 25-74 years with complete information on smoking and socio-demographic covariates. Given

[2] 43.1% of respondents began smoking at age 16 or younger. Sensitivity analyses found that using either ages 15 or 18 as the cut-point produced similar findings (available from author by request).

that the prevalence of cigarette smoking rose and fell on a cohort basis throughout the twentieth century (Preston and Wang 2006), I examine differences in the age at smoking initiation and the proportion of never, former, current light, and current heavy smokers for three separate birth cohorts: born 1930-1944, 1945-1959, or 1960-1974. These three birth cohorts are similar to those defined by Carlson (2008) as the Lucky Few (born 1929-1945), Baby Boomers (born 1946-1964), and Generation X (born 1965-1982) for his comparisons of educational attainment, occupational opportunity, and military service across U.S. birth cohorts. While the 1997-2004 NHIS Adult Sample files contain information on adults born 1912-1986, I exclude respondents born before 1930 given that mortality selection among the oldest smokers would likely underestimate the prevalence of ever smoking and overestimate the mean age at smoking initiation among ever smokers. Christopoulou et al. (2011) showed that in the United States, mortality selection begins to bias population smoking prevalence estimates beyond age 80 for males. The lower age limit of 25 years ensures that respondents completed the life course stages when smoking initiation is most common and education is generally concluded. A cohort perspective is particularly important when discussing U.S. smoking prevalence given that these three 15 year birth cohorts experienced historical periods with distinct attitudes, knowledge, and behaviors regarding cigarette use. For instance, men born early in the 20th century exhibited the highest prevalence of cigarette smoking and likely had engrained habits when the dangers of smoking became apparent. Likewise, members of the 1945-1959 birth cohort were children or adolescents when the Surgeon General issued the first report warning of the health risks attributed to smoking in 1964. The 1960-1974 cohort have lived through a time period characterized by anti-smoking campaigns and tobacco marketing restrictions.

I adjust for adult social statuses associated with smoking behavior and mortality risk in hazard models to determine whether early-life cigarette use directly affects mortality risk (imprint) or operates indirectly through adult characteristics (pathway). Individuals with greater levels of educational attainment are better prepared to gather information and make decisions on the range of available health behavior options and navigate social institutions to secure the resources that will increase their health and well-being (Hummer and Lariscy 2011; Lynch, Kaplan and Salonen 1997; Mirowsky and Ross 2003; Pampel, Krueger and Denney 2010). Additionally, Wadsworth (1997) identified education as a primary pathway linking early-life adversity to later-life respiratory health. Thus, I adjust for educational attainment in mortality analyses to observe whether education (as a proxy for adulthood socioeconomic achievement) mediates the association between life-long smoking patterns and mortality. Educational attainment is coded as less than a high school diploma, high school diploma (reference group), and at least some college education. Marriage is associated with both better health behaviors and reduced mortality risk (Lillard and Waite 1995; Smith and Zick 1994; Zhang and Hayward 2006). Thus, I adjust for marital status as widowed, divorced/separated, never married, and married (reference group).

I adjust for two adult health behaviors other than smoking status: alcohol use and body weight. Evidence suggests that the association between alcohol use and adult mortality risk is U-shaped; individuals who drink excessively or refrain from alcohol are at a greater risk of death during follow-up than are individuals who consume alcohol moderately (Fuller 2011; Himes 2011). Therefore, I include four alcohol consumption groups in analyses: never, former, moderate (reference group), and heavy drinkers. While body weight is not a health behavior per se, it reflects diet and physical activity. I measure body weight as body mass index (BMI),

calculated as weight in kilograms divided by height in meters squared. I code BMI into four groups: underweight (BMI < 18.5), healthy weight (18.5 ≤ BMI < 25), overweight (25 ≤ BMI < 30), and obese (BMI ≥ 30). Adjustment for body weight is particularly important in analyses of smoking status and adult mortality risk since smoking suppresses the effect of high BMI on death from circulatory diseases and cancers (Krueger et al., 2004).

Analytic Approach

Analyses are performed in two stages. First, I calculate racial/ethnic/nativity, sex, and birth cohort differences in age at smoking initiation and percentage distribution of adults in each smoking status group. Second, Cox proportional hazard regression models estimate the relative risk of mortality for adults 25-74 years old. I use an internal moderator approach to interact adult smoking status with age at smoking initiation (Mirowsky 1999). This approach allows examination of the magnitude and significance of early initiation to smoking for each adult smoking status except never smokers (who have no value for age at smoking initiation). The analytic sample consists of 154,963 respondents, 5,960 of whom died during follow-up. SAS survey commands produce point estimates and standard errors that account for the eligibility criteria and geographic stratification of the NHIS-LMF.

RESULTS

Table 1 shows the mean or percentage distribution of all independent variables separately for survivors and deaths during follow-up. Never smokers are over-represented among survivors while smokers (particularly heavy and former smokers) are over-represented among deaths. Interestingly, the mean age at smoking initiation is only 0.2 years greater for survivors compared to decedents. One potential explanation for this is that some respondents who begin smoking in

adolescence quit smoking while later initiators continue smoking through a greater portion of their life. This explanation conforms with the weathering hypothesis proposed by Geronimus and colleagues (1993) to explain black-white differences in smoking prevalence.

TABLE 1 ABOUT HERE

Figure 1 presents mean ages of smoking initiation by race/ethnicity/nativity among three birth cohorts. Because sex differences in smoking trends are so distinct, results are shown separately for females (Panel 1a) and males (Panel 1b). For both sexes, Whites generally begin smoking at the youngest ages. Among the most recent cohort, mean age at smoking initiation among Black smokers is the highest for males and females. For both males and females, foreign-born and U.S.-born Hispanics do not differ greatly from each other. Both groups' mean ages of smoking initiation are somewhat greater than that of Whites, particularly for females. Females generally initiate cigarette use at older ages than do their male peers, although the mean age for females converges toward the male mean across cohorts. The absolute decreases in age at smoking initiation among females could be misleading given that respondents in the younger cohorts may still initiate smoking at a later age. For instance, a respondent born in 1974 and interviewed in 1999 would only be 25 years old. However, this risk of underestimating mean age of smoking initiation is low, as 95% of ever smokers begin at age 26 or younger. Additionally, selective mortality of smokers in the oldest birth cohort (1930-1944) potentially biases mean age of smoking initiation upward in the incidence analysis and smoking prevalence downward in the prevalence analysis below.

FIGURE 1 ABOUT HERE

Since the mean age at smoking initiation only provides information on the early-life health behavior of a portion of the sample (ever smokers), prevalence estimates are needed to

show what proportion of adults begin smoking. Table 2 gives the percentage distribution of adults reporting that they are current heavy, current light, former, and never smokers, separately by sex, race/ethnicity/nativity, and birth cohort. Among both males and females, foreign-born Hispanic females have the highest percentage of never smokers for each birth cohort except the most recent (born 1960-1974), for whom Blacks replace foreign-born Hispanics as the group with the greatest percentage of never smokers. For each birth cohort and racial/ethnic/nativity group, smoking prevalence is higher among males than among females, although the sex gap appears to converge in the younger cohorts. Of particular interest is the sex gap among Whites born 1960-1974; their percentages of never-smokers are only one percentage point apart, compared to a 21 percentage point gap in the 1930-1944 birth cohort. Taken together, the incidence and prevalence analyses indicate that Hispanics and Blacks are advantaged relative to Whites both in terms of starting later and being less likely to smoke.

TABLE 2 ABOUT HERE

Table 3 presents hazard ratios of mortality for both childhood and adulthood smoking behavior. Model 1 compares mortality risk of six combinations of age at smoking initiation (early [≤16] versus late [>16]) and adult smoking status (current heavy smoker, current light smoker, and former smoker) relative to never smokers. Smoking status * early initiation interaction terms allow examination of the magnitude and significance of the effect of age at initiation beyond the effect of adult smoking status. To interpret the hazard ratios for smoking history, the hazard ratio for the non-interaction smoking status represents the mortality risk for smokers of that status who initiated age 17 or older relative to never smokers while the hazard ratio for smokers of that smoking status who initiation early (the interaction term) represents the

additional risk of mortality introduced by early smoking initiation.[3] For current heavy and current light smokers, the relative risk for early initiators is higher than that of late initiators, as demonstrated by significant interaction terms. The former smoker * early initiation interaction term is never significant, possibly due to the heterogeneity of the former smoker group. A graded dose-response relationship between cigarette use and mortality risk is apparent; compared to never smokers, mortality risk increases with higher cigarette consumption and earlier age of initiation, net of sex, age, and birth cohort. For instance, the greatest mortality risk is among current smokers who began smoking at age 16 or earlier and smoke 20 or more cigarettes per day on average; they are more than three times more likely to die during follow-up than never smokers. Interestingly, the hazard ratio for current light smokers who initiated early is greater than that of heavy smokers who initiated later; supplemental analyses indicate this difference is significant (not shown, available from author). Former smokers, both early and late initiators, have a greater mortality risk than never smokers. This finding indicates that the body does not forget exposure to cigarette smoke earlier in life. Smoking cessation may reduce mortality risk but it is not a "risk-factor elimination" that reverses the scarring sustained from earlier exposure (Ferraro and Kelley-Moore 2003). Also, former smokers may quit smoking only after being diagnosed with a smoking-attributable ailment (Hummer et al. 1998; Leffondré et al., 2002).

Model 2 shows baseline racial/ethnic/nativity differences in mortality risk prior to inclusion of smoking history. Compared to Whites, the mortality risk during follow-up is not statistically different for foreign-born Hispanics, 18% higher for U.S.-born Hispanics, and 90%

[3] The hazard ratio of the early initiation group can be recovered by taking the logarithm of both the first order and second order terms, adding them together, and then multiplying them. For example, in Model 1, to recover the hazard ratio for current heavy smokers who initiated smoking early, take the logarithm of 2.52 and 1.33, add them together, and exponentiate the result. The resulting hazard ratio will be 3.36.

greater for Blacks. Model 3 considers the influence of smoking history and race/ethnicity/nativity on mortality risk together. When Hispanics' low smoking prevalence and later age at smoking initiation are controlled for, mortality risk relative to Whites increases for both nativity groups; the hazard ratio of foreign-born Hispanics becomes greater than 1.0 but remains not statistically different from whites and U.S.-born Hispanics are now 25% more likely to die during follow-up relative to Whites. This indicates that the low smoking prevalence and later age at initiation of Hispanics reduce their mortality risk. Additionally, with the inclusion of race/ethnicity/nativity, hazard ratios for current heavy and former smokers increase slightly while hazard ratios for current light smokers decrease slightly. This finding indicates that racial/ethnic minorities and foreign-born respondents who smoke are generally light smokers. Adjustment for adult educational attainment, marital status, and adult health behavior in Model 4 reduces the relative risk of each childhood/adulthood smoking status group, but all smoking history hazard ratios that were previously significant remain highly significant. The hazard ratio among both Hispanic groups when educational attainment, marital status, and other health behaviors are held constant, given that educational attainment is substantially lower among Hispanics relative to Whites. Foreign-born Hispanics are 13% less likely to die during follow-up than Whites and the mortality risk of U.S.-born Hispanics is not statistically different from that of Whites.

TABLE 3 ABOUT HERE

Models 5 and 6 reports hazard ratios of all-cause mortality among adults who have never smoked. As reported in Table 2, a greater proportion of Hispanics and Blacks than Whites are in the "never smoker" category. Thus, Models 5 and 6 includes a selectively healthy group of Whites relative to Hispanics and Blacks. Comparison of Model 5 to Model 2 provides additional

evidence that the mortality risk of Hispanics and non-Hispanic blacks relative to non-Hispanic whites benefits from their lower levels of smoking throughout the life course. For never smokers ages 25-74 years, foreign-born Hispanics, U.S.-born Hispanics, and Blacks have a greater risk of dying during follow-up compared to Whites, net of sex, age, and birth cohort. With adjustment for educational attainment, marital status, and adult health behaviors, the mortality risk for foreign-born and U.S.-born Hispanic never-smokers reduces and is not statistically different from than that of Whites. Adjustment for adult conditions reduces the relative risk for black never smokers, but they are 61% more likely to die during follow-up than non-Hispanic white never smokers.

DISCUSSION AND CONCLUSIONS

Exposure to cigarette smoke is one of the greatest dangers to population health and longevity. Tobacco consumption or secondhand smoke exposure during childhood inflicts indelible damage on the structure and functioning of organ systems. This paper investigates how cigarette smoke exposure in early-life affects mortality risk in later-life. Results indicate that a young age of smoking initiation increases mortality risk net of adult socioeconomic and marital status and other health behaviors. Simultaneous examination of childhood and adulthood cigarette use also reveals how smoking behaviors influence mortality differences based on other social and demographic factors. Even with controls for life-long smoking behavior, educational attainment, marital status, and adult health behaviors, foreign-born Hispanics continue to experience a mortality advantage relative to Whites.

Although the NHIS-LMF provides a unique opportunity to examine the effect of early age at smoking onset on adult mortality risk with detailed smoking information and measures for a number of covariates, the current study has two substantial limitations: incomplete smoking

biography and no information on other childhood conditions. Accurate measurement of smoking patterns is difficult given that a person may quit and restart smoking or their smoking frequency can vary over time. A life history calendar approach (Axinn, Pearce and Ghimire 1999) would likely improve measurement of transitions into and out of smoking statuses throughout the life course, but is currently not available for any population-level data sources with mortality follow-up. For example, inclusion of smoking duration into a model containing smoking status, age at initiation, and smoking intensity improves model fit in predicting lung cancer diagnosis (Leffondré et al. 2002). NHIS data also lack measurement of second-hand smoke exposure. Mother's tobacco use or her exposure to her husband's or partner's smoke during pregnancy for the cohorts represented in this data are likely non-trivial; in the National Longitudinal Survey of Youth 1979 (NLSY79) Children and Young Adults study, roughly one third of all mothers smoked during pregnancy (Case and Paxson 2010). Rates of maternal smoking may exceed this amount among earlier birth cohorts, when the dangers of smoke exposure were poorly understood. Since 1998, the Health and Retirement Survey (HRS) includes a measure of parental smoking in addition to age at smoking initiation and adult smoking status. Thus, HRS will allow examination of smoke exposure during the earliest stage of the life course.

The linked lives principle of the life course theoretical framework emphasizes the importance of the family and household context on health behavior (Elder 1998). That is, initiating smoking at an early age may indicate intergenerational transmission of health risk behavior. For instance, low childhood socioeconomic status may lead to a greater likelihood of smoking in adulthood (Hayward and Gorman 2004; van de Mheen et al. 1998), parents expose children to prenatal and postnatal passive (second hand) smoke exposure (Case, Fertig and Paxson 2005), and children of parents who smoke may adopt their parents' behavior (Gilman et

al., 2009). In this way, smoking acts as a mechanism by which children inherit health disadvantages from their parents.

The NHIS was not specifically designed to test the influence of childhood conditions on adult health. The 1997-2004 NHIS respondents did not report information regarding any other childhood conditions except age at smoking initiation. Information on parental education, father's occupation, childhood exposure to infectious disease, farm residence, nutrition, or other childhood health behaviors would allow researchers to determine whether early-life cigarette use has a unique effect on adult mortality that is distinct from the more traditionally studied early-life conditions. However, measurement of age at smoking initiation as an item of a nationally-representative survey assessing population smoking patterns provides the unique opportunity to examine the effect of early-life health behavior on an adult health outcome.

This study reinforces the importance of state- and national-level policies aimed at delaying or eliminating cigarette smoking among children, adolescents, and young adults. Structural barriers to cigarette use, including clean air laws, excise taxes on cigarette packs, counter marketing, marketing and vending machine restrictions, state- and national-level tobacco control agencies, and smoking cessation assistance (i.e., gums, lozenges, helplines, and support groups), limit availability of tobacco products to adolescents and young adults and inhibit genetic tendencies toward smoking behavior (Boardman 2009). The convergence of female smoking patterns toward those of males across birth cohorts shown in the incidence and prevalence analyses highlight an emerging challenge for public health policymakers. Increasing prevalence and earlier initiation among females may reflect increases in social stress experienced by females entering the labor force and balancing work-family obligations (Waldron 1993).

This study indicates a number of potential follow-up studies that would further elucidate the association between life-long cigarette use and adult health. The conceptual frameworks guiding both life course and health behavior research stress examination of mortality risk by cause. Prior studies suggest that individuals who experience adverse childhood circumstances may be particularly vulnerable to respiratory diseases, causes of death with etiologies linked to cigarette use (Blackwell, Hayward and Crimmins 2001; Elo and Preston 1992). Also, international comparisons with nations characterized by greater smoking burdens among men (Russian Federation, Indonesia, China, and Japan) may explain to what extent life-long cigarette use determines observed sex difference in life expectancy (World Health Organization 2009). Reduction in smoking prevalence across U.S. birth cohorts will likely improve the U.S. life expectancy relative to other developed countries.

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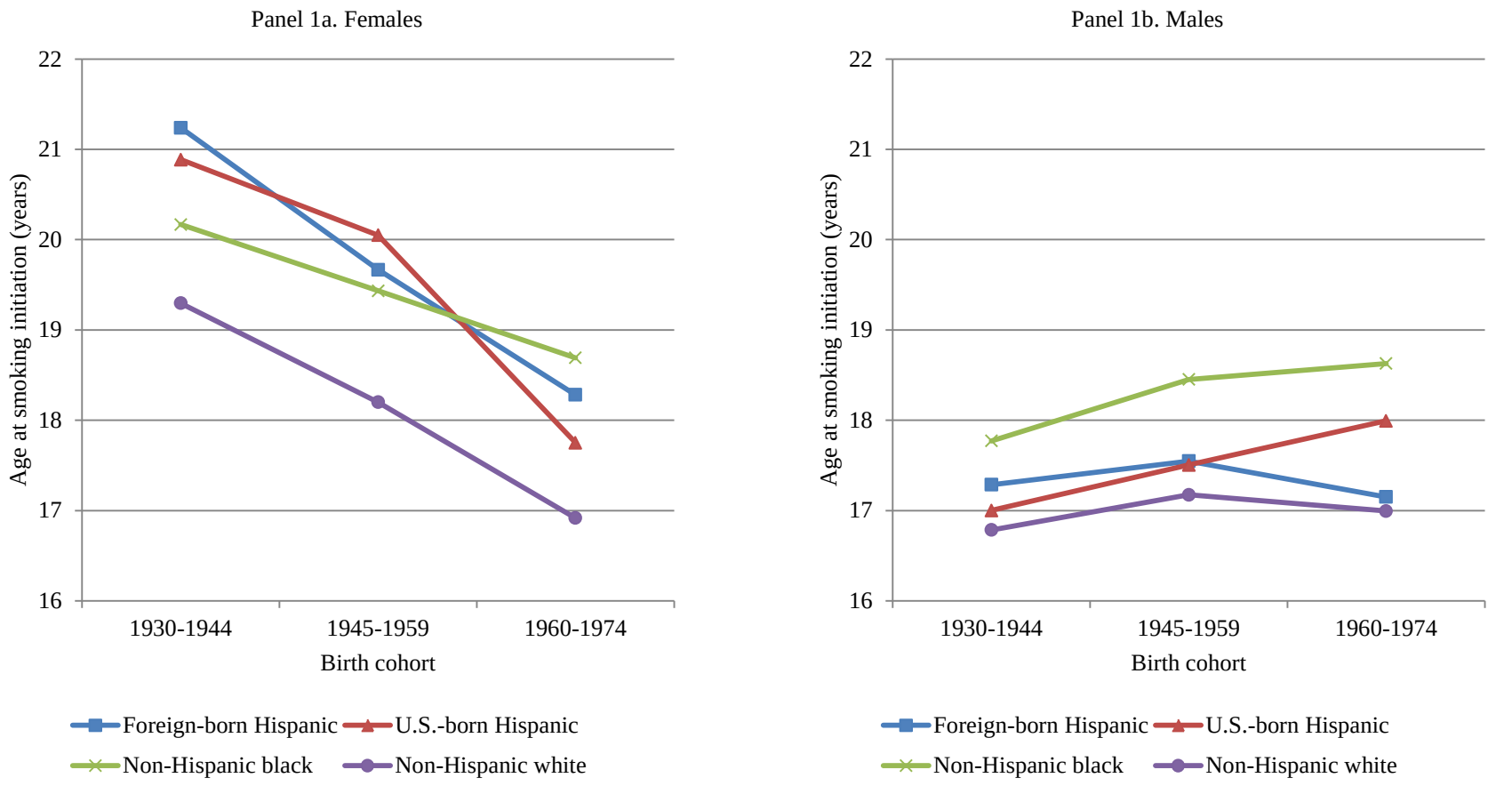
Table 1. Distributions of Smoking Variables and Covariates by Vital Status

	Survivors	Deaths
Age at smoking initiation, mean (SD)[a]	17.7 (4.7)	17.5 (5.2)
Adult smoking status		
Current heavy smokers	11.8	22.2
Current light smokers	12.6	15.9
Former smoker	23.5	32.1
Never smoker	52.5	29.9
Race/ethnicity/nativity		
Foreign-born Hispanic	7.3	4.6
U.S.-born Hispanic	4.2	3.7
Non-Hispanic black	10.8	16.6
Non-Hispanic white	77.7	75.2
Sex		
Female	51.2	40.5
Male	48.8	59.5
Age, mean (SD)	45.1 (11.7)	56.1 (10.4)
Birth cohort		
1930-1944	20.3	59.4
1945-1959	38.8	30.9
1960-1974	40.9	9.8
Educational attainment		
Less than high school diploma	14.3	28.8
High school diploma	30.2	33.6
At least some college	55.4	37.6
Marital status		
Never married	13.5	11.5
Divorced/separated	15.4	21.5
Widowed	3.3	9.9
Married	67.8	57.2
Alcohol use		
Heavy drinker	5.0	7.7
Moderate drinker	62.1	42.1
Former drinker	15.4	30.0
Never drinker	17.5	20.2
BMI		
Underweight	1.3	3.4
Normal weight	36.1	33.5
Overweight	37.3	34.6
Obese	25.2	28.5
Unweighted N	148,191	5,791

Source: 1997-2006 National Health Interview Survey Linked Mortality Files.

[a] Values available for ever-smokers only.

Figure 1. Mean Age at Smoking Initiation by Race/Ethnicity/Nativity and Birth Cohort



Source: 1997-2006 National Health Interview Survey Linked Mortality Files.

Table 2. Percentage Distribution and 95% Confidence Intervals of Adult Smoking Status by Sex, Birth Cohort, and Race/Ethnicity

	Females			Males		
	1930-1944	1945-1959	1960-1974	1930-1944	1945-1959	1960-1974
Foreign-born Hispanic (N)	1,512	2,959	4,787	1,086	2,343	4,040
Current heavy smoker (%)	1.8	2.2	0.7	5.5	5.8	2.8
	(1.2, 2.7)	(1.7, 3.0)	(0.5, 1.1)	(4.3, 6.9)	(4.7, 7.2)	(2.3, 3.4)
Current light smoker (%)	6.3	9.2	7.7	13.1	17.5	19.6
	(5.1, 7.9)	(8.0, 10.5)	(6.9, 8.6)	(10.9, 15.8)	(15.6, 19.5)	(18.3, 21.0)
Former smoker (%)	14.9	11.5	6.3	33.8	23.8	14.6
	(13.0, 17.1)	(10.2, 12.9)	(5.5, 7.2)	(30.9, 36.9)	(21.9, 26.0)	(13.3, 16.0)
Never smoker (%)	77.0	77.1	8.5	47.6	52.9	63.0
	(74.7, 77.0)	(75.1, 79.0)	(84.0, 86.5)	(44.0, 51.3)	(50.4, 55.3)	(61.1, 64.8)
U.S-born Hispanic (N)	1,021	1,912	3,178	762	1,498	2,265
Current heavy smoker (%)	4.8	4.5	3.4	6.0	9.2	6.4
	(3.2, 7.2)	(3.5, 5.8)	(2.8, 4.3)	(4.1, 8.7)	(7.8, 10.9)	(5.3, 7.7)
Current light smoker (%)	10.3	15.9	16.4	15.2	19.1	21.3
	(8.2, 12.8)	(14.1, 17.9)	(14.9, 18.0)	(12.4, 18.5)	(16.8, 21.5)	(19.5, 23.3)
Former smoker (%)	21.5	17.7	11.6	46.1	25.5	13.2
	(18.4, 24.9)	(15.7, 19.8)	(10.2, 13.2)	(42.0, 50.3)	(22.9, 28.2)	(11.7, 14.8)
Never smoker (%)	63.4	61.9	68.6	32.7	46.3	59.1
	(59.4, 67.3)	(59.2, 64.5)	(66.4, 70.7)	(28.8, 36.9)	(43.4, 49.2)	(56.8, 61.4)
Non-Hispanic black (N)	2,737	4,656	5,546	1,793	3,117	3,211
Current heavy smoker (%)	4.7	6.6	4.5	11.0	12.3	7.4
	(3.9, 5.7)	(5.9, 7.4)	(3.9, 5.2)	(9.5, 12.8)	(11.0, 13.6)	(6.6, 8.4)
Current light smoker (%)	14.5	22.6	18.3	18.3	24.4	21.5
	(12.9, 16.2)	(20.9, 24.3)	(17.0, 19.8)	(16.4, 20.3)	(22.8, 26.0)	(20.0, 23.1)
Former smoker (%)	24.8	15.6	7.2	40.1	23.0	8.4
	(23.0, 26.7)	(14.4, 16.9)	(6.4, 8.1)	(37.5, 42.8)	(21.3, 24.8)	(7.3, 9.6)
Never smoker (%)	56.1	55.2	70.0	30.6	40.4	62.7
	(53.6, 58.5)	(53.2, 57.2)	(68.2, 71.7)	(28.1, 33.2)	(38.1, 42.7)	(60.8, 64.6)
Non-Hispanic white (N)	14,428	21,169	20,633	11,831	19,084	18,414
Current heavy smoker (%)	9.1	12.9	12.1	12.2	18.7	16.7
	(8.6, 9.7)	(12.4, 13.4)	(11.5, 12.7)	(11.6, 12.9)	(18.0, 19.3)	(16.0, 17.4)
Current light smoker (%)	9.2	11.5	16.0	6.8	9.2	12.9
	(8.7, 9.8)	(11.0, 12.0)	(15.4, 16.6)	(6.2, 7.3)	(8.8, 9.7)	(12.4, 13.5)
Former smoker (%)	30.9	23.8	16.5	51.0	30.2	15.4
	(30.1, 31.7)	(23.1, 24.5)	(15.9, 17.1)	(49.9, 52.0)	(29.4, 30.9)	(14.8, 16.0)
Never smoker (%)	50.8	51.8	55.5	30.1	42.0	55.0
	(49.8, 51.7)	(51.0, 52.6)	(54.6, 56.3)	(29.2, 31.0)	(41.1, 42.9)	(54.2, 55.9)

Source: 1997-2006 National Health Interview Survey Linked Mortality Files.

Note: N's are unweighted. Percentages and 95% confidence intervals are weighted.

Table 3. Hazard Ratios from Cox Proportional Hazard Regression of Mortality Risk on Smoking History and Race/ethnicity/nativity

	Total sample				Never smokers	
Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Smoking history (ref=Never smokers)						
Current heavy smokers	2.52***		2.59***	2.23***		
Current heavy smokers * early initiation	1.33***		1.35***	1.25***		
Current light smokers	2.15***		2.01***	1.92***		
Current light smokers * early initiation	1.30***		1.32***	1.18**		
Former smokers	1.33***		1.36***	1.43***		
Former smokers * early initiation	1.08		1.09	0.99		
Race/ethnicity/nativity (ref=Non-Hispanic white)						
Foreign-born Hispanic		0.92	1.05	0.87**	1.28**	0.97
U.S.-born Hispanic		1.18**	1.25**	1.10	1.53**	1.25
Non-Hispanic black		1.90***	1.91***	1.48***	2.20***	1.61***
Male (ref=Female)	1.47***	1.63***	1.47***	1.70***	1.60***	1.81***
Age (50+)	1.09***	1.09***	1.10***	1.09***	1.09***	1.08***
Birth cohort (ref=Born 1960-1974)						
Born 1930-1944	0.92	1.03	0.91	0.99	1.07	1.12
Born 1945-1959	0.92	1.01	0.92	1.02	1.10	1.19
Educational attainment (ref=High school diploma)						
Less than high school diploma				1.25***		1.24**
At least some college				0.84***		0.75***
Marital status (ref=married)						
Never married				1.74***		1.81***
Divorced/separated				1.48***		1.42***
Widowed				1.43***		1.34**
Alcohol use (ref=Never drinker)						
Heavy drinker				1.47***		1.53**
Former drinker				1.72***		1.72***
Never drinker				1.49***		1.44***
BMI (ref=Normal weight)						
Obese				1.04		1.99**
Overweight				0.85***		0.98
Underweight				2.39***		1.36***
-2*Log-likelihood	115284.7	115,926.9	115,027.9	114,146.8	32,553.4	32,270.3

Source: 1997-2006 National Health Interview Survey Linked Mortality Files.
Note: *** p < .001; ** p < .05; * p < .10