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Impact of declining exposure to secondhand tobacco smoke in public places to decreasing smoking-related cancer mortality in the US population

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ABSTRACT

Background: The major decrease in exposure to secondhand smoke (SHS) in public places in recent decades could have contributed to the decline in smoking-related cancer mortality observed in the US population.
Methods: Prospective study among 11,856 non-smoking adults aged ≥40 years who participated in NHANES 1988–1994 or 1999–2004 and were followed for mortality through 2006. We estimated the amount of change in cancer mortality over time attributed to the intermediate pathway of changes in SHS exposure in public places, after adjustment for risk factors and SHS exposure at home.
Results: The adjusted smoking-related cancer mortality rate ratios (95% CI) for a two-fold increase in serum cotinine and a 1-hour increase in occupational SHS exposure time were 1.10 (1.03, 1.17) and 1.14 (1.06, 1.24) for all-cancer, and 1.13 (1.03, 1.24) and 1.14 (1.02, 1.26) for smoking-related cancer, respectively. The absolute reduction in mortality comparing 1999–2004 to 1988–1994 was 75.8 (−25.5, 177.0) and 77.0 (2.6, 151.4) deaths/100,000 person-years, for all-cancer and smoking-related cancer, respectively. Among these avoided all-cancer deaths, 45.8 (2.8, 89.5) and 18.1 (−1.2, 39.6)/100,000 person-year were attributable to changes in serum cotinine concentrations and occupational SHS exposure time, respectively. The corresponding numbers of smoking-related cancer avoided deaths were 36.4 (0.7, 72.8) and 9.9 (−3.8, 24.9)/100,000 person-year.
Conclusions: Declines in SHS exposure were associated with reductions in all-cancer and smoking-related cancer mortality, supporting that smoking bans in public places may have reduced cancer mortality among non-smoking adults.

1. Introduction

Secondhand tobacco smoke (SHS), the combination of the sidestream smoke emitted from the burning end of a tobacco product and the mainstream smoke exhaled by the smoker, contains > 6000 chemicals and is carcinogenic to humans (International Agency for Research on Cancer (IARC), 2012). The 2006 Surgeon General's Report on the Health Consequences of Involuntary Exposure to Tobacco Smoke concluded evidence is sufficient to infer that SHS exposure causes lung cancer (Moritsugu, 2007). Other health effects causally linked to SHS exposure include ischemic heart disease and stroke in adults, and lower respiratory tract infections and impaired lung function in children (Alberg et al., 2014). For many other diseases, there is suggestive evidence for causality (Moritsugu, 2007).

Exposure to SHS can be assessed by interviews or by measuring the levels of cotinine (a nicotine by-product in the body) in biological samples. According to self-reported information and serum cotinine concentrations, exposure to SHS in the United States has declined intensely during the past three decades (Disparities in secondhand smoke exposure–United States, 2008). This phenomenon has been attributed to downward trends in smoking rates and intensity, and to increases in the number of states with smoke-free policies in public places. As of June 30, 2016, a total of 29 US states and the District of Columbia have enacted statewide bans on smoking in all public places and workplaces,

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including bars, restaurants and private worksites (CDC (Centers for Disease Control and Prevention)).

Cancer death rates in the US, including those that arise at the most common sites (i.e. lung and bronchus, colon and rectum, breast or prostate), have also fallen during the last decades, with an estimated overall decrease of 23% from 1990 to 2012 (Howlader et al., 2016). This decline has been attributed to reductions in tobacco consumption, as well as to better surveillance and treatment options. Little is known, however, about the potential contribution of reductions in SHS exposure to the decline of cancer mortality among nonsmokers. Most studies on the health benefits of smoke-free policies have focused on acute cardiovascular and respiratory effects (Tan & Glantz, 2012; Lin et al., 2013), and on birth outcomes (Peelen et al., 2016). Moreover, a recently published Cochrane review reported evidence of reduced cardiovascular and respiratory mortality after introduction of national smoke-free bans (Frazer et al., 2016). Recently, though, an ecological study within EU countries reported no statistically significant changes in lung cancer mortality trends after the introduction of smoke-free legislation (Lopez-Campos et al., 2017). Although SHS is known to be carcinogenic (International Agency for Research on Cancer (IARC), 2012), the long latency period that is required for most cancers to develop makes it difficult to link changes in SHS exposure to changes in cancer rates.

The objective of this study was to evaluate the hypothesis that population changes in the distribution of SHS exposure in public places explain changes in smoking-related cancer mortality over time in two samples of the non-smoking US population, the U.S. National Health and Nutrition Examination Survey (NHANES) 1988–1994 and 1999–2004. For this purpose, we used individual information on serum cotinine and made adjustments for SHS exposure at home, so that our analyses reflect SHS exposure not coming from private settings. Moreover, to differentiate between occupational SHS exposure and SHS exposure from other public places, we also evaluated self-reported occupational SHS exposure time (hours/day). To address the study objectives, we implemented a causal inference mediation approach (Lange & Hansen, 2011) using study period as a major determinant of cancer mortality and changes in SHS exposure as a potential mediator of the relation between period and cancer mortality (see Supplemental Fig. S1). This method allows us to estimate the proportion of the decline in smoking-related cancer mortality rates among nonsmokers recruited in 1988–1994 and 1999–2004 that can be independently attributed to changes in SHS exposure.

2. Methods

2.1. Study population

NHANES is a program of studies designed to assess the health and nutritional status of the general population in the United States. The survey uses a complex multistage sampling design to obtain representative samples of the non-institutionalized US population (Centers for Disease Control and Prevention (CDC)), and it is unique in that it combines interviews and physical examinations. Blood and urine specimens are obtained from NHANES participants who give consent for their specimens to be used in future research studies. Serum specimens are available from NHANES III (1988–1994) up to the present time (Centers for Disease Control and Prevention (CDC)).

In this study we used data from NHANES III and NHANES 1999–2004. Because each survey period provides a snapshot of the US population health status, comparing these two periods we can assess how population changes over time in SHS exposure can explain temporal trends in cancer mortality. We initially included 15,973 non-smoking adults (i.e. both never and former smokers) aged ≥40 who completed the NHANES clinical examination. Because self-reported non-smokers or former smokers with serum cotinine levels above 10 ng/mL were considered current smokers, these were not included in the analyses. From these, we excluded pregnant women ($n = 4$ in NHANES 1988–1994 and $n = 7$ in NHANES 1999–2004), as well as participants with missing values in serum cotinine ($n = 1183$ and 1190, respectively), occupational SHS exposure ($n = 63$ and n = 1, respectively) or SHS exposure at home ($n = 9$ and $n = 58$, respectively). Supplemental Fig. 2 shows the flow chart of participant exclusions by study period, which left 11,856 participants for analysis.

2.2. Secondhand tobacco smoke measures

Serum cotinine was measured by an isotope-dilution high-performance liquid chromatography/atmospheric pressure chemical ionization tandem mass spectrometric method at the Division of Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention (CDC). The limits of detection (LOD) for serum cotinine were 0.05 ng/mL in NHANES III and 0.015 ng/mL in NHANES 1999–2004. For the 11% and 13% of participants < LOD, respectively, cotinine concentrations were replaced by the LOD divided by the √2 (Hornung & Reed, 1990).

Exposure at home was defined as the presence of one or more smokers at home. Occupational SHS exposure time was defined as the self-reported daily number of hours of exposure to SHS at the workplace on the basis of the participant's main paid job within the last week before the interview. The following questions were asked: 1) “At work, how many hours per day are you close enough to people who smoke so that you can smell the smoke?” in NHANES III, and 2) “At this job or business, how many hours per day can you smell the smoke from other people's cigarettes, cigars and/or pipes?” in NHANES 1999–2004. Individuals who were not working 2 weeks before the interview ($N = 3533$ in NHANES III and $N = 3051$ in NHANES 1999–2004) were considered unexposed at work.

2.3. Mortality risk factors

NHANES collected information on age, sex, race/ethnicity (Non-Hispanic White (“White”), Non-Hispanic Black (“Black”), Mexican-American, other), education (≥high school, < high school), smoking status (never, former smokers), body mass index (< 30, ≥30 kg/m (Moritsugu, 2007)), alcohol consumption (never, former, current drinkers), physical inactivity (no, yes), and exposure to SHS at home (no, yes). Former smokers were participants who had smoked ≥100 cigarettes in life but were not current smokers (i.e. did not answered yes to the question “Do you smoke cigarettes now?” nor had serum cotinine levels > 10 ng/mL) (Centers for Disease Control and Prevention (CDC)). According to their alcohol intake, participants were classified as never (< 12 drinks in life), former (at least 12 drinks in life but < 12 drinks during the previous year), or current drinkers (at least 12 drinks during the previous year). Leisure time physical activity questions included information on the type and frequency of physically active hobbies, sports, or exercises. Four open-ended questions assessed information on physical activities not previously listed. Participants who responded “no” to all leisure time physical activity questions were classified as physically inactive. Obesity was defined as a body mass index (BMI) ≥30 kg/m.

2.4. Mortality follow-up

Cause-specific cancer mortality follow-up data in NHANES 1988–1994 and 1999–2004 was publicly available through December 31, 2006. Vital status and cause of death were determined by probabilistic matching between NHANES records and death certificates from the National Death Index (NDI) based on identifying data elements (Centers for Disease Control and Prevention (CDC)). Cause of death was determined using the underlying cause listed on death certificates, and was coded using the *International Classification of Diseases, 10th Revision* (ICD-10) (World Health Organization, 2014). The primary study