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Tobacco smoke overload and ethnic, state, gender, and temporal cancer mortality disparities in Asian-Americans and Pacific Islander-Americans

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Abstract

Background. Asians and Pacific Islanders (APIs) are important populations nationally and globally. So we assessed cumulative tobacco smoke overexposure (smoke overload)/cancer mortality associations across states, ethnicities, years, and genders among API-Americans.

Methods. Death rates were adjusted to the 2000 United States age standard, lung cancer death rates used as a smoke overload bio-index, and lung/non-lung cancer death rate linear regressions run. Cancer death rate smoking-attributable fractions (SAFs) are equal to 1 − estimated unexposed rate/observed rate.

Results. The two lowest smoke overload and non-lung cancer death rates were in South Asian (Indo)-Californian females and males. The highest were in Korean-Californian males. Non-lung cancer death rates were tightly and steeply associated with smoke overload across ethnicity, state, year, or gender. Cancer death rate smoking-attributable fractions ranged from 0 in female and 6% in male Indo-Californians, to 39% in female and 57% in male API-Americans in 2002, to 71% in Korean-Californian and 69% in API Hawaiian males.

Discussion. Many API American cancer death rate disparities across genders, ethnicities, states, or years can be explained by smoke overload disparities. Tobacco control may greatly reduce cancer death rates and disparities among API-Americans and, likely, others.
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Keywords: Smoking; Asian-Americans; State governments; Time factors; Sex characteristics; California; Hawaii; New York; Illinois; New Jersey; Gender; Ethnicity

Introduction

Asians and Pacific Islanders are a large, heterogeneous population whose exposure/cancer associations merit more study. Asians and Pacific Islanders represent over 60% of the global and 4% of the United States (US) populations (Ezzati et al., 2005; SEER∗Stat Database, 2005). Cancer is the leading cause of death among API-Americans, despite their wide, unexplained all-sites cancer death rate disparities across gender, ethnicity, place, and time (SEER∗Stat Database, 2005). For example, in 1997–2001, API-Californian all-sites cancer death age-adjusted rates (rates)/100,000 ranged nearly four-fold from 57.8 in South Asian (Indo)-Californian females to 203.4 in Korean-Californian males (Kwong, 2004).

Cumulative tobacco smoke overexposure (smoke overload) disparities can account for most temporal and regional cancer mortality variation in African-American males (Leistikow and Tsodikov, 2005), but have not been studied in Asian populations. So we assessed smoke overload/cancer death rate associations and cancer death smoking-attributable fractions (SAFs) in API-Americans stratified by time, place, ethnicity, and gender.

Methods

We used published 1980, 1990–2002 (Health, United States, 2004, 2005; SEER∗Stat Database, 2005), and 1997–2001 lung and all-sites cancer death rates adjusted to the 2000 US age standard (Kwong, 2004; Kwong et al., 2005). Subtracting lung cancer rates from all-sites rates gave us "non-lung" rates.

Lung cancer death rates were used as a smoke overload bio-index (Leistikow and Tsodikov, 2005). Given many states' high API death rate standard errors, we limited state analyses to the 5 lowest lung cancer death rate standard error states across all the available data (1990–2002) (SEER∗Stat Database, 2005). Those 5 states (Table 1) had 63% of total US API 1990–2002 person-years (SEER∗Stat Database, 2005).

Lung/non-lung cancer rate linear regressions were run. SAFs were calculated using the formula SAF = 1 − (estimated unexposed cancer death rate) / (observed rate), based on the linear regression and respective best, upper, and lower bound estimated lung cancer rates in the unexposed of 3, 5 (the rate

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seen in Indo-Californian women in 1997–2001), and 2/100,000 (a rate seen in Japanese women in the early 1950s). Those unexposed rate estimates are slightly lower than the rates of 4, 7, and 3 used in past African-American male SAF estimates (Leistikow and Tsodikov, 2005). Lower and upper 95% confidence interval (CI) limit values for the slope and intercept were used in calculating the respective upper and lower bound SAFs. The resulting negative SAF best estimate in Indo-Californian women was changed to zero, since smoking is not known to protect people from cancer.

Results

The first, second, and third highest all-sites cancer death rates and SAFs studied were in 1997–2001 Korean-Californian, 1990–2002 API-Hawaiian, and 1997–2001 Japanese-Californian males, respectively (Table 1). The first, second, and third lowest all-sites cancer death rates and SAFs were in Indo-Californian females and males in 1997–2001, and female API-Americans in New Jersey in 1990–2002, respectively.

Lung and non-lung cancer death rates were tightly, steeply, and significantly associated across each stratification studied except within US API female annual data. That includes across year, ethnic, or place-specific data in males (Fig. 1), in females (Fig. 2), and in the combined above-mentioned male and female points ($P < 0.02$ and $R^2 > 0.68$ for each gender-stratification-specific association except US female annual data ($P = 0.24$)). The lung/non-lung cancer death rate slope across all points studied was 1.57 (95% CI 1.41–1.73), $R^2 = 0.89$. The regression line formula was non-lung rate = 1.57 * lung rate + 51.6.

Based on the above regression line, estimated all-sites cancer death rate SAFs were 57% for API males and 37% for API females nationally in 2002, and ranged from 0 and 6% in South Asian females and males in 1997–2001, respectively, to 71% in Korean-Californian males in 1997–2001 and 69% in API-Hawaiians in 1990–2002 (Table 1).

Discussion

The observed strong API lung/non-lung cancer death rate associations across diverse stratifications and death rates suggest that the same thing, most likely tobacco smoke overload, may cause most prematurely fatal cancers in API-American males, and many such female deaths. These API population-level associations, and the increasing frequency of lung and non-lung cancers in the same API individual (Liu et al., 2002), resemble the Kaposi's sarcoma/pneumocystosis associations seen in the early 1980s US HIV epidemic (Gottlieb et al., 1983; Michalany et al., 1987). High HIV loads were eventually shown to have caused the co-occurrence of Kaposi's sarcoma and pneumocystosis in individuals and populations (Schneider et al., 2005).

The present associations suggest high (25%–71%) SAFs of all-sites cancer death rates in each group studied except Indo-Californians. Even API-Californian females who averaged a 7% phone self-reported smoking prevalence in the 1990s (Adult Smoking, 2005) had an estimated 44% cancer death rate SAF for 1990–2002, perhaps due to known mis-sampling or mis-reporting in prevalence surveys and/or heavy secondhand smoke exposures among API females (Tobacco Use among U.S. Racial/Ethnic Minority Groups: A Report of the Surgeon General, 1998).

The lung/non-lung cancer associations suggest diverse smoking-attributable fractions for the different subgroups studied (Table 1). This study provides the first published evidence of lung/non-lung cancer death associations in females, across states, across Chinese-, Indian-, Japanese-, Korean-, Filipino-, Vietnamese-, or API-Americans, or in Asians.

This study has strengths. Strong, consistent, positive smoke overload/cancer death associations were seen within and across four distinct stratifications—time, ethnicity, place, and gender. Smoke overload variation provides a more parsimonious and empirically supported (Leistikow and Tsodikov, 2005) explanation for the disparities seen than would arguments that ethnic mix determines the state and temporal differences while Y-chromosome-linked genetic disparities determine the gender disparities. The US API time-series covered both cancer death rate rises from 1980, falls in the latter 1990s, and is consistent with findings from 1950 to 2001 in African-American males (Leistikow and Tsodikov, 2005). At least the epidemic rises in Filipina- and Korean-Californian female and possibly Indo-Californian male lung cancer death rates (Cockburn et al., 2004) and rises and falls in African-American cancer death rates and disparities are not consistent with solely genetic explanations. The great majority, or in the time-series, all, of API-Americans were studied (Watanabe and Wride, 2004). API-American groups differing in ethnicity, socioeconomic status, nativity, age, etc., were studied. Our recent, representative data include multiple stratifications with comprehensive smoke overload assessment concurrent with outcome assessment. This contrast with most cohort studies' outdated, unrepresentative (healthy volunteer; Froom et al., 1999; Taylor et al., 2002) data and poor, point exposure measures (often one-time smoking self-report) (The World Health Report, 2002) assessed sometimes decades before outcomes occur (Rockhill, 2005). The lung/non-lung cancer associations seen are roughly consistent with time-series and cross-region associations seen in US African-American males (Leistikow and Tsodikov, 2005).

Theories suggesting that parallel increases in detection of both lung and non-lung cancer deaths account for the epidemics seen are severely flawed. Such theories are inconsistent with at least: (1) 1990s cancer death rates declines; (2) strong dose–response smoker lung and non-lung cancer death relative risks in cohorts with concurrent, careful cause of death classification; and (3) over 40 years of expert conclusions that smoke exposure epidemics cause lung cancer epidemics (Samet et al., 2004).

This study has at least 6 weaknesses. (1) Extrapolation from population-level associations like these has produced inaccurate individual relative risk estimates (the ecologic fallacy). (2) Lower numbers of lung cancer deaths in Indo-Californians in particular and female Asian-Californians in general likely caused wider rate CIs than in the other groups studied. Those CIs are neither available nor well reflected in the regression slopes, SAFs, and their SRs. (3) US API death rates for 1996 were estimated to be too low by 11% (Rosenberg et al., 1999).

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