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Rising incidence of oral tongue cancer among white men and women in the United States, 1973–2012

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ABSTRACT

Background: Despite significant reductions in tobacco use in the US, oral tongue cancer incidence has reportedly increased in recent years, particularly in young white women. We conducted age-period-cohort analyses to identify birth cohorts that have experienced increased oral tongue cancer incidence, and compared these with trends for oropharyngeal cancer, a cancer caused by human papillomavirus (HPV) that has also recently increased.

Methods: We utilized cancer incidence data (1973–2012) from 18 registries maintained by the NCI SEER Program. Incidence trends were evaluated using log-linear joinpoint regression and age-period-cohort modeling was utilized to simultaneously evaluate effects of age, calendar year, and birth year on incidence trends.

Results: Incidence of oral tongue cancer increased significantly among white women during 1973–2012 (0.6% annual increase, $p < 0.001$) and white men during 2008–2012 (5.1% annual increase, $p = 0.004$). The increase was most apparent among younger white individuals (<50 years; annual increase of 0.7% for men [$p = 0.02$] and 1.7% for women [$p < 0.001$] during 1973–2012). Furthermore, the magnitude of the increase during 1973–2012 was similar between young white men and women (2.3 vs. 1.8 cases per million, respectively). Incidence trends for oropharyngeal cancer were similar to trends for oral tongue cancer and similar birth cohorts (born after the 1940s) experienced rising incidence of these cancers (p-value: white men = 0.12, white women = 0.42), although the magnitude of increase was greater for oropharyngeal cancer.

Conclusions: The incidence of oral tongue and oropharyngeal cancer has significantly increased among young white men and women within the same birth cohorts in the US.

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Introduction

Cancers of the oral cavity and pharynx have traditionally been considered a single etiologic entity. However, recent studies show significant differences in the etiology and epidemiology for individual anatomic sites [1–3]. Reduction in smoking in the United States has resulted in significant declines in the incidence of most oral cavity cancers, including lip, gum, floor of mouth, hard palate, buccal mucosa, and vestibule cancers [4–8]. In contrast, the incidence of oral tongue cancers (anterior 2/3 of the tongue) has reportedly increased in recent years [7–12]. The incidence of oropharyngeal cancers has also increased, and descriptive and molecular epidemiologic studies have identified human papillomavirus (HPV) infection as the cause [2,3]. However, the reasons for the increase in oral tongue cancers are unknown, and molecular studies indicate that HPV does not play a major etiologic role [13–16].

Descriptive studies of oral tongue cancer trends have reported that incidence has significantly increased among young (ages 18–44 years), white individuals, and primarily in women [7,8,10–12]. It is, however, unclear whether oral tongue cancer incidence has increased in similar magnitude among young, white men [7–12]. Furthermore, prior studies have not identified the specific

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birth cohorts that have experienced an increase in oral tongue cancer incidence, which could provide important etiologic clues. For example, the identification of birth cohorts of men that went through the sexual revolution as the primary demographic subgroup that has experienced the rise in oropharyngeal cancer incidence, in part, enabled the identification of HPV as the cause [2,3,5,17,18].

The main objective of this study was to identify the demographic subgroups and birth cohorts that have experienced an increase in oral tongue cancer in the United States. We also aimed to compare and contrast our findings for oral tongue cancer with oropharyngeal cancer to determine if similar birth cohorts have experienced an increase in these cancers. This comparison may provide clues regarding the possible role of a sexually transmitted infection or other environmental exposure in the development of oral tongue cancer.

Methods

Data source

Cancer incidence information from the US National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program database were obtained for the years 1973–2012 [19–21]. We included data from the SEER 9 Registries Database [19] for the years 1973–1991 (Atlanta, Connecticut, Detroit, Hawaii, Iowa, New Mexico, San Francisco-Oakland, Seattle-Puget Sound, and Utah), SEER 13 Registries Database [20] for the years 1992–1999 (SEER 9 regions plus Los Angeles, San-Jose Monterey, Rural Georgia, and Alaska Native Tumor Registry), and the SEER 18 Registries Database [21] for the years 2000–2012 (SEER 13 regions plus Greater California, Kentucky, Louisiana, New Jersey, and Greater Georgia), covering 14%, 17%, and 28% of the US population, respectively.

Classification of anatomic sites

For all analyses, oral cavity cancer sites were subdivided into two groups: oral tongue cancer, including the dorsal surface, border, ventral surface, and anterior 2/3 of the tongue (International Classification of Diseases for Oncology version-3 [ICD-O-3] topography codes C020-023) and other oral cavity cancer sites, including lip, gum, floor of mouth, and other/unspecified parts of the mouth (ICD-O-3 codes C000-009, C030-039, C040-050, and C060-069). HPV-associated oropharyngeal cancers were also evaluated, and these sites included base of tongue, lingual tonsil, soft palate and uvula, tonsil, oropharynx, and Waldeyer ring (ICD-O-3 codes C019, C024, C051-052, C090-099, C100-109, and C142) [5]. Cancers classified as tongue, not otherwise specified (ICD-O-3 code: C028) were excluded due to potential ambiguity regarding origin within the oral tongue vs. base of tongue. All histologic subtypes were included in our primary analysis for each cancer site. This study did not use personal identifying information from the SEER data or involve interaction with human subjects, therefore informed consent and institutional review was not required.

Statistical analysis

All analyses were conducted separately by sex and race: white (both non-Hispanic and Hispanic), black, and other races (American Indians and Alaska Natives, Asian/Pacific Islanders, and unknown). For each site, annual incidence rates (1973–2012; age-standardized to the US 2000 standard population) were obtained using SEER*Stat version 8.2.1 software. Temporal trends were evaluated using log-linear joinpoint regression, incorporating cubic regression splines selected based on the Akaike information criterion. Trends are expressed as the annual percentage change (APC) in incidence [22]. For years with zero cases for a particular stratum, the annual incidence rate and standard error were taken from the preceding year, or closest year (preceding or following) when there were consecutive years with zero cases. In addition to presenting the APC (a measure of relative change in incidence), we also calculated the incidence rate difference (a measure of the absolute change in incidence) between 1973 and 2012, and difference in the average incidence rate between late (1973–77) and recent (2008–12) time periods.

To simultaneously evaluate the effects of age, calendar year/period, and birth year/cohort on incidence rates, age-period-cohort modeling was applied using 5-year age groups (20 to 24, ..., 80 to 84), 5-year calendar periods (1973 to 1977, ..., 2008 to 2012), and 5-year birth cohorts (1893, ..., 1988). These models may be useful for detecting changes in exposure experiences (indicated by cohort effects), or changes in reporting, coding, screening and/or diagnostic practices (indicated by period effects) [23–25]. We specifically focused on two aspects of birth cohort effects in these models – cohort deviations and age-specific temporal trends (or net drifts) – that can enable identification of birth cohorts and age groups that have experienced significant changes in incidence rates. Differences in age-specific net drifts and cohort deviations were compared across cancer sites for the same sex (oral tongue cancer vs. oropharyngeal cancer and oral tongue cancer vs. other oral cavity cancer) and across sex for the same site using a 1-*df* Wald test. For oral tongue cancer and oropharyngeal cancer, temporal trends were then reevaluated focusing on young and older age groups separately, with the age cut point identified based on the observed age-specific net drifts.

Given the relative rarity of oral tongue cancers, we combined the three SEER registries for enhanced statistical power. Nonetheless, to ensure robustness of our results as well as potential comparability of incidence rates across the SEER databases, we conducted two sensitivity analyses. First, we reevaluated all incidence trends using SEER 9 registries. Second, for overlapping calendar years (2000–2012), we compared incidence trends for oral tongue and oropharyngeal cancers across SEER 9, 13, and 18. We also conducted sensitivity analyses for oral tongue cancers restricted to squamous cell histologies.

Table 1
Characteristics of cases of oral tongue, oropharyngeal, and other oral cavity cancers during 1973–2012.

Patient characteristic	Oral tongue (n = 16,206) no. (%)	Oropharyngeal (n = 67,789) no. (%)	Other oral cavity (n = 56,168) no. (%)
Age at diagnosis, years[a]			
Mean	60.2	60.0	63.0
Standard deviation	13.5	11.5	13.1
Sex			
Male	9483 (58.5)	50,748 (74.9)	37,074 (66.0)
Female	6723 (41.5)	17,041 (25.1)	19,094 (34.0)
Race/ethnicity			
White	13,772 (85.0)	56,977 (84.0)	49,556 (88.2)
Black	1014 (6.2)	7858 (11.6)	3867 (6.9)
Other	1292 (8.0)	2611 (3.9)	2117 (3.8)
Unknown	128 (0.8)	343 (0.5)	628 (1.1)
Year of diagnosis			
1973–1982	1742 (10.7)	5906 (8.7)	9360 (16.7)
1983–1992	2043 (12.6)	7571 (11.2)	9944 (17.7)
1993–2002	4400 (27.2)	16,363 (24.1)	15,170 (27.0)
2003–2012	8021 (50.5)	37,949 (56.0)	21,694 (38.6)

[a] Restricted to individuals aged 0–84 years at time of cancer diagnosis (oral tongue: n = 15,350; oropharyngeal: n = 65,923; other oral cavity: n = 51,524).

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