



Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology

ORAL AND MAXILLOFACIAL PATHOLOGY Editor: Mark W. Lingen

Oral squamous cell carcinoma incidence by subsite among diverse racial and ethnic populations in California

Lihua Liu, PhD,^a Satish K. S. Kumar, BDS, MDS,^b Parish P. Sedghizadeh, DDS, MS,^c Abheer N. Jayakar, BDS,^d and Charles F. Shuler, DMD, PhD,^e Los Angeles, CA
UNIVERSITY OF SOUTHERN CALIFORNIA, LOS ANGELES

Objective. The aim of this report was to examine the oral cancer incidence by sex, race/ethnicity, and anatomical subsite. **Study design.** Data from the California Cancer Registry (CCR) were used to calculate the age-adjusted incidence rates of invasive oral squamous cell carcinoma (OSCC) by sex, race/ethnicity, and anatomical subsite among residents in California during 1988 to 2001.

Results. Although non-Hispanic (NH) black men have the highest overall incidence rate for OSCC, NH whites and NH blacks have similar incidence patterns by subsite, but the male-to-female (M:F) rate ratio is higher among NH blacks. The OSCC incidence rates for Hispanics are much lower than those for NH whites and NH blacks and similar to those of Asians. The Asian ethnic groups display dramatic variations in terms of the subsite-specific incidence rates and M:F rate ratios.

Conclusion. The findings illustrate the heterogeneity and complexity of oral cancer by anatomical location and the importance of cultural habits and behavioral factors in the development of oral cancer. (**Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105:470-80**)

Cancer of the oral cavity is an important global health concern accounting for an estimated 275 000 cases and 128 000 deaths annually.¹ Its incidence varies markedly by geographic region and occurs more frequently among men than women. Two thirds of all cases are observed in developing countries. The Indian subcontinent accounts for one third of the global burden. The striking geographic and ethnic variations in the inci-

dence of oral cancer underline the importance of studying the disease by race/ethnicity. Epidemiological investigations describing the distribution of disease between different populations are crucial in evaluating etiology, pathogenesis, and treatment so as to design possible preventive measures, screening, and early detection and implementation of relevant health policies.²

Cancer incidence data used in this report have been collected by the California Cancer Registry of the California Department of Health Services as part of the statewide cancer-reporting program mandated by California Health and Safety Code Section 210 and 211.3.

Support has been provided by the Division of Cancer Prevention and Control, National Cancer Institute, U.S. Department of Health and Human Services, under contract N02-PC-15105, the Centers for Disease Control and Prevention National Program of Cancer Registries under contract U75/CCU910677, the Tobacco Tax and Health Promotion Act of 1988, and the Breast Cancer Act of 1993.

The ideas and opinions expressed herein are those of the authors and endorsement by the State of California, Department of Health Services, the National Cancer Institute, and the Centers for Disease Control and Prevention or their contractors and subcontractors is not intended nor should be inferred.

^aResearch Scientist, Los Angeles Cancer Surveillance Program, Department of Preventive Medicine, Keck School of Medicine, University of Southern California.

^bSenior Resident, Orofacial Pain and Oral Medicine Center, Division of Diagnostic Sciences, School of Dentistry, University of Southern California.

^cAssistant Clinical Professor, Division of Diagnostic Sciences and Center for Craniofacial Molecular Biology, School of Dentistry, University of Southern California.

^dDental Student, School of Dentistry, University of Southern California.

^eDirector, Center for Craniofacial Molecular Biology, George and Mary Lou Boone Chair of Craniofacial Molecular Biology, School of Dentistry, University of Southern California.

Received for publication Feb 6, 2007; returned for revision Jun 21, 2007; accepted for publication Jul 3, 2007.

1079-2104/\$ - see front matter

© 2008 Mosby, Inc. All rights reserved.

doi:10.1016/j.tripleo.2007.07.007

Oral squamous cell carcinoma (OSCC) constitutes approximately 94% of all malignant lesions in the oral cavity,³ and because of this great dominance, the term *oral cancer* is almost synonymous with OSCC.⁴ Each year more than 22000 new cases of OSCC and more than 5000 deaths from OSCC occur in the United States alone.^{5,6} Oral cancer is one of several cancers showing decreasing incidence trends in both males and females in the United States since the 1980s.⁷ Despite this fact and the advances made in therapeutic modalities via multidisciplinary approaches, survival rates for OSCC have not significantly improved and racial disparities persist in the United States, with 5-year survival rates approaching approximately 60% in white Americans and only 40% in African Americans.^{6,8,9}

Compared with other major cancers, our understanding of oral cancer is limited. The many anatomical subsites of cancer involvement within the oral cavity contribute to the complexity and prognosis of disease. The relatively low incidence of oral cancer requires a large database for meaningful studies that take into consideration of differences such as anatomical subsite of tumor involvement and race/ethnicity of patients—factors that are important to our understanding of the disease. The availability of data from population-based cancer registries across the globe has increased significantly in the last 40 years and has made significant contributions in reducing the overall cancer burden through identification of risk factors and implementation of cancer control programs.¹⁰ However, only 21% of the world's population is under surveillance of these registries and most are found in developed countries. The diverse racial/ethnic populations in the United States and well-established population-based central cancer registry systems provide the opportunity to examine the cancer incidence rates among different racial/ethnic populations.¹¹

The California Cancer Registry (CCR) was established in 1988 as a statewide population-based cancer surveillance program. With 12% of the US population (over 33 million in the 2000 census)—36% of all US Asians and 31% of all US Hispanics/Latinos living in California¹²—the CCR database serves as an excellent data source for epidemiological investigations.¹³ It allows studies of cancer incidence among many different racial/ethnic populations in California. Asian Americans represent an ethnic group with large internal cultural diversity and varied immigration history. It is most informative to distinguish the differences in risk exposures and disease rates by examining the individual Asian ethnic groups separately.

MATERIAL AND METHODS

Using the CCR data, we examined the oral cancer incidence rate by anatomical subsite and race/ethnicity among residents in California during 1988 to 2001. The cancer cases were classified into the following mutually exclusive racial/ethnic categories: non-Hispanic (NH) white, NH black, Hispanic, Chinese, Japanese, Filipino, Korean, South Asian (including Asian Indian, Pakistani, Bangladeshi, and Sri Lankan), and Vietnamese. The corresponding annual population estimates were obtained by linear interpolation between the 1990 census counts and 2000 census counts, and extrapolation of the 1990 to 2000 trends to obtain the 1988 to 1989 and 2001 annual population counts for each of these population groups by sex and by 5-year age group. Because the 2000 census allowed multiple identifications of race, each racial/ethnic group has 2 counts: the minimum (consists of 1 single race alone) and the maximum (consists of 1 race alone and in combination with any other race). The average of the 2 was taken as the 2000 population counts for a specific racial/ethnic group in the estimates.

We classified anatomical subsites within the oral cavity by using site codes of the *International Classification of Diseases for Oncology*, 3rd edition (ICD-O-3)¹⁴: oral tongue (C02.0-C02.9), gum (C03.0-C03.9), floor of mouth (C04.0-C04.9), palate (C05.0-C05.9), and other mouth (C06.0-C06.9) that included other subsites of the mouth such as the buccal cavity. We limited the cases to invasive squamous cell carcinoma by using ICD-O-3 histology codes (8070/3-8073/3, 8076/3, and 8078/3). Age-adjusted (2000 US Standard) incidence rates by sex, race/ethnicity, and subsite were calculated and compared.

The SEER*Stat software, version 6.2.4 (National Cancer Institute, Bethesda, MD) was used for statistical calculations of age-adjusted incidence rates, rate ratios, and 95% confidence intervals (CIs) for the rate or rate ratio estimates.

RESULTS

As shown in Table I, 12177 (7096 males, 5081 females) OSCC cases were reported to the CCR during 1988 to 2001. The overwhelming majority of cases were NH white (5565 [78%] males, 4072 [80%] females). For the oral cavity as a whole, NH black males have the highest age-adjusted incidence rate (AAIR) for OSCC among males (4.86/100 000), followed by NH whites males (4.71/100 000). For women, the highest AAIR is among South Asians (2.97/100 000), followed by NH whites (2.76/100 000). The OSCC incidence rate for Hispanics is much lower than that of the NH whites or blacks (2.52/100 000 men, 1.38/100 000 women). The differ-

Table 1. Oral squamous cell carcinoma incidence counts and age-adjusted rates (2000 US standard), California, 1988-2001*

Subsite	Male		Female		M:F	
	Count	AAIR	Count	AAIR	Rate ratio	95% CIs
Oral cavity						
NH white	5565	4.71	4072	2.76	1.71	1.6-1.8
NH black	484	4.86	217	1.75	2.77	2.3-3.3
Hispanic	619	2.52	400	1.38	1.83	1.6-2.1
Chinese	74	1.63	77	1.38	1.18	0.8-1.7
Japanese	47	2.06	58	1.97	1.05	0.7-1.6
Filipino	56	1.38	79	1.58	0.87	0.6-1.3
Korean	25	2.21	16	0.91	2.43	1.1-5.5
South Asians	38	3.56	25	2.97	1.20	0.6-2.5
Vietnamese	37	2.42	22	1.76	1.38	0.7-3.0
All races combined†	7096	4.17	5081	2.43	1.71	1.7-1.8
Tongue						
NH white	2333	1.97	1629	1.13	1.74	1.6-1.9
NH black	186	1.87	81	0.63	2.96	2.2-4.0
Hispanic	259	1.01	203	0.63	1.60	1.3-2.0
Chinese	38	0.76	52	0.9	0.84	0.5-1.4
Japanese	28	1.25	31	1.04	1.20	0.7-2.2
Filipino	28	0.66	40	0.8	0.82	0.5-1.4
Korean	17	1.41	10	0.51	2.77	1.0-8.0
South Asians	20	1.94	18	1.94	1.00	0.4-2.6
Vietnamese	22	1.17	11	0.76	1.54	0.6-4.9
All races combined†	3010	1.74	2130	1.02	1.70	1.6-1.8
Gum						
NH white	511	0.44	569	0.37	1.21	1.1-1.4
NH black	28	0.28	25	0.21	1.32	0.7-2.5
Hispanic	40	0.18	61	0.24	0.76	0.5-1.2
Chinese	11	0.30	8	0.14	2.09	0.7-6.4
Japanese	5	0.24	10	0.36	0.67	0.2-2.5
Filipino	5	0.15	6	0.11	1.42	0.3-5.9
Korean	4	0.34	3	0.19	1.81	0.2-18.9
South Asians	3	0.17	1	0.18	0.92	0.0-120.4
Vietnamese	2	0.10	2	0.16	0.62	0.0-26.9
All races combined†	617	0.38	697	0.33	1.16	1.0-1.3
Floor of mouth						
NH white	1675	1.40	1006	0.70	2.01	1.9-2.2
NH black	182	1.83	62	0.50	3.64	2.7-5.0
Hispanic	208	0.82	52	0.18	4.46	3.2-6.3
Chinese	6	0.11	2	0.05	2.41	0.4-29.7
Japanese	8	0.32	5	0.19	1.70	0.4-7.7
Filipino	16	0.39	9	0.18	2.19	0.8-5.9
Korean	1	0.10	1	0.05	2.10	0.0-298.6
South Asians	2	0.26	2	0.17	1.54	0.0-64.5
Vietnamese	4	0.23	3	0.34	0.67	0.1-10.4
All races combined†	2137	1.24	1157	0.56	2.22	2.1-2.4
Palate						
NH white	698	0.59	577	0.39	1.50	1.3-1.7
NH black	106	1.03	40	0.33	3.17	2.2-4.8
Hispanic	89	0.40	49	0.18	2.24	1.5-3.4
Chinese	12	0.25	4	0.07	3.81	1.0-17.6
Japanese	3	0.12	3	0.10	1.16	0.1-12.5
Filipino	1	0.02	17	0.34	0.07	0.0-0.6
Korean	2	0.16	0	0.00	—	—
South Asians	2	0.50	2	0.49	1.02	0.0-17.1
Vietnamese	7	0.48	0	0.00	—	—
All races combined†	196	0.12	234	0.11	1.10	0.9-1.3
Other mouth						
NH white	906	0.78	696	0.46	1.69	1.5-1.9
NH black	69	0.67	41	0.34	1.97	1.3-3.1
Hispanic	90	0.39	59	0.23	1.70	1.2-2.5
Chinese	15	0.38	12	0.24	1.57	0.7-3.9

Table I. Continued

Subsite	Male		Female		M:F	
	Count	AAIR	Count	AAIR	Rate ratio	95% CIs
Japanese	5	0.21	11	0.35	0.59	0.1-2.3
Filipino	7	0.18	9	0.18	0.96	0.3-3.1
Korean	2	0.26	2	0.17	1.58	0.1-29.7
South Asians	11	0.69	2	0.19	3.72	0.4-51.4
Vietnamese	7	0.73	6	0.49	1.49	0.3-8.0
All races combined†	1136	0.69	863	0.41	1.67	1.5-1.8

AAIR, age adjusted incidence rate; CI, confidence interval; NH, non-Hispanic.

*2000 US Standard.

†Includes above listed race/ethnicities as well as other remaining racial/ethnic groups.

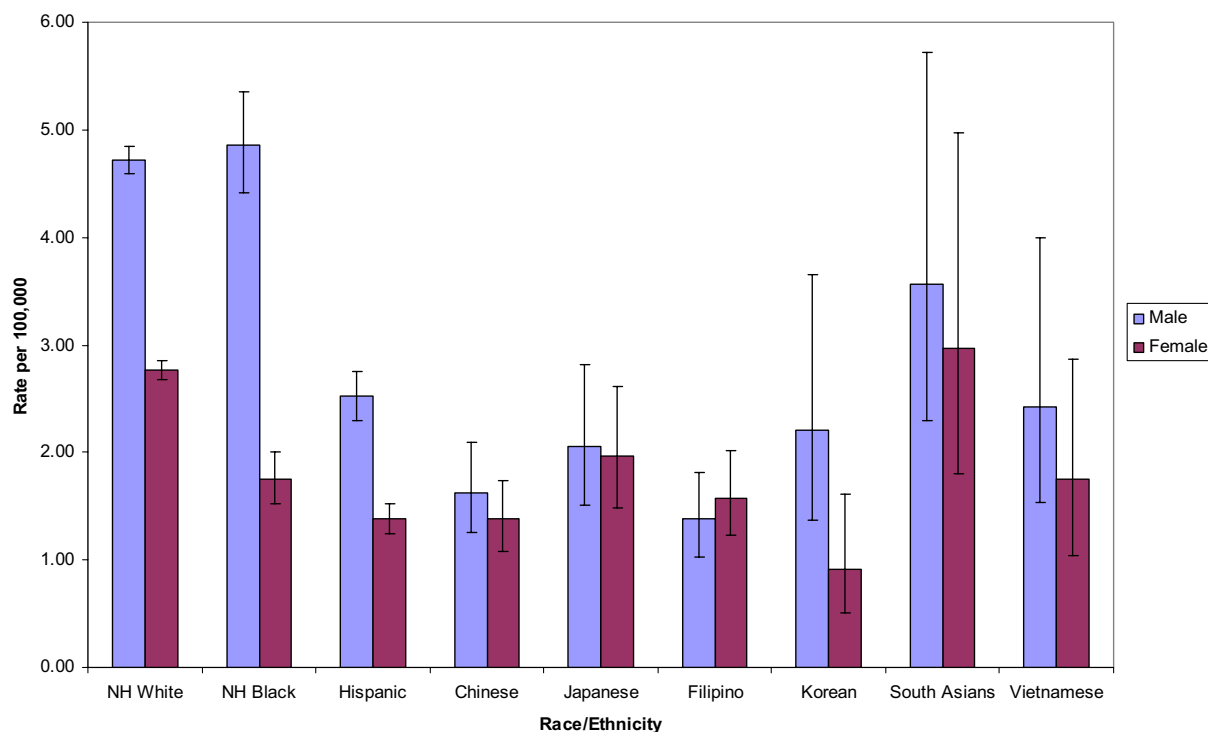


Fig. 1. Age-adjusted incidence rates (2000 US Standard) with 95% confidence intervals (CIs) of oral squamous cell carcinoma (OSCC) by sex and race/ethnicity in California, 1988-2001, oral cavity.

ences between the AAIR for Hispanics and that of NH white or NH black are statistically significant, as indicated by the nonoverlapping 95% CIs of the rate estimates (Fig. 1). The highest M:F rate ratio is found in NH blacks (2.77). The Asian populations display a range of OSCC incidence rates from 3.56 per 100 000 among South Asian men to 0.91 per 100 000 among Korean women. Due to the small number of incident cases in each of the Asian subgroups, the differences in their rate estimates do not show statistical significance according to the 95% CIs (Fig. 1). The M:F rate ratio also varies greatly, the highest is among Koreans (2.43) and the lowest is among Filipino (0.87). However, when examined by the subsite within the

oral cavity, the patterns of racial/ethnic-specific AAIR and M:F rate ratio show dramatic changes.

Tongue

The oral tongue is the most common subsite for OSCC in every race/ethnicity (Table I and Fig. 2). NH white men have a slightly higher AAIR than NH black men for tongue cancer (1.97/100 000 and 1.87/100 000, respectively). The difference in AAIR between NH white women and NH black women is much more pronounced (1.13/100 000 vs. 0.63/100 000). The M:F rate ratio for NH blacks is 2.96, as compared with 1.74 of NH whites. South Asians show same level of tongue cancer AAIR

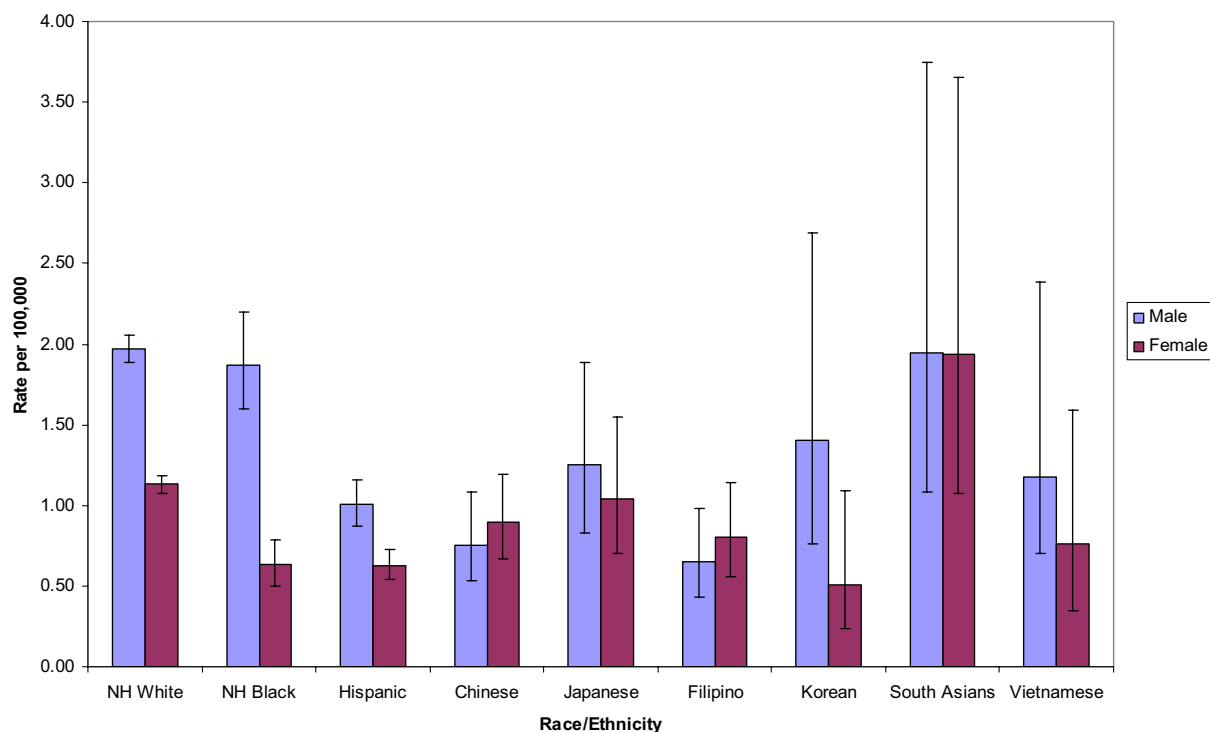


Fig. 2. Age-adjusted incidence rates (2000 US Standard) with 95% CIs of OSCC by sex and race/ethnicity in California, 1988-2001, tongue.

between men and women, which is the highest among Asian groups but similar to that of NH white and NH black men. Tongue cancer occurs more frequently among Filipino and Chinese women than men.

Gum

The gum (gingiva) is the least common subsite for OSCC (Table I and Fig. 3). NH whites appear to have the highest incidence rates in both men (0.44/100 000) and women (0.37/100 000). Compared with other oral subsites, the M:F rate ratio of OSCC in the gum is less pronounced for many racial/ethnic populations, especially for NH whites (1.21), NH blacks (1.32), and Hispanics (0.76).

Floor of mouth

The floor of mouth is the second most common subsite for OSCC (Table I and Fig. 4). NH black men have the highest AAIR (1.83/100 000), followed by NH white men (1.40/100 000) and Hispanics (0.82/100 000). Filipinos and Japanese display higher AAIRs among the Asians. The M:F rate ratio is most striking in Hispanics (4.46) and NH blacks (3.64).

Palate

Similar to the gum, the palate is a less frequent subsite for OSCC (Table I and Fig. 5). No palate

cancers were reported among Korean and Vietnamese women during the study period. The highest AAIR for OSCC of the palate, 1.03 per 100 000, is observed among NH black men; the M:F rate ratio for blacks is 3.17. Chinese have the highest M:F rate ratio, 3.81.

Other mouth

This category largely represents the OSCC in the buccal cavity. This is the second most common OSCC subsite after oral tongue for all Asian groups except Filipinos (Table I and Fig. 5). South Asian and Vietnamese men share similar AAIR as NH white and NH black men (around 0.7/100 000). The AAIR for Vietnamese women (0.49/100 000) is similar to that of NH white women (0.46/100 000). The highest M:F rate ratio of 3.72 for OSCC in this category is found among South Asians, whereas the lowest is among Japanese (0.59).

The patterns of AAIRs of OSCC by race/ethnicity for each subsite included in the study (Figs. 1-6) demonstrate that NH whites and blacks have similar incidence rates, but the M:F ratio is higher among NH blacks. The OSCC incidence rates for Hispanics are much lower than those for NH whites and NH blacks. The Asian subgroups display dramatic variations in terms of subsite distribution, incidence rate levels, and M:F rate ratios.

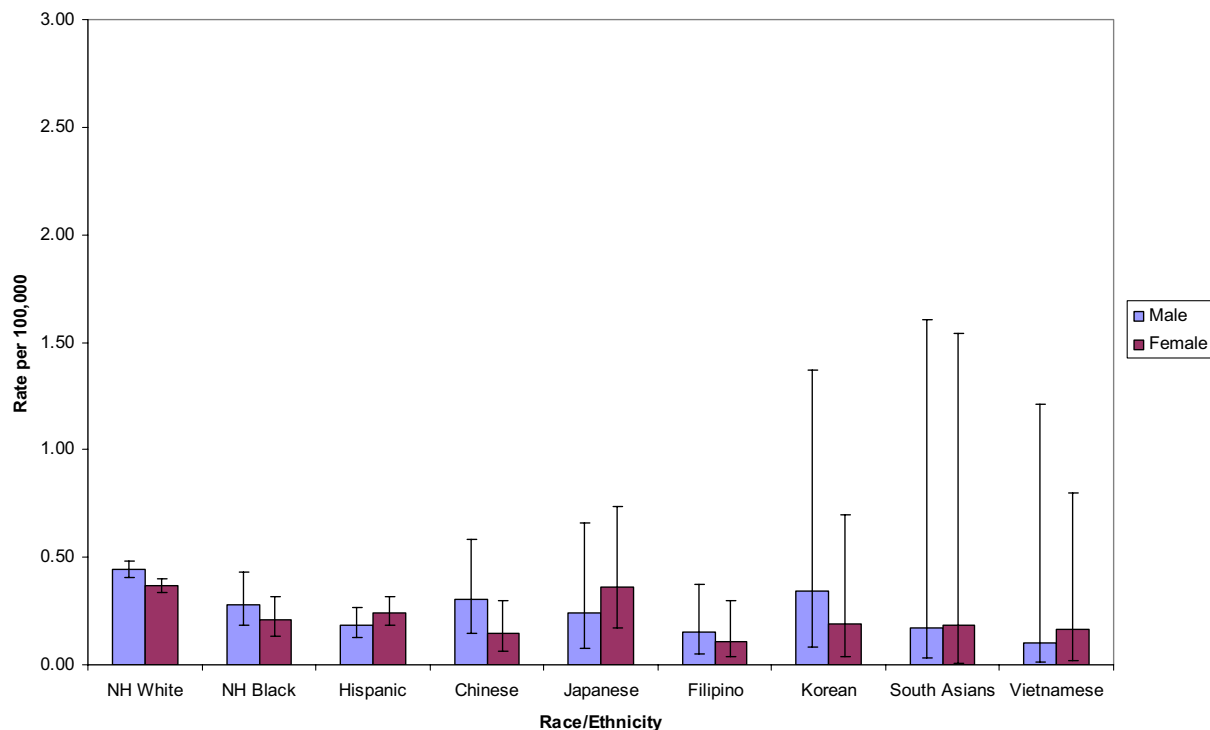


Fig. 3. Age-adjusted incidence rates (2000 US Standard) with 95% CIs of OSCC by sex and race/ethnicity in California, 1988-2001, gum.

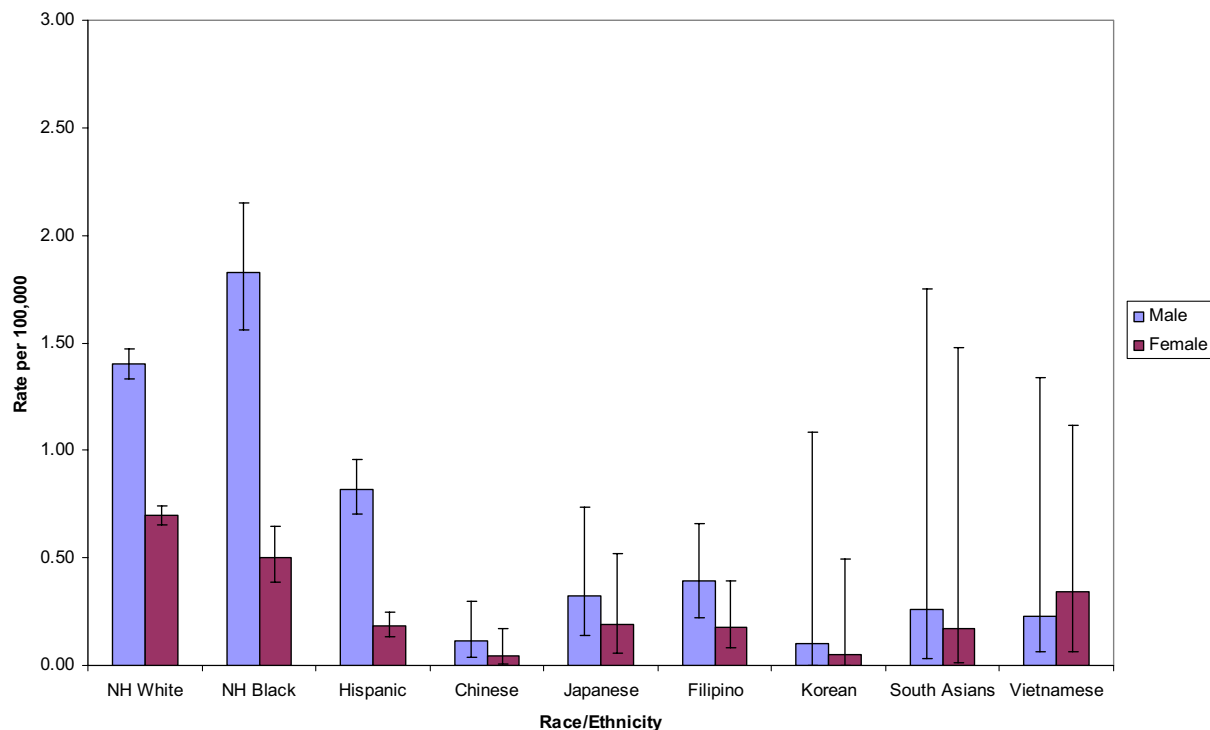


Fig. 4. Age-adjusted incidence rates (2000 US Standard) with 95% CIs of OSCC by sex and race/ethnicity in California, 1988-2001, floor of mouth.

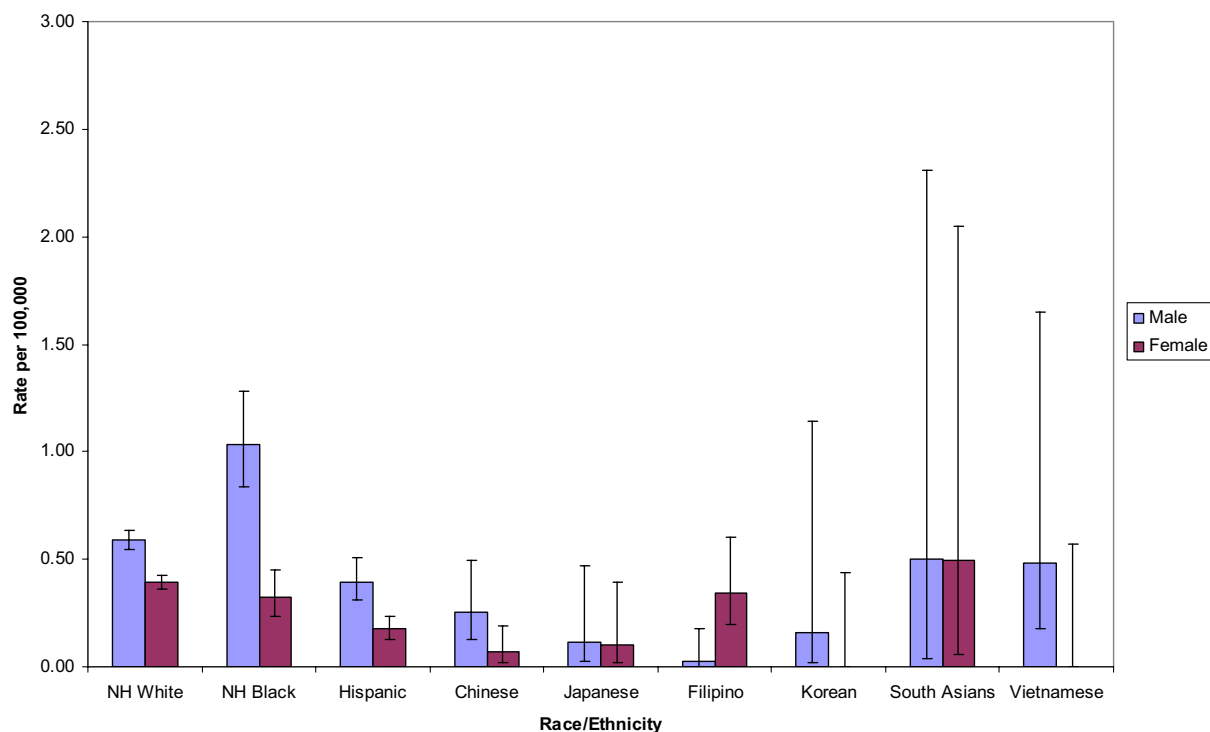


Fig. 5. Age-adjusted incidence rates (2000 US Standard) with 95% CIs of OSCC by sex and race/ethnicity in California, 1988-2001, palate.

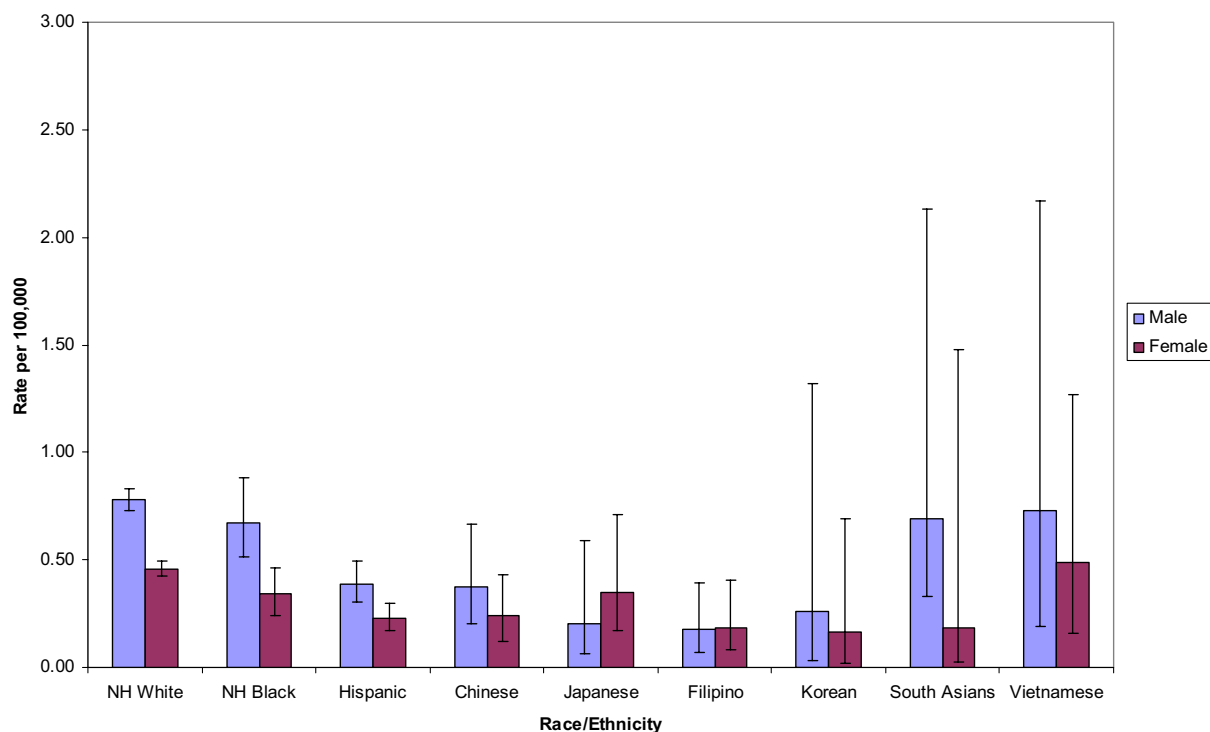


Fig. 6. Age-adjusted incidence rates (2000 US Standard) with 95% CIs of OSCC by sex and race/ethnicity in California, 1988-2001, other mouth.

DISCUSSION

This report represents our efforts to use centralized population-based cancer registry data to examine oral cancer incidence by sex, subsite, and detailed race/ethnicity. The data clearly demonstrate that (1) there are marked differences in oral cancer risk (as indicated by AAIR) between different racial/ethnic populations and (2) the risk of developing cancer varies by the location inside the oral cavity. These differences contain valuable clues regarding the causes, biologic mechanisms, and development of disease.

Although many lifestyle factors are associated with the development of oral cancer (e.g., poor nutrition, suppressed immune system, human papilloma virus [HPV] infection, and dental irritation), about 75% of oral cancer is attributable to tobacco use and alcohol consumption.^{15,16} Tobacco use and heavy alcohol consumption have been proven to increase the risk of oral cancer.¹⁷⁻¹⁹ Regardless of the different modalities of tobacco consumption (whether it is cigarettes, cigars, pipes, chewing tobacco, or snuff), tobacco use is more strongly associated with oral cancer than drinking alcohol.²⁰⁻²² People who use both alcohol and tobacco are at an especially high risk of developing oral cancer due to synergistic effects.¹⁹ The dehydrating effect of alcohol on cell membranes enhances the ability of tobacco-associated carcinogens to permeate mouth tissues; in addition, nutritional deficiencies associated with heavy drinking can lower the body's natural ability to use antioxidants to prevent the formation of cancer.²³

The racial/ethnic and sex differences in oral cancer risk observed in this study may largely reflect different cultural behaviors and lifestyle factors among various populations, especially with regard to tobacco use and alcohol drinking behaviors.²⁴ According to a recent survey conducted by the California Department of Health Services, despite the dramatic decline in tobacco consumption since late 1980s, racial/ethnic differences still exist in adult smoking prevalence.²⁵ In 2002, African-American men had the highest smoking prevalence in California (21.4%) as compared with NH white men (19.4%), Hispanic men (19.0), and Asian/Pacific-Islander men (17.7%). Among women of these racial/ethnic groups, the numbers are 17.0%, 15.2%, 7.4%, and 6.8%, respectively. Data collected by the National Longitudinal Alcohol Epidemiologic Survey²⁶ revealed that the US Asian/Pacific-Islander populations had the highest percentage of nondrinkers (63.3%), followed by African Americans (48.7%), Hispanics (46.8%), and whites (30.7%). Among drinkers, the proportion of heavy drinkers was highest among African Americans (21.3%) and lowest in Asian/Pacific Islanders (10.5%). Women were found twice as likely than men to be nondrinkers (45.3% and 21.7%, respectively).²⁶ These survey data

provide the background information regarding the major risk factors (i.e., smoking and drinking) that may explain in part, if not entirely, the observed higher incidence rates of OSCC in NH whites and NH blacks as compared with Hispanics and Asians.

In our data, the OSCC incidence rates for Hispanics are much lower than those for NH whites and NH blacks. This finding correlates with national data.^{11,27} This disparity has been attributed to the diversity of the Hispanic population, which arises from more than 20 different countries with various cultural, socioeconomic, and political backgrounds.²⁸ In general, Hispanics in the United States have substantially low incidence of cancers at many anatomical subsites except the stomach, liver, gall bladder, and cervix.²⁹ In addition, the origin of Hispanic populations varies across regions of the United States; for example, Hispanics in the west and the south United States are mainly of Mexican origin, those in the southeast are mainly Cuban, and those in the northeast are mainly Puerto Rican. Each of these Hispanic groups has different levels of exposure to oral cancer risk factors.^{28,30} One example is that of Puerto Ricans, who have higher smoking and alcohol drinking rates than Mexicans, and thus they have higher oropharyngeal cancer incidence rates than other Hispanics and non-Hispanic whites.²⁸ In California, Hispanics are mainly of Mexican origin, which could explain their relatively low OSCC incidence rates in California.

As the lower prevalence of smoking and drinking among Asians may help to explain the differences in OSCC incidence rates between Asians and other racial/ethnic groups, the higher socioeconomic status of Asian Americans also contributes to their health profile. On average, Asians in the United States have higher levels of education and higher family income than other racial/ethnic populations.^{31,32} Smoking and drinking are both inversely related to socioeconomic status.^{33,34} Powered with knowledge, information, and resources, people of high socioeconomic status tend to have heightened awareness of health-related issues and are more adaptive to healthy behaviors.³⁵ This phenomenon may partly explain the overall lower OSCC incidence rates among Asians.

The variations in subsite-specific incidence rates among different race/ethnicity, especially among Asian subgroups, are of particular interest. Specific locations within the oral cavity are associated with unique anatomical structure, cellular characteristics, and biophysiological functions. The differences in susceptibility to carcinogens and molecular changes at each of the subsites are of great value for further investigation and have already been shown to be linked to specific risk factors.³⁶

Immigrants tend to carry their cultural traditions with

them to their new homeland. Chewing tobacco is a form of smokeless tobacco consumption and is a customary habit in many populations. The other form of smokeless tobacco use is snuff, which is applied, dipped, or sucked in moist form and is also available as dry snuff that is used nasally by few population groups. Several names used to denote different smokeless tobacco products include *plug*, *gutkha*, *khiwam*, *khaini*, *iq'milk*, *zarda*, *naswar*, *nass*, *chimo*, *toombak*, *shamma*, *gudhaku*, *gul*, *mishri*, *maras*, and *moist snus*.^{20,22} The customary habit of smokeless tobacco chewing, especially among South Asians, is associated with development of precancerous oral lesions and oral cancer.^{17,20,22,37,38} With this habit, cancer commonly develops in the cheek or buccal mucosa (represented partly by "Other Mouth" in our data). Our data show that South Asians have a much higher percentage of cases in these subsites as compared with other racial/ethnic groups. The buccal mucosa is the most common cancer site in Pakistan, where the highest incidence of oral cancer in the world is reported.^{39,40}

In the Philippines, reverse smoking is a common practice among women.^{41,42} As compared with conventional smoking, reverse smoking involves inhaling the lit end of a cigarette inside the mouth after it is lit. This practice can cause cancer of the palate, which is thought to be due to the palate being exposed to higher levels of heat in addition to being exposed to nonfiltered carcinogens in the smoke that would normally be filtered to some extent during standard smoking. Accordingly, in our data, there were 17 palatal OSCC cases in Filipino females and just 1 case in males.

Among Asians, the highest M:F ratio for OSCC was seen in Koreans, and this may well be attributable to the markedly high smoking prevalence among Korean males compared with females.⁴³ In California, Korean men were found to have a smoking prevalence of 27.9%, which is 46% higher than general-population men in California (19.1%) and more than 5 times higher than Korean women (4.3%).⁴⁴

Acculturation definitely plays an important role in the changing behaviors of immigrants with regard to cancer risk factors. Studies on major cancer sites, such as lung, prostate, breast, colon, liver, and stomach, have shown that the cancer incidence rates among ethnic immigrant populations tend to approach those of the white population, over time.⁴⁵⁻⁴⁷ Surveys from California shed some light on how behavior changes toward the mainstream norm³³:

- 19.8% of the least assimilated Chinese men smoke compared with 8.8% of their most assimilated counterparts; whereas the reverse is true for women, with

the least assimilated smoking at 1.3% and the most assimilated at 4.7%

- Asian Indian men and women born in westernized countries were almost 4 times more likely to report ever using tobacco products than those born elsewhere (38.9% compared with 9.6% among those not born in a westernized country)
- only 3.3% of first-generation Korean women were current smokers, compared with 13.6% of second-generation or higher women

It is evident that the assimilation process produces different risk behaviors among different immigrant populations.

Although oral cancer shows declining incidence rates in the United States and even in high-incidence nations like India, there has been an intriguing increase in the incidence rates of tongue cancer.⁴⁸⁻⁵² In the United States, the increasing incidence trends in tongue cancer have been especially strong in young adults (<40 years of age).^{48,52} This trend has also been noted in European populations and in India.^{49,53} The exact reason for this increasing trend in tongue cancer incidence rates is not known yet. Studies have shown that tobacco use and alcohol intake do not completely explain tongue cancer incidence rates, and factors such as HPV infection may also contribute to the rise in tongue cancers.^{48,54-59} HPV-16, which is one of the most common types of HPV causing cervical cancer, has also been the most common type causing oral neoplasia besides HPV-18. The causal relationship between HPV and oral cancer and the mechanisms by which HPV induces oral cancer are being studied widely.^{48,58}

Oral cancer is a group of malignant tumors occurring in different locations in the oral cavity. It is a vicious disease that severely affects the basic human living functions. Alterations in saliva or taste, significant pain, oral and dental infections, mucosal and bone necrosis, and difficulties with mastication, swallowing, and speech severely affects patients and often reduces their quality of life significantly. Nonetheless, oral cancer is a largely preventable disease through lifestyle choices that reduce one's risk for the disease. It is also an easily detectable cancer that can be visualized at a very early stage by trained health care professionals through routine intraoral examinations. Therefore, the morbidity and mortality associated with oral cancer can be largely reduced by early detection and prevention.^{60,61} The characterization of incidence rates by race/ethnicity and sex, such as in this study, will help to identify high-risk populations for targeted education and prevention programs.

Limitations

The CCR database provides high-quality data in terms of completeness of case registration and accuracy

of case identification. However, as with most epidemiological studies, the reliability of rate estimates presented in this study is subject to several factors, including the accurate reporting of race/ethnicity, small numbers when classifying cases by multiple characteristics, and the accuracy of population estimates. Collecting racial/ethnic information is a challenge for the population-based cancer registries, especially for minority populations. There may well be underreporting of cases in specific subgroups despite best efforts by hospitals and registries. Therefore, the rate estimates for the specific racial/ethnic groups may be lower or higher than the actual rates, but the incidence patterns by subsite are not likely to be affected. Small case numbers are the common obstacle for studying oral cancer. However, we believe that it is important to show the different risk patterns by subsite and race/ethnicity. With 14 years data from the CCR, we were able to obtain meaningful numbers that could not have been achieved without a population-based registry database. The stability of the rate estimates based on small numbers of cases should always be interpreted with caution. Because no official annual population estimates are available for the Asian subgroups, we adopted the well-accepted methodology of estimating the annual population based on census results. Given the large population size of the state of California, our annual estimates should be reasonably reliable.

CONCLUSION

Our data show the complexity of oral cancer. The differences in incidence rate by sex, race/ethnicity, and subsite illustrate the heterogeneity of the disease by anatomical location, as well as the impact of varied cultural and behavioral factors in the development of the disease in different ethnic populations. This report demonstrates the value of using population-based cancer registry data to study cancers with relatively low incidence to generate hypotheses and target education and prevention programs.

We thank California's cancer registrars and other persons responsible for cancer data collection, and Dr. Dennis Deapen and Dr. Myles Cockburn at the Los Angeles Cancer Surveillance Program, Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, for their critical reading of the manuscript and statistical advice.

REFERENCES

1. International Agency for Research on Cancer [homepage on the Internet]. Lyon (France): The Agency; 2006 [cited 2006 Nov 7; last updated 2007 July 11]. Available from: <http://www-dep.iarc.fr/>.
2. Parkin DM, Bray F, Ferlay J, Pisani P. Estimating the world cancer burden: Globocan 2000. *Int J Cancer* 2001;94:153-6.

3. Neville BW, Damm DD, Allen CM, Bouquot JE. Epithelial pathology. In: Neville BW, Damm DD, Allen CM, Bouquot JE, editors. *Oral and maxillofacial pathology*. 2nd ed. Philadelphia: W.B. Saunders Company; 2002. p. 356-66.
4. Batsakis JG. Clinical pathology of oral cancer. In: Shah JP, Johnson NW, Batsakis JG, editors. *Oral cancer*. London: Martin Dunitz, an imprint of the Taylor & Francis Group; 2003.
5. Cancer Facts and Figures 2006 [database on the Internet]. Atlanta, GA; American Cancer Society; 2006 [cited 2006 Nov 7]. Available from: <http://www.cancer.org/downloads/STT/CAFF2006PWSecured.pdf>.
6. Jemal A, Siegel R, Ward E, Murray T, Xu J, Smigal C, et al. Cancer statistics, 2006. *CA Cancer J Clin* 2006;56:106-30.
7. Howe HL, Wu X, Ries LA, Cokkinides V, Ahmed F, Jemal A, et al. Annual report to the nation on the status of cancer, 1975-2003, featuring cancer among U.S. Hispanic/Latino populations. *Cancer* 2006;107:1711-42.
8. Morse DE, Kerr AR. Disparities in oral and pharyngeal cancer incidence, mortality and survival among black and white Americans. *J Am Dent Assoc* 2006;137:203-12.
9. Ries LAG, Harkins D, Krapcho M, Mariotto A, Miller BA, Feuer EJ, et al. SEER cancer statistics review, 1975-2003. Bethesda (MD): National Cancer Institute; 2006.
10. Parkin DM. The evolution of the population-based cancer registry. *Nat Rev Cancer* 2006;6:603-12.
11. Surveillance, Epidemiology and End Results [homepage on the Internet]. Bethesda (MD): National Cancer Institute; 2006 [cited 2006 Oct 28; last updated 2007 April 15]. Available from: <http://seer.cancer.gov/>.
12. Population Finder [database on the Internet]. Washington, DC; U.S. Census Bureau; 2006 [cited 2006 Oct 28]. Available from: <http://www.census.gov/>.
13. California Cancer Facts and Figures 2006 [database on the Internet]. Sacramento, CA; California Cancer Registry; 2006 [cited 2006 Oct 28]. Available from: <http://www.ccrca.org/>.
14. Fritz A, Percy C, Jack A, Shanmugaratnam K, Sobin L, Parkin DM, Whelan S (eds). *International Classification of Diseases for Oncology, Third Edition (ICD-O-3)*. Geneva, Switzerland: World Health Organization; 2000.
15. Blot WJ, McLaughlin JK, Winn DM, Austin DF, Greenberg RS, Preston-Martin S, et al. Smoking and drinking in relation to oral and pharyngeal cancer. *1988;48:3282-7*.
16. Negri E, La Vecchia C, Franceschi S, Tavani A. Attributable risk for oral cancer in northern Italy. *Cancer Epidemiol Biomarkers Prev* 1993;2:189-93.
17. Johnson N. Tobacco use and oral cancer: a global perspective. *J Dent Educ* 2001;65:328-39.
18. Hindle I, Downer MC, Moles DR, Speight PM. Is alcohol responsible for more intra-oral cancer? *Oral Oncol* 2000;36:328-33.
19. Ogden GR. Alcohol and oral cancer. *Alcohol* 2005;35:169-73.
20. Warnakulasuriya S. Smokeless tobacco and oral cancer. *Oral Dis* 2004;10:1-4.
21. Hashibe M, Brennan P, Benhamou S, Castellsague X, Chen C, Curado MP, et al. Alcohol drinking in never users of tobacco, cigarette smoking in never drinkers, and the risk of head and neck cancer: pooled analysis in the International Head and Neck Cancer Epidemiology Consortium. *J Natl Cancer Inst* 2007;99:777-89.
22. Warnakulasuriya KA, Ralhan R. Clinical, pathological, cellular and molecular lesions caused by oral smokeless tobacco—a review. *J Oral Pathol Med* 2007;36:63-77.
23. Figuero Ruiz E, Carretero Pelaez MA, Cerero Lapidra R, Esparza Gomez G, Moreno Lopez LA. Effects of the consumption of alcohol in the oral cavity: relationship with oral cancer. *Med Oral* 2004;9:14-23.

24. Zain RB. Cultural and dietary risk factors of oral cancer and precancer—a brief overview. *Oral Oncol* 2001;37:205-10.
25. Adult Smoking Prevalence [database on the Internet]. Sacramento, CA; California Department of Health Services; 2006 [updated 2006 Aug; cited 2007 May 27]. Available from: <http://www.dhs.ca.gov/tobacco/documents/pubs/AdultSmoking06.pdf>.
26. Stinson FS, Yi HY, Grant BF, Chou P, Dawson DA, Pickering R. Drinking in the United States: main findings from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES). U.S. alcohol epidemiologic data reference manual. Washington DC: U.S. Government Printing Office; 1998.
27. Howe HL, Carozza S, O Malley C, Dolecek TA, Finch JL, Kohler B, et al. Cancer in U.S. Hispanics/Latinos, 1995-2000 [database on the Internet]. Springfield, IL: North American Association of Central Cancer Registries; 2003 [updated 2003 Dec; cited 2007 May 27]. Available from: <http://www.naaccr.org/filesystem/pdf/CIUSHL%202003%20incd.sec0.pdf>.
28. Cruz GD, Salazar CR, Morse DE. Oral and pharyngeal cancer incidence and mortality among Hispanics, 1996-2002: the need for ethnoregional studies in cancer research. *Am J Public Health* 2006;96:2194-200.
29. Eschbach K, Mahnken JD, Goodwin JS. Neighborhood composition and incidence of cancer among Hispanics in the United States. *Cancer* 2005;103:1036-44.
30. Perez-Stable EJ, Ramirez A, Villareal R, Talavera GA, Trapido E, Suarez L, et al. Cigarette smoking behavior among US Latino men and women from different countries of origin. *Am J Public Health* 2001;91:1424-30.
31. We the Americans: Asians. Washington, DC; U.S. Census Bureau; 1993 [cited 2006 Nov 7]. Available from: <http://www.census.gov/apsd/wepeople/we-3.pdf>.
32. We the People: Asians in the United States. U.S. Census Bureau; 2004 [cited 2006 Nov 7]. Available from <http://www.census.gov/prod/2004pubs/censr-17.pdf>.
33. Stellman SD, Resnicow K. Tobacco smoking, cancer and social class. In: Kogivenas M, Pearce N, Susser M, editors. *Social inequalities and cancer*. Lyon (France): IARC; 1997.
34. Moller H, Tonnesen H. Alcohol drinking, social class and cancer. In: Kogivenas M, Pearce N, Susser M, editors. *Social inequalities and cancer*. Lyon, France: IARC; 1997.
35. Link BG, Phelan JC. Understanding sociodemographic differences in health—the role of fundamental social causes. *Am J Public Health* 1996;86:471-3.
36. Jovanovic A, Schulten EA, Kostense PJ, Snow GB, van der Waal I. Tobacco and alcohol related to the anatomical site of oral squamous cell carcinoma. *J Oral Pathol Med* 1993;22:459-62.
37. Ahluwalia KP. Assessing the oral cancer risk of South-Asian immigrants in New York City. *Cancer* 2005;104(12 Suppl):2959-61.
38. Changrani J, Gany F. Paan and Gutka in the United States: an emerging threat. *J Immigr Health* 2005;7:103-8.
39. Bhurgri Y. Cancer of the oral cavity—trends in Karachi South (1995-2002). *Asian Pac J Cancer Prev* 2005;6:22-6.
40. Khawaja MI, Shafiq M, Nusrat R, Khawaja MR. Preventing the oral cavity cancer epidemic. *Asian Pac J Cancer Prev* 2005;6:420.
41. Mercado-Ortiz G, Wilson D, Jiang DJ. Reverse smoking and palatal mucosal changes in Filipino women. *Epidemiological features*. *Aust Dent J* 1996;41:300-3.
42. Ortiz GM, Pierce AM, Wilson DF. Palatal changes associated with reverse smoking in Filipino women. *Oral Dis* 1996;2:232-7.
43. Leistikow BN, Chen M, Tsodikov A. Tobacco smoke overload and ethnic, state, gender, and temporal cancer mortality disparities in Asian-Americans and Pacific Islander-Americans. *Prev Med* 2006;42:430-4.
44. New Data Show California Military, Korean Men and LGBT Populations Smoke Much More than Others in the State [database on the Internet]. Sacramento, CA; California Department of Health Services; [cited 2007 May 27]. Available from: http://www.dhs.ca.gov/tobacco/documents/press/PressReleaseSept6_05.pdf.
45. Gomez SL, Le GM, Clarke CA, Glaser SL, France AM, West DW. Cancer incidence patterns in Koreans in the US and in Kangwha, South Korea. *Cancer Causes Control* 2003;14:167-74.
46. Le GM, Gomez SL, Clarke CA, Glaser SL, West DW. Cancer incidence patterns among Vietnamese in the United States and Ha Noi, Vietnam. *Int J Cancer* 2002;102:412-7.
47. Luo W, Birkett NJ, Ugnat AM, Mao Y. Cancer incidence patterns among Chinese immigrant populations in Alberta. *J Immigr Health* 2004;6:41-8.
48. Shiboski CH, Schmidt BL, Jordan RC. Tongue and tonsil carcinoma: increasing trends in the U.S. population ages 20-44 years. *Cancer* 2005;103:1843-9.
49. Elango JK, Gangadharan P, Sumithra S, Kuriakose MA. Trends of head and neck cancers in urban and rural India. *Asian Pac J Cancer Prev* 2006;7:108-12.
50. Canto MT, Devesa SS. Oral cavity and pharynx cancer incidence rates in the United States, 1975-1998. *Oral Oncol* 2002;38:610-7.
51. Carvalho AL, Nishimoto IN, Califano JA, Kowalski LP. Trends in incidence and prognosis for head and neck cancer in the United States: a site-specific analysis of the SEER database. *Int J Cancer* 2005;114:806-16.
52. Schantz SP, Yu GP. Head and neck cancer incidence trends in young Americans, 1973-1997, with a special analysis for tongue cancer. *Arch Otolaryngol Head Neck Surg* 2002;128:268-74.
53. Annertz K, Anderson H, Biorklund A, Moller T, Kantola S, Mork J, et al. Incidence and survival of squamous cell carcinoma of the tongue in Scandinavia, with special reference to young adults. *Int J Cancer* 2002;101:95-9.
54. D Souza G, Kreimer AR, Viscidi R, Pawlita M, Fakhry C, Koch WM, et al. Case-control study of human papillomavirus and oropharyngeal cancer. *N Engl J Med* 2007;356:1944-56.
55. Llewellyn CD, Linklater K, Bell J, Johnson NW, Warnakulasuriya S. An analysis of risk factors for oral cancer in young people: a case-control study. *Oral Oncol* 2004;40:304-13.
56. Hansson BG, Rosenquist K, Antonsson A, Wennerberg J, Schildt EB, Bladstrom A, et al. Strong association between infection with human papillomavirus and oral and oropharyngeal squamous cell carcinoma: a population-based case-control study in southern Sweden. *Acta Otolaryngol* 2005;125:1337-44.
57. Syrjanen S. Human papillomavirus (HPV) in head and neck cancer. *J Clin Virol* 2005;32(Suppl 1):S59-66.
58. Herrero R, Castellsague X, Pawlita M, Lissowska J, Kee F, Balaram P, et al. Human papillomavirus and oral cancer: the International Agency for Research on Cancer multicenter study. *J Natl Cancer Inst* 2003;95:1772-83.
59. Tang X, Jia L, Ouyang J, Takagi M. Comparative study of HPV prevalence in Japanese and North-east Chinese oral carcinoma. *J Oral Pathol Med* 2003;32:393-8.
60. Mignogna MD, Fedele S. Oral cancer screening: 5 minutes to save a life. *Lancet* 2005;365:1905-6.
61. Mignogna MD, Fedele S, Lo Russo L. The World Cancer Report and the burden of oral cancer. *Eur J Cancer Prev* 2004;13:139-42.

Reprint requests:

Lihua Liu, PhD
 Los Angeles Cancer Surveillance Program
 Department of Preventive Medicine
 Keck School of Medicine
 University of Southern California
 1540 Alcazar Street, CHP-204
 Los Angeles, CA 90033
 lihualiu@usc.edu