Head and Neck Cancer Incidence Trends in Young Americans, 1973-1997, With a Special Analysis for Tongue Cancer

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Objective: To examine the temporal changes in head and neck cancer in young adults in the United States.

Methods: Using the cancer surveillance database from the National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) Program, we calculated ageadjusted incidence rates for head and neck cancers. Using the joinpoint regression model, we described tongue cancer incidence trends and established the statistical significance of temporal changes. We also compared changes in 5-year survival rates for tongue cancer.

Results: From 1973 to 1997, there were 63409 patients with head and neck cancer in the 9 SEER registries. Of these, 3339 patients were younger than 40 years. The incidence of head and neck cancer remained stable in groups older than 40 years comparing the 1973-1984 and 1985-1997 data. In contrast, tongue cancer in adults younger than 40 years increased approximately 60% during the same period. We detected a significant increase

until 1985, the estimated annual percentage change being 6.7% (95% confidence interval, 2.7%-10.8%; P<.001). After 1985, incidence rates stopped rising but remained steadily high. The change in tongue cancer incidence rates for young adults was related to birth cohorts between 1938 and 1948. The absolute increase in 5-year survival for tongue cancer ranged from 11.7% (<40 years old) to 6.6% (40-64 years old) between 1973-1984 and 1985-1997, with the most significant improvement occurring in young Americans with regional or distant disease (27% and 21%, respectively).

Conclusions: A sharp increasing trend in tongue cancer in young Americans may be attributed to persons born after 1938. The reason for the increase is uncertain. Improved survival rates in young patients suggest the emergence of a distinct disease process that is apparent in white but not black populations.

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HE RESULTS of many studies1-7 suggest that head and neck cancer, particularly oral tongue cancer, is increasing in young adults internationally. In the United States, Depue⁴ found an increase in the tongue cancer mortality rate in adults younger than 30 years. The increase was cited as beginning in the mid-1970s. In addition, Chen et al⁷ reported that between the 1960s and the mid-1980s, men aged 30 to 39 years had a nearly 4-fold increase in oral cancer incidence in Connecticut. Results of a recent clinical analysis8 indicated that the percentage of young patients in the total tongue cancer population seen at the M.D. Anderson Cancer Center (Houston, Tex) increased from 4% in 1971 to 18% in 1993. Franchesci et al⁵ examined mortality trends from 24 European countries based on death certificates recorded between 1955 and 1989. Ten countries showed greater than a 2-fold increase in oral cancer for men younger

than 44 years, and 3 countries showed a similar increase for women. The countries represented in this increase were principally those in central Europe, including Austria, Germany, Hungary, Poland, and Bulgaria.⁵

Because incidence rates for overall head and neck cancer have remained stable and have even shown a declining trend since the 1970s, ^{6,9} the increase in the number of young adult patients is concerning. Factors that may account for oral cancer in young adults remain unknown. Suspected etiologic agents include smokeless tobacco, ^{10,11} various forms of drug abuse, ¹² virus, ¹³ and host susceptibility factors. ¹⁴ However, no clear evidence exists to support the significance of any single determinant, including the role of tobacco. ¹⁵

The goal of this study is to update and confirm the changes of trend in the incidence of young adult oral tongue cancer and other head and neck cancers using a large cancer surveillance database in the

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MATERIALS AND METHODS

Data for the analysis were obtained from the Surveillance, Epidemiology, and End Results (SEER) Program Public-Use CD-ROM (1973-1997) (National Cancer Institute, Bethesda, Md, Division of Cancer Control and Population Sciences, Cancer Surveillance Research Program, Cancer Statistics Branch, released April 2000) based on the August 1999 submission. We applied information on all head and neck cancers diagnosed in the residents of 9 population-based registries, ie, 5 states (Connecticut, Hawaii, Iowa, New Mexico, and Utah) and 4 standard metropolitan statistical areas (Atlanta, Ga; Detroit, Mich; San Francisco—Oakland, Calif; and Seattle—Puget Sound, Wash). These registries cover approximately 10% of the US population.

The SEER Program uses the International Classification of Diseases for Oncology (second edition) to code site (topography), histologic features (morphology), and behavior for all cancers. 16 The site codes for tongue cancer cases used in this article are C019 (base of tongue), C020 (dorsal surface of tongue), C021 (border of tongue), C022 (ventral surface of tongue), C023 (anterior of tongue), C024 (lingual tonsil), C028 (overlapping lesion of tongue), and C029 (tongue, not otherwise specified). Only tongue cancer cases considered to be invasive (malignant), that is, with a behavior code of 3, are included in this analysis. Oral tongue cancers with histologic codes 9590 to 9989 (lymphomas) were excluded. The site grouping included in this analysis is consistent with that published previously by the SEER Program. 17 Between 1973 and 1997, in SEER Program areas, there were 76 patients whose race was unknown with tongue cancer. To keep the totality of cases, we included them to calculate overall incidence and survival rates.

Incidence rates are expressed per 100000 population and are age adjusted using the direct method to the 1970 US population. We classified incidence data into 3 age groups (<40, 40-64, and ≥65 years) and calculated age-adjusted rates within the groups according to every calendar year from 1973 to 1997. To better describe tongue cancer data, we also calculated race- and sex-specific age-adjusted incidence rates in 5-year intervals beginning in 1973-1977 and extending to 1993-1997.

We applied the joinpoint regression model to characterize trends in tongue cancer incidence to determine

when the incidence started rising, when it peaked, and the degree to which it has returned to the background trend before the change. 18,19 The approach allows more flexible and accurate analyses in detecting temporal changes in incidence over time, thus reducing the effect of possible variable observations. In the joinpoint model, the response variables for analysis are the natural logarithms of age-adjusted rates, and the independent variables are the calendar years of diagnosis. For tongue cancer data, we coded the values of years as 0 to 24, representing a calendar year range from 1973 to 1997. We fitted the heteroscedastic/ uncorrelated error joinpoint model and conducted a series of permutation tests based on 1000 Monte Carlo replicates. The models were estimated by use of weighted least squares, with the weights proportional to the inverse of variance of age-adjusted rates. For each hypothesized model, the best-fitting joinpoints were found by use of a grid search algorithm. In the courses, we first tested H₀ (no joinpoints) vs H_{α} (3 joinpoints). The testing then proceeded sequentially and decreased the number of joinpoints under the alternative hypothesis by 1 if 3 joinpoints are insignificant. The statistics derived from these models are the estimated annual percentage change (EAPC), 95% confidence intervals (CIs) for the EAPCs, and any possible joinpoints (calendar years) at which there is a change in the trends.

To explore the possible effects of birth cohort on tongue cancer incidence rates over time, we calculated 5-year birth cohort incidence rates according to the method of Robertson and Boyle. We stratified the incidence data into 22 birth cohorts (1883-1887, 1888-1892, ..., 1983-1987, and 1988-1992), based on SEER Program data with 18 age groups (0-4, 5-9, ..., and ≥85 years) and 5 periods (1973-1977, 1978-1982, ..., and 1993-1997). All individuals 85 years and older were assumedly classified as the 85- to 89-year-old group. In the stratification, each age group was split into 2 constituent cohorts.

In this descriptive analysis, we further calculated sex-, race-, and stage-specific 5-year relative survival rates according to 2 periods, 1973-1984 and 1985-1997. The analysis excluded patients with a tongue cancer diagnosed as a second or later cancer, patients whose cancer was identified only by a death certificate or autopsy report, and patients with unknown survival time. Classification of tumor stage was based on the standard of SEER Program historic stage A.²¹

United States. A unique feature of our analysis is the use of a new statistical method, *joinpoint regression*, to describe incidence trends and to establish the statistical significance of changes in trend.

RESULTS

The SEER Program reported 63409 patients with head and neck cancer (oral and pharynx: C000-C148) between 1973 and 1997. Of these, 3339 patients were younger than 40 years. Between the 1973-1984 and 1985-1997 periods, the overall incidence for head and neck cancer was stable. A slight decrease could be seen in 1985-1997 in groups aged 40 to 64 years and 65 years and older. However, the number of patients with head and neck cancer increased among Americans younger than 40 years

during the same period. The increase seemed to be mainly caused by increased tongue cancer. Tongue cancer in young Americans ranked second to salivary gland cancer in all head and neck cancers and increased 62% comparing 1985-1997 and 1973-1984. Laryngeal cancer showed no significant change in incidence during the 2 periods (**Table 1**).

Table 2 gives race- and sex-specific age-adjusted incidence rates for tongue cancer according to 3 age groups and five 5-year intervals. Overall age-adjusted rates significantly increased in young Americans in both sexes. Rates increased only slightly in men aged 40 to 64 years and in women 65 years and older. Age-adjusted incidence increased almost 1-fold in the young white population during the past 25 years; from 0.11 in 1973-1984 to 0.19 in 1985-1997 in young white men and from 0.06

Table 1. Observed Incident Cases and Rates for Oral, Pharyngeal, and Laryngeal Cancers by Age and Period, SEER Program, 1973-1997*

	No. (Rate)†						
	0-39 y		40-64 y		≥65 y		
Site of Tumor (<i>ICD-0-2</i> Code)	1973-1984	1985-1997	1973-1984	1985-1997	1973-1984	1985-1997	
Lip (C000-C009)	192 (0.12)	200 (0.10)	1923 (3.10)	1300 (1.58)	2456 (9.68)	2507 (6.85)	
Tongue (C019-C029)	204 (0.13)	413 (0.21)	2886 (4.65)	3485 (4.23)	2212 (8.72)	3348 (9.15)	
Salivary gland (C079-C089)	381 (0.24)	489 (0.25)	897 (1.44)	1241 (1.51)	997 (3.93)	1646 (4.50)	
Floor of mouth (C040-C049)	47 (0.03)	58 (0.03)	2065 (3.33)	1783 (2.16)	1202 (4.74)	1466 (4.01)	
Gum and other mouth (C030-C039, C050-C059, and C060-C069)	188 (0.12)	256 (0.13)	2311 (3.72)	2375 (2.88)	2234 (8.80)	3117 (8.52)	
Nasopharynx (C110-C119)	275 (0.17)	365 (0.19)	848 (1.37)	1055 (1.28)	424 (1.67)	646 (1.77)	
Tonsil (C090-C099)	65 (0.04)	100 (0.05)	1690 (2.72)	2010 (2.44)	998 (3.93)	1347 (3.68)	
Oropharynx (C100-C109)	10 (0.01)	12 (0.01)	440 (0.71)	455 (0.55)	299 (1.18)	448 (1.22)	
Hypopharynx (C129 and C130-C139)	18 (0.01)	30 (0.02)	1518 (2.44)	1565 (1.90)	1172 (4.62)	1634 (4.47)	
Other buccal cavity and pharynx (C140, C142, and C148)	17 (0.01)	19 (0.01)	496 (0.80)	539 (0.65)	385 (1.52)	650 (1.78)	
Total oral and pharynx (C000-C148)	1397 (0.87)	1942 (1.01)	15 074 (24.28)	15 808 (19.18)	12 379 (48.77)	16 809 (45.94)	
Larynx (C320-C329)	181 (0.11)	220 (0.11)	6516 (10.50)	6389 (7.75)	4848 (19.10)	7128 (19.48)	

^{*}SEER indicates Surveillance, Epidemiology, and End Results; ICD-0-2, International Classification of Diseases for Oncology, Second Edition. †Expressed per 100 000 population.

Table 2. Race- and Sex-Specific Age-Adjusted Incidence Rates for Tongue Cancer by Age and 5-Year Interval* Male/Female White Time Interval Black Other Total 0- to 39-Year-Olds 1973-1977 0.11/0.06 0.10/0.03 0.00/0.17 0.10/0.06 1978-1982 0.14/0.09 0.13/0.10 0.24/0.05 0.10/0.06 1983-1987 0.17/0.11 0.26/0.05 0.28/0.07 0.19/0.10 1988-1992 0.18/0.11 0.10/0.08 0.16/0.14 0.18/0.11 1993-1997 0.19/0.10 0.07/0.12 0.15/0.17 0.18/0.12 40- to 64-Year-Olds 1973-1977 5.69/2.71 9.77/3.65 3.91/1.80 5.95/2.78 1978-1982 5.94/2.76 12.92/4.66 3.13/1.41 6.32/2.84 1983-1987 6.65/2.65 12.29/3.77 2.72/0.93 6.87/2.64 1988-1992 6.06/2.42 14.17/3.12 3.46/1.26 6.55/2.40 1993-1997 6.62/2.69 10.59/3.02 3.61/1.41 6.78/2.66 65 Years and Older 1973-1977 13.84/4.88 6.20/1.65 13.39/4.66 13.40/2.87 1978-1982 13.02/6.26 13.14/2.65 13.30/4.27 13.02/5.96 1983-1987 14.01/6.76 14.68/4.94 6.33/6.02 13.63/6.58 1988-1992 13.68/6.29 15 62/3 28 8 36/5 84 13 42/6 04 13.33/6.34 12.64/4.14 11.10/3.86 13.20/6.01 1993-1997

to 0.10 in young white women. Rates fluctuated in the nonwhite population, although an obvious upward incidence with time could be seen in young black women.

Figure 1 displays incidence trends for tongue cancer and the fitting curves of the joinpoint regression model. Incidence trends for young and old Americans changed at different rates. In Americans younger than 40 years, there was a significant upward trend from 1973 through 1997 (Figure 1A). The overall EAPC was 2.69 (95% CI, 1.4-4.1; *P*<.001) during these 25 years. A sharp acceleration was seen from 1973 until 1985, with the EAPC being 6.68 (95% CI, 2.7-10.8; *P*<.001). The significant joinpoint was at the year 1985 (95% CI, 1979-1989). Af-

ter 1985, the incidence stopped rising but remained steadily high (EAPC, 0.002; 95% CI, –2.7-2.9). In contrast to young Americans, however, the age-adjusted incidence trends for Americans older than 40 years remained level (Figure 1B-C). The EAPCs in slope were 1.13 for the group aged 40 to 64 years and 0.31 for those 65 years and older from 1973 to 1997.

Examining changes in cohort-specific incidence rates by age groups show increased incidence rates in individuals born between 1943-1947 in all workable age groups (ie, 30-34, 35-39, 40-44, and 45-49 years) (**Figure 2**). The effect of birth cohort was particularly evident in the incidence curve of the group aged 35 to 39 years. A large

stRates are expressed per 100 000 population.

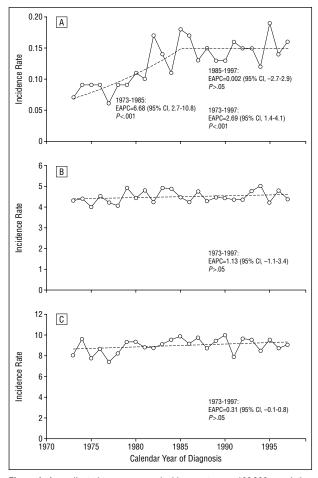


Figure 1. Age-adjusted tongue cancer incidence rates per 100 000 population in 9 National Cancer Institute Surveillance, Epidemiology, and End Results Program registries, 1973-1997, in Americans younger than 40 years (A), 40 to 64 years (B), and 65 years and older (C). The dotted line segments are obtained from the fits of the joinpoint regression models. EAPC indicates estimated annual percentage change; Cl, confidence interval.

acceleration began in the 1938-1942 cohort and peaked in the 1943-1947 cohort (Figure 2A). Cohorts born after 1947 also showed an upward trend among Americans younger than 40 years. Although we calculated cohort-specific rates for the group 65 years and older according to subgroups aged 65 to 69 years, 70 to 74 years, 75 to 79 years, 80 to 84 years, and 85 years and older, we could not present these curves owing to overlapping incidence rates. In the group 65 years and older, the earliest birth cohort was 1883-1887 and the latest was 1923-1927; their incidence rates were 8.35 and 8.43, respectively.

Table 3 gives 5-year survival rates for patients with tongue cancer by age group and period. During 1985-1997, survival declined with increasing age: 70.6% for the group younger than 40 years, 49.8% for those aged 40 to 64 years, and 45.8% for those 65 years and older. From 1973 to 1997, 5-year survival rates for tongue cancer increased in all age groups. The absolute increase in survival was largest in Americans younger than 40 years (11.7%; *P*=.009). Survival improvement was more pronounced in young patients who were male, white, and at an advanced tumor stage. However, survival rates somewhat decreased in young nonwhite patients. Five-year relative survival in 1985-

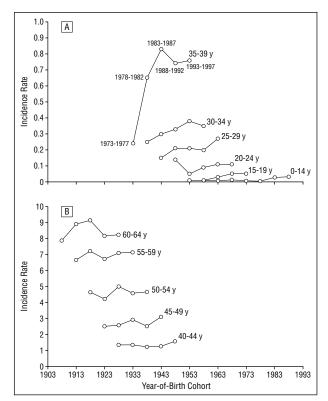


Figure 2. Tongue cancer incidence rates per 100 000 population by age group and birth cohort, National Cancer Institute Surveillance, Epidemiology, and End Results Program, 1973-1997. A, Americans younger than 40 years. B, Americans aged 40 to 64 years.

1997 for young patients with local stage, regional stage, and distant stage tumor were 83.6%, 52.7%, and 44.3%, respectively. More than 20% survival improvement was seen in young patients with regional or distant stage compared with data from 1973-1984. The absolute increase in survival was only 3% in young patients with localized disease. Among older Americans, the increased survival rates were similar in different sexes, races, and stages, except for the black population aged 40 to 64 years, which showed a decreased survival rate.

Between 1973-1984 and 1985-1997, the stage distribution of tongue cancer did not significantly change in the 3 age groups (**Table 4**). Fifty-five percent of young patients were at the localized stage. The proportion of local-stage disease was approximately 30% to 40% higher for younger patients than for older patients.

COMMENT

The present study exhibits a significantly increased trend of tongue cancer in Americans born after 1938. The acceleration began in 1973, peaked in 1985, and subsequently has been stable. In contrast to oral tongue cancer, the incidence rates of other head and neck cancers in younger Americans, such as cancers of the pharynx and larynx, remained relatively stable during the same period. The marked increase in tongue cancer in young Americans is particularly impressive compared with the stable incidence in older populations. Tongue cancer is a rare malignancy in young adults; therefore, we need to interpret the trends with caution. We do not think that

Table 3. Sex-, Race-, and Stage-Specific 5-Year Survival for Patients With Tongue Cancer by Age and Period*

Characteristic	0-39 y			40-64 y			≥65 y		
	1973-1984	1985-1997	Absolute Increase	1973-1984	1985-1997	Absolute Increase	1973-1984	1985-1997	Absolute Increase
Sex									
F	67.01	72.99	5.98	48.36	55.52	7.16	44.27	53.95	9.68
M	54.57	68.98	14.41	40.73	47.54	6.81	34.45	39.87	5.42
Race									
White	62.34	74.55	12.21	44.72	53.34	8.62	38.29	45.78	7.49
Black	27.10	23.24	-3.86	31.08	27.97	-3.11	21.73	31.67	9.94
Other	69.59	66.16	-3.43	51.35	55.68	4.33	57.56	59.82	2.26
Stage									
Localized	80.61	83.61	3.00	62.60	70.56	7.96	60.00	67.45	7.45
Regional	25.29	52.73	27.44	36.09	40.73	4.64	27.09	33.72	6.63
Distant	23.24	44.28	21.24	13.64	19.87	6.23	6.80	14.98	8.18
Unstaged	55.01	76.59	21.58	38.30	46.45	8.15	30.46	30.09	-0.37
Total	58.90	70.57	11.67	43.19	49.77	6.58	38.15	45.81	7.66

^{*}Data are given as percentages.

Table 4. Stage Distribution of Tongue Cancer by Age at Diagnosis and Period*								
	0-3	9 y	40-	64 y	≥	65 y		
Stage	1973-1984	1985-1997	1973-1984	1985-1997	1973-1984	1985-1997		
Localized	112 (54.9)	232 (56.2)	1120 (38.8)	1282 (36.8)	877 (39.6)	1431 (42.7)		
Regional	54 (26.5)	127 (30.8)	1172 (40.6)	1561 (44.8)	820 (37.1)	1243 (37.1)		
Distant	13 (6.4)	28 (6.8)	375 (13.0)	344 (9.9)	250 (11.3)	304 (9.1)		
Unstaged	25 (12.3)	26 (6.3)	219 (7.6)	298 (8.6)	265 (12.0)	370 (11.1)		

^{*}Data are given as number (percentage).

rate fluctuations, caused by relatively small numbers in several single years, would change the direction of the trend owing to true randomness. On the other hand, joinpoint regression modeling actually accounts for the limited robustness of data and allows us to improve accuracy in trend estimates. It seems unlikely that certain artefactual factors, for instance, improved diagnostic methods, various screening activities, and changes in tumor coding, have led to an increased incidence trend for tongue cancer in young Americans. First, diagnostic methods have not significantly changed for tongue cancer since the 1970s. Second, no specific screening strategies for head and neck cancer in the SEER Program registries have existed during the past 25 years. In addition, the SEER Program has not instituted any changes in either anatomic or histologic coding.

Our analysis reveals that the most significant increase in tongue cancer in young Americans was related to birth cohorts between 1938 and 1948. If birth cohorts since 1938 are primarily responsible for the increase in young adult tongue cancer, then a similar cohort-specific increase should also emerge in other age groups as the population ages. In fact, we found an increase in cohort-specific incidence rates in the age groups of 40 to 44 and 45 to 49 years when corresponding birth cohorts are available (Figure 2). This suggests that birth cohorts since 1938 may have experienced a changing carcinogenic effect on the oral tongue, carcinogen(s) distinct from those contained within tobacco. According

to the National Health Interview Survey, the prevalence of cigarette smoking has markedly decreased among Americans since the mid-1960s.²² Similarly, cigarette smoking prevalence among youths also declined sharply in the 1970s, although the decline slowed significantly in the 1980s.²³ Similar to cigarette smoking, alcohol use shows a decline since the 1970s. 24,25 Results of other studies9,26,27 indicate that smoking-related cancers such as those of the lung, larynx, and oral cavity are actually declining in North America. Age-specific analyses of lung cancer revealed that rates in males first declined at younger ages and then for each older age group successively over time.26 Our analysis of upper aerodigestive tract cancers other than tongue cancer would support that conclusion (Table 1). Thus, some time-dependent factors associated with changing behavior and environmental exposures should be reviewed in relation to the decreasing effect of tobacco use and alcohol consumption.

One such factor may be smokeless tobacco use (moist snuff and chewing tobacco). There has been strong evidence that smokeless tobacco can cause oral cancer and precancerous oral lesions (leukoplakia).^{7,28-31} Smokeless tobacco use has increased dramatically in the United States during the past 30 years.^{28,32} An unexplainable fact, however, is that young American women showed significant increases in tongue cancer from 1973 to 1997 but their prevalence of smokeless tobacco use is extremely low, only 0.3%,³³ and has not significantly changed since the 1970s. Likewise, smokeless tobacco should induce can-

cer in regions where it is held in contact, such as the cheek or gum, but the incidence of these nontongue oral cancers has remained relatively stable.

Another significant factor may be marijuana use. Approximately 31% of the US population 12 years or older in 1992 had ever used marijuana.34 The prevalence of marijuana use sharply increased in the United States in male and female teenagers and young adults in the mid- to late-1960s, that is, in cohorts born between 1941 and 1955 (Figure 3).34-36 Assuming that marijuana use is associated with cancer risk with an induction or latency period of 20 to 30 years, persons born between 1941 and 1955 will be the earliest possible group to experience and clinically manifest elevated risks of tongue cancer. The assumption seems consistent with our findings (ie, a higher incidence of tongue cancer in birth cohorts beginning in 1938). The carcinogenic effect of marijuana has been concluded from a series of case report, laboratory, and experimental evidence.³⁷⁻⁴⁰ Recently, Zhang et al⁴¹ reported a dose-dependent association between marijuana smoking and head and neck cancer risk. The risk was more pronounced in young patients.⁴¹ However, current studies could not prove causation between marijuana use and tongue cancer; therefore, a well-designed study is necessary in the future.

A third suspected factor may be human papillomavirus (HPV) infection. There is evidence for the correlation of HPV infection with oral cavity carcinoma. 42 Positivity for HPV in oral neoplastic tissues varies from 14% to 91%. 43-45 An approximately 5-fold higher expression of HPV can be seen in oral squamous carcinoma than in normal oral mucosa. 43 Two types of HPV (types 16 and 18) seem to be carcinogenic and show a close correlation with p53 mutation.46 Results of laboratory studies show that HPV integration into the oral epithelial cell genome will lead to cellular immortalization and set the stage for enhancing the carcinogenic potential of relevant chemical compounds. 47 To date, however, we are not clear whether HPV infection increases over time in young Americans, and the increase is one of the responsible factors for the rising trend of oral tongue cancer.

In our analysis, the stage and survival characteristics of tongue cancer in individuals born since the early 1940s suggest a distinct disease process. In these younger Americans, the disease typically presents itself in earlier stages and is associated with a higher 5-year determinant survival rate than in older adults. This observation supports the conclusions of Lacy et al,48 who, likewise, concluded in their analysis of a hospital registry that young adults have a more favorable disease process. Approximately 56% of the young adults in the SEER Program presented with localized disease compared with 37% and 43% localized disease in the 40 to 64 year olds and those 65 years and older, respectively. We found that 5-year survival of 70.5% in young patients in 1985-1997 reported by 9 SEER Program registries was comparable with that seen in several other hospital-based observations.^{8,49,50} The 5-year survival rate did not appreciably change in young patients with localized disease between 1973-1984 and 1985-1997. In contrast, 5-year survival has improved more than 20% in young patients with regional and metastatic disease. The improved survival rate and earlier staged disease were not

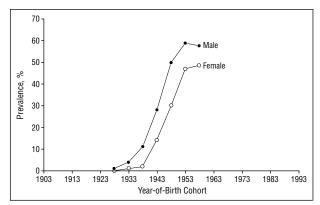


Figure 3. Lifetime prevalence of marijuana use in the US population according to birth cohort. Data from Johnson and Gerstein.³⁴

observed in the young black population, which has a dismal prognosis and a far greater percentage of individuals dying of metastatic disease. This suggests the possibility that various socioeconomic strata are contributing to the emergence of a distinctly different disease process. The survival disadvantage for young black patients with tongue cancer needs further investigation.

CONCLUSIONS

There has been a sharply increasing trend in the incidence of tongue cancer in young Americans since the mid-1970s. The increase may be attributed to persons who were born between 1938 and 1947 and is associated with a less virulent disease course. One contributing factor for the rising trend of tongue cancer in young Americans may be the use of marijuana. Its possible association should continue to be monitored. In addition, other etiologic clues may be gleaned from the changing incidence patterns associated with race and, most likely, socioeconomic status. One must look to lifestyle factors that changed during the 1940s that seem to be more prevalent in the higher socioeconomic strata. Potential factors are diverse and could reflect changes in dental care, diet, food processing, nutritional supplementation, sexual habits, etc. The potential for interaction of each of these factors on biologic processes involving oral tongue mucosa must also be considered. The concern is that observed trends are a "tip-of-the-iceberg" phenomenon. As the current North American population ages, will the incidence of this changing tongue cancer disease process also increase? Continued vigilance involving this phenomenon is required.

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REFERENCES

 Macfarlane GJ, Boyle P, Scully C. Rising mortality from cancer of the tongue in young Scottish males [letter]. Lancet. 1987;2:912.

- 2. Macfarlane GJ, Boyle P, Scully C. Oral cancer in Scotland: changing incidence and mortality. BMJ. 1992;305:1121-1123.
- 3. Shemen LJ, Klotz J, Schottenfeld D, Strong EW. Increase of tongue cancer in young men [letter]. JAMA. 1984;252:1857.
- 4. Depue RA. Rising mortality from cancer of the tongue in young white males [letter]. N Engl J Med. 1986;315:647.
- 5. Franchesci S, Levi F, Lucchini F. Trends in cancer mortality in young adults in Europe, 1955-1989. Eur J Cancer. 1994;30A:2096-2118.
- 6. Shiboski CH, Shiboski SC, Silverman SJ. Trends in oral cancer rates in the United States, 1973-1996. Community Dent Oral Epidemiol. 2000;28:249-256.
- 7. Chen JK, Eisenberg E, Krutchkoff DJ, Katz RV. Changing trends in oral cancer in the United States, 1935 to 1985: a Connecticut study. J Oral Maxillofac Surg. 1991;49:1152-1158.
- 8. Myers JN, Elkins T, Roberts D, Byers RM. Squamous cell carcinoma of the tongue in young adults: increasing incidence and factors that predict treatment outcomes. Otolaryngol Head Neck Surg. 2000;122:44-51.
- 9. McKean-Cowdin R, Feigelson HS, Ross RK, Pike MC, Henderson BE. Declining cancer rates in the 1990s. J Clin Oncol. 2000;18:2258-2268.
- 10. Mattson ME, Winn DM. Smokeless tobacco: association with increased cancer risk. NCI Monogr. 1989;16:13-16.
- 11. Sankaranarayanan R, Mohideen MN, Nair MK, Padmanabhan TK, Ainslie N. Aetiology of oral cancer in patients less than or equal to 30 years of age. Br J Cancer. 1989;59: 439-440.
- 12. Endicott JN, Skipper P, Hernandez L. Marijuana and head and neck cancer. Adv Exp Med Biol. 1994;335:107-113.
- 13. Das C, Schantz SP, Shillitoe EJ. Antibody to a mutagenic peptide of herpes simplex virus in young adult patients with cancer of the head and neck. Oral Surg. 1983:75:610-614
- 14. Schantz SP, Hsu TC, Ainslie N, Moser RP. Young adults with head and neck cancer express increased susceptibility to mutagen-induced chromosomal damage. JAMA. 1989:262:3313-3315.
- 15. Schantz SP, Byers RM, Goepfert H, Shallenberger RS, Beddingfield N. The implication of tobacco use in the young adult with head and neck cancer. Cancer. 1988:62:1374-1380
- 16. Percy C, Van Holten V, Muir C. International Classification of Diseases for Oncology. 2nd ed. Geneva, Switzerland: World Health Organization; 1990.
- 17. Ries LAG, Eisner MP, Kosary CL, et al. SEER Cancer Statistics Review, 1973-1997. Bethesda, Md: National Cancer Institute; 2000.
- 18. Kim HJ, Fay MP, Feuer EJ, Midthune DN. Permutation tests for joinpoint regression with applications to cancer rates. Stat Med. 2000;19:335-351.
- 19. Hankey BF, Feuer EJ, Clegg LX, et al. Cancer surveillance series: interpreting trends in prostate cancer, part I: evidence of the effects of screening in recent incidence, mortality, and survival rates. J Natl Cancer Inst. 1999;91:1017-1024.
- 20. Robertson C, Boyle P. Age, period and cohort models: the use of individual records. Stat Med. 1986;5:527-538.
- 21. National Institutes of Health. Summary Staging Guild for the Cancer Surveillance, Epidemiology and End Results Reporting (SEER) Program. Bethesda, Md: National Institutes of Health; 1986.
- 22. US Department of Health and Human Services. Preventing Tobacco Use Among Young People: A Report of the Surgeon General. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking on Health; 1994.
- 23. US Department of Health and Human Services. Tobacco Use Among U.S. Racial/ Ethnic Minority Groups. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking on Health;
- 24. The Gallup Organization. Percentage of Americans who drink alcoholic beverages, 1939-2000. Available at: http://www.gallup.com/poll/indicators/indalcohol.asp. Accessed November 20, 2001
- 25. Johnson LD, O'Malley PMOH, Bachman JG. University of Michigan, Institute for Social Research, National Survey Results on Drug Use From the Monitoring the Future Study, 1975-1997, Volume I: Secondary School Students. Rockville, Md: National Institutes of Health; 1999.
- 26. Wingo PA, Ries LA, Giovino GA, et al. Annual report to the nation on the status of cancer, 1973-1996, with a special section on lung cancer and tobacco smoking. J Natl Cancer Inst. 1999;91:675-690.

- 27. Cole P, Rodu B. Declining cancer mortality in the United States. Cancer. 1996;
- 28. Cullen JW, Blot W, Henningfield J, Boyd G, Mecklenburg R, Massey MM. Health consequences of using smokeless tobacco: summary of the Advisory Committee's report to the Surgeon General. Public Health Rep. 1986;101:355-
- 29. Martin GC, Brown JP, Eifler CW, Houston GD. Oral leukoplakia status six weeks after cessation of smokeless tobacco use. J Am Dent Assoc. 1999;130:945-
- 30. Grady D, Greene J, Daniels TE, et al. Oral mucosal lesions found in smokeless tobacco users. J Am Dent Assoc. 1990;121:117-123.
- 31. Schantz SP, Byers RM, Goepfert H. Tobacco and cancer of the tongue in young adults. JAMA. 1988;259:1943-1944.
- 32. US Department of Health and Human Services. Spit Tobacco and Youth. Washington, DC: US Dept of Health and Human Services, Office of the Inspector General: 1992.
- 33. Centers for Disease Control and Prevention. Use of smokeless tobacco among adults-United States, 1991. MMWR Morb Mortal Wkly Rep. 1993;42:263-
- 34. Johnson RA, Gerstein DR. Initiation of use of alcohol, cigarettes, marijuana, cocaine, and other substances in US birth cohort since 1919. Am J Public Health. 1998:88:27-33
- 35. Johnston LD, O'Malley PMOH, Bachman JG. National Survey Results on Drug Use From Monitoring the Future Study, 1975-1994, Volume I: Secondary School Students. Washington, DC: National Institute on Drug Abuse; 1995.
- 36. Johnston LD, O'Malley PMOH, Bachman JG. National Survey Results on Drug Use From Monitoring the Future Study, 1975-1994, Volume II: College Students and Young Adults. Washington, DC: National Institute on Drug Abuse;
- 37. Fligiel SE, Roth MD, Kleenup EC, Barsky SH, Simmons MS, Tashkin DP. Tracheobronchial histopathology in habitual smokers of cocaine, marijuana, and/or tobacco. Chest. 1997;112:319-326.
- 38. Tashkin DP. Is frequent marijuana smoking harmful to health? West J Med. 1993; 158.635-637
- 39. Barsky SA, Roth MD, Kleenup EC, Simmons MS, Tashkin DP. Histopathologic and molecular alterations in bronchial epithelium in habitual smokers of marijuana, cocaine, and/or tobacco. J Natl Cancer Inst. 1998;90:1198-1205.
- 40. Firth NA. Marijuana use and oral cancer: a review. Oral Oncol. 1997;33:398-401.
- 41. Zhang ZF, Morgenstern H, Spitz MR, et al. Marijuana use and increased risk of squamous cell carcinoma of the head and neck. Cancer Epidemiol Biomarkers Prev. 1999;8:1071-1078.
- 42. Gillison ML, Koch WM, Shah KV. Human papillomavirus in head and neck squamous cell carcinoma: are some head and neck cancers a sexually transmitted diseases? Curr Opin Oncol. 1999;11:191-199.
- 43. Miller CS, Johnstone BM. Human papillomavirus as a risk factor for oral squamous cell carcinoma: a meta-analysis, 1982-1997. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;91:622-635.
- 44. Bouda M, Gorgoulis VG, Kastrinakis NG, et al. "High risk" HPV types are frequently detected in potentially malignant and malignant oral lesions, but not in normal oral mucosa. Mod Pathol. 2000;13:644-653.
- 45. Adler-Storthz K, Ficarra G, Woods KV, Gaglioti D, Dipietro M, Shillitoe EJ. Prevalence of Epstein-Barr virus and human papillomavirus in oral mucosa of HIVinfected patients. J Oral Pathol Med. 1992;21:164-170.
- 46. Shima K, Kobayashi I, Saito I, et al. Incidence of human papillomavirus 16 and 18 infection and p53 mutation in patients with oral squamous cell carcinoma in Japan. Br J Oral Maxillofac Surg. 2000;38:445-450.
- 47. Li SL, Kim MS, Cherrick HM, Doniger JP, Park NH. Sequential combined tumorigenic effect of HPV-16 and chemical carcinogens. Carcinogenesis. 1992;13: 1981-1987.
- 48. Lacy PD, Piccirillo JF, Merritt MG, Zegueira MR. Head and neck squamous cell carcinoma: better to be young. Otolaryngol Head Neck Surg. 2000;122:253-
- 49. Lydiatt DD, Robbins KT, Byers RB, et al. Treatment of stage I and II oral tongue cancer. Head Neck. 1993;15:308-312.
- 50. Friedlander PL, Schantz SP, Shaha AR, Yu GP, Shah JP. Squamous cell carcinoma of the tongue in young patients: a matched-pair analysis. Head Neck. 1998; 20:363-368