Published in final edited form as:
Cancer Causes Control. 2010 April ; 21(4): 567–575. doi:10.1007/s10552-009-9486-9.

Oral health and risk for head and neck squamous cell carcinoma: the Carolina Head and Neck Cancer Study

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Abstract

Objective—Recent reports have linked oral health and periodontal disease indicators with increased risk of squamous cell carcinoma of head and neck (SCCHN). Thus far, evidence has been inconclusive; our objective was to study the association between oral health and SCCHN risk in the context of a large population-based study.

Methods—A population-based case–control study of incident SCCHN, the Carolina Head and Neck Cancer Study was carried out in 2002–2006 in 46 counties in North Carolina. Controls (n = 1,361) were frequency matched with cases (n = 1,289) on age, race, and gender. Oral health was

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assessed using interview data on tooth loss and mobility, mouthwash use, and frequency of dental visits.

Results—Subjects were 26–80 years old (median age = 61). The distribution of tooth loss among controls was 0–5 teeth = 60%; 5–14 = 15%; and 16–28 = 25%. After controlling for covariates, tooth loss did not yield any notable association with SCCHN (16–28 vs. 0–5 lost teeth: OR: 1.21, 95% CI: 0.94, 1.56). Self-reported history of tooth mobility was moderately associated with increased SCCHN risk (OR: 1.33, 95% CI: 1.07, 1.65); however, the association did not persist among never smokers. Routine dental visits were associated with 30% risk reduction (OR: 0.68, 95% CI: 0.53, 0.87).

Conclusions—These data provide support for a possible modest association of periodontal disease, as measured by self-reported tooth loss indicators, but not tooth loss per se, with SCCHN risk.

Keywords

Case–control studies; Population-based studies; Head and neck cancer; Oral cancer; Oropharyngeal cancer; Laryngeal cancer; Risk factors; Oral health; Periodontal disease; Dental attendance

Introduction

Squamous cell carcinoma of the head and neck (SCCHN) includes cancer of oral cavity, pharynx, and larynx and has been recognized as a significant component of the global burden of cancer [1–3]. These cancers are characterized by marked geographic, racial, and gender disparities [2,4,5]. For oral and pharyngeal cancer, incidence rates in the United States were 15.7/100,000 men and 6.1/100,000 women per year for 2001–2005, whereas annual mortality rates were 3.8/100,000 for white and 6.7/100,000 for black men, for the same period. Based on these data, it is estimated that one out of 99 men and women born in the United States today will be diagnosed with oral/oropharyngeal cancer (OPC) during their lifetime [6]. Notably, oral cancer was recently included among the World Health Organization's (WHO) priorities for action [7,8].

Tobacco use and alcohol consumption have been well established as the predominant etiologic factors for SCCHN, with their population-attributable risk for OPC in the United States estimated to be 74% [9]. Human papillomavirus (HPV) infections have also been implicated in the etiology of SCCHN [10]. These infections may have a substantial role among non-smoker and non-drinker cases [11,12], and viral types identified as high risk in cervical cancer studies (HPV-16) have been shown to confer greater risk [13,14]. Conversely, dietary factors, specifically fruit and vegetable intake and several micronutrients have been consistently associated with decreased SCCHN risk [15–21].

Multiple studies have examined the potential association of oral health parameters with SCCHN risk [16,17,20–41], but the evidence has been inconclusive. Earlier investigations reported substantially increased risk estimates (typically odds ratios between 2 and 5) for higher strata of tooth loss (15 or more lost teeth) [17,25–28,33,36], but were based on small samples. Notably, tooth loss and mouthwash use were found to be associated with increased with for SCCHN in a recent large multi-center study [24] but a prospective investigation from the health professionals follow-up study did not report any important risk increases for tooth loss [23]. Other studies have also implicated alcohol-containing mouthwash use as a risk factor for SCCHN [22,35]. Furthermore, in the study of Day et al. [40] heterogeneity was observed between African-Americans and whites for tooth loss risk estimates.

The objective of the present investigation was to examine the association of oral health indicators with the risk for oral, pharyngeal and laryngeal cancer within a large population-based study of squamous cell head and neck cancer, the Carolina Head and Neck Cancer Study (CHANCE), in North Carolina. Secondary aims were to determine whether African-Americans have a distinct oral health risk profile and explore potential interactions between oral health variables and other factors such as tobacco and alcohol use.

Materials and methods

CHANCE is population-based case–control study of incident SCCHN that was conducted between 1 January 2002 and 28 February 2006 in North Carolina. Cases were adults aged 20–80, residents of a 46 county region of North Carolina, with newly diagnosed first primary invasive squamous cell carcinoma of the oral cavity and pharynx (ICD-O-3 topography codes C0.00 to C14.8) and larynx (C32.0 to C32.9). Case ascertainment relied on rapid identification of newly diagnosed cancer cases through the North Carolina Central Cancer Registry (NCCCR). The cancer registrars of 54 hospitals in the study area were contacted monthly to identify potentially eligible cases. Pathology reports and sociodemographic data were sent to the coordinating center at the University of North Carolina at Chapel Hill, usually within 4–8 weeks of diagnosis. Controls were adults aged 20–80, residents of the same 46 county region of North Carolina, who had never been diagnosed with head and neck cancer. Potentially eligible controls were identified through the North Carolina Department of Motor Vehicle records and were frequency matched with cases using random sampling with stratification on age, race, and sex. Potentially eligible study subjects were mailed a brochure describing the purpose of the study, and upon consent, a study nurse conducted an at-home in-person interview. Contact and cooperation rates were 98 and 82% for cases, and 80 and 61% for controls, respectively. Demographic, lifestyle, oral health, dietary, and other common risk factor information was collected using a structured questionnaire during an in-home visit, which was completed by the study subject or by a proxy if the subject was deceased, and biological samples (blood or mouthwash samples) were obtained for future analyses. The study was approved by the Institutional Review Board (IRB) of the University of North Carolina at Chapel Hill, and all participating institutions.

Self-reported oral health variables included (1) self-reported number of natural teeth lost excluding third molars and teeth extracted due to orthodontic reasons (range = 0–28), (2) history of self-reported tooth mobility, or “teeth loose in their socket due to disease” (dichotomous, referent category: no), (3) regular use of mouthwash (dichotomous, referent category: no), (4) one or more routine (non-emergency) dental visits during the decade prior to SCCHN diagnosis (dichotomous, referent category: no). For purposes of the present analysis, tooth loss was coded as a three-level indicator variable, with 0–5 lost teeth as the referent category, and 6–15 and 16–28 lost teeth as the moderate and severe tooth loss groups, respectively.

Terms for the matching factors age, sex, and race, as well as their pairwise product terms were included in all models. Additional covariates were education level, smoking status and intensity, drinking status, cumulative lifetime alcohol consumption, and fruit and vegetable intake (servings per week divided by total caloric intake). The linearity assumption was evaluated graphically, by plotting the corresponding ln(odds) of SCCHN risk for covariate categories and was confirmed with formal tests of linearity (Wald X^2; $\alpha = 0.05$). Age was coded as a seven-level indicator variable [20–49 (referent), 50–54, 55–59, 60–64, 65–69, 70–74 and 75–80 years]. Race included whites, African-Americans, and others. Education was coded as a three-level categorical variable: less than college (referent), college, and more than college education. Indicator terms were used to classify subjects as never-, ex-, or

current smokers and drinkers. Smoking intensity was coded with a four-level categorical variable with terms for never smoker, 0–19, 20–39, and 40+ cigarettes per day. Cumulative lifetime alcohol consumption (ethanol grams) was calculated as the sum of duration by frequency of consumption products for individual alcoholic beverages (beer, liquor, wine) and was entered in the models as a continuous variable. We adjusted for dietary behavior using a quintile-categorical variable derived from total fruit and total vegetable weekly intake divided by total caloric intake, according to the nutrient-density method [42].

Odds ratios for the association between oral health variables and risk of SCCHN were obtained using unconditional logistic regression. The final logistic model included all oral health variables, the cross-product terms for the matching variables, and education level, smoking status and intensity, drinking status, cumulative alcohol consumption, and fruit and vegetable density intake. The rationale for mutual adjustment for the oral health variables lies on the complex network of associations between oral health status (periodontal disease presence), the number of natural teeth present (natural teeth provide more sites for bacterial adhesion and infection) and dental visits (may be considered either as an ‘upstream’ or ‘downstream’ dental behavior). We also examined oral health factor effects stratified by cancer site (oral, pharyngeal, laryngeal), race (whites, African-Americans), smoking and drinking status (never/ever smokers, never/ever drinkers, never/ever smokers and drinkers) and tooth loss level. Potential interactions between tooth loss, history of tooth mobility, and covariates were evaluated on the additive scale, by estimating interaction contrast ratios/ relative excess risk due to interaction (ICR/RERI) across strata of exposure and covariates [43]. Data analyses were conducted with Stata version 10.1 software (StataCorp LP, College Station, TX).

Results

Interview data were available for 1,389 cases and 1,396 controls. For purposes of the present analysis, 21 cases of lip cancer (1.5% of all cases) were excluded, as well as proxy interview data (69 observations or 2.5% of the total sample), subjects of ‘other’ race (46 or 1.7%) and those with extensive missing covariate information (four or more variables: 38 subjects or 1.4%). The final dataset included 2,650 subjects, with 1,289 cases and 1,361 controls (93 and 97% of eligible cases and controls, respectively). Among cases, 76% were males, and 74% were whites. At the time of the interview, cases had mean (median) age 58.9 (59), and controls 61.5 (63). The distribution of cases by primary cancer site was 692 (54%) oral, 136 (10%) pharyngeal, and 461 (36%) laryngeal. The distribution of sociodemographic, tobacco use, and alcohol consumption data are presented in Table 1.

Results from the analysis of the oral health variables are presented in Table 2. Marked differences in the prevalence and severity of tooth loss were noted between cases and controls: among cases, 44% had lost 0–5 and 39% had lost 15–28 teeth, while these proportions were 60 and 25% among controls. The attenuation of the minimally adjusted estimates (minimally adjusted model A) presented in the Table 2 was attributed primarily to adjustment for smoking status and intensity. A model adjusting for these variables, as well as the matching factors and education (minimally adjusted model B), resulted in attenuated tooth loss estimates [5–14 missing teeth: OR: 1.14 (95% CI: 0.88, 1.49); 15–28 missing teeth: OR: 1.26 (95% CI: 0.99, 1.59)]. The estimates of tooth loss, tooth mobility and preventive dental visits were minimally attenuated when all covariates were included in the model. Tooth loss did not show any pattern of association with the exception of the severe tooth loss category (16 or more lost teeth vs. 5 or less) that was associated with a weakly elevated estimate: OR: 1.21 (95% CI: 0.94, 1.56). History of tooth mobility was associated with a 33% increase in risk, whereas dental visits were associated with a 32% decrease in risk. Mouthwash use was also not associated with SCCHN. A weak ‘more than additive’

interaction was observed between moderate tooth loss (6–15 lost teeth) and mobility (ICR: 0.53, 95% CI: −0.48, 1.53). Tooth mobility was consistently associated with increased cancer risk for all sites (Table 3). Consistent use of mouthwash was not associated with any notably SCCHN risk change, except for a 32% decreased risk for pharyngeal cancer (Table 3).

Stratified results by smoking and drinking history are presented in Table 4. Within the stratum of never smokers, there was no association between tooth loss and mobility with SCCHN risk. In order to evaluate possible interaction between these factors, based on the full model, we created an interaction table with the stratum of never smokers and no mobility as referent (OR: 1.00; cases = 144), which yielded the following estimates: ex- or current smokers and no mobility OR: 1.31 (95% CI: 0.98, 1.74; cases = 657); never smokers and reported mobility OR: 0.69 (95% CI: 0.40, 1.17; cases = 26); ex- or current smokers and reported mobility OR: 2.03 (95% CI: 1.45, 2.84; cases = 455). Therefore, superadditive interaction ICR: 1.03 (95% CI: 0.44, 1.62) was noted between tooth mobility and smoking status (ever vs. never smoker). This estimate corresponds to the excess risk experienced by those who reported both tooth mobility and were ever smokers, relative to the risk experienced by those with no tooth mobility and no history of smoking. There was no important interaction between mobility and drinking status (ICR: 0.29, 95% CI: −0.37, 0.95). Finally, even though some variability was observed, dental visits were consistently associated with decreased SCCHN risk, particularly in strata of smokers and drinkers.

Further stratification by tooth loss status is presented in Table 5. History of tooth mobility did not result in any risk increase among those with no tooth loss, whereas the association of dental visits was more pronounced among those with no or minimal tooth loss (0–5 lost teeth: ICR: −0.44, 95% CI: −1.03, 0.16). With regard to race, tooth mobility emerged as a modest risk factor among whites (OR: 1.42, 95% CI: 1.11, 1.82), whereas an elevated risk estimate for mouthwash use was only noted among African-Americans (OR: 1.50, 95% CI: 0.86, 2.62) (Table 6). Some of the effect estimates were imprecise.

Discussion

Our results do not provide strong support for any positive association between tooth loss and SCCHN risk, in contrast to some recent case control studies employing tooth loss as a measure of oral health have reported [22,24,25,27,28]. With regard to the etiology of tooth loss, it has been well established that periodontal disease is the major cause among older adults [44], although caries reportedly retains a substantial role in younger adults [45]. However, recent evidence indicates that a substantial proportion of teeth, up to 30–40%, are extracted in older age patient groups because of caries and its sequelae, not periodontal disease [46]. In a sample of US adults aged 40–59 with dental insurance, 51% of extractions were due to periodontal disease, 35% due to caries, and 10% for a combination of the two, whereas when considering patients as the unit of analysis, 58% of the patients had an extraction because of caries [47]. In order to disentangle the effects of caries and periodontal disease from tooth loss, we included in our analysis an additional oral health variable ‘history of tooth mobility’ or ‘pathologic tooth mobility, not as a result of trauma.’ The latter is the immediate precursor of periodontitis-related extractions [48], and we believe that is more sensitive in capturing periodontal disease effects, compared to tooth loss alone. Notably, that variable was associated with weakly elevated risk for SCCHN in our study, thus providing support for a possible association of periodontal disease with head and neck cancer risk [49]. The superadditive interaction of tooth mobility with smoking history provides support to possible mechanistic synergistic effects of these factors, whereas a null association between mobility and SCCHN risk was observed within strata of never smokers.

Proposed mechanisms linking periodontal disease with the risk of SCCHN include systemic and local pathways. Periodontitis is a relatively common chronic inflammatory disease that affects approximately 10–15% of the adult population in its severe form [44,50]. In the United States, results from the NHANES III study indicate that at least 35% of the dentate US adults aged 30 to 90 have periodontitis; with 22% having a mild form and 13% having a moderate or severe form [51]. Periodontitis is associated with elevated inflammatory markers [52–55] and thus may increase the risk for numerous adverse health outcomes [56,57], including vascular disease [58]; coronary heart disease and cardiovascular events [59,60]; stroke [60,61]; adverse pregnancy outcomes [56,62]; and total cancer [23]. This chronic infection and resultant low-grade systemic inflammatory response may be one pathway of carcinogenesis promotion [63,64], although alteration of DNA methylation patterns may also play a role [65]. Other investigators have proposed that oral ecological shifts accompanying periodontal disease are characterized by proliferation of ketone-producing and nitrate-reducing microorganisms. The latter may contribute to increases in carcinogen concentrations [24,66], which is consistent with evidence of oral metabolism of alcohol to acetaldehyde [67,68]. Recent evidence indicates that common cariogenic microorganisms such as oral *Streptococci* may contribute substantially to increased acetaldehyde concentration in the oral cavity [69]. Hooper et al. [41] in their systematic review surmised that there is sufficient evidence to suggest that epidemiological and etiological links between microbial infection in the oral cavity and oral cancer could exist.

Our stratified analyses showed a 53% risk increase of laryngeal cancer for moderate (6–15) and 49% increase for severe (16–28) tooth loss, but these associations were imprecise. Similar elevated estimates for tooth loss (6–15 missing teeth vs. 5 or less) and risk for laryngeal cancer were reported by Guha for the Latin American part of that study (OR: 1.7, 95% CI: 1.2, 2.4), while the estimates for oral and pharyngeal sites where lower (OR: 0.87, 95% CI: 0.56, 1.4, and OR: 1.4, 95% CI: 1.0, 2.0, respectively) [24]. Despite the imprecise estimates, we believe that these associations deserve attention and warrant additional study.

The finding that routine dental visits are associated with substantial decrease in the risk of SCCHN is in concordance with previous investigations [20,22,24,25,[28],29,33,34,38] and has important public health implications. In recent years, the oral health professionals' role in the early detection of oral cancer and management of pre-cancerous lesions has been emphasized by international organizations [7,8]. It must be noted, however, that oral health and dental attendance [70], as well as SCCHN [71], are all strongly associated with socioeconomic status and there exists the potential of observing a spurious association. Even if dental attendance may be merely an indicator of favorable self-care attitudes and socioeconomic status, it can be argued that professional dental checkups and interventions contribute to a healthier oral ecology with less pathogenic microflora.

Our data did not support a strong link of mouthwash use with SCCHN risk change, which is consistent with previous investigations [37,72–74]. Other reports have related alcoholic mouthwash use with increased risk for oral and pharyngeal cancers [22,24,75]. However, a critical review of the evidence linking specifically alcohol-containing mouthwashes with SCCHN risk did not support this hypothesis [76]. It has been suggested that spurious findings regarding mouthwash use may arise due to underascertainment of exposure and residual confounding from alcohol and tobacco use [77]. Generally, any finding regarding mouthwash use should be treated with caution because there is usually scarce information on specific products used and their alcoholic content, as well as comprehensive usage frequency data. Of note, however, is our finding that marginally elevated estimates for consistent mouthwash use were observed among African-Americans and within strata of never smokers and never drinkers, which is similar to the findings of Winn [72] and Blot [74].

It is evident from the inconclusive evidence in the literature, as well as from the present study, that more specific and valid oral health indicators are necessary for epidemiologic studies investigating the potential role of oral conditions with SCCHN risk. Tooth loss is the end-point of a series of events initiating in childhood and may be confounded by a variety of factors, including self-care attitudes, insurance status, access to care, and level and quality of dental care received [23]. Moreover, discrepancies between self-reports of periodontal health and actual clinical status have been documented in previous investigations [78,79]. For example, in a prospective cohort study of male health professionals (HPFS—Health Professionals Follow-up Study), although risk estimates were elevated [hazards ratio (HR): 1.2; 95% CI: 0.69, 2.0] for moderate and (HR: 1.6; 95% CI: 0.84, 3.0) for severe tooth loss, the hazard ratio for self-reported periodontal disease with bone loss was lower (HR: 1.2, 95% CI: 0.73, 1.8) [23]. In spite of this, the use of self-reported tooth loss in epidemiologic studies has been proposed as valid approach for estimating clinical dental findings [80]. Despite some variation in self-reports [81], a congruence between self-reported remaining teeth and professional clinical examinations has been established [79,82–89]. Specifically, Buhlin [79] detected a (mean = 1.4; SD = 1.7) difference of self-reported versus clinically confirmed lost teeth, and in an earlier investigation, Douglass [85] reported a 0.97 correlation coefficient between the two measures. The incorporation of self-reported history of tooth mobility as a periodontal disease-specific indicator should be regarded as a minor improvement compared to previous investigations.

In conclusion, our data provide support for a possible modest association of periodontal disease, as measured by self-reported tooth loss indicators but not tooth loss per se, with SCCHN risk. The observed interaction between tooth mobility and smoking provides ground for additional research on mechanistic pathways of these two factors. The finding that routine dental visits were associated with a marked decrease in risk adds to the oral-systemic health interaction hypothesis and underlines the importance of oral disease prevention from a public health perspective. Development and application of more comprehensive indicators of oral health for population-based studies are warranted.

Abbreviations

CHANCE	Carolina Head and Neck Cancer Study
CI	Confidence interval
HR	Hazard ratio
ICR	Interaction contrast ratio
OR	Odds ratio
OPC	Oral/oropharyngeal cancer
SCCHN	Squamous cell carcinoma of the head and neck

Acknowledgments

This study was supported in part by grants R01-CA90731 from the National Cancer Institute and P30ES10126 from National Institute of Environmental Health Sciences.

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Table 1

Demographic and dietary characteristics of the CHANCE study subjects

	Cases		Controls	
	n[a]	%	*n*[a]	%
Sex				
Male	984	76.3	945	69.4
Female	305	23.7	416	30.6
Age (years)				
20–49	253	19.6	156	11.5
50–54	200	15.5	160	11.8
55–59	216	16.8	206	15.1
60–64	217	16.8	205	15.1
65–69	174	13.5	241	17.7
70–74	141	10.9	227	16.7
75–80	88	6.8	166	12.2
Race				
White	959	74.4	1,100	80.8
African-American	330	25.6	261	19.2
Education				
High school or less	798	61.9	540	39.7
Some college	307	23.8	406	29.8
More than college	184	14.3	415	30.5
Smoking status				
Never smoker	170	13.2	521	38.3
Ex-smoker	382	29.6	572	42.0
Current smoker	737	57.2	268	19.7
Smoking intensity (cigarettes per day)				
Never smoker	170	13.2	521	38.3
0–19	225	17.5	332	24.4
20–39	580	45.1	380	27.9
40+	311	24.2	128	9.4
Alcohol use status				
Never drinker	121	9.4	289	21.3
Ex-drinker	440	34.2	318	23.4
Current drinker	724	56.3	751	55.3
Cumulative alcohol consumption (ethanol grams)				
Median	574,430		70,182	
Mean (SD)	1,377,719	(2,375,966)	389,263	(926,246)
Vegetable consumption quintiles (mean servings per day)				
Q1: 1.00	228	18.6	296	21.8
Q2: 1.64	210	17.1	304	22.4
Q3: 2.10	244	19.9	281	20.7

	Cases		Controls	
	n[a]	%	*n*[a]	%
Q4: 2.67	257	20.9	253	18.7
Q5: 3.00	289	23.5	222	16.4
Fruit consumption quintiles (mean servings per day)				
Q1: 0.52	307	25.0	222	16.4
Q2: 1.23	273	22.2	237	17.5
Q3: 2.04	244	19.9	282	20.8
Q4: 3.00	197	16.0	310	22.9
Q5: 5.08	207	16.9	305	22.5

[a]Column totals may not add up to the total number of subjects, due to missing values

Table 2

Minimally and fully adjusted odds ratios (95% confidence intervals) of SCCHN in the CHANCE study for the oral health variables

	Min. adjusted-A[a]		Min. adjusted-B[b]		Full model[c]	
	OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Tooth loss						
0–5	1.0	Referent	1.0	Referent	1.0	Referent
6–15	1.42	1.11, 1.82	1.14	0.88, 1.49	1.07	0.81, 1.42
16–28	1.72	1.38, 2.15	1.26	0.99, 1.59	1.21	0.94, 1.56
Tooth mobility						
No	1.0	Referent	1.0	Referent	1.0	Referent
Yes	1.55	1.28, 1.89	1.40	1.14, 1.71	1.33	1.07, 1.65
Routine dental visits						
No	1.0	Referent	1.0	Referent	1.0	Referent
Yes	0.49	0.39, 0.61	0.59	0.47, 0.74	0.68	0.53, 0.87
Mouthwash use						
No	1.0	Referent	1.0	Referent	1.0	Referent
Yes	0.95	0.80, 1.13	0.92	0.77, 1.11	0.95	0.78, 1.15

Oral health variables estimates are adjusted for all other oral health variables

[a] Adjusted for age, sex, race, and education

[b] Adjusted for age, sex, race, education, and smoking status and intensity

[c] Adjusted for age, sex, race, education, smoking status and intensity, drinking status, cumulative ethanol consumption, and fruit and vegetable consumption

Table 3

Fully adjusted odds ratios (95% confidence intervals) of SCCHN in the CHANCE study for the oral health variables, stratified by cancer site

Cases:	**Oral 692**	**Pharyngeal 136**	**Laryngeal 461**
Tooth loss			
0–5	Referent	Referent	Referent
6–15	0.98 (0.70, 1.38)	0.78 (0.38, 1.60)	1.53 (1.03, 2.28)
16–28	1.05 (0.77, 1.42)	1.18 (0.66, 2.13)	1.48 (1.04, 2.12)
Tooth mobility	1.22 (0.94, 1.59)	1.27 (0.77, 2.10)	1.52 (1.13, 2.05)
Routine dental visits	0.72 (0.53, 0.97)	0.58 (0.34, 1.00)	0.60 (0.43, 0.83)
Mouthwash use	0.97 (0.78, 1.22)	0.68 (0.43, 1.08)	1.00 (0.75, 1.34)

Adjusted for age, sex, race, education, smoking status and intensity, drinking status, cumulative alcohol consumption, fruit and vegetable consumption, and all other oral health variables

Table 4

Fully adjusted odds ratios (95% confidence intervals) of SCCHN in the CHANCE study for the oral health variables, stratified by smoking and drinking status

***n* (Cases):**	**Never smokers 691 (170)**	**Ever smokers 1,959 (1,119)**	**Never drinkers 410 (121)**	**Ever drinkers 2,233 (1,164)**	**Never smokers nor drinkers 250 (50)**	**Ever smokers and drinkers 1,795 (1,045)**
Tooth loss						
0–5	Referent	Referent	Referent	Referent	Referent	Referent
6–15	0.81 (0.38, 1.72)	1.13 (0.83, 1.56)	1.15 (0.51, 2.59)	1.05 (0.77, 1.42)	1.90 (0.56, 6.39)	1.17 (0.84, 1.62)
16–28	0.85 (0.41, 1.73)	1.23 (0.92, 1.63)	1.71 (0.86, 3.41)	1.14 (0.86, 1.51)	1.39 (0.47, 4.09)	1.17 (0.87, 1.58)
Tooth mobility	0.98 (0.53, 1.80)	1.45 (1.14, 1.85)	1.22 (0.67, 2.22)	1.37 (1.08, 1.73)	0.80 (0.28, 2.32)	1.43 (1.11, 1.85)
Routine dental visits	0.84 (0.40, 1.75)	0.69 (0.52, 0.90)	0.90 (0.46, 1.76)	0.66 (0.50, 0.86)	0.99 (0.30, 3.21)	0.66 (0.49, 0.89)
Mouthwash use	1.10 (0.73, 1.65)	0.93 (0.74, 1.16)	1.03 (0.62, 1.72)	0.93 (0.75, 1.15)	0.96 (0.44, 2.12)	0.90 (0.71, 1.14)

Adjusted for age, sex, race, education, smoking status and intensity, drinking status, cumulative alcohol consumption, fruit and vegetable consumption, and all other oral health variables, when appropriate

Table 5

Fully adjusted odds ratios (95% confidence intervals) of SCCHN in the CHANCE study for tooth mobility, dental visits, and mouthwash use, stratified by level of tooth loss

n **(Cases):**	**No tooth loss 456 (185)**	**Some tooth loss 1,651 (748)**	**Complete tooth loss 528 (343)**
Tooth mobility	0.94 (0.36, 2.47)	1.36 (1.03, 1.80)	1.35 (0.86, 2.12)
Routine dental visits	0.38 (0.16, 0.94)	0.62 (0.44, 0.88)	0.95 (0.60, 1.50)
Mouthwash use	0.92 (0.59, 1.45)	0.96 (0.74, 1.23)	0.82 (0.51, 1.31)

Adjusted for age, sex, race, education, smoking status and intensity, drinking status, cumulative alcohol consumption, fruit and vegetable consumption, and all other oral health variables

Table 6

Fully adjusted odds ratios (95% confidence intervals) of SCCHN in the CHANCE study for the oral health variables, stratified by race

***n* (Cases):**	**Whites 2,059 (959)**	**African-Americans 591 (330)**
Tooth loss		
0–5	Referent	Referent
6–15	1.23 (0.89, 1.70)	0.74 (0.39, 1.40)
16–28	1.18 (0.88, 1.58)	1.56 (0.85, 2.86)
Tooth mobility	1.43 (1.11, 1.83)	1.19 (0.72, 1.98)
Routine dental visits	0.72 (0.53, 0.97)	0.74 (0.44, 1.24)
Mouthwash use	0.92 (0.75, 1.13)	1.50 (0.86, 2.62)

Adjusted for age, sex, education, smoking status and intensity, drinking status, cumulative alcohol consumption, fruit and vegetable consumption, and all other oral health variables