



Published in final edited form as:

Sleep Breath. 2016 September ; 20(3): 1095–1102. doi:10.1007/s11325-015-1310-z.

Tooth loss and obstructive sleep apnea signs and symptoms in the US population

Anne E. Sanders¹, Aderonke A. Akinkugbe², Gary D. Slade¹, and Greg K. Essick³

Anne E. Sanders: anne_sanders@unc.edu

¹Department of Dental Ecology, School of Dentistry, University of North Carolina at Chapel Hill, 385 S. Columbia Street, Room 4502, Chapel Hill, NC 27599-7455, USA

²Department of Epidemiology, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, USA

³Department of Prosthodontics and Center for Pain Research and Innovation, School of Dentistry, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599-7455, USA

Abstract

Purpose—The aim of this study is to investigate the relationship between tooth loss and signs and symptoms of obstructive sleep apnea (OSA) in a representative sample of the general US population.

Methods—Data were from 7305 men and women aged 25 years participating in the 2005–2008 National Health and Nutrition Examination Survey. Tooth loss, occlusal contacts, and denture use were determined by dental examination. Four cardinal OSA signs and symptoms were evaluated by questions based on American Academy of Sleep Medicine criteria. Adults with 2 signs/symptoms of OSA were classified at high-risk of OSA. Prevalence ratios (PR) and 95 % confidence limits (CL) from log binomial regression models estimated the strength of association between tooth loss and high-risk for OSA, adjusting for demographic characteristics, body mass index, dentures, and sleep duration.

Results—Prevalence of high-risk for OSA increased 2 % for each additional lost tooth (PR = 1.02, 95 % CL, 1.01, 1.03) among adults aged 25 to 65 years. When tooth loss was modeled as an ordinal variable with 0–4 lost teeth as the referent category, adjusted prevalence of high-risk for OSA was as follows: 25 % greater in those missing 5–8 teeth (PR = 1.25, 95 % CL, 1.07, 1.46); 36 % greater in those missing 9–31 teeth (PR = 1.36, 95 % CL, 1.06, 1.73); and 61 % greater in the edentulous (PR = 1.61, 95 % CL, 1.11, 2.33).

Conclusion—Tooth loss may be an independent risk factor for OSA.

Correspondence to: Anne E. Sanders, anne_sanders@unc.edu.

Compliance with ethical standards

Data collection protocols were approved by the Centers for Disease Control and Prevention/National Center for Health Statistics Ethics Review Board and all participants gave informed consent.

Conflict of interest

The authors declare that they have no competing interests.

Keywords

Epidemiology; Population; Tooth loss; Oral health; Sleep-disordered breathing; NHANES

Introduction

Oral and pharyngeal factors play a prominent role in the pathophysiology of obstructive sleep apnea (OSA). With sleep onset, changes occur in the tonic and phasic contraction of muscles of the upper airway that increase its propensity to collapse, obstructing the passage of air. One of the mechanisms of action in oral appliance therapy is to offset this upper airway collapsibility during sleep [1]. Collapsibility is further exacerbated by excess cervical adipose tissue which narrows the airway space. Compounding these effects, airway space is restricted by enlargement of the tongue, tonsils, and uvula, and narrowing of the airway by the lateral pharyngeal walls [2].

The potential effect of tooth loss on OSA has received less attention. It is well established that the complete loss of teeth—edentulism—leads to morphological changes in the orofacial region that can impact airway patency. There is reduction in the vertical and horizontal dimensions of the alveolar ridges [3], upward rotation of the mandible with decreased lower facial height [4, 5], and retraction of the tongue at rest. [6] In addition, edentulism leads to disuse atrophy of the masseter muscle [7] and to soft tissue changes of the lower lip and chin [8] that increase likelihood of mouth breathing. Chronic mouth breathing shortens the distance between the mandible and hyoid bone, and reduces the retropalatal and retroglottal areas [9], yielding a net reduction in upper airway space [10]. Collectively, these changes predispose to OSA by restricting or obstructing the upper airway. Indeed, spirometry measured during wakefulness suggests that airflow rates are lower when edentulous adults sleep without than with their dentures [11]. Furthermore, morning levels of exhaled nitric oxide and oral nitric oxide are higher when edentulous people sleep without their dentures, indicative of greater airway and oropharyngeal inflammation [12]. Not unexpectedly then, edentulism is associated with OSA [12, 13].

While edentulism is a likely putative factor in upper airway dynamics, only 4.9 % of the US population is edentulous and prevalence is projected to decline [14]. Of greater public health relevance is whether *partial tooth loss* elevates risk for OSA. The effects of airways space following premolar extraction for orthodontic treatment remains inclusive, but the evidence leaves open the possibility that extractions and anterior teeth retraction in adult bimaxillary protrusion cases could promote narrowing of the upper airway [15]. However, more teeth are lost to disease than from orthodontic treatment and this relationship has not been studied. Most studies are restricted to patient groups and evidence is lacking from studies of the general population. We questioned whether any tooth loss—regardless of the reason, location in the arch, and irrespective of prosthetic replacement—is associated with OSA susceptibility. Accordingly, the aim of this study was to evaluate the association between tooth loss and OSA signs and symptoms in a representative sample of the US population.

Materials and methods

Study design, study population, and data collection

The cross-sectional National Health and Nutrition Examination Survey (NHANES) uses a complex, multistage, probability sampling methodology to obtain a representative sample of the civilian non-institutionalized US population. This analysis used data from the 2005–2006 and 2007–2008 cycles of NHANES because these two cycles contained a more comprehensive Sleep Disorders questionnaire than earlier or later cycles. Data were collected during in-home interviews and physical examinations conducted in mobile examination centers. Data collection protocols were approved by the Centers for Disease Control and Prevention/National Center for Health Statistics Ethics Review Board and all participants gave informed consent.

OSA signs and symptoms, susceptibility for OSA, and sleep duration

The Sleep Disorders questionnaire asked about snoring, daytime tiredness, and witnessed apneas (i.e., gasping/choking), which along with hypertension, are four signs/symptoms recommended by the American Academy of Sleep Medicine for OSA screening [16]. NHANES selected these questions based on their administration in highly regarded population-based studies. The questions were adapted from the Sleep Habits Questionnaire used by the Sleep Heart Health Study [17]. This questionnaire in turn was guided by well tested and validated self-reported questions used in the Wisconsin Sleep Cohort Study and the Cleveland Family Study. The Blood Pressure questionnaire asked participants whether they had ever been told by a doctor or other health professional that they had hypertension (yes/no). Participants were classified high-risk for OSA if they reported two or more of the following: hypertension, snoring, daytime tiredness, and witnessed apneas. To meet this classification, participants had to experience snoring, daytime tiredness, and witnessed apneas “frequently”. This classification is consistent with the STOP screening questionnaire for OSA [18], which assigns a status of “high-risk” for OSA to 2 positive responses. We also classified anyone with a doctor’s diagnosis of sleep apnea as high-risk for OSA. Otherwise participants were classified as low-risk. As a measure of sleep duration, participants were asked how much sleep they usually get on weekdays or workdays, to which responses were recorded in whole hours.

Tooth loss and occlusal contacts

Trained health technicians recorded the presence of each of the 32 permanent teeth. In analysis, the number of absent teeth counted was a continuous variable that we refer to as lost teeth. We also created categories of tooth loss: 0–4 lost teeth; 5–8 lost teeth; 9–31 lost teeth; and 32 teeth lost, i.e., being edentulous.

Among adults aged ≥ 25 years, NHANES examiners counted the number of occlusal contacts, to evaluate contacts between opposing teeth. Both left and right posterior regions (premolars and molars) have a maximum of eight zones of contact, yielding up to 16 posterior contacts in total. Inter-rater reliability statistics for technicians’ assessment were excellent. Kappa scores ranged between 0.93 and 1.00 for tooth retention, and between 0.86 and 1.00 for functional contacts.

Covariates

Potential confounding factors were demographic characteristics of age in years, sex, and race/ethnicity. Body mass index (BMI) is strongly associated with OSA and was therefore included in multivariable models. Categories were based on examiner measured height and weight: underweight or healthy <25 kg/m²; overweight 25–<30 kg/m²; and obese ≥30 kg/m². C-reactive protein, a biomarker of systemic inflammation, was explored as a potential explanatory variable. It was classified: low <0.2 mg/dL; intermediate 0.2–<0.5 mg/dL; and high ≥0.5 mg/dL. For adults aged ≥25 years, technicians recorded the presence of a removable complete or partial maxillary and/or mandibular denture. We classified denture status as: having one or more dentures; versus not having a denture.

Statistical analysis

All analyses were conducted in Stata/SE 13.1 (StataCorp. 2013. Stata Statistical Software: Release 13. College Station, TX: StataCorp LP), accounting for the complex sampling design and subpopulation analysis. The binary outcome was being classified high-risk for OSA, i.e., reporting ≥2 signs/symptoms of OSA. A non-parametric test for trend examined the relationship between high-risk for OSA across tooth loss categories. In univariate analysis the Pearson χ^2 test tested the statistical significance of differences ($P < 0.05$) across categories. Effect measure modification was assessed in multivariable modeling via multiplicative interactions between the tooth loss categories and covariates with significantly different stratum specific estimates. Significant effect modification was tested quantitatively using an adjusted Wald test and presented visually with graphics. Because age significantly modified the effect of tooth loss on OSA susceptibility, age-stratified analyses were conducted. Presence of confounding was evaluated by comparing the unadjusted and adjusted effect estimate, accepting a relative post-adjustment change in the beta coefficient (which is the log of the prevalence ratio) greater than 0.10 as probable confounding.

Log binomial regression models estimated prevalence ratios (PR) and 95 % confidence limits (CL) to quantify the strength of association between tooth loss and risk for OSA, adjusting for demographic characteristics, BMI, denture status, and average sleep duration.

Results

Of the 20,497 participants in NHANES 2005–2008, those younger than 25 years ($n = 10,578$) were excluded as they had not been assessed for denture status or number of functional occlusal contacts. Only individuals with complete information about tooth loss and data to classify OSA susceptibility were included, yielding an analytic sample of 7305.

Overall, 20.3 % were classified high-risk for OSA. Prevalence ranged from 18.9 % among 25–64 year olds to 27.4 % among ≥65 year olds. The mean number of teeth lost was 8.1 overall, ranging from 6.6 among 25–64 year olds to 15.9 among the ≥65 year olds.

There was significant effect modification ($P < 0.001$) by age of the relationship between tooth loss and being high-risk for OSA after adjustment for potential confounders. The relationship between tooth loss and high-risk for OSA was strongest in adults aged less than 50 years and attenuated at older age (Fig. 1).

Among 25–64 year olds, a dose–response association was observed between levels of tooth loss and being high-risk for OSA (test for trend, $P < 0.001$, Table 1). Being high-risk for OSA was more than twice as common among the edentulous (31.7 %) than among adults with a full dentition (14.7 %). By contrast, above the age of 64 years, OSA signs and symptoms were not associated with tooth loss. Participants younger than 65 years were strikingly different in other respects as well (Table 1). Having a denture, short duration of sleep, and elevated CRP were associated with being high-risk for OSA, but only below 65 years of age. Despite marked differences, younger and older groups shared certain characteristics. Men, diabetics, and heavier people were more likely than their counterparts to be high-risk for OSA, irrespective of age. In the absence of association with tooth loss in adults aged 65 and older, the remaining analyses were restricted to the 25–64 year olds.

There was no specific configuration of tooth loss that distinguished people being high-risk for OSA (Fig. 2). Rather, tooth loss in adults at risk for OSA was generalized throughout the mouth. These adults had approximately 5–10 % greater probability of having lost molars, premolars, and incisors than adults at low-risk for OSA.

Among adults aged 25–64 years, the unadjusted PR for being high-risk for OSA was 2.16 (95 % CL, 1.62, 2.88) in edentulous individuals relative to the fully dentate (Table 2, model 1). **Another way of expressing this is that OSA risk was more than twice as high in edentulous adults than in the fully dentate.** Although adjustment for covariates attenuated the association (Table 2, model 2), the association with tooth loss remained statistically significant and dose-responsive. Compared with the fully dentate, prevalence of high-risk for OSA was elevated 25 % in those with 5–8 lost teeth (PR = 1.25, 95 % CL, 1.07, 1.46), elevated 36 % in those with 9–31 lost teeth (PR = 1.36, 95 % CL, 1.06, 1.73), and elevated 61 % in the edentulous (PR = 1.61, 95 % CL, 1.11, 2.33). Prevalence of being high-risk for OSA increased 36 % per decade of older age (PR = 1.36, 95 % CI, 1.26, 1.48). **Longer duration of sleep was significantly protective (Table 2, model 2).** Additional adjustment for CRP did not further attenuate the effect size for tooth loss groups (results not tabulated).

In the subset for whom functional tooth contacts were assessed ($n = 2350$), retaining a higher number of posterior occlusal contacts was significantly protective against OSA risk, even after adjusting for lost teeth, age, and sex (Table 3). For each additional posterior functional contact present, prevalence odds of being high-risk for OSA decreased 4 % (PR = 0.94, 95 % CI, 0.94, 0.99).

When the ordinal tooth loss variable was substituted in the fully adjusted model for a continuous count of lost teeth, adjusted odds of being high-risk for OSA increased 2 % (PR = 1.02, 95 % CI, 1.01, 1.03) for each additional lost tooth (results not tabulated).

Discussion

Main findings

In this general population sample of US adults, there was a significant graded association between tooth loss and risk for OSA, after adjustment for confounders. Prevalence of high-risk for OSA increased 2 % for each additional tooth lost. Even relatively minor levels of

tooth loss—between five and eight lost teeth—were associated with 25 % greater prevalence of being high-risk for OSA. The strength of association between tooth loss and high-risk for OSA varied across the age spectrum, being strongest in younger adults.

Consistency with previous findings

Two studies [12, 13] report a significant association between edentulism and OSA as measured by the apnea hypopnea index (AHI). Specifically, Endeshaw et al., reported a strong association between edentulism and AHI 15 with OR = 6.29, 95 % CL = 1.71, 23.22 [13]. Likewise, Bucca et al., reported a mean AHI of $17.4 \pm$ standard error of 3.6 for edentulous participants who sleep without dentures and 11.0 ± 2.3 when these same participants slept with their dentures [12]. Consistent with these studies, and using population-based data, we found greater prevalence of OSA susceptibility among edentulous compared to the fully dentate. However, it is not clear whether wearing dentures during sleep protects against a higher AHI in all edentulous adults. One study of 23 elderly edentulous OSA patients found that adults who wore dentures during sleep had higher polysomnogram-determined AHI than adults who removed their dentures for sleep, but that effect was seen only among adults with mild OSA. Use of dentures during sleep was not associated with AHI among edentulous adults with moderate or severe OSA [19].

In a large ($n = 5424$) representative sample of the general population in northern Sweden, Larsson et al., reported greater crude odds of self-reported snoring and witnessed apnea among men than women [20]. Our findings concur with those earlier results and build on them by adjusting for potential confounding. We found that in comparison to women, prevalence of OSA signs and symptoms were 50 % higher in men (adjusted PR 1.5 (95 % CL 1.29, 1.75)).

Mechanisms

Multiple morphological changes that follow complete loss of teeth also occur, albeit less profoundly, in partial tooth loss. A systematic review of bone dimension change to the alveolar ridge following tooth extraction, summarized evidence as a mean mid-buccal height loss of 1.67 mm and a mean reduction in width of 3.87 mm at extraction sites [21]. Moreover, use of a partial denture results in decreased lower face height, but to a lesser degree than that observed for edentulous individuals wearing complete dentures [22]. Similarly, the gonial angle is increased in partially edentulous, compared to dentate individuals, but not as greatly as for those individuals who are fully edentulous [23]. A large gonial angle is found to be associated with obstructive sleep apnea in observational studies [24]. There are changes in the masticatory muscles that accompany partial as well as complete loss of the teeth. For example, mass of the masseter muscle decreases with increasing loss of teeth [25]. This is attributed to the associated reduction in mandibular stability and masticatory function with progressive loss of teeth, and it is possible that similar reductions occur in the size and strength of other, submental muscles that contribute to both mastication and airway stability during sleep [26]. All considered, the same morphological changes that are thought to contribute to OSA in edentulous individuals may contribute to sleep-disordered breathing in individuals who have lost only some of their teeth.

Another possible mechanism may involve obesity. Increased consumption of refined carbohydrates leads to both tooth decay/loss and weight gain. However, in analysis of the present data, the association between tooth loss and OSA susceptibility persisted after adjusting for BMI in the multivariable model, demonstrating that BMI did not fully account for the apparent association. Apparent enlargement (lateral spreading) of the tongue secondary to tooth loss may reduce the retrolingual space and compromise airway patency regardless of a true increase in tongue mass [12, 27].

In people with missing teeth, a denture may offer partial protection against loss of muscle strength by restoring the number of teeth and occlusal support regions. Maintenance of occlusal support protects against atrophy of masseter muscle fiber thickness and volume, especially in the premolar region [25]. Moreover, dentures restore the vertical dimension of occlusion, lower facial height, retropharyngeal and posterior airway spaces, and peak inspiratory flow rates [11, 28].

The current findings are consistent with a protective effect of denture wearing on risk for OSA, although we caution that this cross-sectional finding provides only weak evidence for efficacy of dentures. Moreover, denture wearers are often instructed not to use the prostheses continuously, viz, during sleep [29] and some changes induced by tooth loss, such as tongue retraction, do not appear to revert upon dental rehabilitation with dentures [6]. In principle, definitive evidence would be needed from a randomized controlled study, although it is unlikely that people would willingly enroll in a study where the wearing, or not, of dentures at least during the daytime was allocated at random.

Strengths and limitations

The NHANES is administered by the National Center for Health Statistics and is the only survey to conduct oral health examinations for a representative sample of the US population. As such, it provides the best population-based estimates of the association between these two conditions. Despite these strengths, the subjective nature of OSA screening by questionnaire inevitably introduces misclassification. However, because the same screening instrument is administered to all participants, regardless of tooth retention, misclassification is likely non-differential, biasing estimates of the association toward the null. The STOP screening items are simple to administer in the general population. They have high sensitivity ($se = 95\%$) to identify people at elevated risk for OSA and although specificity is low ($sp = 16\%$) [30], it is comparable to other OSA screening questionnaires and adequate when used in conjunction with other predictors such as gender and BMI.

These cross-sectional data preclude inference about temporal sequence or causality. We assumed that tooth loss increases risk for OSA, because there is no evidence that OSA increases risk for tooth extraction. However, longitudinal investigation of this relationship is a valid next step, along with assessment of OSA with overnight sleep tests.

Acknowledgments

Akinkugbe A.A. was supported by the National Institute of Health NRSA T90 Training Grant NIH/NIDCR (5T90DE021986).

References

1. Ng AT, Gotsopoulos H, Qian J, Cistulli PA. Effect of oral appliance therapy on upper airway collapsibility in obstructive sleep apnea. *Am J Respir Crit Care Med*. 2003; 168(2):238–241. DOI: 10.1164/rccm.200211-1275OC [PubMed: 12724125]
2. Schellenberg JB, Maislin G, Schwab RJ. Physical findings and the risk for obstructive sleep apnea. The importance of oropharyngeal structures. *Am J Respir Crit Care Med*. 2000; 162(2 Pt 1):740–748. DOI: 10.1164/ajrccm.162.2.9908123 [PubMed: 10934114]
3. Cawood JI, Howell RA. A classification of the edentulous jaws. *Int J Oral Maxillofac Surg*. 1988; 17(4):232–236. [PubMed: 3139793]
4. Douglass JB, Meader L, Kaplan A, Ellinger CW. Cephalometric evaluation of the changes in patients wearing complete dentures: a 20-year study. *J Prosthet Dent*. 1993; 69(3):270–275. [PubMed: 8445557]
5. Tallgren A, Lang BR, Walker GF, Ash MM Jr. Roentgen cephalometric analysis of ridge resorption and changes in jaw and occlusal relationships in immediate complete denture wearers. *J Oral Rehabil*. 1980; 7(1):77–94. [PubMed: 6987348]
6. Kotsiomi E, Farmakis N, Kapari D. Factors related to the resting tongue position among partially and completely edentulous subjects. *J Oral Rehabil*. 2005; 32(6):397–402. DOI: 10.1111/j.1365-2842.2005.01444.x [PubMed: 15899017]
7. Bhojar PS, Godbole SR, Thombare RU, Pakhan AJ. Effect of complete edentulism on masseter muscle thickness and changes after complete denture rehabilitation: an ultrasonographic study. *J Investig Clin Dent*. 2012; 3(1):45–50. DOI: 10.1111/j.2041-1626.2011.0088.x
8. Tallgren A, Lang BR, Miller RL. Longitudinal study of soft-tissue profile changes in patients receiving immediate complete dentures. *Int J Prosthodont*. 1991; 4(1):9–16. [PubMed: 2012676]
9. Lee SH, Choi JH, Shin C, Lee HM, Kwon SY. How does open-mouth breathing influence upper airway anatomy? *Laryngoscope*. 2007; 117(6):1102–1106. DOI: 10.1097/MLG.0b013e318042aef7 [PubMed: 17464234]
10. Kim EJ, Choi JH, Kim KW, Kim TH, Lee SH, Lee HM, Shin C, Lee KY. The impacts of open-mouth breathing on upper airway space in obstructive sleep apnea: 3-D MDCT analysis. *Eur Arch Otorhinolaryngol*. 2011; 268(4):533–539. DOI: 10.1007/s00405-010-1397-6 [PubMed: 20957487]
11. Bucca CB, Carossa S, Colagrande P, Brussino L, Chiavassa G, Pera P, Rolla G, Preti G. Effect of edentulism on spirometric tests. *Am J Respir Crit Care Med*. 2001; 163(4):1018–1020. DOI: 10.1164/ajrccm.163.4.2005022 [PubMed: 11282782]
12. Bucca C, Cicolin A, Brussino L, Arienti A, Graziano A, Erovigni F, Pera P, Gai V, Mutani R, Preti G, Rolla G, Carossa S. Tooth loss and obstructive sleep apnoea. *Respir Res*. 2006; 7:8.doi: 10.1186/1465-9921-7-8 [PubMed: 16417639]
13. Endeshaw YW, Katz S, Ouslander JG, Bliwise DL. Association of denture use with sleep-disordered breathing among older adults. *J Public Health Dent*. 2004; 64(3):181–183. [PubMed: 15341142]
14. Slade GD, Akinkugbe AA, Sanders AE. Projections of U.S. edentulism prevalence following 5 decades of decline. *J Dent Res*. 2014; 93(10):959–965. DOI: 10.1177/0022034514546165 [PubMed: 25146182]
15. Hu Z, Yin X, Liao J, Zhou C, Yang Z, Zou S. The effect of teeth extraction for orthodontic treatment on the upper airway: a systematic review. *Sleep Breath*. 2015; 19(2):441–451. DOI: 10.1007/s11325-015-1122-1 [PubMed: 25628011]
16. Epstein LJ, Kristo D, Strollo PJ Jr, Friedman N, Malhotra A, Patil SP, Ramar K, Rogers R, Schwab RJ, Weaver EM, Weinstein MD. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med*. 2009; 5(3):263–276. [PubMed: 19960649]
17. Nieto FJ, Young TB, Lind BK, Shahar E, Samet JM, Redline S, D'Agostino RB, Newman AB, Lebowitz MD, Pickering TG. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep heart health study. *JAMA*. 2000; 283(14):1829–1836. [PubMed: 10770144]

18. Chung F, Elsaid H. Screening for obstructive sleep apnea before surgery: why is it important? *Curr Opin Anaesthesiol.* 2009; 22(3):405–411. DOI: 10.1097/ACO.0b013e32832a96e2 [PubMed: 19412094]
19. Almeida FR, Furuyama RJ, Chaccor DC, Lowe AA, Chen H, Bittencourt LR, Frigeiro ML, Tsuda H. Complete denture wear during sleep in elderly sleep apnea patients—a preliminary study. *Sleep Breath.* 2012; 16(3):855–863. DOI: 10.1007/s11325-011-0587-9 [PubMed: 21938436]
20. Larsson LG, Lindberg A, Franklin KA, Lundback B. Gender differences in symptoms related to sleep apnea in a general population and in relation to referral to sleep clinic. *Chest.* 2003; 124(1): 204–211. [PubMed: 12853524]
21. Van der Weijden F, Dell'Acqua F, Slot DE. Alveolar bone dimensional changes of post-extraction sockets in humans: a systematic review. *J Clin Periodontol.* 2009; 36(12):1048–1058. DOI: 10.1111/j.1600-051X.2009.01482.x [PubMed: 19929956]
22. Tallgren A. The reduction in face height of edentulous and partially edentulous subjects during long-term denture wear. A longitudinal roentgenographic cephalometric study. *Acta Odontol Scand.* 1966; 24(2):195–239. [PubMed: 5225747]
23. Ohm E, Silness J. Size of the mandibular jaw angle related to age, tooth retention and gender. *J Oral Rehabil.* 1999; 26(11):883–891. [PubMed: 10583739]
24. Lowe AA, Santamaria JD, Fleetham JA, Price C. Facial morphology and obstructive sleep apnea. *Am J Orthod Dentofacial Orthop.* 1986; 90(6):484–491. [PubMed: 3098087]
25. Tetsuka M, Saga T, Nakamura M, Tabira Y, Kusakawa J, Yamaki K. Relationship between masseter muscle form and occlusal supports of remaining teeth. *Kurume Med J.* 2012; 59(1–2):5–15. [PubMed: 23257633]
26. Hollowell DE, Suratt PM (1991) mandible position and activation of submental and masseter muscles during sleep. *J Appl Physiol.* 1985; 71(6):2267–2273. [PubMed: 1778922]
27. Cohen AM, Vig PS. Lateral tongue spreading. *J Dent.* 1973; 2(1):32–34. [PubMed: 4521718]
28. Gupta P, Thombare R, Pakhan AJ, Singhal S. Cephalometric evaluation of the effect of complete dentures on retropharyngeal space and its effect on spirometric values in altered vertical dimension. *ISRN Dent.* 2011; 2011:516969.doi: 10.5402/2011/516969 [PubMed: 21991477]
29. Felton D, Cooper L, Duqum I, Minsley G, Guckes A, Haug S, Meredith P, Solie C, Avery D, Chandler ND. Evidence-based guidelines for the care and maintenance of complete dentures: a publication of the American College of Prosthodontists. *J Am Dent Assoc.* 2011; 142(Suppl 1):1–20.
30. Pataka A, Daskalopoulou E, Kalamaras G, Fekete Passa K, Argyropoulou P. Evaluation of five different questionnaires for assessing sleep apnea syndrome in a sleep clinic. *Sleep Med.* 2014; 15(7):776–781. DOI: 10.1016/j.sleep.2014.03.012 [PubMed: 24891079]

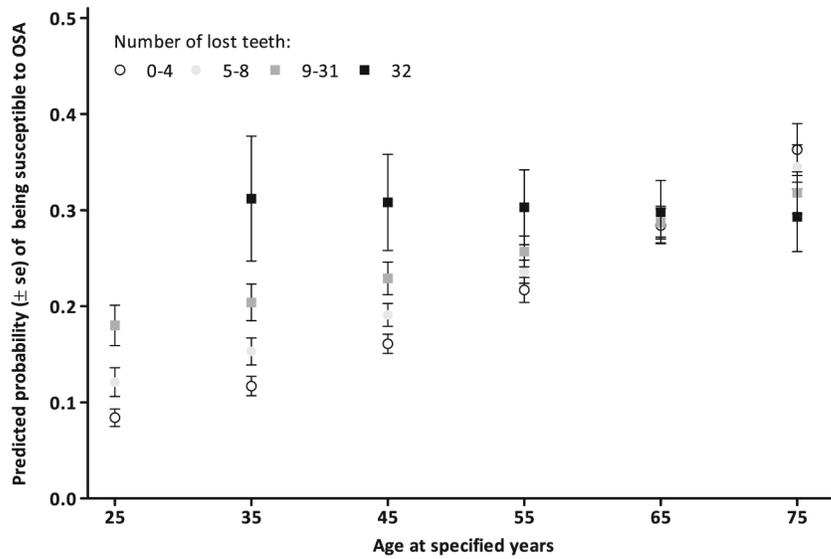


Fig. 1. Effect modification by age ($P < 0.001$) of the association between tooth loss and predicted probability of being high-risk for OSA. Multivariable-adjusted probabilities were obtained from a log binomial regression model in which age was modeled as a continuous predictor variable; covariates were sex, race/ethnicity, body mass index, denture status, and average sleep duration. The *lines* represent a fitted linear model of the association at specified ages in 10-year increments from 25 to 75 years. The convergence of the *lines* provides visual depiction of effect modification, interpreted as a stronger association among younger than older adults. NHANES, 2005–2008 ($n = 7305$ adults aged 25 years and older). NB: The value is suppressed for the one participant aged 25 years with 32 missing teeth

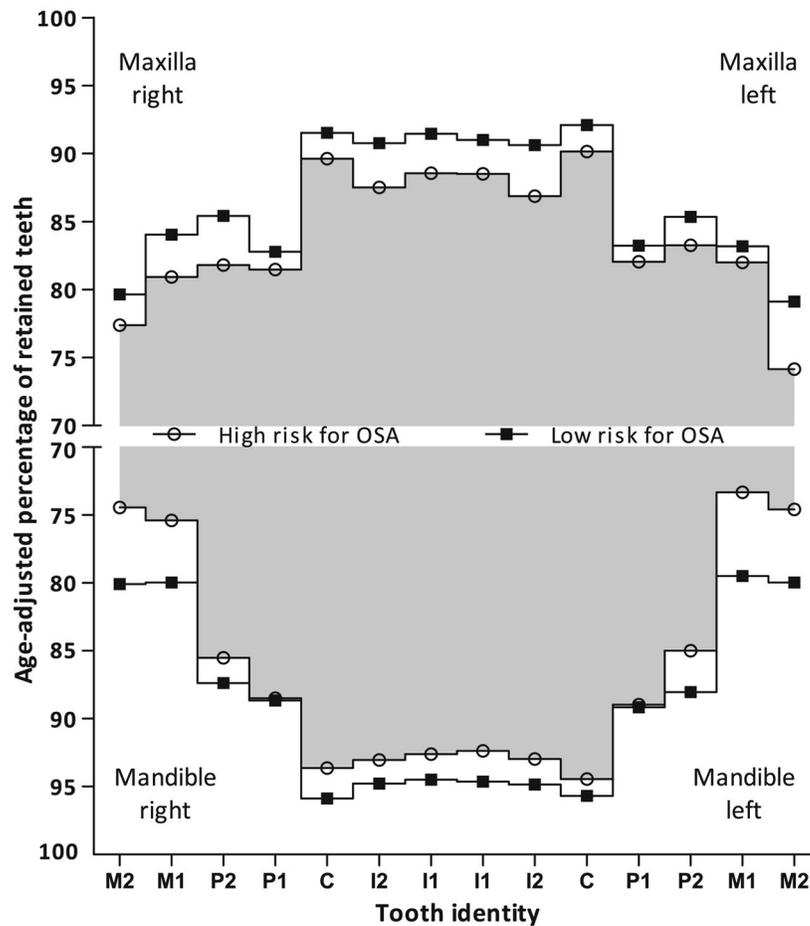


Fig. 2. Stylized oral cavity plotting the age-adjusted mean percentage of retained teeth for participants high-risk versus low-risk for OSA. The gap between the two series of *connected lines* highlights the net increase in lost teeth among those susceptible to OSA. This is interpreted as greater tooth loss throughout the mouth among adults at high risk for OSA. Specifically, these adults had approximately 5–10 % greater probability of having lost molars, premolars, and incisors than adults at low-risk for OSA. Third molars (i.e., wisdom teeth) were not plotted as their retention was considerably lower for both groups. *M2* and *M1* refer to second and first molars, *P2* and *P1* refer to second and first premolars, *C* refers to canines, and *I2* and *I1* refer to second and first incisors. NHANES, 2005–2008 ($N = 5451$ adults aged 25 to 64 years)

Table 1

Age-stratified participant characteristics and percentage at high-risk for obstructive sleep apnea (OSA), NHANES, 2005–2008 (n = 7305)

Characteristic	Participants aged 25 to 64 years		Participants aged 65 years		P
	Unweighted N total = 5451 (weighted col %)	High-risk for OSA ^a n = 1062 (18.9 %)	Unweighted N total = 1854 (weighted col %)	High-risk for OSA ^a n = 495 (27.4 %)	
Number of lost teeth					
0–4	2782 (54.4)	14.7	230 (16.5)	29.1	0.582
5–8	1355 (26.3)	21.2	364 (21.4)	25.5	
9–31	1092 (16.1)	26.6	821 (40.6)	28.9	
32 (edentulous)	222 (3.2)	31.7	439 (21.5)	25.0	
Sex					
Male	2703 (49.5)	22.4	1019 (47.4)	31.7	0.003
Female	2748 (50.5)	15.4	835 (52.6)	23.5	
Age (years)					
25–34	1444 (24.7)	8.3	n.a.	n.a.	
35–44	1406 (27.4)	18.0	n.a.	n.a.	
45–54	1389 (29.5)	20.9	n.a.	n.a.	
55–64	1212 (18.5)	30.9	n.a.	n.a.	
65–74	n.a.	n.a.	990 (56.5)	30.6	0.031
75	n.a.	n.a.	864 (43.5)	23.2	
Race/ethnicity					
Non-Hispanic White	2515 (70.5)	19.4	1188 (84.0)	27.1	0.305
Non-Hispanic Black	1172 (10.8)	20.4	328 (7.8)	25.1	
Mexican American	1511 (12.6)	14.5	297 (5.7)	30.5	
Other	253 (6.1)	19.3	41 (2.5)	37.0	
Body mass index ^b					
Underweight/healthy (<25)	1519 (31.1)	7.4	524 (29.5)	16.9	<0.001
Overweight (25–<30)	1846 (33.4)	16.3	706 (40.1)	25.8	
Obese (≥ 30)	2051 (35.5)	31.1	571 (30.4)	39.6	
Denture status					
No denture	4704 (89.1)	18.1	889 (51.5)	27.7	0.752

Characteristic	Participants aged 25 to 64 years		Participants aged 65 years		P
	Unweighted N total = 5451 (weighted col %)	High-risk for OSA ^a n = 1062 (18.9 %)	Unweighted N total = 1854 (weighted col %)	High-risk for OSA ^a n = 495 (27.4 %)	
Has denture	747 (10.9)	25.1	965 (48.5)	27.0	
Average sleep duration ^c					
5 h	918 (14.2)	31.5	246 (10.2)	31.6	0.178
>5 and < 9 h	4247 (81.1)	16.8	1385 (77.6)	27.7	
9 h	280 (4.7)	16.2	220 (12.3)	21.9	
C-reactive protein (mg/dL) ^d					
<0.2	2531 (50.9)	15.2	805 (47.1)	24.6	0.006
0.2–<0.5	1406 (25.0)	20.9	524 (26.9)	26.6	
0.5	1287 (20.6)	26.8	451 (23.1)	35.0	
Missing	227 (3.5)	10.6	74 (2.9)	19.0	

n.a. not applicable

^aValues are column percentages. The percent not susceptible to OSA (i.e., <2 OSA symptoms) is the inverse of the percent susceptible

^bA value for BMI is missing for 35 adults aged 25–64 years and for 53 adults aged 65 years

^cAverage sleep duration is missing for six adults aged 25–64 years and for three adults aged 65 years

^dAs a large number of participants was missing CRP, this category was included as missing in analyses

Table 2

Prevalence ratios (PR) and 95 % confidence limits (CL) for the association between tooth loss and high-risk for obstructive sleep apnea, in participants aged 25 to 64 years, NHANES, 2005–2008 ($n = 5410$)

	Model 1 unadjusted PR (95 % CL)	P value	Model 2 adjusted PR (95 % CL)	P value
Number of lost teeth				
0–4 (full dentition)	Referent		Referent	
5–8	1.44 (1.26, 1.65)	<0.001	1.25 (1.07, 1.46)	0.006
9–31	1.81 (1.46, 2.23)	<0.001	1.36 (1.06, 1.73)	0.016
32 (edentulous)	2.16 (1.62, 2.88)	<0.001	1.61 (1.11, 2.33)	0.014
Age per decade (i.e., age divided by 10)				
			1.36 (1.26, 1.48)	<0.001
Sex				
Male			1.50 (1.29, 1.75)	<0.001
Female			Referent	
Race/ethnicity				
Non-Hispanic White			Referent	
Non-Hispanic Black			0.91 (0.78, 1.06)	0.207
Mexican American			0.86 (0.71, 1.05)	0.135
Other			1.08 (0.85, 1.37)	0.536
Body mass index				
			1.05 (1.04, 1.06)	0.000
Denture status				
No denture			1.26 (1.02, 1.55)	0.030
Has denture			Referent	
Average sleep duration (hours)				
			0.84 (0.80, 0.87)	<0.001
Intercept	0.14 (0.12, 0.16)	<0.001	0.02 (0.01, 0.04)	<0.001

Table 3

Relationship between number of posterior occlusal contacts and prevalence ratios (PR) and (95 % confidence limits (CL)) for being high-risk for obstructive sleep apnea, adjusted for tooth loss, age, and sex, among participants aged 25 to 64 years ($n = 2350$), NHANES, 2005–2008

	PR (95 % CL)	P value
Number of posterior occlusal contacts	0.96 (0.94, 0.99)	0.005
Number of lost teeth	1.01 (0.99, 1.02)	0.398
Age per decade	1.37 (1.24, 1.52)	<0.001
Sex		
Male	1.63 (1.27, 2.10)	<0.001
Female	Referent	
Intercept	0.05 (0.22, 0.09)	<0.001

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript