Effects of Smoking on Periodontal Tissues and Halitosis

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Abstract

The purpose of this study was to assess the effects of smoking on periodontal tissues and halitosis. The study group consisted of 287 individuals. They received full mouth periodontal examinations and measurements of volatile sulfur compounds in mouth. The study group consisted of 76 non-smokers, 63 former smokers and 148 current smokers. The results showed that the mean clinical attachment level in non-smokers, former smokers and current smokers were 2.22±0.67 mm, 2.43±0.74 mm and 2.76±1.17 mm, respectively. Smokers had higher mean periodontal probing depth, mean clinical attachment level, mean number of sites with probing depth 4-5 mm and ≥ 6 mm than those of non-smokers ($p < .05$). The degree of association between the smoking status and the risk for halitosis was investigated using logistic regression analysis. Current smokers and former smokers were not at a higher risk for halitosis, compared to non-smokers. Heavy smokers (≥ 30 packyears) and moderate smokers (15 - 29.9 packyears) were not at a higher risk for halitosis, compared to light smokers (< 15 packyears).

In conclusion, smoking had an adverse effect on periodontium, in terms of increasing probing depth and periodontal attachment loss. However, current smokers and former smokers were not at a greater risk for halitosis, compared to non-smokers.

Key words: halitosis; smokers; volatile sulfur compounds

Introduction

Worldwide smoking remains one of the most important public health problem. The harmful effects of smoking and tobacco use on oral health are well recognized. Oral cancers, pre-cancerous lesions, periodontal diseases and poor wound healing are the most detrimental effects of smoking on oral health.[1,2] Moreover, smoking is directly related to tooth staining, soft tissue changes and halitosis which involve esthetic and social impact to smokers.[3] A significantly greater frequency of disease sites and a significantly greater reduction of alveolar bone height is found among current smokers, compared to non-smokers.[4,5,6] The periodontal health condition of former smokers lies between current smokers and non-smokers.[7]

Halitosis is a general term used to describe an unpleasant or offensive odor emanating from the oral cavity.[8] Several extraoral pathological conditions have been related to oral malodor including infection of both upper and lower respiratory tracts, the gastrointestinal tract and some metabolic diseases involving the liver or kidneys.[9] However, recent epidemiological studies have shown that around 90% of all bad breath odors originate in the mouth.[8] Oral halitosis or oral malodor is the term used to define halitosis or malodor with an origin within the oral cavity.[10] It usually derives from oral microbial metabolism. The pathogenesis of oral halitosis is associated with protein degradation by oral microbial, followed by a subsequent breakdown of certain amino acids (e.g., methionine, cysteine, tryptophan, and lysine), producing malodorous volatile products (e.g., methyl mercaptan, hydrogen sulfide, indole, skatole, and cadaverine.[11,12] The major elements of oral halitosis are volatile sulfur compounds (VSCs), especially hydrogen sulfide (H_2S), methyl mercaptan (CH_3SH) and dimethyl sulfide ($(CH_3)_2S$).[13]

Many studies suggested that the presence and severity of periodontal disease may contribute to the intensity of halitosis.[14] The concentrations of hydrogen sulfide and methyl mercaptan in mouth breathe were higher in patients with a probing depth greater than 4 mm than in healthy controls.[15,16] In addition, the percentage of sites per subject with high levels of sulfides measured in moderate (4-6 mm) and deep ($\geq$ 7 mm) periodontal pockets was found to be significantly higher among smokers, compared to non-smokers.[17] VSCs are directly toxic to epithelial tissues and may contribute to the destruction of tissue and the progression of periodontitis. Increased levels of VSCs production may represent a further mechanism of increased risk to periodontitis in smokers. The increased amount of VSCs production in the periodontal pocket of smokers is likely to be associated with oral malodor.

Nowadays populations in many countries around the world are increasingly aware of oral halitosis due to the importance of social interactions in contemporary society.[17] Halitosis has acquired increasing attention in recent years and the relationship between smoking and halitosis has been documented previously. However, further research into the clinical significance of elevated sulfide concentrations, and the source and the process for increased VSCs production in smokers are needed.[18] The purpose of this study was to assess the effects of smoking on periodontal tissues and halitosis.

Materials and methods

Ethical clearance for this study was approved by the Ethical Review Committee of Department of Health, Bangkok Metropolitan Administration. All participants were explained regarding the research process. Informed consent from each participant was received prior to data collection. Of the 287 individuals who participated in this study, 248 participants were government officers and employees in Ratchathevi District Office, Bangkok, Thailand, while the remainder were government officers and employees in Military Medical Department, Bangkok, Thailand. The subjects were required to exhibit a minimum of 10 teeth. The participants presenting with a history of concurrent systemic diseases or pregnancy were excluded from the study. In the study population, nearly all smokers and former smokers were males so it is necessary to limit the number of females in the non-smoker group to avoid confounding caused by the difference in sex distribution among different smoking status.

Oral Examination

Oral examinations were performed by four dentists at Ratchathevi District Office and the Military Medical Department in mobile dental units. Calibration for periodontal measurements was carried out among four examiners and between four examiners and one experienced periodontist before the study. All teeth were examined, except third molars and retained roots. Clinical recordings included: Simplified Debris Index,[19] Simplified Calculus Index,[19] Gingival Index,[20] Winkel Tongue Coating Index,[17] gingival recession and probing depth (PD). Gingival recession and PD were measured using a PC-PUNC 15 probe on six sites per tooth; mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, disto-lingual. These measurements were made in millimeters and were rounded to the nearest millimeters. Clinical attachment level (CAL) was calculated as the addition of probing depth and gingival recession and represented the distance from the cemento-enamel junction (CEJ) to the base of the probing pocket depth.The weight kappa coefficients (within ±1 mm) between each pair of examiners ranged from 0.73 to 0.89 for PD and from 0.68 to 0.79 for CAL.

Measurements of volatile sulfur compounds (VSCs)

Measurements of VSCs were performed at the laboratory of Faculty of Dentistry, Mahidol University. A halimeter® was used to assess the concentration of VSCs in mouth air. The manufacturer's

instructions were followed for all measurement procedures with the halimeter. The monitor was calibrated to zero on ambient air before each measurement. Subjects were asked to close their mouths for 1 minute prior to measurements and instructed to protrude their tongues. A disposable straw was placed at the mid-dorsal posterior part of the tongue and fixed until the maximum peak value of VSCs was recorded. The subjects were asked to breathe through their noses during measurements. Peak VSCs level was registered in parts per billion. Three independent and consecutive measurements were taken. The mean of all scores represented the individual VSCs score. If the first 2 measurements were extremely low or high, the subjects were replaced with new subjects to be measured to test the validity of the halimeter. Halitosis was evaluated based on the standard mentioned in the manufacturer's instructions (http://www.halimeter.com/halcal.htm).

Smoking status assessment

Smoking status was assessed by a self-reported questionnaire. Smoking status was classified as non-smokers, former smokers and current smokers. Current smokers were persons who were smoking at the time of the examination. Former smokers were persons who had quit smoking at least one year. Non-smokers were persons who had never used tobacco products. Current smokers and former smokers were questioned about the number of cigarettes consumed per day and the number of years they smoked. Former smokers were asked to report the number of years they had quit smoking. Smoking exposure was expressed in term of packyears (pkyr), which is calculated by the multiplication of the number of packs of cigarettes smoked per day by the number of years they smoked.

Statistics

Statistical analysis was carried out using SPSS 11.0 for Windows software. One-way analysis of variance (ANOVA) with post hoc the Bonferroni's multiple comparison was used to determine the mean PD, CAL, mean number of site with PD $\leq$ 3 mm, 4-5 mm and $\geq$ 6 mm and WTCI values among all different smoking groups (for variables with equal variances among groups). For variables with unequal variances among groups (mean VSCs), the significant difference between groups was tested using the Welch test and post hoc multiple comparisons were performed according to the Tamhane test. Pearson's correlation was used to determine the association between VSCs and different smoking groups. Logistic regression was used to address the association between VSCs and different smoking groups. The variables of Simplified Debris Index (DI-S), Simplified Calculus Index (CI-S), Simplified Oral Hygiene Index (OHI-S), Gingival Index (GI), Winkel Tongue Coating Index (WTCI), CAL and PD were included in logistic regression model. Statistical significance was determined at $p \leq .05$.

Results

The distribution of the study population according to smoking status, gender, age and occupation is presented in Table1. The proportions of males to females among non-smokers, former smokers and current smokers were 82.9%, 98.4% and 94.6%, respectively. Age ranged from 18 to 67 years (mean±s.d. = 38.69±9.95). Non-smokers and former smokers had a similar proportion of occupation (government officers 46.0-47.4% and government employees 52.6-54.0%) while current smokers had a higher proportion of government employees than non-smokers and former smokers. In former smokers, 57.1%, 23.8% and 19.1% had quit smoking for less than 10 years, 10-19 years and more than 20 years, respectively.

GI, DI-S and CI-S are shown in Table 2. Current smokers and former smokers had a much higher proportion of subjects who had DI-S $\geq$ 1 and CI-S $\geq$ 1 than non-smokers. It means that non-smokers had better oral hygiene than current smokers and former smokers. Current smokers and former smokers had a little higher proportion of subjects who had GI-S $\geq$ 1 than non-smokers.

Periodontal variables, mean WTCI and mean VSCs were assessed according to smoking status (Table 3). Current smokers had deeper mean probing depth, greater mean clinical attachment level, less mean numbers of sites with PD of 3 mm, more mean numbers of sites with PD of 4-5 mm and PD of more than 6 mm than former smokers and non-smokers. The values for former smokers were between those of current smokers and non-smokers.

The difference of periodontal variables was statistically significant only for the pair of current smokers versus non-smokers. The difference of mean numbers of sites with PD of 4-5 mm and PD of more than 6 mm between non-smokers and former smokers, and between former smokers and current smokers was not statistically significant. The difference of WTCI and VSCs between all pairs of smoking groups, including current smokers versus non-smokers, current smokers versus former smokers and former smokers versus non-smokers, were not statistically significant.

Table 1 *Distribution of study population according to smoking status, gender, age and occupation (N = 287)*

Characteristic	N (%)		
	Non-smokers	Former smoker	Current smoker
Gender			
Male	63 (82.9)	62 (98.4)	140 (94.6)
Female	13 (17.1)	1 (1.6)	8 (5.4)
All	76 (100.0)	63 (100.0)	148 (100.0)
Age (years)			
18 - 30	21 (27.6)	10 (15.9)	39 (26.4)
31 - 40	18 (23.7)	15 (23.8)	49 (33.1)
41 - 50	29 (38.2)	23 (36.5)	46 (31.1)
51 - 67	8 (10.5)	15 (23.8)	14 (9.5)
All	76 (100.0)	63 (100.0)	148 (100.0)
Occupation			
Government officer	36 (47.4)	29 (46.0)	42 (28.4)
Government employee	40 (52.6)	34 (54.0)	106 (71.6)
All	76 (100.0)	63 (100.0)	148 (100.0)

Table 2 *Gingival Index(GI), Simplified Debris Index (DI-S), Simplified Calculus Index (CI-S) according to smoking status*

Characteristic		N (%)		
		Non-smokers	Former smoker	Current smoker
GI	< 1	14 (18.4)	8 (12.7)	19 (12.8)
	≥ 1	62 (81.6)	55 (87.3)	129 (87.2)
DI - S	< 1	40 (52.6)	23 (36.5)	53 (35.8)
	≥ 1	36 (47.4)	40 (63.5)	95 (64.2)
CI - S	< 1	59 (77.6)	33 (52.4)	70 (47.3)
		17 (22.4)	30 (47.6)	78 (52.7)

Table 3 *Periodonatal variables, WTCI and VSCs according to smoking status (Mean±s.d.)*

Variables	Mean ± s.d.		
	Non-smokers	Former smoker	Current smoker
Mean PD (mm) †	2.09 ± 0.40	2.23 ± 0.49	2.41 ± 0.66
Mean cal (mm) †	2.22 ± 0.67	2.43 ± 0.74	2.76 ± 1.17
Mean site with PD ≤ 3 mm (site/person)‡	144.24 ± 21.36	139.60 ± 24.60	131.54 ± 35.82
Mean site with PD 4 -5 mm (site/person)‡	6 ± 9.69	10.43 ± 15.10	12.97 ± 15.45
Mean site with PD ≥ 6 mm (site/person)‡	0.63 ± 1.75	1.38 ± 3.57	2.57 ± 6.97
Mean WTCI	7.79 ± 3.64	7.32 ± 3.91	7.86 ± 3.89
Mean VSCs (ppb)	165.82 ± 239.14	166.11 ± 200.29	115.51 ± 93.44

† Significant difference between current smokers and non-smokers at $p < .001$

‡ Significant difference between current smokers and non-smokers at $p < .05$

†, ‡ using ANOVA and Bonferroni post hoc analysis

Table 4 ***Halitosis according to smoking group***

Halitosis	N (%)		
	Non-smokers	Former smoker	Current smoker
No halitosis (VSC ≤ 110ppb)	24 (31.6)	26 (41.3)	62 (41.9)
Halitosis (VSC > 110 ppb)	52 (68.4)	37 (58.7)	86 (58.1)
All	76 (100.0)	63 (100.0)	148 (100.0)

Table 5 *Level of cigarette consumption according to smoking status*

Packyear	N (%)	
	Former smoker	Current smoker
< 15	48 (84.2)	107 (72.3)
15-29.9	8 (14.0)	31 (20.9)
≥ 30	1 (1.8)	10 (6.8)

Table 6 *Results of logistic regression analysis of volatile sulfur compounds (VSCs) concentration (N = 287 ; VSCs : 0: ≤110 ppb, 1 : >110 ppb)*

Variables	Adjust OR	95 % CI	*p*-value
Smoking status			
Non-smokers	1*		
Former smoker	1.61	0.84-3.07	0.15
Current smoker	1.22	0.61-2.42	0.57
Cigarette consumption			
< 15 packyear	1*		
15 - 29.9 packyear	0.98	0.24-3.98	0.98
≥ 30 packyear	0.56	0.12-2.61	0.46
Clinical attachment level			
< 2.5 mm	1*		
2.5 - 3.9 mm	3.92	0.69-22.20	0.12
≥ 4.0 mm	5.09	0.89-29.15	0.07

Odd ratios and their 95% confidence interval (95% CI) are from logistic regression analysis and are adjusted for age, DI-S, CI-S and WTCI.

* Reference group.

Table 4 shows halitosis conditions among non-smokers, former smokers and current smokers. Halitosis was defined when VSCs were more than 110 ppb, following manufacturer's instruction. The proportion of subjects who had halitosis in non-smokers was higher than that of former smokers and current smokers. The proportion of subjects who had halitosis problem in former smokers was similar to that of current smokers. However, no significant differences of halitosis was found among different smoking groups.

Level of cigarette consumption was classified into 3 groups: <15 pkyr (light smokers), 15-29.9 pkyr (moderate smokers) and ≥30 pkyr (heavy smokers). Former smokers and current smokers had the highest proportion of light smokers (84.2% and 72.3%, respectively) and the lowest proportion of heavy smokers (1.8% and 6.8%, respectively).

The degree of association between the risk for halitosis and different smoking status, level of cigarette consumption and clinical attachment level was determined using logistic regression analysis as shown in Table 6. Other factors that might affect halitosis, including age, DI-S, CI-S and WTCI, were included in the model. Both current smokers and former smokers had no increased risk for halitosis, compared to non-smokers. Smoking status, level of cigarette consumption and clinical attachment level were not associated with VSCs.

Discussion

The results from this study, in accordance with many publications in various populations, showed the adverse effect of smoking on periodontium, in terms of increasing probing depth and increased periodontal attachment loss.[5,21,22] Most of current smokers and former smokers in this study had moderate (4-5 mm) periodontal pocket depth. Mean number of sites with pocket depth 4-5 mm in current smokers and former smokers was 12.97(±15.45) and 10.43 (±15.10) sites/person, respectively. Few current smokers and former smokers had deep (≥6 mm) periodontal pocket, because they were light smokers. This explains why current smokers and former smokers in our study had less mean probing depth, less mean numbers of sites with

PD of 4-5 mm and PD of more than 6 mm, less mean clinical attachment level, more mean numbers of sites with PD of 3 mm than those in other studies. The present study showed the dose-effect relationship between cigarette consumption and pocket formation. This was consistent with previous studies.[7,23]

Previous studies have mentioned the etiology of halitosis including retention of odorous meal particles between the teeth, tongue coating, gingivitis, acute necrotic ulcerative gingivitis, periodontal diseases, dehydration after physical activity, caries, badly finished prosthesis, premenstrual periods, surgical healing or tooth extraction wounds, intestinal dyspepsia, esophagus reflux, sinusitis and rhinosinusitis.[24] Moreover, halitosis can be related to some metabolic diseases involving the liver or kidneys.[9] However, the etiology of halitosis is usually (around 90%) an oral cavity phenomenon. Therefore, the participants presenting with a history of concurrent systemic diseases or pregnancy were excluded from the study. Other factors that might affect halitosis, including age, DI-S, CI-S and WTCI, were measured and included in the bivariable logistic regression analysis model. The purpose of this study was to investigate the association of cigarette smoking on halitosis. Current smokers and former smokers were not at a greater risk for halitosis, compared to non-smokers. Our results were similar to previous investigations in finding that smoking does not appear to contribute to the incidence of halitosis.[25,26,27]

Tobacco smoke itself consists of VSCs.[28] The adverse effect of smoking on periodontal tissue may influence halitosis[7]. Several publications have demonstrated a significant association between VSCs and periodontal disease. VSCs in mouth air increased with the increase of the number and depth of periodontal pockets (>3 mm).[29] An association was found between the amounts of hydrogen sulfide in the gingival crevice and the depth of corresponding periodontal pockets.[30] The percentage of sites per subject with high levels of VSCs measured in moderate (4-6 mm) and deep (≥7 mm) periodontal pockets was significantly higher among smokers, compared to non-smokers in Khaira's study.[17] The instrument measuring VSCs in that study was the Research/International model of Diamond Probe®/ Perio2000® system, different from our study, and the probe of instrument was placed into the periodontal pockets of the subjects to measured VSCs. Therefore, the VSCs measured in Khaira's study were VSCs in the periodontal pockets, while the VSCs measured in this study was VSCs in mouth air. Periodontal pockets are putrid when probed or scaled. However, many pockets are relatively sealed, so a small fraction of malodor inside pockets comes into the mouth air.[31]

VSCs are directly toxic to epithelial tissues and may contribute to the destruction of tissue and the progression of periodontitis.[30,32] Increased production of VSCs may, therefore, represent an increased risk of periodontitis in smokers.[17] However the subjects in the present study were light smokers and had only moderate periodontitis (pocket depth 4-5 mm). It may be the reason smokers in this study produced less VSCs in periodontal pocket than general smokers.

The inflamed pockets (with bleeding on probing) had significantly higher total sulfide than non-inflamed pockets (without bleeding on probing).[33] Periodontal disease progresses in a series of relatively short, acute "burst" of rapid tissue destruction, followed by some tissue repair with long periods of remission, following the burst theory of periodontitis.[34] Halitosis in periodontitis is related to inflammation of periodontitis. The other indexes, such as biomarkers, that can reflect inflammation of periodontal tissue should be used to determine the association between halitosis and smoking. More researches should be conducted to evaluate the relationship between VSCs in periodontal pocket and in mouth air.

Conclusion

There was an adverse effect of smoking on periodontium, in terms of increasing probing depth and increased periodontal attachment loss. However, current smokers and former smokers were not at a greater risk for halitosis, compared to non-smokers.

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บทวิทยาการ

ผลของบุหรี่ต่อเนื้อเยื่อปริทันต์และการมีกลิ่นปาก

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แหล่งทุน: สำนักงานกองทุนสนับสนุนการสร้างเสริมสุขภาพ และทันตแพทยสภา (ภายใต้โครงการกลยุทธ์วิชาชีพทันตแพทย์ในการควบคุมการบริโภคยาสูบ)

บทคัดย่อ

การศึกษานี้มีวัตถุประสงค์เพื่อศึกษาถึงผลกระทบของการสูบบุหรี่ต่อเนื้อเยื่อปริทันต์และการมีกลิ่นปาก การศึกษาดำเนินการโดยการตรวจกลุ่มตัวอย่างจำนวน 287 คน โดยตรวจสภาวะปริทันต์ฟันทุกซี่ในช่องปากและวัดปริมาณสารประกอบซัลเฟอร์ที่ระเหยได้ในช่องปาก กลุ่มตัวอย่างประกอบด้วยผู้ไม่สูบบุหรี่ จำนวน 76 คน ผู้เคยสูบบุหรี่ จำนวน 63 คน และผู้สูบบุหรี่ จำนวน 148 คน ผลการศึกษาพบว่าค่าเฉลี่ยระดับยึด (ของอวัยวะปริทันต์) ทางคลินิกในกลุ่มผู้ไม่สูบบุหรี่ กลุ่มผู้เคยสูบบุหรี่ และกลุ่มผู้สูบบุหรี่ เท่ากับ 2.22±0.67 มม. 2.43±0.74 มม. และ 2.76±1.17 มม.ตามลำดับ กลุ่มผู้สูบบุหรี่มีค่าเฉลี่ยร่องลึกปริทันต์ ระดับยึด (ของอวัยวะปริทันต์) ทางคลินิก ค่าเฉลี่ยจำนวนตำแหน่งที่มีร่องลึกปริทันต์ 4-5 มิลลิเมตรและตั้งแต่ 6 มิลลิเมตรขึ้นไป สูงกว่าผู้ไม่สูบบุหรี่อย่างมีนัยสำคัญทางสถิติ ($p < .05$) ระดับความสัมพันธ์ระหว่างการเกิดกลิ่นปากกับการสูบบุหรี่ที่วิเคราะห์ โดยใช้สถิติการวิเคราะห์ความถดถอยโลจิสติก พบว่ากลุ่มผู้ที่สูบบุหรี่และผู้ที่เคยสูบบุหรี่มีความเสี่ยงที่จะเกิดกลิ่นปากไม่แตกต่างจากผู้ไม่สูบบุหรี่ ผู้ที่สูบบุหรี่มาก (≥30 ซองปี) และผู้สูบบุหรี่ปานกลาง (15-29.9 ซองปี) มีความเสี่ยงต่อการเกิดกลิ่นปากไม่แตกต่างจากผู้ที่สูบบุหรี่น้อย (<15 ซองปี) โดยสรุปการสูบบุหรี่มีผลเสียต่อเนื้อเยื่อปริทันต์โดยเพิ่มร่องลึกปริทันต์และการยึดติดของเหงือก แต่อย่างไรก็ตาม ผู้สูบบุหรี่และผู้เคยสูบบุหรี่ไม่มีความเสี่ยงต่อการมีกลิ่นปากมากกว่ากลุ่มผู้ไม่สูบบุหรี่