

Periodontal disease and halitosis

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بجر الفم مصطلح عام يستعمل لوصف الرائحة الكريهة المتبعثة من الفم. وتعتبر ذات تأثيرات صحية واجتماعية بالنسبة للمعائين منها. يعود سبب هذه الحالة إلى أسباب جهازية وموضعية في معظم الحالات. ويعتبر غاز الكبريت الطيار المسؤول الأول عن بجر الفم، ويتشكل من جراء تفسخ الجراثيم السلبية الجرام. وتعتبر بعض المواقع مثل ظهر اللسان والجيوب حول السنية من أهم مصادر بجر الفم. ولوحظ وجود علاقة شدة بجر الفم وكلا من كمية اللويحة الجرثومية المتواجدة على سطح اللسان والتهاب الأنسجة المحيطة بالأسنان. تهدف هذه المقالة إلى مراجعة الحقائق المتوفرة والربط بين التهاب الأنسجة المحيطة بالأسنان وشدة الإصابة ببجر الفم وتأثير العوامل المسبب لبجر الفم على تطور الأمراض المحيطة بالأسنان.

Halitosis is a general term used to describe an unpleasant or offensive odor emanating from the oral cavity. It is a condition that has health and social implications in the life of those who suffer from it. The origin of halitosis is related to both systemic and oral conditions although the oral causes predominate. Volatile sulfur compound is the primary gas responsible for halitosis. They are formed as a result of gram-negative bacterial putrefaction. The major sites for oral halitosis are the dorsum of the tongue and periodontal pockets. There is a correlation between the amount of plaque on the tongue and periodontitis with the severity of halitosis. The aim of this article was to review the data and correlate periodontitis with severity of halitosis and the effect of halitosis- inducing factors on the progress of periodontal diseases.

INTRODUCTION

Halitosis is a common condition which affects most of the population. Tessier *et al.*,¹ Bosy² and Nachnani³ reported that halitosis affects more than 50% of population. It may cause a psychological or social handicap to those suffering from it.⁷

TERMINOLOGY AND DEFINITIONS

Many terms were used to describe oral malodor and these different terms may lead to confusion, lack of clarity or ambiguity as to the kind of smell, its etiology, interpretation and significance. The term malodor is used to describe a foul smell emanating from the mouth. However, it is general term which does not imply any source or causation. There are several terms used to distinguish the source and the required treatment.

Ozostomia (*ozo* means smell, *stomai* means mouth) refers to a putrid smell originating from the upper respiratory tract in particular, nasal, sinus cavities, the pharynx and larynx. Any inflammation in these sites like rhinitis, sinusitis, pharyngitis, laryngitis and tonsillitis

would cause ozostomia.^{4,5} Stomatodysodia (*stoma* means mouth, *dysodia* means bad odor) which refer to foul breath originating from local areas in the lower respiratory tract like bronchitis, pulmonary abscess and tuberculosis. Halitosis (*halitus* means breath, *osis* means condition) refers to a breath that is offensive to others, caused by variety of reasons. It could be a result of systemic metabolic conditions, as well as generalized pathophysiological condition. Fetor oris or fetor ex ore (*fetor* means offensive odor, *oris* plural of ora which means mouth) which describes an offensive odor emitting from the mouth.

SOURCES OF HALITOSIS

Oral halitosis can be caused by different localized and systemic disorders.

Non-oral Sources

Normal physiological processes and behaviors can cause oral malodor which is usually transitory. These non-pathological oral malodor may due to empty stomach, low level of salivation during sleep, food debris, smoking and volatile hematologically borne substance which are released into the lungs from food like herbs, spices or selected vegetable like onion or garlic, or from some drinks like coffee or tea.^{4, 8}

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Some systemic pathological conditions may cause oral malodor such as diabetic ketosis, gastrointestinal conditions, irregular bowel movement, uremia, hepatic and renal failure.^{3,9}

Oral Sources

Oral cavity is considered as one of the major sources of oral halitosis. Delanghe *et al.*¹⁰ reported after examination of hundreds of patients with bad breath that around 87% of oral malodor originated from the oral cavity, whereas 5 – 8 % were from ear – nose – throat (ENT) causes. Other reports indicated that 80 – 90 % of all oral halitosis were from oral sources.⁸

Some oral diseases and conditions which are associated with oral halitosis are gingivitis, periodontitis, acute herpetic gingivostomatitis, erythema multiforme, benign mucous membrane pemphigoid, acute necrotizing ulcerative gingivitis and periocoronitis.^{4,11} Other oral conditions that are associated with oral malodor include aphtous ulcers, dental abscesses, candidiasis, oral cancer and xerostomia.⁹ Although large carious lesions which can trap food could produce halitosis, caries itself is not an etiological factor of halitosis.¹²

Compounds Associated with Halitosis

There are various compounds that could produce unpleasant smell in the oral cavity such as hydrogen sulfide (H₂S), methyl mercaptan (CH₃SH), dimethyl sulfide (CH₃SCH₃), dimethyl disulfide (CH₃SSCH₃), methanethanol ndodecanol, phenol, n-tetradecanol, pyridine.¹³⁻¹⁵ Several studies have demonstrated that hydrogen sulfide (H₂S), methyl mercaptan (CH₃SH), dimethyl sulfide (CH₃SCH₃) are the main causes of oral halitosis and account for about 90 % of the total volatile sulfur compounds (VSCs) found in mouth air.^{8,16-18} VSCs has been shown to result from bacterial putrefaction of peptides,

salivary mucin, blood, gingival crevicular fluid and lysed neutrophils. VSCs are also formed as a result of bacterial putrefaction of protein with sulfur-containing amino acids like cysteine, cystine and methionine which are derived from exfoliated human epithelial cells and white blood cells debris.^{15,19-21} These sulfur compounds are known to be toxic at low concentrations and therefore, they are not only associated with halitosis but may also contribute to the etiology and further progress of gingivitis and periodontitis.¹⁵

Tonzetich reported that VSC can penetrate the pocket epithelium and damage the underlying tissue. Thus, it is possible that oral VSCs formation may be an important aspect in the etiology of periodontal diseases.^{21,22} VSCs could be increased in certain situations. Yaegaki *et al.*²³ found that VSC level and the CH₃SH/H₂S ratio in patients with periodontal diseases were 8 times higher than in healthy control subjects. They also found that tongue coating in patients with periodontal involvement were 4 times higher than the control group.

Smoking was found to be associated with increase oral halitosis. Khaira *et al.*²⁴ did a human clinical trial to evaluate the effectiveness of smoking on VSC level in periodontal pockets of two groups of patients with the similar periodontal diseases. They found that the smoker group had more VSC level in their periodontal pockets.

Queiroz *et al.*²⁵ did a human study to determine the relationship between stress, salivary flow rate and oral VSCs. The results suggested that stress can be a predisposing factor for the increase of VSC level in the mouth air but the mechanism of this increase could not be related to the reduction of the salivary flow. Stress also has been found to increase the level of VSC. In a study of animals, Kurihara *et al.*²⁶ demonstrated positive relation between stress and increase of VSC level in male wistar rats.

Microbiota Associated with Halitosis

Many oral bacteria, especially gram-negative anaerobic species which are found in subgingival plaque and dorsum of the tongue can cause halitosis by producing malodorous compound as by-product of their metabolism like VSC and other products.²⁷ McNamara *et al.*²⁸ demonstrated that the formation of halitosis from incubated saliva correlated with a shift in the microbial flora from a predominately gram-positive to a predominately gram-negative anaerobic flora. With this shift in flora, a depletion of carbohydrates was observed and a rise in the pH level of the stagnant saliva. As the carbohydrates are depleted, the acidogenic gram-positive flora is suppressed and the gram-negative microorganisms become progressively more dominant. Evaluated individually, none of the gram-positive microorganisms produced an unpleasant odor whereas all of the gram-negative microorganisms (*Fusobacterium polymorphum*, *Veillonella alcalescens*, *Bacteroides fundiliformis*, *Klebsiella pneumoniae*) produced oral malodor. Solis-Gaffar *et al.*²⁹ reported that only the gram-negative microorganisms (*Veillonella alcalescens*, *Fusobacterium nucleatum*, *Bacteroides melanogenicus* and *Klebsiella pneumoniae*) produced VSC. The production of VSC was accompanied by an oral malodor and a rise in the pH level of the saliva mixture.

Many bacteria in the periodontal pocket are known to produce VSCs, eg., *Porphyromonas gingivalis*, *Prevotella intermedia*, *Fusobacterium nucleatum*, *Peptostreptococcus micros*, *Bacteroides forsythus*, *Campylobacter rectus*, *Eikenella corrodens*, *Desulfovibrio* species.^{19,30}

These bacteria can be identified in plaque samples when detecting the presence of an enzyme that degrades benzoyl-DL-arginine-a-naphthamide

(BANA) using BANA® test that detects the presence of this enzyme in plaque and tongue samples.³¹ BANA test can be used to correlate the levels of some VSCs. Your levels Morita *et al.*³² have shown that the BANA test correlated significantly with the PS level (H₂S, CH₃SH) found in the gingival sulcus which indicated that a positive plaque BANA test reflected the PS activity of the plaque.

Tongue Coating and Halitosis

A lot of research reports suggested that the tongue is the primary site responsible for halitosis. The dorsoposterior surface of the tongue has been identified as the principal location for the intraoral generation of VSCs.^{1,2,9,33} The shape and topography of the tongue is favorable for the growth of microorganisms since the papillary nature of the dorsum of the tongue creates a unique ecological site that provides an extremely large surface area, favoring the accumulation of oral bacteria. The proteolytic anaerobic bacteria that reside on the tongue which retain considerable amount of desquamated epithelial cells and dead leukocytes play an essential part in the development of oral malodor.³⁴

Tongue coating is more susceptible to increase in patients with chronic periodontitis. Yaegaki *et al.*²³ collected the tongue coating from patients with periodontitis and healthy individuals using a tongue scraper and measured it. They found that tongue coating was greater in periodontal patients than in healthy individuals. They also reported that VSCs production by the periodontal patients' tongue coating was 4 times higher than the control group. In another study, De Boever *et al.*³¹ reported positive relationship between the level of volatile sulfur compounds founded in the breath and tongue coating or deep fissures on the tongue.

Relationship Between Periodontitis and Halitosis

Periodontitis is considered one of the major etiological factors of halitosis. In an old study done by Sulser *et al.*,³⁵ he reported that saliva collected from individuals with periodontal disease putrefied more rapidly than that from healthy individuals. Berg *et al.*³⁶ showed the same correlation by collecting saliva of 100 periodontally involved patients and 100 periodontally healthy individuals. After incubation, saliva of periodontitis patients showed higher amount of hydrolysis, indole and sulfide than that from healthy individuals. Consequently, more oral malodor was produced from the saliva of periodontitis patients. Sharman *et al.*³⁷ found that the prevalence of oral malodor is significantly higher in periodontal patients than in healthy individuals. They also reported that patients with oral malodor had more tooth sites with pockets 5 mm than periodontal patients without oral malodor. Figueiredo *et al.*³⁸ did a study on the relationship of periodontitis with oral malodour. They concluded that VSC level was significantly higher in subjects with probing depth >3 mm and noticed the gingival inflammation contributed the intensity of oral malodor.

In case of periodontitis, the presence of inflammation and number of pockets seems to be more important than the pocket depth itself. Rizzo³⁹ evaluated the production of hydrogen sulfide in the periodontal pockets. He used filter paper strips impregnated with lead acetate. A positive correlation was noted between the amounts of hydrogen sulfide in the gingival crevice and the depth of corresponding periodontal pockets. Coil *et al.*⁴⁰ compared the differences between deep inflamed crevicular sites and shallow non-inflamed sites. They found that the ratio of CH₃SH/H₂S was higher in deep inflamed crevicular sites compared to shallow non-inflamed sites. They

also found that the total sulfur in deep inflamed crevicular sites was significantly higher than the shallow non-inflamed sites. Miyazaki *et al.*⁴¹ proposed that the presence of active inflammation in the periodontal tissue is more associated with oral malodor than the existence of periodontal pockets. They also found that oral malodor might be caused by the tongue coating in younger individuals and the combination of periodontal disease and tongue coating in older individuals.

Morita *et al.*³² evaluated the sulcular sulfide level in subjects with periodontal diseases. They found that there was weak association between low to moderate periodontal disease and oral malodor while there was no correlation between oral malodor and severe bone loss. They suggested that the VSCs within the deep pockets was not released into the oral cavity. They showed that bleeding index was more associated with oral malodor rather than probing depth.

Lee *et al.*⁴² reported that in patients with high level of CH₃SH, 73% had inflammation and pockets deeper than 4 mm while in low level, CH₃SH group 38 % had pockets deeper than 3 mm.

However, some studies have failed to demonstrate positive correlations between halitosis and periodontitis. De Boever³¹ found that tongue odor was negatively correlated with probing depths suggesting an inverse relationship between malodor and periodontal parameters.

Effect of Volatile Sulfur Compounds in Periodontal Tissues

Hydrogen sulfide (H₂S), methyl mercaptan (CH₃SH) can play an important role in periodontal pathogenesis. Both hydrogen sulfide and methyl mercaptan are capable of altering permeability of the gingival tissues which will allow bacterial antigens such as lipopolysaccharide (LPS) to penetrate the gingival tissues

and initiate inflammation.³⁹ Ng *et al.*⁴³ reported that the permeability of porcine sublingual mucosa increased 75% and 103% respectively, following exposure to hydrogen sulfide and methyl mercaptan.

CH₃SH has the ability to induce secretion of interleukin-1 (IL-1). It also has the ability when used alone or in combination with IL-1 to enhance human gingival fibroblasts to secrete prostaglandin E₂, cAMP and procollagenase. These secreted substances may contribute to the increased production of collagenase which caused tissue destruction leading to periodontal disease.⁴⁴

Periodontal ligament cell can be affected by methyl mercaptan. A study done by Lancero *et al.*⁴⁵ showed that exposing periodontal ligament cells to methyl mercaptan will change their intracellular pH and make them become more acidic. There were also decrease in protein synthesis, decrease in motility and alteration of collagen metabolism.

CONCLUSIONS

Halitosis is a common condition which means an unpleasant or offensive odor emanating from the oral cavity. Majority of halitosis cases originates from the oral cavity especially from the tongue and periodontal pockets. Some oral conditions may also produce halitosis. It can also originate from systemic sources or ENT conditions or sometimes due to normal physiological processes.

Volatile sulfur compounds are the major components of oral malodor. Oral VSCs formation is caused by bacterial putrefaction of peptides, mucin found in saliva, blood and gingival crevicular fluid. Different gram-negative anaerobic bacteria have been identified in dorsum of the tongue and periodontal pockets that are capable of producing VSCs.

Several reports have examined the relationship between oral malodor and

periodontal diseases. There are clear indications that the VSC levels in mouth air correlate with the extent of periodontal breakdown. It has been shown that the VSC level increase with the presence of inflammation and number of periodontal pockets. Some authors have indicated the relation of VSCs with the presence of inflammatory process regardless of pocket depth. However, some studies failed to report any correlation.

It is well established that VSCs can penetrate pocket epithelium and damage the underlying tissue. Thus, it is possible that oral volatile sulfur compounds formation may be an important aspect in the etiology of periodontal disease. It appears that methyl mercaptan especially may be involved in the pathogenesis of periodontal disease and other inflammatory diseases. Volatile sulfur compounds can also contribute in forming collagenase which cause tissue destruction leading to periodontal disease and alter collagen metabolism.

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