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Halitosis: Its Intraoral Factors and Mechanism Relating to Formation

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Abstract

Various factors contribute to halitosis, such as smoking, food smell, etc., but intraoral factors are significant. There are many niches for bacteria in oral cavity, so they are suitable for the growth of microorganism. Oral anaerobes can produce volatile sulfur compounds (VSCs) including hydrogen sulfide, methyl mercaptan, dimethyl sulfide and minor components including amines and acids, which may be associated with malodor. Hydrogen sulfide and methyl mercaptan are considered as major ingredients of VSCs responsible for halitosis. This review of literature illustrates the formation and the affiliated intraoral factors to halitosis.

Keywords

Halitosis; Intraoral factor; Mechanism; Oral anaerobes; Volatile sulfur compounds

Introduction

Halitosis, alternatively named as oral malodor (foul breath, breath malodor, and foetor oris) [1], is an abnormal symptom characterized by a foul or offensive odor emanating from the oral cavity with breath air [2]. It is considered as indicators of health status and oral cleanliness with the problems of producing social and psychological handicap [3]. From Greek and Roman times, it has been well recognized in the literature and its remedies can be traced to Hippocrates [4]. With the improvement of life quality and the enhancement of awareness for oral health, more and more people pay attention to halitosis, visiting dentists as a chief complaint and spent a lot of hundred millions in developed countries on breath fresheners. Several studies reported that no significant differences had been found between male and female participants in terms of oral malodour values [5,6]. One recent study shows that halitosis has a worldwide occurrence with a prevalence range of 22% to 50% [7] whereas severe cases are restricted to 5% [8]. Some estimation suggests that approximately 50% the North American population [9], 24% of Japanese individuals [10], 27.5% Chinese population [11], and 15% among Brazilian population [12] are suffered from halitosis epidemiologically. From the recent Switzerland survey, 0–10% of all Europeans have chronic halitosis according to the majority of the surveyed medical professional groups and 11%–20% according to dentists and dental hygienists opinion [13]. The proportion of oral malodor caused by oral factors statistically is about 80-90% [7,14,15], of which about 60% exists that oral microbes occupy the surface of the tongue [16,17].

The symptom of halitosis is generated by oral floras metabolism, food remnants putrefaction and epithelial cells desquamation with copious amounts of volatile sulfur compounds (VSCs) including hydrogen sulfide (H_2S), methyl mercaptan (CH_3SH), dimethyl sulfide and minor components like amines and acids from diverse substances produced by oral microorganisms associated with halitosis [4,8,18]. Furthermore, other compounds such as indole, methyl indole and cadaverine, etc. are affiliated to halitosis [8,10,19,20]. Because of the multi-factors and complexity in the formation of halitosis, its mechanisms, however, are still obscure.

The purpose of this review is to reflect the present knowledge focusing on the formation and intraoral pathologic factors regarding halitosis such as VSCs-producing oral microorganisms, periodontal diseases, tongue and saliva. Another aim is to inspire more and more investigations on this area.

The Formation of Halitosis

Many researchers focused on the formation mechanisms of halitosis and found that the VSCs-producing microorganism mainly contributed to it [20,21,22]. It was demonstrated that the predominant composition of halitosis is VSCs and that some 82 commensal species of microorganisms in oral environment that were named as VSCs-producing bacteria could engender H_2S, CH_3SH and acids in the process of metabolism that are in first place in composite of VSCs [20,23]. Of which, some have a close bearing on halitosis, principally, *Fusobacterium, Veillonella, T.denticola* (*T.d*), *Porphyromonas gingivalis* (*P.g*), *Bcteroides forsythus* (*B f*) and *Peptostreptococcus,* etc. [16,24]. In the oral cavity of elderly people, *Fusobacterium* and *P. melaninogenica* were illustrated to be involved in the production of H_2S [25]. Furthermore, oral gram-negative anaerobes were verified to be responsible for halitosis, such as *P.g, Veillonella parvula, Prevotella intermedia* (*P.i*), *Actinomycetes* (*A.a*) [16,21], *Peptostreptococcus, Eubacterium, Selenomonas, Centipeda, Bacteroides, Fusobacterium* and *T.d* [26]. All pathogens to halitosis can give out VSCs when they metabolize by taking advance of their especial enzyme such as trypsinlike, collagenic hydrolase, L-cysteine desulfhydrase and L-methionine-α-deamino-γ-mercaptomethane-lyase (METase) [18,20,21,27]. The halitosis-associated bacteria metabolize protein [15], cysteine [18,28], L-methionine and other amino acids as substrates to produce H_2S and CH_3SH that are converted to VSCs, but some authors showed that the ratio of methionine to whole free amino acids was significantly higher than that of cysteine [17]. There were remarkable differences of anaerobes between halitosis patients and healthy people, especially *P.g*, which could use sulphur-containing amino acids as substrates to produce a great deal of H_2S [29,30] with collagenic hydrolase and gingivain [21]. Persson, Edlund et al. [26] reported that the genera *Peptostreptococcus, Eubacterium, Selenomonas, Centipeda, Bacteroides* and *Fusobacterium* formed significant amounts of H_2S from L-cysteine while CH_3SH from L-methionine was formed by some members of the genera *Fusobacterium, Bacteroides, Porphyromonas* and *Eub*acterium [26]. The enzymatic reactions involved in the formation of H_2S and CH_3SH are predicted to be as follows: L-cysteine→$^{\text{L-cysteine desulfhydrase}}$→pyruvate +

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ammonia + H_2S, L-methionine→^{METase}→α-ketobutyrate + ammonia + CH_3SH [20]. Moreover, these oral gram-negative anaerobes are able to cause a corresponding decrease in oxidation-reduction potentials (Eh) and at the same time a low Eh favours oral putrefaction and halitosis occurrence [28].

Additionally, *H. pylori* have been shown to produce H_2S and CH_3SH, which could contribute to the development of halitosis [31]. *H. pylori* presence in the oral cavity may be only transient due to the gastric reflux [32]. A recent study showed clear association between gastric *H. pylori* and occurrence of halitosis and coated tongue, and eradication of this infection on the contrary significantly improve halitosis and coated tongue [33]. Other recent studies have indicated a possible involvement of *H. pylori* in the pathogenesis of halitosis and glossitis, and even periodontitis as well [34].

Intraoral related factors to halitosis

The mouth harbors hundreds of bacterial species with various nutritional preferences and provides various microbiological niches in human ecology, such as saliva, dental plaque, periodontal pocket, gingival crevicular fluid (GCF), gingival pocket and tongue etc. [2,16,23,35]. Recently, some researchers [2,29,22] found that the process of developing bad breath is similar to that noted in the progression of gingivitis/periodontitis. Meanwhile, many investigations and experiments focused on the intraoral factors of halitosis respectively.

Intraoral niches for VSCs-producing flora

The main anatomical sources of VSCs identified in the oral cavity are the tongue and gingival sulcus [36]. Researchers suggest that the tongue plays an important role in the production of halitosis [7,16,27]. The surface morphologic characteristics of the tongue dorsum provide additional irregularities such as fissures, grooves and depapillated areas that may serve as retention areas for harboring bacteria, and the papillary structure on the dorsum represents a unique ecological niche in the oral cavity, offering a large surface area that favors the accumulation of oral debris and microorganism [16,20,37]. Furthermore, there is a visible white-brownish layer adhered to the dorsum of the tongue, which is named tongue coating or tongue biofilm, and is considered as a particular niche on the dorsum of tongue composed of desquamated epithelial cells, blood cells and metabolites, nutrients and bacteria [20,38]. Tongue biofilm is anyhow much more of importance by a wide variability and diversity of bacteria, with a high proportion of anaerobic bacteria [39], including *P.g, A.a, Spirochaetes, P.i* [38], *E. Corrodens* and *oral spirochetes* [40]. Gordon et al. [41] were the first to analyze the tongue microbiota and they identified several anaerobic species (*Bacteroides, Fusobacteria spp, Peptococcus* and *Peptostreptococcus*) among the prominent cultivable microorganisms [42]. In 80%-90% of cases, bacterial activities especially on the dorsum of the tongue are implicated [7], so that tongue coating is the major factor in halitosis [16,38]. Thus, with its anatomic and formative characteristics, tongue coating is referred to as one of the major sources of VSCs responsible for main components of halitosis [7,20,34,43,44]. In addition a Brazilian study [44] has found that high tongue coating levels were related with more VSCs counts and found a significant correlation between VSCs and tongue coating scores for all age groups, which was the similarly reported by Hinode et al. [45] and Liu et al. [11] whom also found that no statistically significant relation was observed among the VSCs levels considering age, bleeding and periodontal pockets sites. In a Japanese study, the numbers of total bacteria and H_2S-producing bacteria in the tongue biofilm in the malodorous group were statistically higher than those in the non-odorous group [16,43]. Another research investigated the relationship between halitosis and tongue microbiota in tongue biofilm samples via real-time PCR and found that anaerobes like *P.g, Tannerella forsythia, P. i, Prevotella nigrescens* and *T.d* might contribute greatly to VSCs production [46], while other study reported that an increase in the number of H_2S-producing bacteria such as *Prevotella spp* as well as *Veillonella* and *Actinomyces* in tongue coating sample responsible for oral malodour [47]. The most studies showed a positive correlation between the clinical parameters of halitosis and total bacterial numbers, as well as that and *Prevotella intermedia, Fusobacterium nucleatum* and *Campylobacter rectus* concentrations in the tongue coating samples. However, there was no similar correlation with respect to the saliva samples [5]. Some researchers stated that the tongue dorsum and tongue coating played a more important role in the formation of halitosis than periodontal diseases [6,27]. José R et al. [48] also reported that tongue biofilm seems to be directly involved in the production of oral halitosis and may have an important role in the success of periodontitis therapy since it is a potential reservoir for periodontal pathogens. Other structures on the tongue dorsum were confirmed that the presence of deep fissures was related to the twice counts of bacteria and to the noticeable higher mouth and tongue odor scores [16,27].

Saliva played a positive role in the formation of halitosis [49]. Saliva, containing a multitude of desquamated oral epithelial cells, leukocytes, bacteria, food residues and a little of proteins and enzymes, is a feasible culture for oral bacteria. As a result, external degeneration of saliva provides substrates to be oxidized as VSCs [49,50]. Besides, the pH, Eh and the quantity of saliva also are effective in the formation of malodor [51]. The higher density of hydrogen ions and the larger quantity of saliva refrain or mitigate oral malodorous formation, whereas neutrality and alkalinity of saliva promote production of malodor. The pH value on the surfaces of oral mucosa is mainly determined by the fermentative and putrefactive activities of the adhered bacteria, and these acid-base processes are of importance for regulating the formation of halitosis [49]. According to microbiota in saliva, the previous reports suggested that *P. g* and *P. i* in saliva contribute to oral malodour [52]. However, recently it has been found that there was no positive correlation between specific oral bacterial species in resting saliva and VSCs concentrations [5]. The comparison of microbial profile with VSCs levels showed a weak positive correlation of *P. g, F. n* and *T. f* count with Organoleptic and Tanita score. A quantitative analysis of *P. g, F. n, T. f* and *T. d* in the saliva, on the tongue coating, and in the subgingival plaque of patients with oral malodor using real time PCR showed correlation between increased VSCs levels and increased levels of *P. g* and *T. f* in subgingival sample and *F. n* in tongue coating [53].

Dental plaque especially materia alba, gingival pocket, periodontal pocket, and gingival crevicular fluid serve the formation of halitosis as important niches for VSCs-producing bacteria. These niches provide suitable microbial ecosystem for the development and growth of diverse and plentiful oral flora.

Periodontal diseases and halitosis

By the examination of gas chromatography, the amount of VSCs and CH_3SH/H_2S ratio in mouth air from patients with periodontal involvement were 8 times greater than those of control subjects [17] and these two parameters increased in proportion to the bleeding

index and probing depth in another investigation [38]. Many studies confirmed that the primary causative microbes for halitosis were similar to the periodontopathic bacteria to some extent [16,41,48], such as *P. g, P. i, A. a*, and *T. d* [2,21,29,54], so that periodontal disease–associated bacteria are capable of producing large amounts of VSCs [20]. Furthermore, gingival diseases and periodontitis might lead to halitosis [3,17,54] due to the maturation of subgingival plaque suitable for the growth of anaerobes in periodontal pocket. Meanwhile, VSCs could destroy periodontal tissues in turn [29,55]. An increasing volume of evidence is demonstrating that extremely low concentrations of many of these compounds are highly toxic to tissues [55]. It can increase the permeability of the oral mucosa [56] and decrease protein or collagen synthesis [57,58]. These compounds are highly toxic, especially CH_3SH [10]. H_2S, CH_3SH and other substances could prevent fibroblasts or collagen from synthesis [22,55] and inhibit cell migration in periodontal ligament cells [59], then invade into deeper tissue directly to make kinds of toxin devastate periodontal tissues. The presence of CH_3SH within a periodontal pocket was involved in the induction or progression of periodontal disease [22]. The increase in the ratio of CH_3SH to H_2S in human gingival crevicular sites was correlated with deeper pockets or bleeding pockets [60]. In addition, CH_3SH also increases permeability of intact mucosa and stimulates production of cytokines which have been associated with periodontal disease [55]. Consequently, some periodontopathic bacteria produce VSCs, which cause halitosis, aggravate periodontal diseases as well in the same time. Some studies, however, showed that there was a much weaker association between VSCs and periodontal conditions [44,61]. These observations tend to suggest that oral malodour is not caused by periodontal disease alone, but by a combination of factors, including periodontal conditions and tongue coating.

Oral healthcare is obviously paying more and more attention along with more attention nowdays. Due to the psychological and physical harm as a kind of oral disease, halitosis is drawing more and more attention from dentists and researchers. From mouth access, external supplies of various foods are passed and nearly 400 species of microorganisms could be identified [29]. Oral cavity provides a favorable condition for the survival and development of diverse and numerous oral flora, owing to its proper temperature, humidity and food remnants. Of these oral floras, anaerobes such as *P.g, Veillonella parvula, P.i* and *Actinomycetes,* etc. are found in dental plaque, periodontal pocket, tongue coating and saliva, and confirmed as the pathogens for halitosis to engender H_2S, CH_3SH, indole, etc. in the progress of metabolism [2]. Recently it has been investigated that the microbial profile of the tongue dorsum showed maximum counts of *F.n* followed by *P.g* and *T.f* [53]. Rotten food residue and decomposed cells play a role in halitosis as well. Candidiasis, mouth dryness, caries, open root canals, neglected prostheses or insufficient oral hygiene also lead to halitosis often [13,62]. So, the most importantly related factors to halitosis are VSCs-producing bacteria, and the action of periodontal pocket, tongue dorsum coating and saliva. Meanwhile, VSCs products make an aggravation in periodontal diseases, and vice versa more VSCs components.

Considering the formation and related factors of halitosis, the initial treatment strategy should focus on the exact cause and on oral hygiene [63]. Except for oral health instruction, necessary treatments are used for the patients with halitosis [64]. The patients with halitosis should destroy oral niches for VSCs-producing, using mechanical and medical means. Most approaches were found to be inefficient and/or short lasting. The most successful treatment involved mechanical debridement (including tooth brushing, flossing, chewing gums and tongue cleaning), possibly combined with the use of an antimicrobial mouth rinse, such as chlorhexidine, cetylpiridinium chloride and zinc lactate [22,48,64,65]. Several previous and recent studies reported that breath odor and tongue coating status, tongue coating scores, concentrations of CH_3SH and total VSCs are improved after instructions on tongue brushing or with patients who had the habit of tongue cleaning [5,66]. Investigators have showed that the mouth rinse is efficient, which contains metal ions, especially zinc, that inhibit odor formation because of its affinity to sulphur compounds [63,67]. Thus, tongue cleaning is an effective method for preventing or improving halitosis.

Better education of both the public and dental professionals as to the most frequent cause of halitosis, insufficient oral hygiene, might elevate the level of compliance by patients [64]. It is necessary to make oral examination regularly for them. Also, the patients with halitosis should avoid smell-producing foods and habit with those such as garlic, onion, smoke, etc...

The formation and mechanism of VSCs for halitosis are partially crystallized according to the current literature. The oral ecology and the interaction among microorganisms and saliva, tongue coating, the dorsum of tongue and embrasure of gingival papilla are expected for further researches on mechanism, prevention and treatment of halitosis.

Halitosis caused by oral pathologic causes is associated with anaerobes in the oral niches including periodontal area, dental plaque, tongue, GCF and saliva, which can produce VSCs in metabolization. This article concludes the formation mechanisms of halitosis and its intraoral related factors, such as niches for VSCs-producing bacteria and periodontal diseases.

References

1. Gokdogan O, Catli T, Ileri F (2015) Halitosis in otorhinolaryngology practice. Iran J Otorhinolaryngol 27: 145-153.
2. Morita M, Wang HL (2001) Association between oral malodor and adult periodontitis: a review. J Clin Periodontol 28: 813-819.
3. Tonzetich J (1978) Oral malodour: an indicator of health status and oral cleanliness. Int Dent J 28: 309-319.
4. Sreenivasan PK, Furgang D, Zhang Y, DeVizio W, Fine DH (2005) Antimicrobial effects of a new therapeutic liquid dentifrice formulation on oral bacteria including odorigenic species. Clin Oral Investig 9: 38-45.
5. Amou T, Hinode D, Yoshioka M, Grenier D (2014) Relationship between halitosis and periodontal disease - associated oral bacteria in tongue coatings. Int J Dent Hyg 12: 145-151.
6. Miyazaki H, Sakao S, Katoh Y, Takehara T (1995) Correlation between volatile sulphur compounds and certain oral health measurements in the general population. J Periodontol 66: 679-684.
7. Akaji EA, Folaranmi N, Ashiwaju O (2014) Halitosis: a review of the literature on its prevalence, impact and control. Oral Health Prev Dent 12: 297-304.
8. Chomyszyn-Gajewska M (2012) Contemporary views on etiology and pathogenesis of halitosis. Przegl Lek 69: 1293-1296.
9. Lee SS, Zhang W, Li Y (2007) Halitosis update: a review of causes, diagnoses, and treatments. J Calif Dent Assoc 35: 258-260, 262, 264-8.
10. Scully C, el-Maaytah M, Porter SR, Greenman J (1997) Breath odor: etiopathogenesis, assessment and management. Eur J Oral Sci 105: 287-293.
11. Liu XN, Shinada K, Chen XC, Zhang BX, Yaegaki K, et al. (2006) Oral malodor-related parameters in the Chinese general population. J Clin Periodontol 33: 31-36.
12. Nadanovsky P, Carvalho LB, Ponce de Leon A (2007) Oral malodour and its association with age and sex in a general population in Brazil. Oral Dis 13: 105-109.

13. Roth B, Oppliger N, Filippi A (2014) Knowledge of different medical and dental professional groups in Switzerland about halitosis. Swiss Dent J 124: 1302-1312.

14. Spielman AI, Bivona P, Rifkin BR (1996) Halitosis. A common oral problem. N Y State Dent J 62: 36-42.

15. Feller L, Blignaut E (2005) Halitosis: a review. SADJ 60: 17-19.

16. De Boever EH, Loesche WJ (1995) Assessing the contribution of anaerobic microflora of the tongue to oral malodor. J Am Dent Assoc 126: 1384-1393.

17. Yaegaki K, Sanada K (1992) Biochemical and clinical factors influencing oral malodor in periodontal patients. J Periodontol 63: 783-789.

18. Hughes FJ, McNab R (2008) Oral malodour - a review. Arch Oral Biol 53:S1-S7.

19. Kleinberg I, Codipilly M (1999) Modeling of the oral malodor system and methods of analysis. Quintessence Int 30:357-369.

20. Nakano Y, Yoshimura M, Koga T (2002) Correlation between oral malodor and periodontal bacteria. Microbes Infect 4: 679-683.

21. Allaker RP (2010) Investigations into the micro-ecology of oral malodour in man and companion animals. J Breath Res 4: 017103.

22. Yoshimura M, Nakano Y, Yamashita Y, Oho T, Saito T, et al. (2000) Formation of methyl mercaptan from L-methionine by Porphyromonas gingivalis. Infect Immun 68: 6912-6916.

23. Takahashi N (2005) Microbial ecosystem in the oral cavity: Metabolic diversity in an ecological niche and its relationship with oral diseases. International Congress Series 1284: 103-112.

24. Rosenberg M, Kozlovsky A, Gelernter I, Cherniak O, Gabbay J, et al. (1995) Self-estimation of oral malodor. J Dent Res 74: 1577-1582.

25. Senpuku H, Tada A, Yamaga T, Hanada N, Miyazaki H (2004) Relationship between volatile sulphide compounds concentration and oral bacteria species detection in the elderly. J Int Dent 54: 149-153.

26. Persson S, Edlund MB, Claesson R, Carlsson J (1990) The formation of hydrogen sulfide and methyl mercaptan by oral bacteria. Oral Microbiol Immunol 5: 195-201.

27. De Boever EH, Uzeda M, Loesche WJ (1995) Role of tongue surface characteristics and tongue flora in halitosis. J Dent Res 74: 127.

28. Kleinberg I, Codipilly DM (2002) Cysteine challenge testing: a powerful tool for examining oral malodour processes and treatments in vivo. Int Dent J 52 Suppl 3: 221-228.

29. Wåler SM (1997) [Bad breath from the oral cavity]. Tidsskr Nor Laegeforen 117: 1618-1621.

30. Waler SM (1997) On the transformation of sulfur-containing amino acids and peptides to volatile sulfur compounds (VSC) in the human mouth. Eur J Oral Sci 105: 534-537.

31. Hoshi K, Yamano Y, Mitsunaga A, Shimizu S, Kagawa J, et al. (2002) Gastrointestinal diseases and halitosis: association of gastric Helicobacter pylori infection. Int Dent J 52 Suppl 3: 207-211.

32. Al-Ahmad A, Kürschner A, Weckesser S, Wittmer A, Rauberger H, et al. (2012) Is Helicobacter pylori resident or transient in the human oral cavity? J Med Microbiol 61: 1146-1152.

33. Zaric S, Bojic B, Popovic B, Milasin J (2015) Eradication of gastric Helicobacter pylori ameliorates halitosis and tongue coating. J Contemp Dent Pract 16: 205-209.

34. Nisha KJ, Nandakumar K, Shenoy KT, Janam P (2016) Periodontal disease and Helicobacter pylori infection: a community-based study using serology and rapid urease test. J Investig Clin Dent 7: 37-45.

35. Krespi YP, Rosenberg M (2004) The relationship between oral malodor and volatile sulfur compounds producing bacteria. Otolaryngology - Head and Neck Surgery 131: 212–213.

36. Rosenberg M (1996) Clinical assessment of bad breath: current concepts. J Am Dent Assoc 127: 475-482.

37. Roldán S, Herrera D, Sanz M (2003) Biofilms and the tongue: therapeutical approaches for the control of halitosis. Clin Oral Investig 7: 189-197.

38. Yaegaki K, Sanada K (1992) Volatile sulfur compounds in mouth air from clinically healthy subjects and patients with periodontal disease. J Periodontal Res 27: 233-238.

39. Loesche WJ, Kazor C (2002) Microbiology and treatment of halitosis. J Periodontol. 28: 256-279.

40. Lee KH, Tanner AC, Maiden MF, Weber HP (1999) Pre- and post-implantation microbiota of the tongue, teeth, and newly placed implants. J Clin Periodontol 26: 822-832.

41. Gordon DF Jr, Gibbons RJ (1966) Studies of the predominant cultivable micro-organisms from the human tongue. Arch Oral Biol 11: 627-632.

42. Goldberg S, Cardash H, Browning H 3rd, Sahly H, Rosenberg M (1997) Isolation of Enterobacteriaceae from the mouth and potential association with malodor. J Dent Res 76: 1770-1775.

43. Washio J, Sato T, Ikawa K (2005) Relationship between hydrogen sulfide-producing bacteria of the tongue coating and oral malodor. International Congress 1284: 199-200.

44. Calil C, Liberato FL, Pereira AC (2009) The relationship between volatile sulphur compounds, tongue coating and periodontal disease. Int J Dent Hyg 7: 251-255.

45. Hinode D, Fukui M, Yokoyama N, Yokoyama M, Yoshioka M, et al .(2003) Relationship between tongue coating and secretory immunoglobulin A level in saliva obtained from patients complaining of oral malodor. J Clin Periodontol 30: 1017-1023.

46. Tanaka M, Yamamoto Y, Kuboniwa M, Nonaka A, Nishida N, et al. (2004) Contribution of periodontal pathogens on tongue dorsa analyzed with real-time PCR to oral malodor. Microbes Infect 6: 1078-1083.

47. Washio J, Sato T, Koseki T, Takahashi N (2005) Hydrogen sulfide-producing bacteria in tongue biofilm and their relationship with oral malodour. J Med Microbiol 54: 889-895.

48. Cortelli JR, Barbosa MD, Westphal MA (2008) Halitosis: a review of associated factors and therapeutic approach. Braz Oral Res 22 Suppl 1: 44-54.

49. Kleinberg I, Westbay G (1992) Salivary and metabolic factors involved in oral malodor formation. J Periodontol 63: 768-775.

50. Tonzetich J (1977) Production and origin of oral malodor: a review of mechanisms and methods of analysis. J Periodontol 48: 13-20.

51. Tonzetich J, Carpenter PA (1971) Production of volatile sulphur compounds from cysteine, cystine and methionine by human dental plague. Arch Oral Biol 16: 599-607.

52. Awano S, Gohara K, Kurihara E, Ansai T, Takehara T (2002) The relationship between the presence of periodontopathogenic bacteria in saliva and halitosis. Int Dent J 52 Suppl 3: 212-216.

53. Kamaraj R D, Bhushan KS (2014) An evaluation of microbial profile in halitosis with tongue coating using PCR (polymerase chain reaction)- a clinical and microbiological study. J Clin Diagn Res 8: 263-267.

54. Yang F, Huang S, He T, Catrenich C, Teng F, et al. (2013) Microbial basis of oral malodor development in humans. J Dent Res 92: 1106-1112.

55. Ratcliff PA, Johnson PW (1999) The relationship between oral malodor, gingivitis, and periodontitis. A review. J Periodontol 70: 485-489.

56. Ng W, Tonzetich J (1984) Effect of hydrogen sulfide and methyl mercaptan on the permeability of oral mucosa. J Dent Res 63: 994-997.

57. Johnson PW, Yaegaki K, Tonzetich J (1992) Effect of volatile thiol compounds on protein metabolism by human gingival fibroblasts. J Periodontal Res 27: 553-561.

58. Johnson P, Yaegaki K, Tonzetich J (1996) Effect of methyl mercaptan on synthesis and degradation of collagen. J Periodontal Res 31: 323-329.

59. Lancero H, Niu J, Johnson PW (1996) Exposure of periodontal ligament cells to methyl mercaptan reduces intracellular pH and inhibits cell migration. J Dent Res 75: 1994-2002.

60. Coli JM, Tonzetich J (1992) Characterization of volatile sulphur compounds production at individual gingival crevicular sites in humans. J Clin Dent 3: 97-103.

61. Bosy A, Kulkarni GV, Rosenberg M, McCulloch CA (1994) Relationship of oral malodor to periodontitis: evidence of independence in discrete subpopulations. J Periodontol 65: 37-46.

62. Nalcaci R, Baran I (2008) Factors associated with self-reported halitosis (SRH) and perceived taste disturbance (PTD) in elderly. Arch Gerontol Geriatr 46: 307-316.

63. Meningaud JP, Bado F, Favre E, Bertrand JC, Guilbert F (1999) [Halitosis in 1999]. Rev Stomatol Chir Maxillofac 100: 240-244.

64. Delanghe G, Ghyselen J, Bollen C, van Steenberghe D, Vandekerckhove BN, et al. (1999) An inventory of patients' response to treatment at a multidisciplinary breath odor clinic. Quintessence Int 30: 307-310.

65. Quirynen M, Zhao H, van Steenberghe D (2002) Review of the treatment strategies for oral malodour. Clin Oral Investig 6: 1-10.

66. Ohmori M, Miyazaki A, Sato H (2005) A study on the effects of tongue cleaning. J Jpn Assoc Periodontol 47: 36-43.

67. Zaric S, Bojic B, Popovic B, Milasin J (2015) Eradication of gastric Helicobacter pylori ameliorates halitosis and tongue coating. J Contemp Dent Pract 16: 205-209.

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