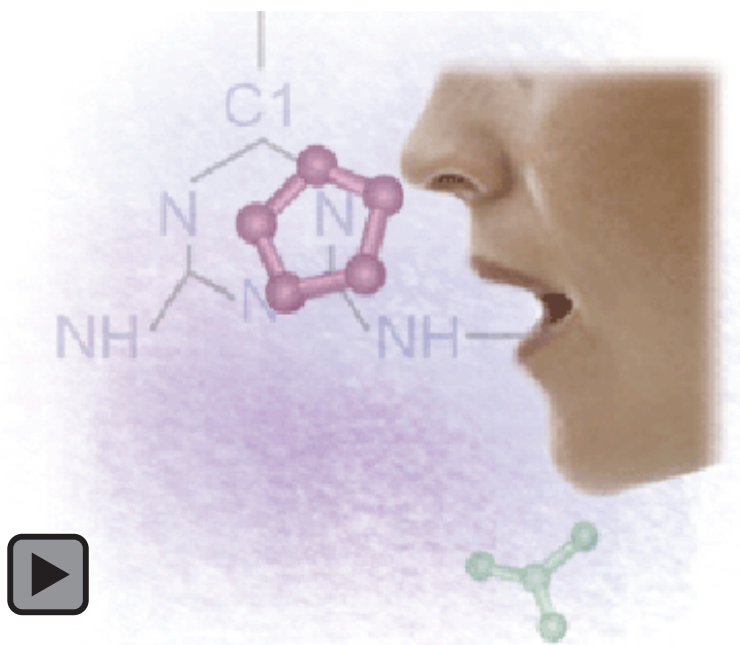


## *Fundamentals of Breath Malodour*

Mariano Sanz, MD, DDS; Silvia Roldán, DDS, MS;  
David Herrera, DDS, MS



### **Abstract**

Breath malodour is a condition that has health and social implications. The origin of breath malodour problems are related to both systemic and oral conditions. The advice of dental professionals for treatment of this condition occurs with regularity since 90% of breath odor problems emanate from the oral cavity. This paper provides a comprehensive review of the etiology of breath odor, its prevalence, diagnosis, and treatment strategies for the condition.

**Keywords:** Breath malodour, oral malodour, halitosis, oral halitosis, high volatile compounds, Halimeter, organoleptic, pseudohalitosis, halitophobia

**Citation:** Sanz M, Roldán S, Herrera D. Fundamentals of Breath Malodour. J Contemp Dent Pract 2001 Nov;(2)4: 001-017.

© Seer Publishing

## Introduction



Breath malodour is a condition that has health and social implications rendering it an area of oral science that spans medical and psychological issues. Current social norms emphasize the importance of personal image and interpersonal relationships. In this context, breath malodour may be an important factor in social communication and, therefore, may be the origin of concern not only for a possible

health condition but also for frequent psychological alterations leading to social and personal isolation.

This being the case, oral malodour's importance goes beyond the knowledge of its cause, diagnosis, and therapy because it interacts with other sociological issues such as culture, religion, race, sex, and social taboos. Knowledge and written reference to this condition dates back to ancient cultures. A clear example comes from the Hebraic liturgics (the Talmud), dating back more than two thousand years ago, which clearly states the terms of a marriage license (the Ketuba) may be legally broken in case of malodour of one of the partners. Similar references can be found in writings from Greek, Roman, early Christian, and Islamic cultures. However, this condition was not studied scientifically until the 1940's and 1950's when Fosnick et al developed an instrument called the osmoscopy, which measures the sources of malodour. They demonstrated this problem could be either physiologic or pathologic, and the source of bad breath could originate from the mouth, the nasopharynx, or various other parts of the body. During the last 30 years, our knowledge of this phenomenon has become much greater, and the sources and causes of malodour have become clearer.

Halitosis is a general term used to describe an unpleasant or offensive odour emanating from the oral cavity. Although several non-oral sites have been related to oral malodour, including the upper and lower respiratory tracts, the gastrointestinal tract, and some diseases involving the kidneys or the liver, it is thought that around 90% of all bad breath odours emanate from the mouth itself.<sup>1,2</sup> Oral halitosis is the specific term used to define halitosis with an origin within the oral cavity.

Oral halitosis is a very common problem in dental patients. In fact, most adult subjects have socially unacceptable bad breath when waking up in the morning. This problem is transitory and attributed to physiologic causes such as reduced saliva flow during sleep. Although these transitory problems are easily controlled, persistent bad breath may be indicative either of oral diseases (i.e., periodontal diseases, the presence of bacterial reservoirs in the mouth) or indicative of systemic diseases (i.e., hiatus hernia, hepatic cirrhosis, or diabetes mellitus). Due to the importance of social interactions in contemporary society, the population in the western countries is becoming more concerned and paying more attention to this problem. This has been reflected in the results of a telephone survey carried out in the United States where 60% of American women and 50% of American men referred to using cosmetic breath-freshening products.<sup>3</sup>

However, in spite of this general concern and the possible pathological implications of halitosis, health professionals, including dental professionals, generally lack adequate training on this condition. Therefore, they are unable to treat or properly advise this population.<sup>4</sup> The aim of this review is to summarize the current knowledge on halitosis and to clarify some frequent misconceptions that lead us to mistreat or fail to treat patients suffering from it. Emphasis will be placed on its etiology and on the role of the general dentist as the most appropriate professional to diagnose and manage this condition.

## Prevalence and Social Importance of Halitosis

Information regarding the prevalence of breath malodour is scarce. It is very difficult to determine the exact number or percentage of the population who have oral malodour since there is a lack of epidemiological studies that address this issue. Moreover, there are no universally accepted standard criteria, objective or subjective, that define a halitosis patient.<sup>5</sup> A large study performed in Japan involving 2,672 individuals indicated that 6-23% of the subjects had oral malodour as measured by volatile sulphur compounds (VSCs) higher than 75 parts per billion (ppb) in expired air at some period during the day.



If these Japanese data reflect the prevalence of oral malodour in other populations, then oral malodour would represent a major oral health concern of the public.<sup>6</sup> Another study in the United States involving individuals older than 60 years found 24% to have been told that they had oral malodour.<sup>7</sup> Another source of indirect information is related to the American Dental Association's 1995 annual session where 92% of the dentists surveyed reported they had patients with chronic bad breath based on the patient's self report. Almost half reported seeing six or more patients weekly with unpleasant breath.<sup>4</sup> On the other hand, bad breath merits concern as virtually all individuals may occasionally experience episodes of malodour. Furthermore, some authors estimate that approximately 50% of middle-aged and older individuals emit socially unacceptable breath, attributed to physiological causes, upon arising in the morning.<sup>8</sup>

The reported incidence ratio between female and male patients with oral malodour is almost the same; no gender-based differences have been found with regard to prevalence and severity of halitosis.<sup>9,10</sup> However, it has been observed that women seek treatment more often than men.<sup>6,9</sup> This could be explained because women are normally more concerned about their health status and appearance. Moreover a significant age-related increase in the mean values of odor-causing VSCs has been reported when different age groups have been assayed.<sup>6</sup>

In spite of this reported high prevalence of breath malodour, only a few patients visit dental clinics seeking treatment. This fact has been termed the "bad breath paradox" since people suffering from bad breath often remain completely unaware of this fact. Whereas, others remain convinced they suffer from oral malodour, although in some circumstances, no objective basis can be found (pseudohalitosis or halitophobia).<sup>11</sup> This fact does not mean that all patients coming to seek treatment present a psychological component. They frequently are pushed to seek therapy by people living in close contact with them such as a spouse, family member, or friend.<sup>12</sup>

Although there is anecdotal and indirect evidence suggesting people have trouble estimating their own bad breath, the first quantitative study to address this question was carried out by Rosenberg and co-workers in a group of 52

subjects, 83% of whom complained of having bad breath.<sup>13</sup> The results of the study demonstrated the subjects studied were generally incapable of scoring their own oral malodour in an objective way. Subjects' preconception scores recorded prior to self-measurement were not associated with the scores of the odour judge, the laboratory tests, or the dental measurements. Self-estimates of whole mouth and tongue malodour were closely related to preconception scores and were similarly subjective. Only in the case of saliva were subjects partially capable of objective self-estimation. Nevertheless, in the subsequent post-measurement self-assessment, participants reverted to subjective scores closely resembling their initial preconception.<sup>13</sup> Moreover, it seems that objective, self-estimation of oral malodour is not an ability that can be acquired with training or experience as was demonstrated by this research group. They demonstrated that despite the initial consultation and instruction, subjects remained unable to self-estimate their own oral malodour in an objective way one year after the consultation.<sup>14</sup> Some gender based differences in regards to the ability to self-estimate the malodour level have been identified, with women tending to overestimate their own malodour. The underlying reasons leading people to believe mistakenly they suffer from bad breath or to exaggerate self-estimations of bad breath are not yet clear.<sup>15</sup>

### Classification of Halitosis

When dealing with the problem of halitosis or with the halitosis patient, it is important to distinguish between "genuine halitosis" and "pseudo-halitosis." "Genuine halitosis" is where the breath malodour is a real problem that can be easily diagnosed either by organoleptic or by physico-chemical means. "Pseudo-halitosis" is where the oral malodour does not exist, but the patient believes that he or she has it. If after successful treatment for either genuine halitosis or pseudo-halitosis the patient still believes that he or she has halitosis, then the diagnosis is termed "halitophobia." This simple classification system includes corresponding treatment needs (Miyazaki et al<sup>16</sup>) and allows the clinician to differentiate between a pathological and a psychological condition. (Table 1)

Genuine halitosis is sub classified as physiologic or pathologic halitosis. Physiologic halitosis, also termed transient halitosis, has its origin in the dorsum of the tongue, is self-limited, does not prevent the patient from carrying out a normal life, and



Table 1: Classification of Halitosis with Corresponding Treatment Needs (TN)

Classification	Treatment Need	Description
I. Genuine halitosis		Obvious malodour, with intensity beyond socially acceptable level, is perceived.
I.A. Physiologic halitosis	TN-1:	<ol style="list-style-type: none"> <li>1. Malodour arises through putrefactive process within the oral cavity. Neither specific disease, nor pathologic condition that could cause halitosis is found.</li> <li>2. Origin is mainly the dorsoposterior region of the tongue.</li> <li>3. Temporary halitosis due to dietary factors (e.g., garlic) should be excluded</li> </ol>
I.B. Pathologic halitosis		
(i) Oral	TN-1 and TN-2:	<ol style="list-style-type: none"> <li>1. Halitosis caused by disease, pathologic condition, or malfunction of oral tissues.</li> <li>2. Halitosis derived from tongue coating, modified by pathologic condition (e.g., periodontal disease, xerostomia) is included in this subdivision</li> </ol>
(ii) Extraoral	TN-1 and TN-3:	<ol style="list-style-type: none"> <li>1. Malodour originates from nasal, perinasal, and/or laryngeal regions.</li> <li>2. Malodour originates from pulmonary tract or upper digestive tract.</li> <li>3. Malodour originates from disorders anywhere in the body, whereby, the odour is bloodborne and emitted via the lungs (e.g., diabetes, hepatic cirrhosis, uremia, internal bleeding).</li> </ol>
II. Pseudo-halitosis	TN-1 and TN-4:	<ol style="list-style-type: none"> <li>1. Obvious malodour is not perceived by others although the patient stubbornly complains of its existence.</li> <li>2. Condition is improved by counselling (using literature support, education and explanation of examination results) and simple oral hygiene measures.</li> </ol>
III Halitophobia	TN-1 and TN-5:	<ol style="list-style-type: none"> <li>1. After treatment for genuine halitosis or pseudohalitosis, the patient persists in believing that he/she has halitosis.</li> <li>2. No physical or social evidence exists to suggest that halitosis is present.</li> </ol>
Taken from <sup>16</sup> Yaegaki K, Coil JM. Examination, classification, and treatment of halitosis; clinical perspectives. J Can Dent Assoc. 2000 May;86(5):257-61. Review.		

usually does not need any therapy. This situation, also termed "morning breath," is more a cosmetic problem than a health-related condition.

On the contrary, pathologic halitosis is permanent, does not resolve by usual oral hygiene methods, and prevents the patient from carrying out a "normal" life. This being the case, pathologic halitosis should be treated and its therapeutic approach will depend on the source of the malodour. Depending on its origin, this pathologic halitosis has been sub classified as follows:

- Oral: the origin of the pathologic condition is in the oral cavity and/or in the posterior dorsum of the tongue

- Extra oral: the origin of the pathologic condition is outside of the mouth (upper/lower respiratory tract, digestive system, systemic disorders, etc.)

A key factor in the management of this problem is the diagnosis of the malodour origin.

### Etiology of Halitosis

Periodontal diseases, in particular, acute necrotizing ulcerative gingivitis (ANUG), severe periodontitis, pericoronitis<sup>17,18</sup>, dry socket, other oral infections<sup>17</sup>, and ulcers<sup>17,19</sup> have been classically associated with oral malodour. This relationship was only established by case reports and clinical experience. Probably one of the most important scientific reports regarding the origin of oral malodour is that of Delanghe et al.<sup>1</sup> In a group of 260 patients visiting their breath odour

clinic, they found that approximately 87% of the cases had halitosis of oral origin, 8% had malodour originating in the ears, nose, and throat (ENT) region, and in 5% of the patients the cause could not be determined. In the group of the patients with an oral origin, 41% had tongue coating, 31% had gingivitis, and 28% had periodontitis. This report supports the results of many investigations and the clinical experience of the experts worldwide that only a minority of halitosis cases diagnosed cannot be treated in a dental clinic. These cases should be referred to their physicians or an ENT specialist for further investigation.

In spite of this low frequency, halitosis may also reflect a serious local or systemic condition. Anaerobic infections localized in the upper respiratory tract, such as chronic sinusitis or tonsillitis, are the most frequent ENT sources of malodour, although lung abscesses or neoplasms may also cause it. Systemic conditions causing halitosis are very rare, although they are important and should not be completely ruled out when dealing with a halitosis patient. Such conditions include diabetic acidosis, hepatic failure/infection, or trimethylaminuria. Conditions related to the digestive system are extremely rare contributors to oral halitosis.

### Factors Involved In The Etiology of Halitosis

Halitosis is due to the presence of odorous gases in the air expelled from the oral cavity, therefore, most of the efforts in studying the etiology of this condition have been devoted to the identification of these gases. VSCs (i.e., hydrogen sulphide, methyl mercaptan, and dimethyl sulphide) are the gases that have demonstrated a higher correlation with halitosis. However, other gases not containing sulphur have also been identified as potential contributors to malodour such as volatile aromatic compounds (indole, skatole), organic acids (acetic, propionic), and amines (cadaverine<sup>20</sup>, putrescine).<sup>21</sup>

VSCs are mainly produced through putrefactive activities of bacteria present in saliva, the gingival crevice, the tongue surface, and other areas.<sup>7,8,22,23</sup> The substrates are sulphur-containing amino acids such as cysteine, cystine, and methionine which are found free in saliva, gingival crevicular fluid, or produced as a result of proteolysis of protein substrates.<sup>17,24</sup> Epithelial cells shed from

different locations of the oral cavity<sup>25,26</sup>, and effused leukocytes are the major sources of such substrates.<sup>26</sup> (Figure 1)

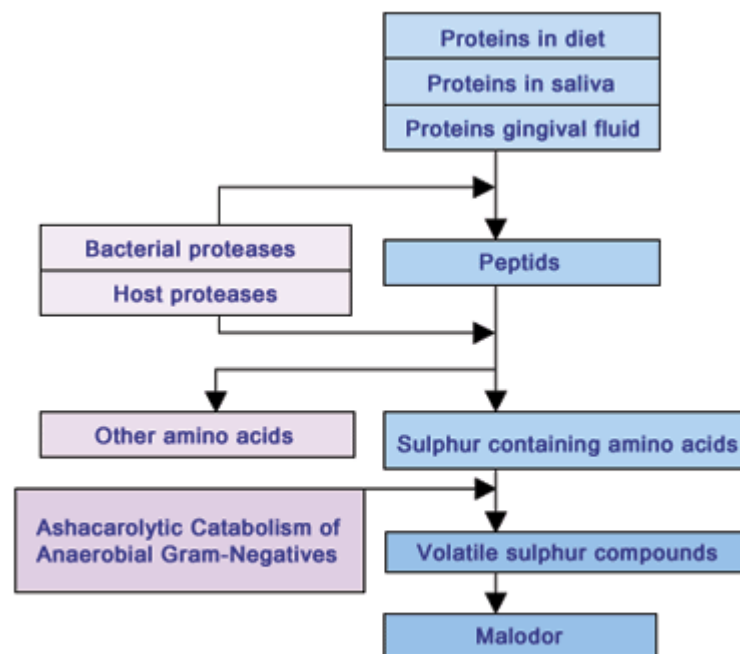


Figure 1. Production of Volatile Sulphur Compounds (VSCs)

Production and release of the VSCs appear to depend on many local factors (approximately 74 total):

- **Bacterial population:** predominance of gram-negative anaerobes.
- **Physical-chemical conditions:** salivary pH and oxygen depletion, for example.
- The substrates available for bacterial metabolism found in saliva, crevicular fluid and at a lower extent, in diet.

### The Role of Bacteria In Oral Halitosis

Oral microorganisms play an important role in the production of malodour. In the absence of microorganisms, the odoriferous components are not generated. Moreover, McNamara et al<sup>27</sup> using in vitro methods demonstrated the formation of malodour components from incubated saliva correlated with a shift in the microflora from a predominately gram-positive to a predominately gram-negative anaerobic flora. Different authors have studied the in vitro capability of different bacteria to generate VSCs. Among the species capable of VSC production are



Peptostreptococcus, Eubacterium, Selenomonas, Centipeda, Bacteroides, and Fusobacterium. From these species, specific microorganisms such as Porphyromonas gingivalis, Treponema denticola, and Porphyromonas endodontalis tend to be associated with periodontitis or periapical infections and are rarely found in a healthy mouth.

The putative malodourous species identified are mainly gram-negative anaerobes. Their main nutrient sources are proteins, peptides, or amino acids that, under specific physico-chemical conditions, are degraded to VSCs and other odouriferous substances. These gram-negative anaerobic bacteria can be isolated from the subgingival plaque in gingivitis and periodontitis patients and from the dorsum of the tongue in periodontally healthy subjects.

### The Role of Physical and Chemical Conditions of the Oral Cavity

Apart from the presence of gram-negative anaerobic bacteria, certain physical-chemical conditions are needed for the production of odouriferous gases. These conditions such as pH, pO<sub>2</sub> (oxygen level), and Eh (Oxidation-reduction potential) are usually determined by the bacterial metabolism. If the main nutrient sources are carbohydrates, their fermentation shifts the environment towards an acidic pH and the VSC formation is inhibited. If, on the contrary, the main nutrient source is protein, its metabolic end products such as nitrogenous compounds (including urea, free amino acids, and amino acids) increase the pH. This neutral or alkaline environment will favor anaerobic bacterial growth and VSC production, thereby, increasing oral malodour.<sup>28</sup>

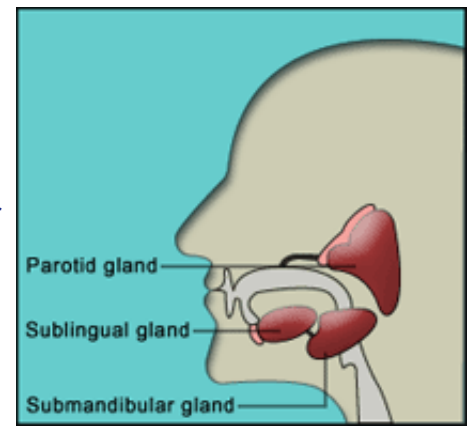
Moreover, in an oxygen-depleted environment the pH is lowered which also favours VSC production.

### The Role of Substrates

Various authors have tried to reproduce the halitosis process in the laboratory by incubating saliva under different conditions. Saliva consists of a complex mixture of secretions from the salivary glands together with multiple species of bacteria, desquamated epithelial cells, leukocytes, and food remnants. Under healthy conditions, saliva does not have an odour. When its pH is increased, however, it turns into an increasingly putrefied odour. Normal saliva is rich in proteins and urea. In contrast, saliva is very poor in free

glucose and carbohydrates since most of them are associated with glycoproteins. These protein components are increased by the different cellular and non-cellular elements coming from the mucosa and gingival crevicular fluid.

This high protein component allows, under ideal pH and Eh conditions, the formation of VSCs and the production of halitosis. The incubation of this aqueous mixture will produce volatile compounds and malodour.



Different authors have demonstrated the conditions leading to the putrefaction of saliva and the production of malodour are enhanced in patients with periodontal disease. This fact has been attributed to a higher number of desquamated epithelial cells, a higher number of gram-negative anaerobic bacteria, and a higher protein substrate from gingival bleeding and gingival crevicular fluid.

Another important factor is salivary flow. Independent from the oral health status of a particular subject, halitosis is more apparent in the morning after a period of sleep ("morning breath"). During sleep, the salivary flow from major salivary glands is minimal, favouring stagnation and the initiation of putrefaction processes.

### The Role of the Dorsum of the Tongue

Recent studies implicate the dorsum of the tongue as the primary source of VSC production both in periodontally healthy and diseased populations.<sup>29</sup> These studies demonstrate (1) that removal of the tongue coating reduces VSC production<sup>30</sup> and (2) when comparisons are performed in samples of mouth air following tongue scraping, tooth brushing, and rinsing with water in subjects with malodour, the longer lasting reductions in VSC levels are followed after tongue scraping.<sup>31</sup>

In 1997, Waller carried out a study in 4 healthy subjects with no previous history of halitosis to

locate the region in the mouth in which VSCs were produced by placing 2mL of cysteine solution in the sublingual area, in the buccal sulcus, and on the dorsum of the tongue. Also, 0.5 ml of freshly collected whole saliva was added to 2mL of cysteine (pH 7.2) and shaken for 10 minutes at 37°C in a closed tube. The results indicated the dorsum of the tongue gave the highest VSC values in all subjects (~1600 ppb). Similarly, the buccal sulcus and the sublingual area gave considerable amounts of VSCs (~900 ppb), whereas saliva showed little production.<sup>32</sup>



Dorsum of the Tongue

In 1995, Miyazaki carried out a study involving 2,672 individuals in the general population without self-awareness of oral halitosis. They evaluated oral malodour using a portable sulphide monitor (Halimeter®) and examined their dental, periodontal health, and tongue coating status. A positive correlation was found between (1) VSC production and tongue coating in all age groups and (2) between VSCs and the periodontal index of periodontal treatment needs (CPITN) in 45 to 54 and 55 to 64 year-old groups. However, they only could find a weak correlation between VSCs and plaque index, tooth brushing, smoking habits, self-awareness of oral malodour, or the number of decayed teeth in any age group.<sup>6</sup>

Similarly, in 1992 Yaegaki and Sanada studied the source of VSC production in periodontally diseased patients compared with healthy controls. They found that the amount of tongue coating measured as wet weight was much higher in the group with periodontal disease (probing depth  $\geq 4$ mm) than in controls. Furthermore, the VSC production from the tongue coating in periodontally diseased patients was more than 4 times higher than in controls.<sup>22</sup>

Tongue coating comprises desquamated epithelial cells, blood cells, and bacteria. In fact, more than 100 bacteria may be attached to a single epithelial cell on the tongue dorsum, whereas, only about 25 bacteria are attached to each cell in other areas of the oral cavity.<sup>16</sup> The morphology of the dorsal surface of the tongue is very irregular with the presence of multiple fissures and mucosal papillae. These fissures and crypts may

create an environment where microorganisms are well-protected from the flushing action of the saliva and where oxygen levels are low enhancing the growth of anaerobic bacteria.

Studies on the bacterial microflora of the tongue are scarce, however, all these studies have identified several malodourous bacteria (*Bacteroides*, *Fusobacteria* spp., *Peptococcus*, and *Peptostreptococcus*) among the prominent cultivable microbiota.<sup>8,33</sup> When comparisons have been made between subjects suffering halitosis with healthy controls, the malodour subjects showed higher total bacterial counts and proportionally higher numbers of gram-negative anaerobes, (ten-fold increase in the numbers of

*Fusobacteria* spp.) than subjects without malodour.<sup>34</sup>

All these factors make tongue coating the ideal microenvironment to produce malodourous compounds.

### Association Between Halitosis and Periodontal Disease

Different lines of evidence have demonstrated this association between halitosis and periodontal disease:

- **State of gingival health:** In experimental gingivitis, the amount of VSCs expelled in mouth air was significantly higher in subjects with gingivitis compared to control subjects. Moreover, salivary VSC production increased with gingival inflammation and conversely decreased with the return to gingival health.<sup>6,35</sup>

- **Severity of periodontitis:** In periodontitis, different studies have shown a correlation between VSC concentration in mouth air and increased pocket depth.<sup>6,13,29</sup> However, some studies have failed to demonstrate positive correlations between periodontitis severity and halitosis. Using a group of 16 patients seeking treatment for oral halitosis, De Boever found that tongue odour was negatively correlated with probing depths suggesting an inverse relationship between malodour and periodontal parameters.<sup>36</sup> Similarly, Bosy et al did not find a relationship between periodontal disease and the prevalence or severity of halitosis.<sup>37</sup> In addition to the lack of

correlation between halitosis and the presence/absence of periodontal disease, the intensity of halitosis based on VSC concentration was 19% less in periodontally healthy subjects (mean 111 ppb) than in subjects with periodontitis (mean=136 ppb). Meta analysis indicated that oral malodour was not associated with periodontitis (odds ratio was 1.2.)<sup>37</sup>

• **Source of VSC production in periodontitis**

**patients:** In 1998, Yaegaki et al carried out a study aimed to investigate the VSC production of tongue coating in relation to the severity of periodontal disease. They analysed the VSCs in mouth air using gas chromatography before and after removing the tongue coating. They concluded the tongue coating might be a main site of oral malodour production in slight or moderate periodontal disease, whereas, the periodontal pocket would be the main origin of VSCs only in severe periodontal disease.<sup>38</sup>

• **Correlation between the presence of a pathogenic microflora in the subgingival microbiota and halitosis:**

In 1981, Pitts et al studied the correlations between odour scores and microbiological findings in crevicular samples of periodontally healthy subjects. They found that odour scores were significantly correlated with the concentration of overall bacterial populations and that higher levels of crevicular bacteria were associated with greater odour scores.<sup>39</sup> In 1994, Bosy et al examined the association of trypsin-like activity detected by the BANA (Benzoyl-DL-arginine-2-Naphthylamide) test in 4 subgingival samples of 127 patients with floss odour.<sup>37</sup> They found a moderately strong correlation between the BANA scores of the four tooth sites with floss odour. They also found when periodontitis was present, 87.5% of tooth sites were BANA positive as compared with 74.4% of tooth sites positive in healthy individuals.<sup>37</sup>

• **Role of VSCs in the pathogenesis of destructive periodontitis:**

VSCs, particularly hydrogen sulphide and methyl mercaptan, are a family of gases which are primarily responsible for halitosis. They have been identified as the main contributors to oral malodour, and they have also been found in increased levels in pockets with bleeding on probing. These products are highly toxic to tissues even at extremely low concentrations and, therefore, may play a role in the pathogenesis of inflammatory conditions affecting the periodontium, such as periodontitis. Different in vitro studies have demonstrated that VSCs alter

the permeability of oral and junctional epithelium.<sup>21</sup> They are toxic to fibroblasts, altering their morphology and function.<sup>7,40,41,42</sup> They also alter the metabolism of fibronectin<sup>43</sup> and interfere in the enzymatic and immunological reactions leading to tissue destruction while showing an increase in the release of interleukin-1 (IL-1) and PGE<sub>2</sub>(Prostaglandin E2)<sup>7</sup>

**Diagnosis of Halitosis**

There are three main methods of quantifying oral malodour: organoleptic measurement, gas chromatography (GC), and sulphide monitoring.

Table 2: Organoleptic Scores

Organoleptic Scale (0-5).	
0= no appreciable odor	3= moderate odor
1= barely noticeable odor	4= strong odor
2= slight but noticeable odor	5= extremely foul odor
Taken from <sup>10</sup> Rosenberg M, Kulkarni GV, Bosy A, et. al. Reproducibility and sensitivity of oral malodor measurements with a portable sulphide monitor. J Dent Res. 1991 Nov;70(11):1436-40.	

Taken from <sup>10</sup> Rosenberg M, Kulkarni GV, Bosy A, et. al. Reproducibility and sensitivity of oral malodor measurements with a portable sulphide monitor. J Dent Res. 1991 Nov;70(11):1436-40.

• **Organoleptic measurement.** A subjective test scored on the basis of the examiner's perception of a subject's oral malodour. Different semi-quantitative scales have been used, however, at the most recent International Workshop on Oral Halitosis (1999), there was consensus on using a scale ranging from 0 to 5. (Table 2) Before the organoleptic assessment, both patients and examiner must follow some instructions in order to obtain a more reliable result. Patients are instructed to abstain from eating strong foods at least 48 hours before the assessment and to avoid using scented cosmetics for 24 hours before the assessment. Patients must abstain from ingesting any food or drink, omit their usual oral hygiene practices, abstain from using oral rinse and breath fresheners, and abstain from smoking for 12 hours before the assessment. The oral malodour examiner is required to refrain from drinking coffee, tea, or juice and to refrain from smoking and using scented cosmetics before the assessment.<sup>16</sup>

• **Gas chromatography (GC).** GC is considered the gold standard for measuring oral malodour



since it is specific for VSCs, the main cause of oral malodour. The GC equipment is expensive, bulky, and the procedure requires a skillful operator. Therefore, this technology has been confined to research and not to clinical use.

• **Sulphide monitoring.** Sulphide monitors analyse for total sulphur content of the subject's mouth air. Although compact sulphide monitors are inexpensive, portable, and easy to use, most of them are not able to distinguish among the VSCs. For example, the Halimeter® (Interscan Co., Chatsworth, CA) (Figure 2) has high sensitiv-



**Figure 2. Portable Sulphide Monitor: Halimeter® (Interscan Co., Chatsworth, CA)**

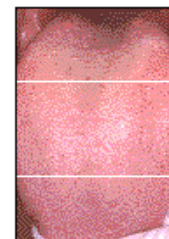
ity for hydrogen sulphide but low sensitivity for methyl mercaptan which is a significant contributor to halitosis caused by periodontal disease.<sup>16</sup>

The most reliable and practical procedure for evaluating a patient's level of oral malodour is still a thorough organoleptic assessment by a trained clinician. Nevertheless, the use of a portable sulphide monitor is of interest, since we can quantify the changes and the patients are able to monitor their evolution through therapy. This is an important factor, especially in those patients with pseudohalitosis or halitophobia.

Apart from the mentioned methods to assess the level of oral malodour, there are other clinical variables that we must evaluate as these data can be useful to design the individual treatment needs and to objectively evaluate the changes in the follow up visits. Among these clinical variables are the patient's periodontal status including oral hygiene levels and the status of tongue coating. Since different indexes and methods have been reported in the literature to evaluate tongue coating, it is recommended that one index be used that allows us to quantify changes in the amount of coating. (Tables 3, 4, and 5)

Table 3. Tongue Coating Index

0- no tongue coating
1- thin coating over 1/3 of the tongue dorsum.
2- thin coating over 2/3 or thick over 1/3.
3- thick coating over 2/3.



Taken from <sup>38</sup> Yaegaki K, Coil JM. Origin of oral malodour in periodontal disease. J Dent Res. 1998 77;1998.

Table 4. Tongue Coating Index: (Winkel E.G. 1998 (personal communication))

W.T.C. INDEX = A+B+C+D+E+F
0 = no coating
1 = light coating
2 = heavy coating



Table 5. Tongue Coating Wet Weight: (Yaegaki K. 1998)

Remove the entire tongue coating and measure its wet weight



### Therapeutic Approaches to the Treatment of Halitosis

Treatment needs (TN) for halitosis in the dental practice have been categorized into 5 classes in order to provide guidelines for clinicians in treating halitosis patients. (Table 6) These guidelines are directly related to a thorough diagnosis of the origin of halitosis.<sup>16</sup> Treatment of physiologic halitosis (TN-1), oral pathologic halitosis (TN-1 and TN-2), and pseudo-halitosis (TN-1 and TN-4) should be the responsibility of a dentist, however, treatment of extra-oral pathologic halitosis (TN-3) or halitophobia (TN-5) should be undertaken by a physician or medical specialist such as a psychiatrist or psychologist.

Table 6. Treatment Needs (TN) for Halitosis

Category	Description
TN-1	Explanation of halitosis and instructions for oral hygiene (support and reinforcement).
TN-2	Oral prophylaxis, professional cleaning, and treatment for oral diseases especially periodontal diseases.
TN-3	Referral to a physician or medical specialist.
TN-4	Explanation of examination data, further professional instructions, education, and reassurance.
TN-5	Referral to a clinical psychologist, psychiatrist, or other psychological specialist

In physiologic halitosis (TN-1), management should be focused on patient self-care. It is important to make the patient aware of his/her halitosis, instruct him/her on the appropriate cleaning of the dorsum of the tongue (Figure 3),

In oral pathologic halitosis (TN-2), patients should carry out the same regime as in TN-1, but the dentist should take care of the underlying oral pathology, especially the treatment of periodontal diseases or any dental therapy needed to treat caries or faulty restorations.

In TN-3, patients exhibit oral malodour but no oral origin can be demonstrated. These patients should be referred to an appropriate medical specialist.

In TN-4, patients need to be counselled by educating them that their problem is psychological through an explanation of their results of diagnostic assessment. For this purpose, the portable sulphide monitors are very useful. Some patients are convinced of not having halitosis after they can see the lack of objective signs of malodour for themselves (pseudo-halitosis), whereas, others remain completely obsessed about their perceived problem in spite of any counselling (halitophobia). In these (TN-5) situations, patients would need assistance from a psychological specialist. Furthermore, patients with genuine halitosis who undergo successful reduction of halitosis by TN-2 or TN-3 and still believe they have the condition should also be referred to a psychological specialist.

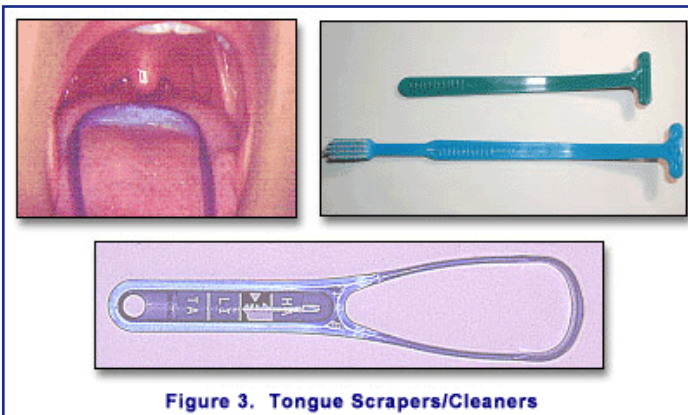


Figure 3. Tongue Scrapers/Cleaners

as well as on the use of adequate interdental oral hygiene measures. In most of the patients, self-performed oral hygiene should be reinforced with an adequate chemical plaque control approach consisting of the use of mouthrinses or dentifrices with proven antibacterial efficacy.

## References

- 1 Delanghe G, Bollen C, van Steenberghe D, et. al. foetor ex ore. *Ned. Tijdsch.Tandheelkd.* 1998;105:314-7.
- 2 Delanghe G, Ghyselen J, Bollen C, et. al. An inventory of patients' response to treatment at a multidisciplinary breath odor clinic. *Quintessence Int.* 1999 May;30(5):307-10.
- 3 Rosenberg M. First international workshop on oral malodor. *J Dent Res.* 1994 Mar;73(3):586-9. No abstract available.
- 4 Meskin LH. A breath of fresh air. *J Am Dent Assoc.* 1996 Sep;127(9):1282, 1284, 1286 passim. No abstract available.
- 5 Newman MG. The role of periodontitis in oral malodour: clinical perspectives. In van Steenberghe D, Rosenberg M, eds. *Bad Breath: A multidisciplinary approach.*, pp 3-14. Leuven: Leuven University Press, 1996.
- 6 Miyazaki H, Sakao S, Katoh Y, et. al. Correlation between volatile sulphur compounds and certain oral health measurements in the general population. *J Periodontol.* 1995 Aug;66(8):679-84.
- 7 van Steenberghe D, Rosenberg M. *Bad Breath: A multidisciplinary approach.* Leuven: Leuven University Press, 1996.
- 8 Rosenberg M. *Bad breath: research perspectives.* Ramat Aviv: Ramot Publishing–Tel Aviv University, 1997.
- 9 Iwakura M, Yasuno Y, Shimura M, et. al. Clinical characteristics of halitosis: differences in two patient groups with primary and secondary complaints of halitosis. *J Dent Res.* 1994 Sep;73(9):1568-74.
- 10 Rosenberg M, Kulkarni GV, Bosy A, et. al. Reproducibility and sensitivity of oral malodor measurements with a portable sulphide monitor. *J Dent Res.* 1991 Nov;70(11):1436-40.
- 11 Rosenberg M. Clinical assessment of bad breath: current concepts. *J Am Dent Assoc.* 1996 Apr;127(4):475-82. Review.
- 12 Johnson PW. The olfactory reference syndrome and halitosis. In van Steenberghe D, Rosenberg M, eds. *Bad Breath: A multidisciplinary approach.*, pp 231-40. Leuven: Leuven University Press, 1996.
- 13 Rosenberg M, Kozlovsky A, Gelernter I, et. al. Self-estimation of oral malodor. *J Dent Res.* 1995 Sep;74(9):1577-82.
- 14 Rosenberg M, Kozlovsky A, Wind Y, et. al. Self-assessment of oral malodor 1 year following initial consultation. *Quintessence Int.* 1999 May;30(5):324-7.
- 15 Rosenberg M, Eli I. Experiences of an israeli malodour clinic. In Rosenberg M, ed. *Bad breath: research perspectives.*, pp 137-48. Ramat Aviv: Ramot Publishing–Tel Aviv University, 1997.
- 16 Yaegaki K, Coil JM. Examination, classification, and treatment of halitosis; clinical perspectives. *J Can Dent Assoc.* 2000 May;66(5):257-61. Review.
- 17 Scully C, el-Maaytah M, Porter SR, et. al. Breath odor: etiopathogenesis, assessment and management. *Eur J Oral Sci.* 1997 Aug;105(4):287-93. Review.
- 18 Ship J. Gustatory and olfactory considerations: examination and treatment in general practice. *J Am Dent Assoc.* 1993 Jun;124(6):55-62. Review.
- 19 Amano A, Akiyama S, Ikeda M, et. al. Oral manifestations of hereditary sensory and autonomic neuropathy type IV. Congenital insensitivity to pain with anhidrosis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1998 Oct;86(4):425-31.
- 20 Goldberg S, Kozlovsky A, Gordon D, et. al. Cadaverine as a putative component of oral malodor. *J Dent Res.* 1994 Jun;73(6):1168-72.
- 21 Reingewirtz Y. Halitose et parodontite; revue de littérature. *Journal de parodontologie & d'implantologie orale* 1999;18:27-35.
- 22 Yaegaki K, Sanada K. Volatile sulfur compounds in mouth air from clinically healthy subjects and patients with periodontal disease. *J Periodontal Res.* 1992 Jul;27(4 Pt 1):233-8.
- 23 Loesche WJ. The effects of antimicrobial mouthrinses on oral malodor and their status relative to US Food and Drug Administration regulations. *Quintessence Int.* 1999 May;30(5):311-8.
- 24 Ratcliff PA, Johnson PW. The relationship between oral malodor, gingivitis, and periodontitis. A review. *J Periodontol.* 1999 May;70(5):485-9. Review.
- 25 Kleinberg I, Westbay G. Salivary and metabolic factors involved in oral malodor formation. *J Periodontol.* 1992 Sep;63(9):768-75. Review.



- 26 Klokkevold PR. Oral malodor: a periodontal perspective. J Calif Dent Assoc. 1997 Feb;25(2):153-9. Review.
- 27 McNamara TF, Alexander JF, Lee M. The role of microorganisms in the production of oral malodor. Oral Surg Oral Med Oral Pathol. 1972 Jul;34(1):41-8. No abstract available.
- 28 Kleinberg I, Codipilly M. The biological basis of oral malodour formation. In Rosenberg M, ed. Bad breath: research perspectives., pp 13-39. Ramat Aviv: Ramot Publishing–Tel Aviv University, 1997.
- 29 Yaegaki K, Sanada K. Biochemical and clinical factors influencing oral malodor in periodontal patients. J Periodontol. 1992 Sep;63(9):783-9. Review.
- 30 Tonzetich J, Ng SK. Reduction of malodor by oral cleansing procedures. Oral Surg Oral Med Oral Pathol. 1976 Aug;42(2):172-81.
- 31 Kaizu T, Tsunoda M, Aoki H, et. al. Analysis of volatile sulphur compounds in mouth air by gas chromatography. Bull Tokyo Dent Coll. 1978 Feb;19(1):43-52. No abstract available.
- 32 Waler SM. On the transformation of sulfur-containing amino acids and peptides to volatile sulfur compounds (VSC) in the human mouth. Eur J Oral Sci. 1997 Oct;105(5 Pt 2):534-7.
- 33 Goldberg S, Cardash H, Browning H, et. al. Isolation of Enterobacteriaceae from the mouth and potential association with malodor. J Dent Res. 1997 Nov;76(11):1770-5.
- 34 Hartley G, El-Maaytah, M, Greenman J. Tongue microflora of subjects with low and high malodour levels. J Dent Res. 1995 74:587. Abstract.
- 35 Ko, YH, Kim YJ, Chung HJ. Methyl Mercaptan Concentration during Experimental Gingivitis in Man. J Dent Res. 1996 75:195. Abstract.
- 36 De Boever EH, Loesche WJ. Assessing the contribution of anaerobic microflora of the tongue to oral malodor. J Am Dent Assoc. 1995 Oct;126(10):1384-93.
- 37 Bosy A, Kulkarni GV, Rosenberg M, et. al. Relationship of oral malodor to periodontitis: evidence of independence in discrete subpopulations. J Periodontol. 1994 Jan;65(1):37-46.
- 38 Yaegaki K, Coil JM. Origin of oral malodour in periodontal disease. J Dent Res. 1998 77;1998.
- 39 Pitts G, Pianotti R, Feary TW, et. al. The in vivo effects of an antiseptic mouthwash on odor-producing microorganisms. J Dent Res. 1981 Nov;60(11):1891-6. No abstract available.
- 40 Johnson PW, Ng W, Tonzetich J. Modulation of human gingival fibroblast cell metabolism by methyl mercaptan. J Periodontal Res. 1992 Sep;27(5):476-83.
- 41 Johnson PW, Yaegaki K, Tonzetich J. Methyl mercaptan modulates collagen processing. J Dent Res. 1996 75;324. Abstract.
- 42 Lancero H, Johnson PW. Methyl mercaptan modulates the expression of alfa5beta1 in periodontal cells. J Den Res. 1996 75;324. Abstract.
- 43 Johnson PW, Lancero H. Function of gingival fibroblasts and periodontal ligament cells in the presence of methyl mercaptan. Quintessence Int. 1999 May;30(5):343-9.

## About the Authors

### Mariano Sanz, MD, DDS



Dr. Sanz holds academic degrees in Medicine and Surgery and received specialist training in Dentistry at the University Complutense of Madrid, Spain and in Periodontics at the University of California Los Angeles. He is a Professor in Periodontics and serves as the Vice-Dean, at the Faculty of Odontology at the University Complutense of Madrid. Dr. Sanz is the Secretary of the European Federation of Periodontology, and President of the Association for Dental Education in Europe. He is the author of more than 100 publications.

e-mail: [marianosanz@odon.ucm.es](mailto:marianosanz@odon.ucm.es)



### Silvia Roldán, DDS, MS



Dr. Roldán, received her dental degree and Masters degrees in Periodontology and Implantology, Oral Medicine from the University Complutense of Madrid, as well as in Oral Pathology from the Hospital Gregorio Marañón, Madrid Spain. Dr. Roldan is a guest professor in the graduate program in Periodontics and Implantology University Complutense of Madrid and is currently involved in research in the fields of periodontics and oral halitosis.



### David Herrera, DDS, MS



Dr. Herrera, received his dental degree Dentistry and his Masters Degree in Periodontology from the University Complutense of Madrid, Spain. He is also a European Doctor of Odontology, an Associate Professor in Periodontics and is involved in research in the fields of Periodontics and Microbiology at the University Complutense of Madrid.

