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Aetiology, diagnosis and management of halitosis: a review

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Halitosis is so common among any adult population group, that some believe this distressing breath condition to be within a range of normal breath odours. However, as halitosis often interferes with social relationships it has rapidly brought awareness among the general population. Assuming that the cause of halitosis is in the oral cavity, many rightly seek therapy from dentists. It therefore becomes necessary for dentists to have an understanding of the oral as well as the systemic factors that cause halitosis. This article aims to address some misconceptions, and reviews the current information on the aetiology, diagnosis and management of various types of halitosis, with an emphasis on halitosis originating in the oral cavity.

■ Introduction

Halitosis is the term used to describe any disagreeable odour in the exhaled air of an individual, regardless of whether the odour originates in the oral cavity or elsewhere from within the body. References to halitosis are present from the time of ancient Greek, Roman, Hebrew, Christian and Islamic cultures. However, not until Howe[1] described halitosis in 1874 did it become a clinical entity. Other terms such as oral malodour, bad or foul breath, and fetor oris are often used to describe the same condition. In this article the two terms halitosis and oral malodour will be used interchangeably to mean the same condition.

Halitosis is not a disease but rather a symptom of underlying oral, systemic or psychological conditions. The primary cause of halitosis is due to the release of odoriferous volatile sulphur compounds (VSC) in the exhaled air[2]. VSC are released following putrefactive activity of anaerobic bacteria present in the oral or nasal cavity. The VSC may also be absorbed by the blood stream from a remote part of the body, such as from a cirrhotic liver, and transferred to the pulmonary alveoli to be exhaled through the nostrils or

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mouth as malodorous breath. If VSC are present in an objectionable concentration in the breath, it is perceived as halitosis.

Halitosis affects a large part of any population, especially the adult population[3], and surveys conducted in various countries indicate that people are concerned about oral malodour[3-5]. Those in close contact with halitosis sufferers, including relatives, friends and colleagues, may at first be reluctant to point out the problem, but in due course when they begin to take avoiding action it often leads to social embarrassment. Although the prevalence and incidence ratios between males and females have been reported to be the same, women seem to seek treatment more often than men[6]. There are also some individuals who have halitosis but are totally unaware of it. This may be as a result of the affected person becoming subjected to a sensory phenomenon known as adaptation, in which the specialised olfactory neurons become constantly occupied with the otherwise offensive odour, making the person insensitive to the odour[7]. There is also another widely held view that there is a divergence of the pathways of the inhaled and exhaled air, thereby reducing the chance for the person to detect his/her malodour from the exhaled air.

In recent years the aetiology of halitosis has become increasingly clear, and it is now known that halitosis originates more from within the oral cavity than elsewhere in the body[8]. Therefore, more than any other health professional, dentists ought to be well informed on halitosis in order to provide effective treatment and proper advice to the significant proportion of the general population affected by this condition. This review summarises current knowledge with an aim to address misconceptions and aid in understanding the aetiology, diagnosis and management of halitosis.

■ Aetiology of halitosis

Three factors are involved in the aetiology of halitosis, namely the bacteria that produce the malodourous compounds, the substrates that the bacteria utilise to release the odour compounds, and the malodourous compounds themselves. In the absence of any one of these factors halitosis is unlikely to occur.

■ Bacteria

The oral cavity is an ideal location for many microorganisms to flourish. It has various sheltered areas such as interdental spaces, dental caries, gingival sulcus and deeper layers of papillae on the dorsum of the tongue, which function as bacterial reservoirs. Microorganisms thrive not only in these sheltered areas but also on all the surfaces in the oral cavity, including the non-shedding surfaces such as on teeth and restorations. They are also present in saliva and in the gingival crevicular fluid (GCF). However, the primary site where halitosis-causing bacteria accumulate is on the dorso-posterior surface of the tongue. The tongue's papillary nature creates a unique ecological site that provides an extremely large and protected surface area, favouring the accumulation of oral bacteria. With nutrients always available, it is not surprising that more than 500 different species of bacteria have been identified within the oral cavity, with at least 150 different species being present at any given time[9]. However, only a few from the vast array of the oral microbiota are involved in causing halitosis. For example, it was demonstrated in an *in vitro* experiment that malodour compound formation from incubated saliva correlated with a shift in the microflora from a predominantly gram-positive to a predominantly gram-negative anaerobic flora[10]. In another study that investigated the role of the subgingival microflora in halitosis, significant differences were observed between the subgingival microflora of individuals with and without halitosis; individuals with halitosis had a higher percentage of spirochetes and motile rods[11]. Most of the odoriferous compounds that cause bad breath are waste products produced by anaerobic bacteria as they digest proteins. VSC-producing oral microbes have now been identified; while *Porphyromonas gingivalis*, and *Prevotella intermedia* produce methyl mercaptan and hydrogen sulphide, *Fusobacterium nucleatum*, *Treponema denticola* and *Veillonella alcalescens* produce hydrogen peroxide[12,13], the two primary odour compounds associated with halitosis of intraoral origin.

■ The substrates

An organic substance that is acted upon in a biochemical reaction is called a substrate. The consumed food is acted upon in a similar manner by various di-

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Table 1 Breath malodour compounds and their characteristics (adapted from Verschueren[18]).

Name	Formula	Odour characteristics	100% Odour recognition concentration
Allyl mercaptan	$CH_2=CHCH_2SH$	garlic-like	0.05 ppb*
Propyl mercaptan	$CH_3CH_2CH_2SH$	pungent, unpleasant	0.70 ppb
Dimethyl disulphide	CH_3SSCH_3	pungent	7.00 ppb
Methyl mercaptan	CH_3SH	pungent, rotten cabbage	35.00 ppb
Dimethyl sulphide	CH_3SCH_3	unpleasantly sweet	100.00 ppb
Carbon disulphide	CS_3	slightly pungent	900.00 ppb
Hydrogen sulphide	H_2S	rotten eggs	1000.00 ppb
Trimethylamine	$(CH_3)_3N$	fishy, ammoniacal	4000.00 ppb
Dimethylamine	$(CH_3)_2NH$	fishy, ammoniacal	6000.00 ppb
Ammonia	NH_3	pleasantly sweet	55,000.00 ppb

** ppb, parts per billion*

gestive enzymes and in a way becomes the substrate. Bacteria resident in the oral cavity utilise part of the food that is stagnant in the mouth as their substrate. However, only water-soluble nutrients enter the bacteria through pores on the cell wall, and are then digested within the cell. In contrast, complex molecules, such as proteins or complex carbohydrates, are broken down into simpler molecules outside the bacteria by enzymes before they are transported across the cell membrane. Following the proteolytic activity of bacteria on the sulphur-containing amino acid substrates such as cystine, cysteine and methionine, the end products released are the odoriferous VSC that are associated with halitosis[2,13]. However, if a mainly carbohydrate nutrient is present, bacterial putrefaction lowers the pH to an acid medium and VSC formation is inhibited. The substrates for this putrefaction process come from stagnant food, exfoliated epithelial cells, effused leukocytes, stagnant saliva, gingival crevicular fluid and inflammatory exudates[14-16].

Malodour compounds

The human breath has been found to contain more than 200 volatile compounds[17], including VSC, gases not containing sulphur such as amines (cadaverine), volatile aromatic compounds (indole, skatole) and short chain carboxylic acids (SCCA), and organic acids (acetic, proprionic)[18,19]. However, contrary to the traditional belief that ammonia and amines were the main source of halitosis, it was Tonzetich and Richter[20] who first reported that VSC are the main components of halitosis. Of the total VSC found in the mouth air, 90% is made up of hydrogen sulphide, methyl mercaptan and to a lesser extent dimethyl sulphide[2,13]. However, the VSC that cause the halitosis of oral origin differ from VSC found in blood-borne halitosis of extraoral origin[21]. Compounds such as methyl mercaptan and hydrogen sulphide that are associated with oral malodour are not found in blood-borne halitosis.

These compounds when blood-borne are oxidised and irreversibly bind to blood components, and are prevented from being released into the lung air. On the other hand, compounds such as dimethyl sulphide, which is a neutral compound that is also stable in blood, can be blood-borne and released into the lung air[21]. Thus in patients with a cirrhotic liver the odour named fetor hepaticus is caused by dimethyl sulphide[22]. The breath of a person with halitosis of extraoral origin may rarely contain odoriferous amines. Table 1 lists some of the common odour-producing compounds along with their odour recognition threshold concentration[18]. The malodour gases with the lowest recognition threshold concentration such as allyl mercaptan are the most odorous, whereas odour compounds such as ammonia have the least odour.

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Table 2 Classification of halitosis with its corresponding treatment needs (TN).

Classification	Description	TN#
I. Genuine halitosis	Presence of a malodour that is beyond socially acceptable level.	
A. Physiological halitosis	Neither a specific disease nor a pathological condition that could cause halitosis is found, e.g. malodour from putrefactive processes within the oral cavity, temporary halitosis due to dietary factors.	TN 1
B. Pathological halitosis		
(i) Oral	Halitosis caused by disease, pathological condition or malfunction of oral tissues. Halitosis derived from tongue coating, modified by pathological condition, e.g. periodontal disease, xerostomia.	TN 1 & 2
(ii) Extraoral	Malodour originates from nasal, paranasal sinuses, laryngeal regions, respiratory or upper digestive tract. Malodour originates from anywhere in the body whereby the odour is blood borne and emitted through the lungs, e.g. diabetes mellitus, hepatic cirrhosis, uraemia and internal bleeding.	TN 1 & 3
II. Pseudo-halitosis	Obvious malodour is not perceived by others, although the patient complains of its existence. Condition is improved by counselling and simple oral hygiene measures.	TN 1 & 4
III. Halitophobia	No evidence of any halitosis being present. After treatment for genuine halitosis or pseudo-halitosis, the patient fears that he/she has halitosis	TN 1 & 5

■ Types of halitosis

During the last quarter of a century, several classifications of halitosis have been proposed[23-25]. One of the recent and frequently used classifications broadly categorises this condition into three major types: exogenous, endogenous and psychogenic halitosis[25]. In this article an earlier and unique classification is used that not only differentiates the various forms of halitosis (Table 2)[24,26], but also enables clinicians to order the process of diagnosis as well as establish the corresponding treatment needs (Table 6)[24,26].

This classification broadly classifies halitosis into genuine halitosis, pseudo-halitosis and halitophobia. Genuine halitosis is further sub-classified into physiological halitosis or pathological halitosis, and based on the origin of pathological halitosis it is further differentiated into oral and extraoral halitosis. As the name signifies, oral halitosis originates in the oral cavity and extraoral from other parts of the body, such as the nasal cavity and paranasal sinuses, respiratory tract, digestive system and the kidneys.

■ Genuine halitosis

Genuine halitosis is where clearly a disagreeable odour is present in the breath of the person. Genuine halitosis can be physiological or pathological. Depending upon the origin, physiological and pathological halitosis could be of oral or extraoral origin.

■ Physiological halitosis of oral and extraoral origin

Most adults, when waking up in the morning may notice a transient dry mouth accompanied by an unacceptable odour, which is referred to as 'morning breath'. Although this form of halitosis is transient and does not need any special consideration, dentists still need to know its cause. The superficial layer of oral mucosa consists of ready-to-be-shed squames, which are covered with a large number of anaerobic bacteria[14]. Covering the microorganisms is a continually replaced layer of residual saliva that maintains the surface of the oral mucosa in a moist state. While the residual saliva layer provides the easily degradable peptide and protein substrates to the bacteria

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and promotes the process of putrefaction, the outer layer of the residual saliva also restricts the release of odoriferous volatiles into the mouth air. During sleep, due to a reduced flow of saliva, the resulting thinner residual saliva layer makes it easier for the odorous volatiles generated in the mucosal layer to be released into the breath[27,28]. Morning breath disappears soon after intake of food or fluid. This may be explained by the increased saliva flow and the resulting thickening of the residual saliva layer over the oral mucosa, restricting the release of foul smelling VSC. In addition, the movement of the tongue and cheek during mastication and the increased salivary flow removes food debris, desquamated epithelial cells and free-floating bacteria. Although morning breath is transient, in about 10% to 30% of individuals it remains more persistent, lasting long after the transitory xerostomia has disappeared[3]. The origin of this chronic form of physiological halitosis is mainly from the tongue coating present on the dorsoposterior surface of the tongue[29]. The tongue coating consists of desquamated epithelial cells, blood cells and VSC-producing anaerobic bacteria. Even in healthy subjects with no oral lesions, the tongue coating is believed to be the primary source of oral malodour[30,31].

Physiological halitosis may also be caused by tobacco smoking, and by certain foods and medications. Tobacco smoking not only causes a characteristic breath odour, but also may encourage hairy tongue formation, which traps food debris and tobacco odour. Salivary flow is also decreased and the ensuing xerostomia further increases halitosis. When odorous food such as garlic, onions, spices and certain flavouring agents are consumed, they are absorbed in the intestines, metabolised in the liver and the VSC is released into the bloodstream. These diffuse into the lung air and are exhaled as malodorous breath. Halitosis produced by garlic may be of oral or gut origin, and VSC from oral origin were found to be allyl mercaptan and methyl mercaptan, whereas VSC from the gut were found to be allyl methyl sulphide[32]. Alcohol is a potent drug that is absorbed in the gut, and odoriferous compounds are expelled through the lungs. This mechanism is used by the police to analyse the breath of suspected drunk drivers. A number of other drugs have been implicated in causing halitosis. Disulphiram, used to combat abusive use of alcohol, can cause halitosis by the release of carbon disulphide through the exhaled air[33]. Isosorbide dinitrate, a drug taken for angina, has been known to cause objectionable odour[34]. Any drug that reduces salivary flow will indirectly produce halitosis by reducing the self-cleaning ability of the oral cavity. Well known among these are the antidepressant drugs.

Pathological halitosis

Pathological halitosis of oral origin

Putrefaction of nutrients by bacteria resident in the mouth is a physiological process, and the ensuing halitosis is also considered to be physiological. However, when halitosis is enhanced by periodontal disease or other local pathological conditions it is considered to be pathological. Unlike physiological halitosis, which can be resolved by oral hygiene methods, pathological halitosis remains unresolved until the factors causing the condition have been brought under control. Apart from the naturally existing substrate within the oral cavity such as saliva and food debris, the pathological conditions in the oral cavity make available additional substrates such as necrotic tissue debris, blood cells, inflammatory exudates, and gingival crevicular fluid, thus making the halitosis pathological.

It is estimated that in about 90% of patients with pathological halitosis, the odour originates in the oral cavity, with the bacteria in the oral cavity being implicated in the release of VSC[2,8]. It has also been estimated that of the total quantity of VSC produced in the oral cavity of healthy or periodontally involved subjects, approximately 60% are produced on the surface of the tongue[30,35]. Pathological halitosis of oral origin has always been associated with poor oral hygiene, dental caries, periodontal diseases, in particular necrotising ulcerative gingivitis, necrotising periodontitis, periodontitis, pericoronitis, dry socket, other oral infections[15], tongue coating and oral carcinoma[36]. The pathogens strongly associated with the aetiology of periodontal disease are also associated in the production of VSC[12] and therefore it is not surprising that a good correlation between periodontitis and oral malodour has been shown[37,38]. Several aetiological factors are involved in causing periodon-

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tal disease; and how these local and systemic factors initiate, exaggerate and sustain periodontal disease will not be elaborated here. However, the relationship between halitosis, gingivitis and periodontitis will be discussed briefly.

One of the earliest events associated with the initiation of gingivitis occurs in the gingival sulcus; which along with the dorso-posterior surface of the tongue has been identified as the two main anatomical sources of VSC[39]. In gingivitis, the bacteria-resistant epithelial seal around the teeth is lost due to the increased permeability of the gingival sulcus. VSC produces a similar effect on the gingiva. An experiment demonstrated that when no inflammatory response could be initiated on healthy gingiva by topical application of bacterial antigen lipopolysaccharide (LPS), exposure of the same tissue to hydrogen sulphide prior to topical application of LPS resulted in gingival inflammation[40]. Hydrogen sulphide could have caused this by increasing the permeability of the surface epithelium and facilitating increased penetration of LPS into the gingival tissues. Unlike in the mouth air where hydrogen sulphide is present in greater concentration, the gingival sulcus has methyl mercaptan as the predominant compound[13,40]. Methyl mercaptan has been shown to penetrate into the deeper gingival tissues and induce deleterious effects[41]. As a consequence, bacterial products could penetrate deeper into the periodontium and, depending upon the host response, the disease process may progress. In addition methyl mercaptan induces secretion of interleukin-1 beta (IL-1β), and acts synergistically with LPS and IL-1β to increase the secretion of inflammatory mediators such as prostaglandin E2 and collagenase[42]. Methyl mercaptan has also been shown to be toxic in low concentrations to gingival fibroblasts[43]. These studies show that VSC are not just odour-causing compounds but that they might actually participate in initiating gingivitis. In periodontitis, which is always preceded by gingivitis, there is a continuation of all the events that caused gingivitis in addition to a new group of events that lead to periodontitis. The new events are mainly due to the inclusion of the periodontal ligament (PDL) and alveolar bone in the underlying periodontium. PDL cells exposed to methyl mercaptan have exhibited lowered pH, decreased cell migration, alterations in collagen metabolism and lowered protein synthesis; all detrimental to the maintenance or regeneration of mineralised tissues such as the alveolar bone[44]. These are only a few from several studies that have demonstrated that VSC, particularly methyl mercaptan, even in low concentrations are not only associated with causing halitosis, but may also be implicated in contributing to the pathogenesis of periodontal disease[40-42,44,45].

Pathological halitosis of extraoral origin

Only about 10% to 20% of halitosis is of extraoral origin[8,32,39,46] and it is a common belief that it is mainly from the stomach that odorous compounds are expelled through the oesophagus into the mouth. This is due to the fact that odours from the stomach can easily escape into the oral cavity during belching or vomiting. However, under normal conditions odour compounds cannot easily pass through the oesophagus. It is not an open tube and it normally remains collapsed and tightly closed, preventing the VSC produced in the gastrointestinal tract from being released into the mouth[36,47]. In addition there are the upper and lower oesophageal sphincters in the respective ends of the oesophagus, which also restrict the back-flow of stomach contents into the oral cavity. Nevertheless, there are a few case reports of halitosis being caused by gastrointestinal diseases such as pyloric stenosis and gastric *Helicobacter pylori* infection[48,49].

Extraoral halitosis can originate from organs in proximity to or remote from the oral cavity, and many of them have a characteristic odour (Table 3). Malodour may be from anaerobic infections, ulcerations or malignancy in the upper or lower respiratory tracts from where the VSC are directly expelled into the air that is exhaled. Chronic sinusitis and tonsillitis are frequent ENT sources of halitosis, but bronchitis, bronchiectasis, pneumonia, lung abscess and carcinoma of the lung can cause halitosis from the lower respiratory tract. Malodour may also originate from remote locations such as the stomach, intestines, liver or kidney that have been affected by systemic diseases. The odorous volatile compounds from these locations are absorbed by the bloodstream and transferred to the pulmonary alveoli to be expelled into the lung air and exhaled as foul-smelling breath[7,23,36,46].

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Diabetes is perhaps one of the best-known examples of a systemic pathological condition that causes halitosis. Although patients with well-controlled diabetes do not have any detectable odour, in the poorly controlled diabetic with diabetic acidosis or an impending hyperglycaemic coma there is a sweet, fruity acetone odour caused by an abnormal accumulation of ketones in the blood, which diffuses into the lung air[50]. In patients with liver cirrhosis, the extensive shunting of blood around the liver causes an elevated concentration of dimethyl sulphide in the blood, which diffuses into the breath and produces the malodour referred to as fetor hepaticus[22]. Kidney failure or uraemia may produce an odour of urine or ammonia.

Table 3 Systemic diseases and their associated halitosis (adapted from Lu[23]).

Systemic condition	Characteristic odour
Poorly controlled diabetes mellitus or hyperglycemic coma	Acetone, sweet fruity
Renal failure or uraemia	Urine or ammonia
Liver failure or hepatic coma	Fresh cadaver
Liver cirrhosis	Fetor hepaticus
Tuberculosis/lung abscess	Foul, putrefactive
Gastrointestinal disorder	Rotten egg
Internal haemorrhage	Decayed blood
Blood disorders	Decomposed blood
Fever, dehydration	Odour due to xerostomia and poor oral hygiene

■ Pseudo-halitosis

Apparently healthy individuals who have no perceivable halitosis, or any halitosis causing local or systemic factors, but persistently claim to have halitosis suffer from a condition called pseudo-halitosis. Although most of these individuals do not claim to smell their own offensive breath, they assume to have halitosis by misinterpreting the attitudes of people who come in contact with them[51]. They imagine that people deliberately avoid them, or turn their face away to escape from their bad breath.

■ Halitophobia

Halitophobia is a condition in which the patient has an exaggerated fear of having halitosis; those affected may or may not have a previous history of having genuine halitosis. Halitosis becomes an obsession that dominates their life, and some may develop depression secondary to their delusion of having bad breath[52].

This form of halitosis has also been referred to as delusional halitosis and is considered a variant of monosymptomatic hypochondriacal psychosis (MHP)[53]. The halitophobics are obsessive about oral health, and often use odour-masking techniques. In the severe form, halitophobics extract all their teeth, isolate themselves and may even commit suicide.

■ Halitosis assessment

An average person can detect unpleasant smell, and almost everyone has some experience of bad breath in others. Clinical research on halitosis requires more than just detection of odour; odour needs to be quantified. Unlike quantification of intensities of light, sound, smoke or heat, quantification of odour sensation is very difficult; and even more difficult is the controlled presentation of odour to stimulate its perception by the odour judges[54,55]. Another difficulty in odour research is that once a halitosis patient expels breath for estimation by one judge, the odoriferous compound released subsequently for other judges may differ in composition and intensity[56]. Despite these difficulties our knowledge of halitosis has increased owing to a better understanding of its aetiology and improved methods of collecting and analysing odoriferous compounds. However, our knowledge of halitosis is still poor at the very best.

In the cocktail of oral malodour compounds, VSC are the most prevalent and also the main components of halitosis[2,13]. Therefore, VSC concentration is used as an objective measurement of halitosis. There are three main methods of analysing oral malodour: organoleptic measurement, portable sulphide monitoring (both of which are done at patient's chair-side) and gas chromatography (GC), which is carried out in the laboratory.

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Table 4 Organoleptic scoring scale.

Odour Scale	Description
0: Absence of odour	Odour cannot be detected.
1: Questionable odour	Odour is detectable, although the examiner could not recognise it as malodour.
2: Slight odour	Odour exceeds the threshold of malodour recognition.
3: Moderate odour	Malodour is definitely detected.
4: Strong odour	Strong malodour is detected, but can be tolerated by examiner.
5: Severe odour	Overwhelming malodour is detected and cannot be tolerated by examiner.

Organoleptic measurement

Organoleptic measurement is considered to be the most reliable and practical procedure with which one can evaluate oral malodour. It is a test scored on the basis of examiner's perception of a subject's malodour; in short it is referred to as the 'sniffing method'. The examination is simple, does not need any special equipment, except the examiner's nose and a plastic tube or a straw that is inserted into the patient's mouth, and while the patient exhales the examiner judges the odour by sniffing at the other end of the tube or straw. The odour strength is evaluated and given a recognition score on the scale of 0–5 (Table 4).

If the patient's breath receives a score of 2 or more, the patient is diagnosed with genuine halitosis[24,26]. Odour from lung air that is present in the oral cavity may interfere with oral malodour assessment. If a lung condition is suspected to be a contributor to the oral malodour, a precise lung air examination must be performed after brushing the teeth, tongue cleaning and mouth rinsing with chlorhexidine or hydrogen peroxide. However, organoleptic measurement has its drawbacks. Although a good correlation between VSC concentration and organoleptic values has been found[57], it is still a subjective test, and when the examiners are repeatedly exposed to bad odours they become adapted to them and lose sensitivity. Dentists, especially periodontists, may not be ideal judges if they do not use masks on a regular basis[58]. There is also the potential risk of disease transmission to the examiner through the expelled air. This is particularly important with the existence of epidemic bird flu infections and other acute respiratory illnesses. Recently, an indirect organoleptic method of measuring VSC on incubated saliva has shown a strong correlation with direct clinical method[59]. This method may overcome some of the disadvantages of direct clinical organoleptic measurements, and may be used more often in the future.

Table 5 Halimeter monitoring.

Odour category	VSC concentration
Normal	< 100 ppb
Minor	100 – 180 ppb
Chronic	> 180 – 250 ppb

Portable sulphide monitoring

The Halimeter® (Interscan Co., Chatsworth, CA, USA) is a portable, easy to use instrument for measuring VSC concentration in mouth air. With the patient keeping the mouth wide open, the open end of a tube that is connected to the halimeter is placed over the dorsum of the tongue without touching it and the VSC concentration peak is recorded (Table 5). This instrument is particularly useful in monitoring VSC concentration changes during treatment. The device is highly sensitive to hydrogen sulphide, but has a low sensitivity to methyl mercaptan, a VSC that is the primary odour compound implicated in halitosis caused by periodontal disease[24]. It has other disadvantages, such as the need for the instrument to be re-calibrated often, and VSC measurements cannot be made in the presence of other odorous compounds such as ethanol or essential oils[56]. Therefore the VSC measurements may be affected if the subject is wearing perfume, hairspray, deodorant, etc. Although the halimeter is not accepted by some as an accurate instrument for determination of VSC in mouth air, it is a useful and handy instrument in odour research.

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Table 6 Treatment needs for breath malodour.

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

Gas chromatography

To overcome errors of human odour perception such as from organoleptic measurements, odour research laboratories measure the quantity of VSC released in breath air using GC. GC results are highly objective and reproducible, and considered a gold standard for measuring oral malodour. GC is often used in combination with a flame photometer detector[60], and sometimes with flame ionisation detector[61], or mass spectrometry[62]. Although GC is a reliable method for diagnosing halitosis, it consists of a sophisticated piece of equipment that requires an experienced operator. Therefore, it is not practical for most offices to be equipped with the GC apparatus.

Diagnosis of halitosis

A thorough dental examination, along with medical and halitosis history, is essential to determine the cause of the halitosis. The process of diagnosis begins with distinguishing genuine halitosis from pseudo-halitosis and halitophobia by analysing the obtained data. If the patient's breath receives an organoleptic recognition threshold score of 2 or more, or a halimeter reading of more than 100 ppb, the subject is diagnosed as having genuine halitosis. In subjects with genuine halitosis, halitosis of oral origin has to be distinguished from halitosis of extraoral origin by comparing mouth breath, nasal breath[7,39,46] and the lung air. This is not always easy, because malodour originating from the oral cavity sometimes may be perceived as malodour from the nasal breath, and this interference could also occur during lung-air examination. Therefore, nasal breath and lung air are examined after TN-1 and TN-2 (see above for an explanation of treatment needs [TN]). Nasal breath through both nostrils is examined separately, and if nasal breath odour is not detected and lung odour is present, then the odour is caused by systemic conditions.

Treatment of halitosis

Clinical experience and many investigations suggest that only a small number of cases diagnosed with halitosis cannot be treated in the dental clinic. Successful treatment depends on the correct diagnosis and carrying out a cause-related therapy. Treatment needs for halitosis (Table 6) are based on the diagnosis of the type of halitosis (Table 2), and are categorised into five kinds to help employ a precise, sequential treatment protocol. TN-1 is the basic treatment indicated for all types of halitosis; it includes tongue cleaning and mouth rinsing. TN-2 is strictly cause-related therapy, and its aim is to control all oral diseases and all intraoral malodour-causing factors. Patients diagnosed with genuine physiological halitosis are provided with TN-1, whereas patients with genuine pathological intraoral and extraoral halitosis are given TN-1 and TN-2 for improving their oral health.

Patients with pathological extraoral halitosis are referred to the appropriate medical practitioner (TN-3) for management of the systemic condition that is responsible for halitosis. Treatment of pseudo-halitosis, halitophobia and pathological halitosis of extraoral origin is outside the domain of dentists. However, in the beginning all suspected halitophobia patients are diagnosed as having genuine halitosis and are given TN-1 and TN-2, and if needed TN-3, whereas pseudo-halitosis patients are given TN-1 and TN-4. After a confirmed diagnosis, patients with

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pseudo-halitosis or halitophobia should be referred to the appropriate psychologist or psychiatrist (TN-5) for further management.

Effects of tongue cleaning and mouth rinsing on halitosis

Maintaining good oral hygiene and periodontal health are key to preventing halitosis. To this effect it is uncommon to find a person who does not brush or floss their teeth properly. The two most effective methods of controlling the VSC levels in the mouth are tongue cleaning and mouth rinsing.

Tongue cleaning

Cleaning the dorso-posterior surface of the tongue has a significant beneficial effect on all forms of genuine halitosis. As approximately 60% of total VSC in the mouth is from tongue coating, it is important to more than encourage patients with halitosis to prescribe to regular tongue cleaning. Removal of tongue coating by mechanical means has been shown to reduce the VSC concentration by 52% in the mouth air of a periodontally healthy individual[63]. A comparative clinical trial on the effects of tongue cleaning with a toothbrush and a tongue scraper showed that while both methods removed tongue coating, the tongue scraper showed a 75% reduction in VSC, and the toothbrush only achieved a 45% reduction in VSC[64]. However, patients generally seem to prefer tongue brushing to tongue scraping. Brushing the tongue with a regular toothbrush can cause microbleeding and damage to the dorsal surface of the tongue. Cleaning the tongue either with a brush or a scraper often effectively reduces malodour to below recognition threshold[39,65].

Mouth rinsing

Mouth rinsing is a very effective method of reducing oral malodour and is routinely prescribed for treatment of halitosis[66]. No toothpaste has been demonstrated to reduce mouth air VSC as dramatically as some mouthwashes. Antimicrobial components such as chlorhexidine, hydrogen peroxide, cetylpyridium, triclosan, zinc salts, benzalkonium, essential oils and their combinations are some of the commonly used mouth rinses. An ideal mouth rinse should maintain a normal oral flora by eliminating the existing pathogens and prevent the overgrowth of opportunistic pathogens. However, many mouth rinses mask the malodour and have insignificant antimicrobial effects. This is mainly because the mouth rinses are incapable of penetrating into the deeper layers of plaque biofilm and into periodontal pockets, and the antibacterial effects are not sustained for long periods[67].

Chlorhexidine is perhaps the most studied and the most effective anti-plaque mouth rinse. It has the quality of substantivity through which it maintains a prolonged anti-plaque effect. It has been shown to reduce gingivitis by 45%[68] and is very effective in reducing halitosis[66]. However, it cannot be routinely used because of its many side effects such as burning sensation of the oral mucosa, loss of taste sensation, tooth staining and rarely an allergic reaction[69,70]. Hydrogen peroxide has been commonly used and is also effective in reducing the malodour by oxidising odorous VSC to non-odorous salts[46,60]. Zinc salts in acetate, citrate or chloride forms are used as one of the ingredients in mouthwashes and anti-odour lozenges. Zinc-containing mouthwash has been shown to reduce mouth air VSC by 80% to 90% for up to three hours after rinsing[27,71]. It has been found to be effective in reducing halitosis by binding to the VSC[72].

Conclusions

Patients seek therapy more from dentists than from physicians. It is therefore crucial for dentists to have an understanding of all types of halitosis, especially those that arise from the oral cavity. Several misconceptions about halitosis need to be put aside. The available evidence indicates that VSC are not just odoriferous, but that some of them are deleterious to periodontal health. Therefore treatment of halitosis can no longer be considered as just a cosmetic therapy.

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