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# Halitosis and Oral Malodour

*In the second of a series of articles resulting from the work of the FDI's Scientific Commission, Professor Stephen Moss looks at halitosis*

## Introduction

Halitosis, or bad breath, is a commonly reported complaint. Whether in the form of occasional 'morning breath', which nearly every otherwise healthy adult encounters, or rarer and more serious problems ranging from metabolic disorders to chest tumours, halitosis is said to affect nearly 50% of the adult population. To judge from the size of the market for mouthwashes and other

breath fresheners (nearly US\$one billion at last count) it is a personal concern for a great many people<sup>1</sup>.

## Aetiology

Halitosis generally arises as a result of the bacterial decomposition of food particles, cells, blood and some chemical components of saliva. Thus 90% of causes of halitosis arise in the mouth<sup>1</sup>. As proteins and other chemicals in these materials are broken down into simpler components such as amino acids and peptides, many volatile substances (fatty acids and sulphur compounds) related to their decomposition are produced. Among these are propionic acid (smelling of vomit), butyric acid (smelling of rancid butter or rotting meat), valeric acid, acetone, acetylaldehyde, ethanol, propanol, and diacyl<sup>2</sup>.

Other decomposition products themselves become part of the metabolic pathways of bacteria in the mouth, and are further decomposed into volatile compounds. This is particularly true of the sulphur-

containing amino acids such as methionine, cysteine and cystine. The resulting volatile sulphur compounds (VSCs, such as hydrogen sulphide, methyl mercaptan, dimethyl sulphide and dimethyl disulphide) and other chemicals (cadaverine and putrescine, foul-smelling diamines) are at least partially responsible for the odours of which halitosis patients (or the people around them) complain. Four hundred volatile compounds have been detected in human mouth air. Over 300 oral bacteria have been found to cause the detectable VSC concentrations associated with halitosis, with over 80 species from subgingival plaque alone<sup>1</sup>.

VSCs are found in higher concentrations in the mouth gases emitted by patients with gum disease than in the mouth gases of healthy patients<sup>2</sup>. A recent study showed that subjects complaining of oral malodour had significantly more bleeding sites and plaques containing BANA-hydrolysing bacteria than subjects who did not report bad breath<sup>3</sup>. VSCs have recently been implicated in a feedback loop which begins and ends with poor oral health. Poor periodontal hygiene may lead to gum inflammation, creating oxygen-poor pockets in the mouth (e.g. between the gingiva and the teeth) and trapping gram-negative anaerobic bacteria in them. These bacteria then begin the proteolysis of salivary and tissue proteins which leads to the production of VSCs. Apart from their obvious effects on the patient's breath

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odour, these VSCs increase the permeability of the oral mucosa, speed the degradation of collagen, delay the healing of existing wounds, and also affect gingival and other periodontal cell function<sup>1,4</sup>. All of these effects may reinforce or aggravate the original conditions of poor oral health which led to the growth conditions of the halitotic anaerobes in the first place.

## The role of the tongue

The gram-negative anaerobic bacteria implicated in halitosis are also encountered on the tongue's coating, primarily the dorsal third of the tongue, which is not constantly scraped clean through mechanical interaction with the hard palate and the teeth (in contrast to the anterior two-thirds of the tongue). This area is also frequently missed by direct brushing, mouthwashes and antibacterial rinses either because it triggers a gag reflex or helps to close off the nasal cavity to the liquid with which the patient gargles. The tongue is an excellent harbour for anaerobic bacteria because of its large, continuous surface area which features taste and filiform papillae and crevices related to mucous glands and lingual tonsils<sup>1</sup>. Bacterial flourishing on the tongue is not unlike dust accumulation on a large, wrinkled shag carpet; this is why, even though periodontitis is linked with halitosis in one-third of patients (and some studies suggest this is a weak association at best<sup>5</sup>), *more* patients' bad breath is related to plaque on the dorsal third of the tongue than to periodontitis<sup>1</sup> (particularly, one study suggests, among younger subpopulations<sup>6</sup>). Tongue-brushing or tongue-scraping may provide significant relief for those patients whose gag reflex is not too easily stimulated.

## The role of saliva

One might wonder, if the oral cavity provides such a suitable environment for bacterial growth, why chronic halitosis is not rampant among the adult population. The truth is that while the dorsal third of the tongue, the spaces between teeth, and the spaces between the gingiva and teeth provide a rich growth medium *per se*, the mouth is a system in constant thermal, chemical, mechanical, and even biological flux. While salivary proteins are suitable for bacterial proteolysis, saliva also contains mucins, oligosaccharides and other substances constantly excreted into the mouth which facilitate the accumulation and clearing of bacteria through normal fluid motion. Saliva also contains immune factors like specific immunoglobulins. Oxygenation of the saliva inhibits the formation of anaerobic bacteria<sup>7</sup>. Speech and swallowing bring the forward two-thirds of the tongue into contact with the rugae of the palate, which serve to scrape this portion of the tongue clean. Finally, any given pathogen must compete with several hundred other pathogens, anaerobic and aerobic, halitotic and cariogenic, as well as dealing with the environmental changes that these other pathogens create. For example, sugar in the oral cavity is metabolised by aerobic bacteria, many of which produce acids as waste products. These acids lower the system's pH below the range at which the anaerobic halitotic bacteria can feed or reproduce. Sadly, they also cause cavities<sup>2</sup>.

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## Systemic conditions and halitosis

It is important to note that, while poor oral hygiene and poor oral health may often be sufficient conditions for the development of halitosis, they are not necessary conditions. Other causes of halitosis relate to the excretion into saliva of normal (and abnormal) metabolites from non-oral bodily processes and functions; this is the case with trimethylaminuria, or “Fish-Odour Syndrome,” which results from the body’s inability to completely break down choline and leads to an accumulation in the patient’s blood and other bodily fluids of trimethylamine, the compound responsible for the fishy odour of the patient’s breath, urine and sweat. People are probably more familiar with the acetone-smell of diabetic patients’ breath, and the ketone smell of anorexics’ (and some dieters’) breath. Problems with the renal system leading to uremia cause the breath to have a urine smell, while gall bladder problems and liver disorders like cirrhosis give the breath a mousy odour<sup>2</sup>. Leukaemia and other blood disorders have associated effects as well.

Halitosis is a common secondary complaint of patients with xerostomia, or chronic dry mouth, which is often a result of advanced age or of the irradiation of the head and neck which constitutes treatment for many cancers. Many commonly used drugs, such as antihistamines, antihypertensives, and drugs used to fight depression and Parkinson’s disease, can cause xerostomia as a side effect. Xerostomic patients’ *salivary function* is impaired, which either aggravates or precipitates periodontitis or simply raises the salivary concentration of the VSCs so their concentration in exhaled mouth air is increased. A drop in salivary flow associated

with the normal circadian rhythms of sleep is responsible for the ‘morning breath’ with which so many are familiar.

The fact that halitosis is so frequently associated with either xerostomia or sleep (where salivary flow decreases due to circadian rhythms and a cessation of chewing or other oral-mechanical activity) indicates the critical role saliva plays in the control of halitosis. *Bacterial* putrefaction of chemicals in the mouth is largely responsible for halitosis. Saliva contains proteins, carbohydrates and immuno-globulins which interfere with bacterial metabolism and bacterial adherence to oral surfaces, and which in some cases are actually *bactericidal*. Moreover, saliva’s role as a solvent in the oral chemical environment carries over to its role in controlling mouth odour: Volatile compounds dissolved in saliva don’t smell until they’re evaporated into mouth air and our noses evolved to register evaporated chemicals in air. It follows, then, that decreasing their concentrations in saliva by *stimulating salivary flow* makes it more difficult for the decreased vapour pressure in the mouth caused by inhalation or exhalation to evaporate them into mouth air, thus rendering them less detectable to ourselves and others. Breath fresheners tend to be both bactericides and stimulate salivary secretion. The act of chewing gum also stimulates salivary flow (and thus the clearance rate of accumulated bacteria), buffer capacity, and the concentration of critical oral defence factors; sugarless gum is less cariogenic than gum with sucrose or other sugars, but tends to raise the oral pH, making the environment more hospitable to

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halitotic anaerobic bacteria<sup>2</sup>.

Other oral causes include ulceration from diabetes, herpes, or trauma; postoperative complications from tonsillectomy<sup>8</sup> or oral reconstructive surgery<sup>9</sup>; oral cancers, dental abscesses, candidiasis, impacted food, improperly performed restorations to teeth, use of tobacco products, and dirty dentures. Foods rich in sulphur compounds, such as garlic, onions, or curries (which contain both), have been widely recognised for centuries as causes of unpleasant mouth odour<sup>2</sup>. Finally, any number of drugs (among them disulfiram, isosorbide dinitrate, ammonium trichlorotellurate<sup>10</sup>, even tetracycline<sup>11</sup>) can cause halitosis as a side effect.

## Respiratory and Gastro-intestinal Tract Disorders

Respiratory causes for halitosis include sinusitis, tuberculosis, bronchogenic carcinomas, foreign bodies lodged in the sinus cavity, and simple sore throat. Possible gastrointestinal causes for halitosis are a more contentious issue; some researchers argue that because of the gastrointestinal system's functional isolation from the respiratory pathway, and since the normal state of the oesophagus is one of collapse, breath odour should not necessarily indicate anything untoward about gastrointestinal function<sup>2</sup>. Cases have been reported of halitosis associated with hepatitis, cologastric fistula (a rare complication of Crohn's disease)<sup>12</sup>, with aorto-enteric fistula<sup>13</sup>, and with various diverticula<sup>14,15</sup>. Other researchers (in work that has yet to be widely reproduced) studied patterns of halitosis in couples and suggested a possible link between the bacterium *Helicobacter pylori* (implicated in stomach ulcers) and breath

odour<sup>16</sup>. This work would seem to share a basis with the recent development of *H. pylori* breath tests.

An Italian study<sup>17</sup> on the relationship between *H. pylori* infection and halitosis found a highly significant correlation between the elimination of *H. pylori* and the disappearance of halitosis (measured by sulphide compound assay). However, the eradication double therapy that eliminated the *H. pylori* may also have eliminated other, established halitotic bacteria. The study also found that in the presence of *H. pylori*, chlorhexidine rinses may not be completely effective against halitosis.

## Diagnosis

For centuries, diagnosis and measurement of halitosis were made through a human judge's assessment, through own sense of smell, of the odour of the patient's breath or saliva. While experienced odour judges may have developed a degree of acuity in sensing particular compounds (odour judges are still used in current research), the process was highly subjective and now seems unfit as a basis for differential diagnosis. Human sensory processing also places a limit on the ability of a judge to isolate a particular compound, creating a hypo-additive, non-linear relationship between the number of malodorous substances present and the number the judge can detect<sup>2</sup>.

Self-diagnosis seems particularly flawed, since a normally functioning olfactory sense becomes desensitized to odours it encounters continually. Additionally, psychological factors such as paranoia, schizophrenia and obsessive-compulsive disorder may distort the patient's own sense of his personal mouth odour, potentially leading to a form of hypochondria known as 'delusional halitosis'<sup>18</sup>. A

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1996 study found that self-ratings of mouth odour were significantly higher than the ratings assigned by an odour judge and were not associated with periodontal health. Complainants had a relatively higher psychopathological score on an SCL-90 profile than an age- and gender-matched reference group not reporting halitosis<sup>19</sup>.

A recent Japanese clinical study is indicative of the problems with self-diagnosis: It finds that patients entering Japanese dental clinics whose primary complaint was of halitosis were significantly less likely (by a factor of two) to be diagnosed with halitosis than those patients who had a different primary complaint (say, periodontitis or gingivitis) and a secondary complaint of halitosis. The researchers concluded that “the majority of patients with primary complaints of halitosis at the dental clinic did not actually have halitosis, but suffered from an imaginary halitosis due to presumptions based upon others’ attitudes.” Not surprisingly, the researchers found that the patients whose primary complaints of halitosis went unconfirmed by diagnosis at the clinic were more likely to be dissatisfied with the quality of treatment they received<sup>20</sup>.

Another study on self-assessment<sup>21</sup> made use of a new microbiological test to differentiate between psychogenic and organic halitosis. The test involved observing lead sulphide precipitation on an applicator tip imbedded in a specially supplemented anaerobic bacterial growth medium. The test had considerable power for classifying patients whose status (psychogenic or organic halitosis) had previously been established by an interview method.

Among ‘more objective measures’ are the use of portable sulphide monitors, gas

chromatography, flame-photometric detectors, and mass spectrometers. Since these measurement procedures range from the narrow but convenient to the expensive and unwieldy, a sizable body of dental research concerns itself with establishing lower-cost correlates for known clinical markers for halitosis. Levels of substances such as BANA (Benzoylarginine-2-Naphthylamide, hydrolysed by many oral microflora) are used as ‘instruments’ for levels of chemicals implicated in halitosis which are more expensive to detect. BANA testing, for example, has been shown to serve as a useful approach for detecting compounds, which cause halitosis but are *independent of VSC levels*<sup>22</sup>.

A recent study<sup>23</sup> on current measurement techniques reports progress both with zinc-oxide and nitrogen chemiluminescence detectors. The chemiluminescence detector, for example, permits the precise measurement of nitrogen compounds (such as indole and cadaverine) in organic matrices. This may help researchers to determine whether these nitrogen compounds are present in heretofore-undetectable concentrations in mouth air.

A 1996 Japanese study utilising a zinc-oxide thin film semiconductor shows that this sensor technology may be fruitfully employed in the development of a small, easy-to-handle chairside halitosis monitor. The monitor, which detects VSCs in mouth air, had measurements, which significantly correlated with those of odour judges, portable sulphide monitors and a gas chromatograph<sup>24</sup>.

## Treatment

Cost-effective and reliable management of mouth odour should include regular toothbrushing, flossing, and cleaning of the tongue's coat. These techniques aid in the control of the oral microflora that cause both halitosis and dental caries. Research has shown that salivary concentrations of thiols (such as mercaptans), which are precursors of foul mouth odour, can be significantly reduced through the use of hydrogen peroxide solutions or hydrogen peroxide-containing toothpastes. Mouthwashes and mouth rinses containing chlorhexidine gluconate have also proven effective in controlling oral microbes, but not without side effects such as tooth staining. More advanced treatments, such as the use of antibiotics, are rarely indicated, except in the case of postoperative oral infections.

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