# Oral Malodor--A Scientific Perspective

**Louis Z.G. Touyz, BDS, M.Sc. (Dent.), M.Dent. (POM)**

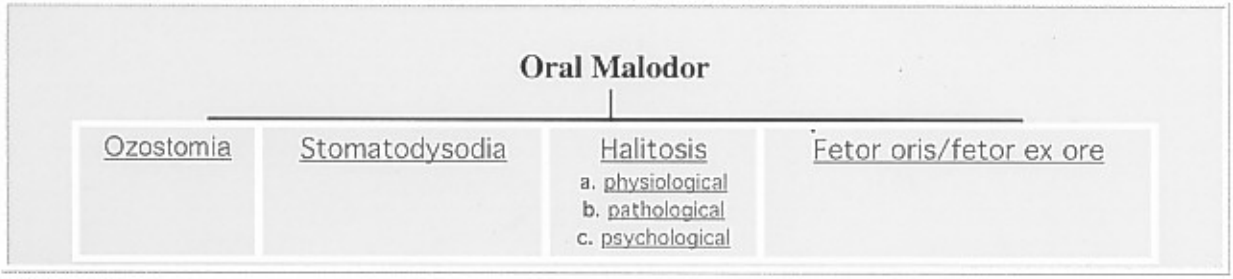
Originally published in the *Journal of the Canadian Dental Association* 59:607-610, 1993

Dr. Louis Touyz is the Director of the Division of Periodontology,
Faculty of Dentistry, McGill University,
Montreal, Quebec, Canada

## Introduction

Oral malodor, which is commonly noticed by patients, is an important clinical sign and symptom that often aids clinicians in establishing a diagnosis of underlying pathology.[1] Many terms are used to describe oral malodor, and many practitioners frequently use these terms imprecisely, with a resultant lack of clarity, confusion or ambiguity as to the kind of smell, its etiology, interpretation and significance. This article reviews the terminology related to the various types of oral malodors and associated conditions, presents a clarifying compendium of significant terms, and discusses some aspects of osmology relative to bad breath in general dentistry.

Oral malodor is a generic descriptor term for foul smells emanating from the mouth. The term does not imply any source or causation, *per se*. It is an aid to diagnosis and includes ozostomia, stomatodysodia, halitosis, and fetor ex ore/fetor oris.[2] It is important for clinicians and dentists to decide which of these oral malodors exist in the patient, as each implies different etiologies and demands different management.




Ozostomia (from the Greek *ozo*, to smell, and *stoma*, the mouth) refers to a putrid smell that is detected from the mouth but derives from the upper respiratory tract, in particular the nasal and sinus cavities, the pharynx and larynx.[3]

The etiology of ozostomia includes rhinitis, sinusitis, pharyngitis, laryngitis and tonsillitis, as well as any other inflammatory, neoplastic or putrefying condition of the upper respiratory tract, *e.g.* a nasal carbuncle, polyps, or carcinoma.[4]

Stomatodysodia (from the Greek *stoma*, mouth, and *disodia*, bad odor) refers to foul breath from the mouth originating from local areas in the lower respiratory tract, particularly below the caryna from the bronchi, bronchioles and alveoli, or other contiguous parts of the lung, *e.g.*, pleura.[3,5]

The etiology of stomatodysodia includes infective/necrotic processes of the lower respiratory tract, such as bronchitis, bronchiectasis, pulmonary abscess, tuberculosis, pneumonia, emphysema, secondary

infection and necrosis of neoplasms, pulmonary infarcts with all its sequelae, stagnation of sputum, and smells from inhaled smoke.

Halitosis (from the Latin *halitus*, breath, and *osis*, condition) refers to a stench on the breath stemming from systemic metabolic conditions, including the gastrointestinal tract and its contents, as well as generalized pathophysiological conditions that initiate transport of smelly substances via the blood supply to the lungs or gut, which then act as an excretory or secretory mechanism for one or more specific products redolent of the cause.

Halitosis may be physiological or pathological.[6,7] Physiological halitosis will be of a temporary nature and exists when volatile odoriferous hematologically borne substances are released into the lungs from food, notably herbs, spices, curries, and selected vegetables such as onions, garlic, radishes, turnips, and leeks; or from flavory drinks, such as tea and coffee, and particularly those containing unique derivatives of water and alcohol soluble esters and polyphenols.These include wine, brandy, whisky, liqueurs, and beer. Physiological halitosis will also occur with dehydration, starvation, constipation, loose stools or other conditions that may affect the gut, most of which are reversible.[7]

Pathological halitosis occurs by essentially the same mechanism as physiological halitosis, namely by pulmonary release of hematologically borne substances. This halitosis is not easily reversible, however, and tends to persist without treatment. It stems from regional or systemic pathology, such as diabetic ketosis (from producing acetoacetic acid, hydroxybutyric acid, acetone and other ketones), uremia, gastritis, gastric ulcer, oesophagitis, pyloric stenosis, or hepatitis.[8,9]

Fetor oris/fetor ex ore (from the Latin *fetor*, an offensive odor, and *os*, gen. *oris*, plural *ora*, mouth) is an offensive odor emanating from the mouth, which arises from sources in the mouth.[3]

**Etiology**

The commonest source of fetor oris is oral microbiota and their action on oral debris, blood or oral tissues. Oral bacterial plaques adhering to the teeth are the major offenders. Consequent conditions that result from various kinds of mature plaques reacting on the teeth or the supporting soft tissues and bone include dental decay, non specific gingivitis, periodontitis, gingival and alveolar abscesses, local soft tissue necrosis, degenerating food residues, acute necrotizing ulceromembranous gingivitis, and calculus adherent on natural teeth and prostheses.[8,10] Many local or systemic pathological states manifest in the mouth, and include conditions such as acute herpetic gingivostomatitis and other oral viral infections, erythema multiforme, benign mucous membrane pemphigoid, hemorrhagic diatheses, oral neoplasms, salivary gland diseases, and healing trauma, all of which are frequently associated with fetor oris. Poor oral hygiene, morning breath, hunger breath, and menstruation breath all result from changes in the oral microbiota and may manifest discernible bad breath.[11-13]

Similarly, changes in the oral microbiota resulting from the use of antibiotics frequently manifest fetor oris.[14] Other clinical findings associated with oral malodor may be detected or related to smell and/or taste at the time of presentation, and defining these findings certainly contributes to the comprehensive description of a presenting case. Descriptive terms include:

**Ozaena:** Any disease characterized by intranasal crusting, mucosal atrophy and a fetid odor from the

nose.

**Fetor narium:** A bad smell coming from the nose.

**Dysosmia:** A perversion of smell, frequently impairment.

**Hyperosmia:** An increased subjective sensitivity to odors.

**Cacosmia:** The subjective, consistent, perception of a real or imagined distinctly putrefactive disagreeable odor. A variety of parosmia.

**Parosmia:** A perversion of the normal sense of smell, usually, but not always, to unpleasant odors that do not exist. Also called parosphresia.

**Anosmia:** The inability to detect any smell.

**Euosmia:** A pleasant fragrance, normal olfaction.

**Hypereuosmia:** The inability to differentiate between foul odors and pleasurable fragrances, perceptions being mainly pleasant.

**Cacogeusia:** A bad taste.

**Dysgeusia:** A perversion of taste.

**Discussion**

Smell and taste are interrelated in that taste or smell in isolation do not function fully if any of the receptors or innervations are blocked to either sensation — *i.e.*, disruption of one sense compromises the function of the other.[15] The olfactory nerve (first cranial nerve) for smell, and the lingual branch of the trigeminal (fifth) nerve, the corda tympanic branch of the facial (seventh), the lingual portion of the glossopharyngeal (ninth) and a branch to the palate and epiglottis from the vagus (tenth) nerve, all must be functionally intact to ensure ideal function of smell and taste.[16] The physiology, structure and function of smell and taste sensations have been comprehensively described and reviewed elsewhere.[16]

Any substance that can be detected from a distance through a perceived olfactory experience is said to smell. The emotional conscious reaction induced, provoking an overall stimulation, acceptance and pleasure, or irritation, revulsion and rejection, dictates whether smells are classified as scents, aromas and fragrances, or as odors, stinks and stenches, respectively.[17]

Aromas and fragrances are a product of numerous substances, individually acceptable, but collectively in the right concentrations evoking an overall very pleasant reaction.[17]

With oral malodor, a number of individually bad smelling substances combine to produce a stink that is unpleasant. Detecting, appreciating and describing the stink can help to define its source and establish a diagnosis. Oral malodor could therefore be a feature of some oral or non oral systemic disease.[1] However,

there are healthy individuals who complain of having bad breath that no one else can smell and for which no local or systemic condition can be found. Although this is sometimes referred to as "delusional halitosis," it would more accurately be described as delusional cacosmia, and is considered a monosymptomatic hypochondriacal disorder.[18] A malodor is often not detected subjectively due to the phenomenon of olfactory adaptation. Approximately 80 per cent of the oral malodor that manifests as fetor oris originates from the mouth,[19] and physicians should readily refer these patients to dentists for appropriate treatment and oral hygiene management.

Hydrogen sulphide ($H_2S$) and mercaptan ($CH_3SH$) are most commonly associated with oral malodor, but other volatile sulphur compounds are also involved, such as dimethylsulphide ($CH_3 - S - CH_3$).[20] The intensity of oral malodor fetor oris increases eight times or more in mouths affected by periodontal disease. This fetor oris is caused by an increase in volatile sulphur compounds and an increased mercaptan/hydrogen sulphide ratio.[21] Ketobutyrate is a product of the essential sulphur containing amino acids, namely cysteine and methionine, and is metabolized to mercaptan. Bacterial enzymes such as L-cystine desulfhydrases and 1-methionine gama-lyase break down cystine, cysteine, and methionine to produce mercaptan.[22]

Many oral microbes found in saliva and mature climax community subgingival bacterial plaques possess these and other similar enzymes that produce mercaptan and other volatile sulphur compounds.[13] Sources other than the mouth produce oral malodor by ozostomia, stomatodysodia or halitosis, as explained above. Breakdown products like indoles, skatoles, ammonia, and urea all contribute to oral malodors.[13]

Bacterial stagnation in grooves, fissures and interpapillary areas on the dorsoposterior part of the tongue is a major source of malodor fetor oris.[23] Invading bacteria flourish under gingival opercula, flaps and tags associated with partially erupted third molars. The removal of these teeth and the offending surrounding soft tissue markedly reduces oral malodor fetor oris.[24]The removal or controlled reduction of bacteria by mechanical or chemical means will reduce oral malodor — for example, zinc containing mouth rinses are effective as are chlorhexidine mouthwashes .[25,26]

Oral malodor is an ubiquitous and common condition. Since this is a distressing symptom for patients, the need to diagnose the underlying cause is very important. Once diagnosed, the appropriate therapy can be instituted. The cause, if possible, should be defined by the treating doctor so as to help the patient.

Management of oral malodor is therefore dependent on diagnosing the foul breath as ozostomia, stomatodysodia, physiological or pathological halitosis, or fetor oris. Therapy should then be appropriately and specifically directed to the cause of the oral malodor to ameliorate or totally eliminate the condition.

Establishing the full character of an oral malodor is not without some slight risk, because viable bacterial and viral infections are known to occur through droplet spread.[27,28] Although the human immunodeficiency virus has not been reported to infect people via this mechanism, in light of the transmissibility of microbiota and viruses, it would be most prudent to detect smells through an orofacial mask. Clear and precise descriptions of oral malodors facilitate good general medical practice, but more research[29]is needed to produce simple and safe bedside clinical tests,[30,31] which could easily detect, classify and quantify malodorous substances so that oral malodor could be used safely and more

successfully in diagnosing, monitoring, and treating disease.

**References**


1. Tessier, J.F. and Kulkarni, G.V. Bad breath, etiology, diagnosis and treatment. *Oral Health* 81:19 24, 1991.

2. Kleinberg, I. and Westbay, G. Oral malodor. *Crit Rev Oral Biol Med* 1:247-260, 1990.

3. Stedman's Medical Dictionary. Ed. 24. Baltimore and London, Williams and Wilkins, 1982.

4. Fletcher, S.M. and Blair, P.A. Chronic halitosis from tonsilloliths: a common etiology. *J LA State Med Soc* 140:7-9, 1988.

5. Hopkins, J.M. The suppurative lung diseases. In: *Oxford Textbook of Medicine*. Ed. 2. D.J. Weatherhall, J.G. Ledingham and D.A. Warrell, eds. Oxford, Melbourne, New York, Oxford University Press, ch. 15:15.100-15.106, 1987.

6. Bogdasarian, R.S. Halitosis. *Otolaryngol Clin North Am* 79:111-117, 1986.

7. Rosenberg, M. and Gabbay, J. Halitosis — a call for affirmative action. *Refuat Ha Shinayim* 5:13-15,1987.

8. Tonzetich, J. Production and origin of oral malodor: a review of mechanisms and methods of analysis. *J Periodont* 48:13-20, 1977.

9. Madarikan, B.A. and Bees, B.I. Halitosis and gastric outlet obstruction in infants. *Br J Clin Pract* 44:419-423, 1990.

10. Kostelc, J.G., Preti, G., Zelson, P.R. et al. Oral odors in early experimental gingivitis. *J Periodont Res* 19:303-312, 1984.

11. Tonzetich, J. Oral malodor: an indicator of health status and oral cleanliness. *Int Dent J* 28:308-319, 1978.

12. Natty, F. Dry mouth and halitosis. *Practitioner* 234:603-606, 1990.

13. McNamara, T.F., Alexander, J.F. and Lee, M. The role of micro-organisms in the production of oral malodor. *Oral Surg* 34:41-48, 1972.

14. Ogunwande, S.A. Halitosis and abuse of antibiotics. Report of a case. *Ceylon Med J* 34:131-133, 1989.

15. Bradley, R.M. Physiology of taste receptors. Physiology of olfactory receptors. In: *Basic Oral Physiology*. R.M. Bradley, ed. Chicago and London, Year Book Medical Publishers. ch. 3, 21-24; ch. 4, 43-57. 1981.

16. Jenkins, G.N. Sensations arising in the mouth. In: *Physiology and Biochemistry of the Mouth*. G.N. Jenkins, ed. Oxford and London. Blackwell Scientific Publications (67 References to Basic Physiology), 1978, pp. 542-570.

17. Noble, A.C., Arnold, R.A., Buchsenstein, J. et al. Modification of a standard system of wine aroma terminology. *Am J Enol Vitic* 38:143-146, 1987.

18. Iwu, C.O. and Akpata, O. Delusional halitosis. Review of the literature and analysis of 32 cases. *Br Dent J* 168:294-296, 1990.

19. Tonzetitch, J. Sources measurement and implication of oral malodor. *J Dent Res* 70:337, 572 (abstract), 1991.

20. Schmidt, N.F., Missan, S.R. and Tarbet, W.J. The correlation between organoleptic mouth-odor ratings and levels of volatile sulfur compounds. *Oral Surg* 45:560-567, 1978.

21. Kostelc, J.G., Zelson, P.R., Preti, G. et al. Quantitative differences in volatiles from healthy mouths and mouths with periodontitis. *Clin Chem* 27:842-845, 1981.

22. Yaegaki, K. and Suetaka, T. Fractionation of the salivary cellular elements by Percoll density gradient centrifugation and the distribution of oral malodor precursors. *Shigaku* 77:269-275, 1989.

23. Rosenberg, M. Bad breath, diagnosis and treatment. *Univ Toronto Dent J* 3:7-11, 1990.

24. Rajashuo, A., Torkko, H. and Meurman, J.H. Effect of extractions of lower third molars on periodontopathogens. *J Dent Res* 70:367, 817 (abstract), 1991.

25. Grossman, E., Meckel, A.M., Isaacs, R.L. et al. A clinical comparison of antimicrobial mouth rinses: effects of chlorhexidine, phenolics and sanguinarine on plaque and gingivitis. *J Periodont* 60:435-440, 1989.

26. Pitts, G., Brogdon, C., Hu, L. et al. Mechanism of action of an antiseptic, antiodor mouthwash. *J Dent Res* 62:738-742,1983.

27. Miller, R.L. and Micik, R.E. Air pollution and its control in the dental office. *Dent Clin N Am* 22:453-476, 1978.

28. Mosley, J.W. Hepatitis infection. *New Engl J Med* 293:729-734, 1975.

29. Rosenberg, M. Halitosis--the need for further research and education. *J Dent Res* 71:424, 1992.

30. Rosenberg, M., Kulkarni, G.V., Bosy, A. et al. Reproducibility and sensitivity of oral malodor measurements with a portable sulphide monitor. *J Dent Res* 70:1436-1440, 1991.

31. Phillips, M. Breath tests in medicine. *Scientific Am* 74-79, July 1992.