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Halitosis: a new definition and classification

ABSTRACT

There is no universally accepted, precise definition, nor standardization in terminology and classification of halitosis. This paper reviews the previous approaches and highlights inconsistencies and redundancies.

A new definition, free from subjective descriptions (fecal, fish odor, etc.), one time sulfide detector readings and organoleptic estimation of odor levels, and excludes temporary exogenous odors (e.g. from dietary sources). Some terms previously used in the literature are revised.

A new etiologic classification is also proposed, dividing pathologic halitosis into Type 1 (oral), Type 2 (airway), Type 3 (gastroesophageal), Type 4 (blood-borne) and Type 5 (subjective). In reality, any halitosis complaint is potentially the sum of these types in any combination, superimposed on the Type 0 (physiologic odor) present in health.

This system allows for multiple diagnoses in the same patient, reflecting the multifactorial nature of the complaint. It represents the most accurate model to understand halitosis and forms an efficient and logical basis for clinical management of the complaint.

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LITERATURE REVIEW

Previous definitions

Halitosis is receiving increasing scientific interest, but still no accepted definition exists. In the literature, definitions include: “the subjective perception after smelling someone's breath, if unpleasant”,[1] “noticeably unpleasant odors exhaled in breathing",[2] "an oral health condition characterized by unpleasant odors emanating consistently from oral cavity",[3] "general term to describe any disagreeable odor of breath, regardless of its origin",[4] and "an unpleasant odor emanating from oral cavity."[5]

Many definitions are inadequate, ignoring the potential emanation of odors via the mouth and nose from the respiratory and gastroesophageal tracts, transfer of volatiles from blood to breath during alveolar gas exchange, and also self-perception of halitosis by the patient. To varying degrees, the breath always has odorant volatiles, originating orally or elsewhere. None set a clear boundary between normal, physiologic breath odor and, pathologic halitosis. Negative identification of an odor requires qualification. Who determines this? The patient, the patient's social environment, or the clinician by sulfide detectors (halitometers)? Some refer to exogenous odorants (e.g. garlic) as halitosis, yet this is not pathologic. To distinguish normality from disease, a more precise definition and classification is needed.

This paper reviews previous attempts at classification and definition of halitosis, and forwards a new scheme. The diagnosis and treatment of halitosis according to this scheme are discussed in a separate publication.

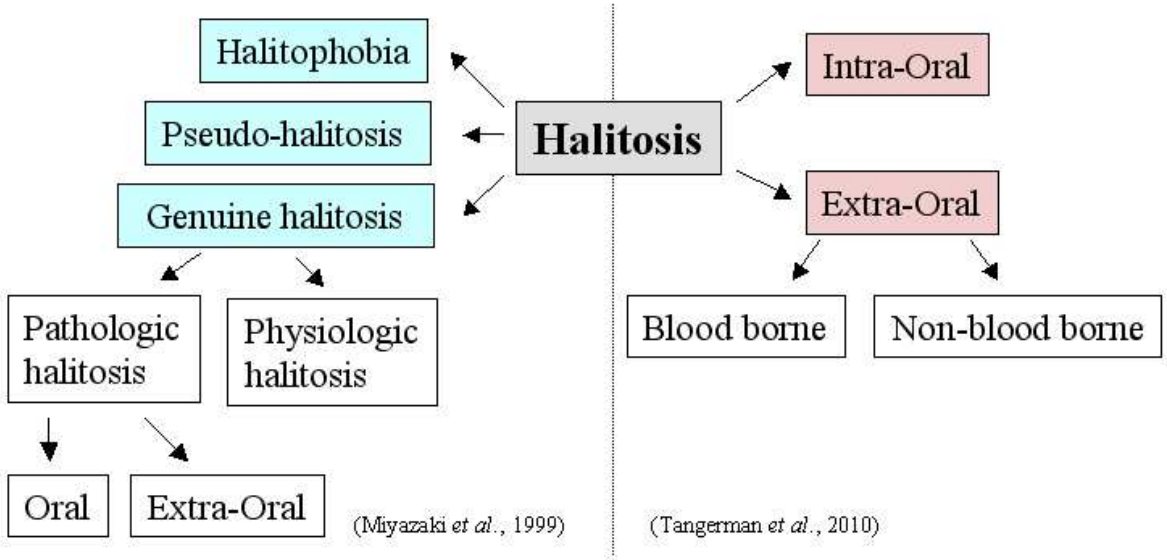

Fig.1 *Two previous classifications. Miyazaki et al. 1999 is generally the most widely used, but neither is universally accepted.*[5]

Previous classifications

Miyazaki *et al.* 1999 suggest genuine halitosis, pseudo-halitosis and halitophobia (Fig 1).[6] Genuine halitosis is divided into physiological or pathological, then the latter is split into oral and extra-oral. This was adapted to North American society with regards halitophobia, and appeared in publications a decade ago.[7-9] This classification is inflexible since multiple diagnoses for one patient are not enabled. The broad category "extra-oral, pathologic halitosis" does not aid referral choice, or help the receiving clinician, and is also poor for researchers who need to precisely classify extra-oral halitosis according to etiology. “Morning breath” is not placed in the oral category, despite manifesting orally. Inappropriately, two out of three categories (pseudo-halitosis and halitophobia) have psychopathologic connotations, and, they are excluded from pathologic halitosis. Subjective halitosis can be caused by psychologic or neurologic factors, which are technically extra-oral processes, yet “extra-oral halitosis” is again excluded from pathologic halitosis. After treatment, whether for genuine halitosis or pseudo-halitosis, if the patients continue to believe they have halitosis, reclassification to halitophobia occurs. This categorizes cases according to treatment outcome, as halitophobia is diagnosed following a treatment failed. This scheme claims to provide treatment needs, but how can these be determined beforehand if they depend on results of treatment? Pseudo-halitosis is misleading when considered alongside other medical terms, e.g. pseudo-Cushing's syndrome, intestinal pseudo-obstruction, pseudo-lymphoma or pseudo-Kaposi's sarcoma. These exist as physical entities which masquerade as their namesake. Pseudo-halitosis implies a genuine, physical condition mistaken for halitosis non-exist. Similarly, halitophobia suggests an irrational fear, but instead refers to where the patients believe their treatment unsuccessful.

Tangerman and Winkel suggest intra- and extra-oral halitosis, the latter then divided into non-blood-borne and blood-borne.[10] An earlier publication divides extra-oral halitosis into blood-borne, upper respiratory tract and lower respiratory tract.[11] They list 4 etiologic mechanisms of blood-borne halitosis: systemic diseases, metabolic disorders, food and medications.[10] These authors use "pseudo-halitosis/halitophobia" to describe no measurable halitosis, whilst retaining their own classification for measurable halitosis.[12] In reality, this classification focuses on oral and blood-borne halitosis, with insufficient categorization of physiologic, sinonasal, laryngopharyngeal, gastroesophageal, or psychologic causes. The significance of blood-borne halitosis relative to other extra-oral mechanisms is unclear, and a broad division into blood-borne and non blood-borne may be inappropriate. Again, this system does not allow for multiple diagnoses, making accurate categorization of some cases difficult, and there is no distinction between pathologic and physiologic halitosis.

Motta *et al.* suggest primary halitosis ("respiration exhaled by the lungs"), and secondary halitosis ("originates in mouth or upper airways").[13] It is unclear if primary halitosis refers to blood-borne halitosis, odor from the lower respiratory tract itself, or both. This is seldom used, perhaps because the clinical utility is limited by not addressing subjective halitosis or gastroesophageal halitosis.

Previous terminology

In some cases, odor is not detected organoleptically and volatile sulfur compound (VSC) levels are normal. There is no local or systemic condition, and no reliable, third party confidants confirming the complaint. This scenario is generally ascribed to psychologic factors, termed imaginary halitosis,[14] delusional halitosis,[15] pseudo-halitosis,[7] non-genuine halitosis,[16,17] chronic olfactory paranoid syndrome,[18] anthropophobia (taijin kyofusho),[19] halitophobia,[20] olfactory reference syndrome (ORS),[21,22] and social anxiety disorder.[23] These terms may easily cause confusion.

The term psychosomatic halitosis is incorrectly when referring to subjective halitosis complaints. Psychosomatic disorders are disorders in which psychologic factors play a significant role, and there are physical symptoms which are detectable clinically. However, the term psychosomatic halitosis is used to describe an odor is that is clinically nonexistent.

Terms which refer to odor character promote confusion for clinicians and patients e.g., sulfurous/fecal, fruity, and ammoniacal/urine-like; respectively attributed to VSC, acetone, and ammonia with other amines.[10] Sweet, musty or fishy are used to describe particular halitosis types. However, fish odor is nonspecific for trimethylaminuria (TMAU),[24] as is acetone for diabetes. All individuals have detectable breath acetone >400 ppb,[25,26] especially when fasting. Fish odor can be perceived as musty, and acetone as sweet. The sweet, musty aroma in liver failure has been termed fetor hepaticus.[27] This is also described as fecal, "the smell of dead mice" or "the breath of the dead".[28]

Other terms include "denture odor",[29] "uremic fetor" in renal failure,[30] and "rotten egg" in poor oral hygiene. All these terms are subjective and open to misinterpretation. There is no standardization in terminology, which has lead to discrepancies developing

where some authors use a term with one definition and others with different meaning.

- Oral malodor, oral halitosis, tongue malodor, odontogenic halitosis, pathological halitosis, objective halitosis, genuine halitosis and intra-oral halitosis are used incorrectly as synonyms for halitosis.[31] E.g. oral malodor includes all odors originating orally, not just the tongue; but not all pathologic/objective halitosis originates orally.
- Pseudo-halitosis, psychosomatic halitosis, halitophobia or self- and imaginary halitosis are also sometimes used interchangeably,[32] as with non-genuine, delusional and phantom halitosis, but they not synonymous, e.g., halitophobia describes a fear; self-halitosis describes clinically existent, self-producing odor; imaginary halitosis describes halitosis produced psychologically; and phantom halitosis is neurologic.
- Morning breath is sometimes used instead of physiologic halitosis, but these are also dissimilar. Not all 'morning breath' is physiologic.

NEW DEFINITION OF HALITOSIS

Objective halitosis has been defined as "malodor with intensity beyond a socially acceptable level perceived".[7] This is independent from halitometric readings and subjective odor descriptions. This should be a basic definition of objective halitosis, but must be qualified with several important points:

- A halitosis complaint may be **objective**, where there is an unpleasant odor endogenously produced anywhere in the body, emitted from the mouth and/or nose and detectable to others; or **subjective**, where there is no detectable odor to others but the patient complains of its presence.
- Anyone who complains of halitosis, objective or subjective, should be considered a "halitosis patient".
- Evidence of objective halitosis is a clinical picture built of (i) reliable reports from the patient's social environment e.g. family members or close friends, (ii) patient's self declaration of halitosis, and to a lesser extent (iii) halitometric readings.
- A lack of complaints from the patient's social environment including family members, suggests that there is no objective halitosis. Furthermore, if there are no complaints from either the patient or his/her social environment, this usually implies that there is no need to diagnose halitosis or treat, even if halitometric measurements appear to indicate the presence of elevated VSC. As a rule, halitometers measure VSC, not halitosis.
- Halitosis is considered unpleasant by the patient and his/her social environment. If the odor is not perceived negatively, it is not halitosis.
- Halitosis is almost always chronic in nature, although it may be intermittent.
- Some diseases (tonsillitis, pharyngitis, etc.) or transient oral flora or metabolic changes in the body may cause bad odor in the short term (< 2 months), which disappears when the condition resolves. Such bad odors are called temporary halitosis.
- Some volatile foodstuffs posses specific odors (e.g. garlic, onion) and

may cause short term halitosis ("dietary odor"), as with certain medications or intoxications. All are called temporary halitosis, managed with reassurance and advice, and further diagnosis or treatment is unnecessary.

NEW CLASSIFICATION OF HALITOSIS

Types 1 - 5 (Fig. 2) represent different odor mechanisms, which may be present in any combination at any time. Potentially, each type of pathologic halitosis (Type 1 - 5) is superimposed on physiologic odor (Type 0). At any given time, pathologic halitosis is the sum of the all these types sources, as well as their respective underlying physiologic contributions.

The relative contributions of these different physiologic and pathologic etiologies is subject to interpersonal variation, and may fluctuate even within hours in the same individual. Sometimes the level of one or more types may be so low as to give negligible contributions to the overall complaint, or there may be multiple contributing factors in the same patient. This can be recorded as Type 1+3, Type 2+4, Type 1+4+5 halitosis, etc. Previous classifications oversimplify halitosis, and this new classification is the most representative model proposed.

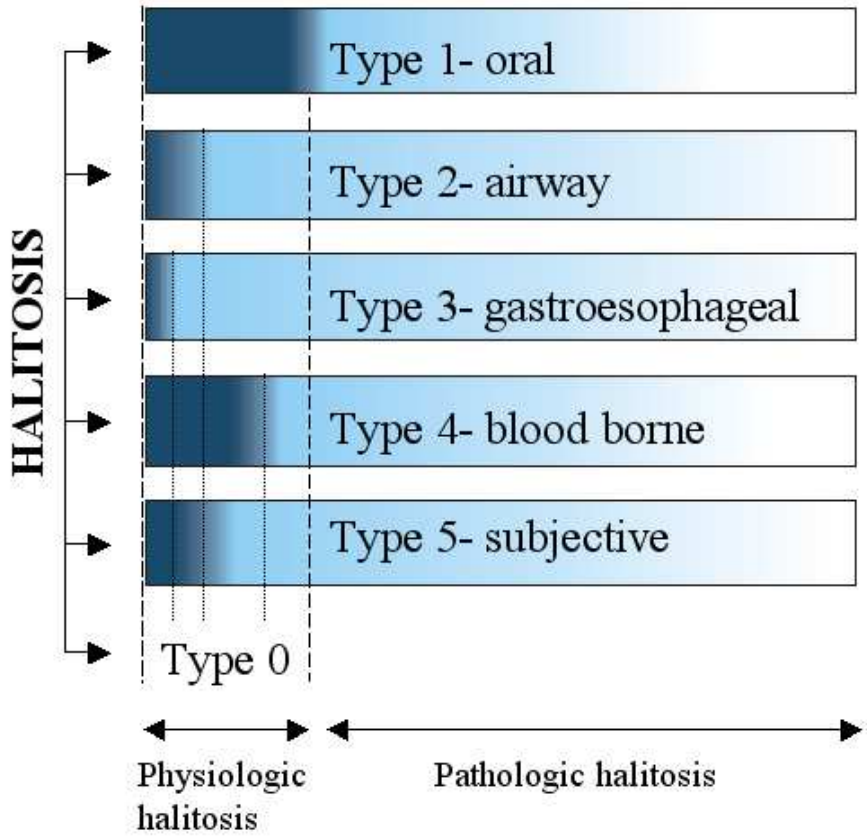

Fig.2 New etiologic classification is proposed.
Physiologic (Type 0) halitosis consists of the sum of the physiologic aspects of oral (Type 1), airway (Type 2), gastroesophageal (Type 3), and blood-borne (Type 4) subjective (Type 5) odors at any particular time, potentially superimposed on halitosis concern.

Type 0 halitosis: *Physiologic halitosis*

Type 0 halitosis represents the sum of the physiologic contributions of oral, airway, gastroesophageal, blood-borne and subjective halitosis that are potentially present in every healthy person, in any combination. All healthy individuals have a certain level of bacterial activity in the mouth and on respiratory tract mucosae. In addition there is a potentially a negligible amount of gas leakage from the gastroesophageal tract, and blood gases are transferred to the exhaled breath during gas exchange in pulmonary alveoli. Therefore, minimal amounts of types 1 - 5 potentially exist in health. The total level of odor, and the relative contributions of these different sources of physiologic odor, is subject to both interpersonal variation, and also variation in the same individual from one occasion to the next.

Table.1 Physiologic halitosis should not be confused with a low level of oral (type 1) halitosis since there are differences

	Type 0	**Type 1**
Duration	Always present; fluctuating	While a cause exits
Originates	Mouth + elsewhere	Mouth only
Detectable on	Mouth air + breath	Mouth air
Offensive?	Possibly	Yes
Treatable?	No	Yes
Preventable?	No	Yes
Detectable by halitometer?	Yes	Yes

One or multiple types may exist in any combination at any time, varying according to many different factors, including hydration, oral hygiene, microbiota, salivary flow rate, nature of last food consumption, biochemical, hormonal, mechanic activity of the body, fasting, sleep, digestive enzyme profile in gut, momentarily amino acid and electrolyte profile in serum etc. It is distinguished from oral halitosis (See Table 1).

Type 1 halitosis: *Oral halitosis*

The gases that contribute to Type 1 (oral) halitosis are (greatest to least): VSC, volatile organic compounds (VOC) and nitrogen containing gases (amines).[33] The main VSC involved are hydrogen sulphide (H_2S), methyl sulfide (CH_3SH, or methyl mercaptan, MM), and dimethyl sulfide ($(CH_3)_2S$, DMS). Nearly 700 different compounds have been detected orally,[35] including indole, skatole, acetic acid and short chain acids (e.g. butyric, valeric, isovaleric, lactic, caproic, propionic and succinic acids). In halitosis patients, the 30 most abundant VOC in mouth air are alkanes or alkane derivatives, and of these the most common are methyl benzene, tetramethyl butane, and ethanol.[34] Alkanes are aromatic breakdown products from reactive oxygen species acting on inflamed tissues.[34] Others report acetone, methenamine, isoprene, phenol, and D-limonene are the most abundant organic compounds in mouth air in oral halitosis patients. The organoleptic level of oral halitosis correlates with VSC,[35] and amines (such as putrescine, cadaverine, and trimethylamine).[36]

The gases responsible for oral halitosis are byproducts of protein and glycoprotein putrefaction by the oral microbiota. The dorso-posterior tongue is the most important halitogenic site, by virtue of having both the largest surface area and the highest bacterial

load, within a densely populated biofilm.[37-39] The most common factors include poor oral hygiene, plaque stagnation areas, gingivitis, tongue coating; and account for ~85% cases.[31] However, a degree of oral bacterial action is continuously present in health, even with impeccable oral hygiene, and this constitutes the physiologic part of Type 1 halitosis.

Specific bacteria, especially anaerobes, are suggested to cause oral halitosis.[40,41] In reality, most oral bacteria are potentially odorigenic, releasing VSC, VOC and/or amines. Depending upon the constituents of the gas produced by oral bacteria and ecologic factors in the mouth (e.g. microbiota compositional fluctuations, available nutritional substrate, bacterial metabolism) momentarily determine the composition and level of odor. Therefore, the diagnostic value of the odor character at any one time is questionable. To consider some bacteria as odorigenic and others as non-odorigenic is oversimplification. In reality every bacterium is odorigenic, and there is a continuous spectrum from low to high degree of odor formation capability.[33,42]

Other possible origins of oral halitosis include: periodontal disease, acute necrotizing ulcerative gingivitis, osteoradionecrosis, large carious cavities, blood/thrombi (e.g. extraction sockets), ulceration, interdental food packing, oral prostheses (dentures, orthodontic appliances, bridges).

Type 2 halitosis: *Airway halitosis*

Type 2 halitosis originates from the respiratory tract itself (rhinosinusitis, tonsillitis, pharyngitis, laryngitis, bronchitis, pneumonia), anywhere from nose to alveoli. Odorous gases produced by various respiratory pathoses, held in the exhaled breath and expressed via the nose or mouth. This should be distinguished from type 4 (blood-borne) halitosis, where volatiles from the systemic circulation are transferred during gas exchange to the breath. Some studies report the proportion of halitosis cases that are due to upper respiratory tract pathology to be between 2.9 and 10%.[31,43-47]

Halitosis is considered a regional symptom of chronic rhinosinusitis, and some report as many as 50-70% will complain of halitosis.[48,49] In pediatric patients, one of most frequent symptoms is halitosis together with cough, rhinorrhea and sniffling,[50] even when nasal obstruction, postnasal exudate, pain, sneezing and secretion are clinically absent.[51] Sinonasal anatomic variations (e.g. agger nasi cells, pneumatization of turbinates or septum; deviated nasal septum) are very commonly found together with mucosal pathoses including rhinosinusitis.[52]

Post nasal drip is where mucus drains onto the dorsal tongue via the nasopharynx.[53] This is related to allergic rhinitis, however the existence of post nasal drip as a clinical entity disputed as this occurs in health and there is no agreed definition or pathologic changes.[54] Mucus stagnation provides a proteinaceous medium for more bacterial putrefaction, but the relationship between halitosis and post nasal drip has not been formally investigated.

Obstructive nasal pathology causes mouthbreathing, possibly resulting in xerostomia and halitosis.[13,55]

Tonsillitis causes edema and hypertrophy, which may obstruct orifices on tonsillar surface. This disrupts the cleansing flow of secretions, and desquamated epithelial and bacterial cells, extracellular matrix and food debris become trapped, leading to

stagnation. Bacteria putrefy local substrate and release VOC and VSC, expressed on the breath as halitosis with a similar mechanism operates on tongue surface. Crypt debris may mineralize, similar to the transformation of dental plaque to dental calculus. These mineralized deposits are termed tonsilloliths (tonsil stones). The presence of tonsilloliths is strongly associated with abnormal VSC levels.[46] They are asymptomatically present in up to 10% of the general population.[56] Anaerobic bacteria detected in tonsilloliths include *Eubacterium, Fusobacterium, Porphyromonas, Prevotella, Selenomonas* and *Tanerella spp.*, all associated with the production of VSCs.[57]

Odorous gases from the mouth or present in oronasal secretions can excite olfactory receptors and be perceived as halitosis,[58] even if no halitosis can be detected halitometrically. This is retronasal olfaction and and is usually misdiagnosed.

"Airway reflux" describes gaseous or liquid gastric contents refluxing to the pharynx, oral cavity, nasal cavity, paranasal sinuses or even the middle ear,[59] and is sometimes said to be a cause of halitosis, however there is little credible evidence for this mechanism.

Other respiratory tract causes of halitosis include: laryngitis, tracheitis, bronchitis, bronchiectasis, pneumonia, tuberculosis, nasal foreign bodies, rhinoliths, atrophic rhinitis (ozena), abscesses (peritonsillar, nasopharnygeal, lung), and carcinomas (nasal, sinuses, pharyngeal, lung).[10,60-65]

Type 3 halitosis: *Gastroesophageal halitosis*

Type 3 halitosis is leakage of odorant volatiles from the stomach via the esophagus to the mouth and nose. This should be distinguished from volatiles in the GI tract being absorbed into the systemic circulation and exhaled (Type 4, blood-borne halitosis). A degree of gastroesophageal reflux is considered normal, occurring in almost all individuals several times per day.[66] In a study of 14 healthy individuals, 1.2 ml/10 min gas leakage from the stomach to the esophagus whilst lying horizontal and 6.8 ml/10 min while sitting was demonstrated.[67] If odorous, this constitutes the physiologic part of gastroesophageal halitosis.

Pathologic level of gastroesophageal halitosis is said to occur due to i) gastroesophageal reflux disease (GERD), ii) *Helicobacter pylori* related gastritis, or iii) other causes e.g. gastrocolic fistulae, Zenker diverticulum and hypopharyngeal diverticulae.[66] Falcao *et al.* argued that certain GI disorders can cause taste disturbance. Taste receptor cells are associated with lingual papillae, but also present on the palate, epiglottis and upper esophagus. Low intensity acid reflux can cause phantom taste sensations, which may manifest as subjective halitosis.[16]

Evidence for GERD-related halitosis is contradictory. Some studies report self reported/subjective halitosis complaints are associated with GERD.[68-71] One study reported gastroesophageal pathology in >50% of patients complaining of halitosis,[72] whilst others report that GI disorders may account for up to 5% of objective halitosis complaints.[31] A systematic review investigated the relationship between GERD and halitosis (among other things). Three studies were included, and the authors concluded halitosis is a possible extra-esophageal symptom of GERD,[73] however 2

of these studies utilized questionnaires (i.e. subjective halitosis). Yoo *et al.* report *H. pylori* infection correlated with elevated VSC in mouth and mucosal erosions ,[74] posing halitosis as a potential biomarker to distinguish between erosive (200 ppb) and (75 ppb) non-erosive GERD.[75] Gas chromatography on gastric juice and biopsies in these subjects found resolved 7.5 ppm significantly higher H_2S and expression of VSC-releasing enzymatic activity in the erosive group, and 0.5 ppm in the non-erosive group.[74] However, another study reported no significant difference in halitosis parameters when comparing erosive and nonerosive GERD.[76] Others have suggested the stomach rarely causes halitosis,[10] gastroscopy in halitosis patients is entirely unnecessary,[12] as the findings do not correlate with halitosis.[77] It has also been argued that there is no evidence that odorous substances are formed in the stomach.[12] Another study reported no statistically significant difference in the prevalence of halitosis symptoms between children with GERD and those without.[78]

H. pylori infection also has a controversial role. *H pylori* possesses a strain-dependent ability to synthesize H_2S and MM from combined cysteine-methionine substrate *in vitro*.[79] Elevated levels of both hydrogen cyanide and hydrogen nitrate were detected on the breath of *H. pylori* infected patients compared to healthy controls,[80] however whether this represents type 3 or type 4 (blood-borne) halitosis is unknown.

Oral *H. pylori* colonisation without gastritis may cause Type 1 (oral) halitosis. PCR detected *H. pylori* in 6.4% (21/326) of saliva samples from non-dyspeptic individuals complaining of halitosis. *H. pylori* was associated with higher MM concentration.[81]

Improvements in halitosis (defined by various methodologies) following eradication therapy are reported.[82-87] Positive correlation between *H. pylori* and halitosis is reported by some studies,[88-90] however some of these can be criticized for relying on self reported halitosis rather than semi-objective breath analysis.[91] Others report no statistically significant correlation.[69,77,91-93]

This mechanism is rarely responsible for halitosis, but cannot presently be dismissed due to several studies which support the idea that GI disease may cause halitosis.

Type 4 halitosis: *Blood-borne halitosis*

Type 4 (blood-borne) halitosis is where volatile chemicals in the systemic circulation can transfer to exhaled breath during alveolar gas exchange and cause halitosis.[94]

Volatiles are endogenously produced, mostly by-products of biochemical metabolic processes.[11] The concentration of volatiles on the exhaled breath reflects their respective arterial concentrations,[24] Healthy subjects' breath contains 3481 VOCs,[95] constituting the phsyologic aspect of Type 4 halitosis (Table 2).

Methylated or low carbon containing alkanes, cyclic hydrocarbons, alcohols and aldehydes have an especially pungent odor when they exceed specific odor thresholds for the individual or his/her social environment, constituting pathologic Type 4 halitosis.

Table 2: Example aromatic gases exhaled in healthy individuals

Breath gas	Normal level (ppb)	Ref#	Associated with
Ammonia	833 422-2390 688	[25] [96] [97]	Protein or amino acid metabolism, nitrogen metabolism
Acetone	477 661.3 462 293-870	[25] [98] [26] [96]	Lipid metabolism
Methanol	461 32-1684	[25] [99]	Abnormal gut flora, renal or pancreatic insufficiency, carbohydrate malabsorption
Ethanol	112 27-153 184 (7-18 age)	[25] [96] [26]	Bacterial overload in gut
Isoprene	106 117.6 212	[25] [98] [100]	Cholesterol synthesis
Propanol	18 20	[25] [26]	Pancreatic insufficiency
Acetaldehyde	22 10 24	[25] [100] [101]	Alcohol metabolism
Butane	2.4	[102]	Protein oxidation /colonic bacteria
Alkanes C13-C20	1.5 x10 $^{-10}$ M/l	[103]	Oxidative stress
Dimethyl Sulfide	0.2 nMol/l	[12, 104]	Hepatic metabolism
Hydrogen*	<10 ppm	[105]	Carbohydrate metabolism in gut

** Hydrogen is odorless, but its elevation >10 ppm in breath may indicate small intestinal bacterial overgrowth syndrome, ileocaecal valve syndrome, ileitis, or carbohydrate malabsorption/intolerance.[106,107] Along with methane, hydrogen is an indicator gas used in disaccharide malabsorption tests to detect intestinal gases exhaled in breath.[105] Odorous breath gases (type 4 halitosis) are potentially present when breath hydrogen is elevated.[33]*

The threshold concentration for any given chemical depends on the change in intensity (odor strength) with concentration and the odor character. There is also interpersonal variation in emotional reactions to detected odor; some may react positively and others negatively.[108] A single volatile chemical can be perceived at lower concentrations than expected when it is combined in a mixture of thousands of VOC like the breath by interaction with other odorants and collective stimulation of olfactory receptors.

Artificial systems containing chemical sensor arrays for the detection of breath volatiles allow for profile readings of multiple compounds instead of single sensors for a single volatile.[109] This is more favorable as breath odors are not limited to a single or a handful of gasses describable according to their individual threshold levels. Rather, breath odors are "olfactory spectra" of breath. Every exhaled odorant gas should be

suspected as potentially contributing, by varying degrees, to the overall perception of breath odor.

In pathologic type 4 halitosis, the concentrations and profile of exhaled gases is significantly different to those seen in health, depending on the pathology. Exhaled breath volatiles are reported in diabetes mellitus, sleep apnea, *H. pylori* infection, sickle cell disease, asthma, breast cancer, lung carcinoma, chronic obstructive pulmonary disease, cystic fibrosis, liver diseases, cirrhosis, uremia, kidney failure and TMAU.[24,94]

Breath alkanes have pungent odor are elevated in intestinal inflammation,[110] e.g. ulcerative colitis,[111,112], Crohn's Disease,[112] in pulmonary tuberculosis,[113] schizophrenia,[114] pneumonia,[115] asbestos-related disorders,[116] stomach cancer,[117] and angina pectoris.[118] Pregnant females or pre-eclampsia patients have a specific breath gas profile, including undecane, 6-methyltridecane, 2-methylpentane, 5-methyltetradecane and 2-methylnonane.[119] DMS, acetone, 2-butanone and 2-pentanone are reported in liver failure, including cirrhosis.[27]

The “fetor hepaticus” of hepatic failure is largely caused by DMS, not ammonia.[28] Elevated blood DMS (“dimethylsulfidemia”),[28] was reported to be responsible for the majority of cases of blood borne halitosis.[12]

Body odor may accompany Type 4 halitosis as the same volatiles are also excreted during perspiration. This is sometimes termed blood-borne body odor and halitosis. An example is TMAU, a rare condition, classically characterized by fish odor in urine, sweat, and breath.

Another potential blood-borne mechanism may contribute to a halitosis complaint when blood-borne odorants stimulate olfactory receptors via their blood supply.[16,120] Strong olfactory receptor responses can be triggered by intravascular injection of odorants in tracheotomized animals. Such odor perceptions are not occurring by the normal air-borne route, so there may not be measurable halitosis.

Type 5 halitosis: *Subjective halitosis*

Subjective halitosis is a halitosis complaint without objective confirmation of halitosis by others or halitometer readings. Type 5 halitosis can be misdiagnosed if there are measurement errors or transient symptoms.

It can be considered normal for even mentally healthy individuals to worry occasionally about to be having halitosis.[121]

Such halitosis concern can rationally dismissed by most healthy people, who have a degree of psychological resilience which is capable of compensating for stressors. This normal level of concern for halitosis constitutes the “physiologic” aspect of Type 5 halitosis.

Pathologic subjective halitosis can be categorized as psychologic or neurologic.

Psychologic causes

Psychologic factors can cause subjective halitosis. This is termed monosymptomatic hypochondriacal psychosis,[16] a type of obsessive-compulsive spectrum disorder,[121] or ORS. 75% of ORS patients present with halitosis complaints,[122] but obsession over other non-existent body odors, often in combination, are included. Others’ behavior (e.g. opening windows, sniffing, touching noses etc.) is misinterpreted as evidence of

halitosis. Employment loss, divorce or suicidal ideation are reported.[123] "Doctor shopping" to find clinicians to treat the non-existent odor may occur. However, some report that TMAU or other genuine odor symptoms can be misdiagnosed as ORS.[124]

It may be the case that the previously black and white thinking of objective halitosis on the one hand and psychologic halitosis on the other is oversimplification. Instead, it might be more accurate to consider a spectrum, with entirely subjective halitosis at one extreme and entirely objective halitosis with no psychologic concern at the other. Most patients will fall somewhere between these two points.

When objective halitosis has not been treated, it may cause the patient distress or social isolation, and eventually over-concern about halitosis may develop. Even after the odor is reduced to physiologic levels, the negative psychosocial sequalae may persist, making these cases difficult to treat. Conversely, oversensitivity to physiologic odor may be the basis of a subjective halitosis with no history of objective halitosis.

Neurogenic causes

Traditionally, subjective halitosis complaints are attributed to psychologic factors, but at least some are neurologic. Nearly 200 disorders can cause chemosensory dysfunction (CSD).[16] Dysosmia (disordered olfaction including parosmia and phantosmia) and dysgeusia (disordered gustation) present extensive differential diagnoses.

Olfaction and gustation are intimately interlinked at the neuronal level in the brain. The definition of subjective halitosis (pseudo-halitosis) has been broadened: "the perception of an alteration in the quality of expired odor air, a symptom perceived only by the patient."[16] Many patients fail to distinguish between bad taste and bad odor. Gustatory stimuli may influence orthonasal and retronasal odorant perception.[58]

Side effects of medication, hypothyroidism, hyposalivation (another extensive differential), nutrient deficiency (zinc, copper, iron, and vitamins A and B12), trauma and tumors involving the olfactory center in the brain, or nerve damage (glossopharyngeal, vagus, chorda tympani, olfactory), neurodegenerative diseases (Parkinson's, Alzheimer's and Huntington's disease), environmental pollutants (e.g. smoking), drug abuse, certain oral hygiene products (e.g. mouthwashes) and certain foodstuffs can all be potentially involved in subjective halitosis complaints, by various mechanisms.[16,125] As described previously, diabetes mellitus, GERD and blood-borne stimulation of taste and smell receptors via the blood circulation may also contribute to subjective halitosis.[16,126]

NEW TERMINOLOGY

Unhelpful terms no longer needed:

- Many of the confusing array of synonyms used to describe a psychologic, subjective halitosis complaint (which would fall within type 5 halitosis) are unneeded, including pseudo-halitosis, non-genuine halitosis, delusional halitosis, olfactory obsession, psico-olfactory sensitivity, olfactory depression, halitosis anxiety and imaginary halitosis.
- Subjective, descriptive terms such as sulfurous, ammoniacal, fecal, fishy or

similar should be discontinued since they invite misunderstanding.

Useful terms that are retained:

- **Objective halitosis** refers to any combination of Types 1-4, but should not refer to type 1 (oral) halitosis exclusively.
- **Morning breath** is a temporarily increased physiologic halitosis during sleep and disappears soon after waking.[127] Xerostomia is largely responsible,[128] resulting from diminished salivary and respiratory secretion during sleep, especially when the mouth remains open. Proteinaceous substrates in saliva allows for microbial action, and release of VSC and other volatiles, thereby enhancing Type 1 and 2 halitosis. Increased breath ammonia, acetone,[97,129] and isoprene,[127] occur after overnight fasting. Intestinal gas builds up in the colon during sleep,[130] possibly due to immobility and microbial fermentation of intestinal contents. More type 4 halitosis might result, or possibly, more gas leakage from the gastroesophageal valve (i.e. type 3). All the above mechanisms may operate during sleep. The resultant halitosis upon waking can be termed morning breath, in reality an enhanced form of Type 0 halitosis.
- **Psychosomatic halitosis** should be retained, but the term should not be misused . Some hypothesize that anxiety enhances oral VSC production.[131] This mechanism is correctly termed psychosomatic, since a physical symptom is being influenced by psychologic factors. This is the uniquely correct usage of the term “psychosomatic halitosis”, rather than previous meanings (see “Previous terminology”).
- **Self halitosis** has been used to describe a lack of objective halitosis even though the patient believes themselves to have halitosis,[32] but it is better used to define endogenously produced, self perceived odor, which is not a detectable odor to others. By true description, self halitosis appears in three forms: retronasal olfaction, olfactory receptor responses triggered by blood-borne odorants, and phantom tastes/odors.
- **Halitophobia** should be retained with correct meaning. It refers to "fear of having halitosis" but not "untreated halitosis".

New terms

- **Exogenous odor** results from consumption of aromatic foodstuffs (e.g. garlic, onion, spicy foods), beverages (e.g. alcohol) or tobacco. Exogenous volatiles may released transiently from residues of food or drink in the mouth, or released unchanged via the blood-borne mechanism after being absorbed. Such odors are distinguished from pathologic halitosis, e.g. garlic smells like garlic. The terms garlic odor, spice odor, etc. seem suitable. Dietary or temporary halitosis are also terms that could be argued to be useful.
- Halitosis is an **endogenous odor** because it is produced in the body

DISCUSSION

The new definition places less importance on organoleptic examination and single occasion halitometric reading, and instead places more emphasis on the declarations of the patient and his/her social environment. The reasoning for this follows.

Organoleptic examination

Organoleptic measurement is carried out by smelling the patient's breath then scoring the level of halitosis.[7] However, the examiner does not smell a pure sample of mouth air, but rather a mixture of mouth air and alveolar air. The organoleptic examination does not distinguish between these, only subjectively assesses the overall odor level.

The perception of odorants depends upon several factors, including constant fluctuations in the clinician's individual threshold level for that specific odor, what was last smelled before the examination, the concentration and volatility of the molecules themselves, room temperature (gases are less volatile in lower temperatures), humidity of exhaled breath, how strongly the breath is blown into the examiner's nose (less forcefully expired breath will consist of less volume of air, and less odorant molecules will be carried to the examiner's olfactory epithelia), and lastly the examiner's concentration at that moment. All these parameters vary from one hour to the next and from one individual to the next, making this a subjective measure which does not reflect the actual level of odor. It can be suggest to self applied organoleptic scoring (self assessment) should be evaluated to monitorize prognosis.

Organoleptic examination is problematic,[132] and objectionable to both dentists and to patients. Dislike or shame is experienced by 50% of patients with this examination (n=283).[133] Some use a privacy screen to prevent the patient from seeing the examiner during the examination.[7] Examiners find it repulsive to smell a halitosis patient's breath.

Self detection of halitosis correlated positively with actual halitosis only when subjects smelt their own saliva isolated from their mouth. Other methods did not correlate.[134] Another study reported less correlation between self detection of halitosis and clinical findings. The sensitivity and specificity of self-perceived oral malodor were 47.2% and 59.2%, respectively.[135] The same author later compared 252 halitosis patients' self-estimation, organoleptic and halitometric results and found that self-estimated corresponded significantly with clinical oral malodor.[136]

Halitometers

Gas chromatography (GC), alone or combined with mass spectrometry (MS), is most frequently utilized for highly sensitive VSC detection (1-10 ppb). Nevertheless, routine application of these clinically is impractical given the expense and complexity, and the expertise required.[94] More practical methods utilize colorimetric hydrogen sulfide sensors engineered both as an optical fibre, capable of measuring reflectance change of an immobilized reagent,[137] and as thin reactive films of chromophores.[138] A bio-electronic nose capable of detecting the oxygen consumption induced by an enzymatic reaction with methyl sulfide has also been developed.[139] The Halimeter,[140] contains an

electrochemical sensor for VSC. The semiconductor gas sensors Breathtron,[141] constructed as a zinc oxide film with specificity to hydrogen sulfide and mercaptans.[142] The GC-based OralChroma,[143] is portable equipment capable of determining combined H_2S, MM and DMS levels, with a 10 min response time and a detection limit of a few ppb. Twin Breasor,[144] Diamond Probe/Perio 2000,[145] Cyranose 320,[146] and B/B Checker,[147] are portable devices for detecting several gases including VSC and other odorous gases in mouth or breath air.[148,149] Their accuracy is poor compared to GC and MS. They cannot distinguish one odor from another, and they have difficulty distinguishing individual compounds from the family of VSC.[132]

Almost all halitosis researchers and specialists use portable sulfide monitors (e.g. Halimeter) to detect oral VSC.[14] Good correlation exists between Halimeter readings and VSC concentration,[35] and sulfur-producing bacteria levels.[150] However, Halimeter readings are imprecise, and misdiagnosis may result.[151] The Halimeter has biexponential response to a constant concentration of VSC. Rapid (peak) and slow (plateau) responses differed. The total VSC in air samples was 2.7 times greater than at its peak concentration, but its plateau phase measurement is 25% greater than the actual concentration. A modified protocol measuring plateau instead of peak values is available, yielding more favorable correlation with the actual level of VSC.[152]

In order to investigate the Halimeter's ability to distinguish between VSC and other gases, having calibrated the Halimeter to ambient air, the aspirating tube was inserted into the headspace of some 250 ml commercial juice cartons immediately after opening. The Halimeter readings for apricot, apple, peach, cherry juices, buttermilk, soda, were 114, 352, 91, 48, 39, 47 ppb VSC respectively. In a similar experiment, the Halimeter reported VSC as if H_2S is emitted from various flowers: daffodil, rose, jasmine were 255, 42, 73 ppb while 104 ppb was read near a sump, and 417 ppb near hand soap. When using another gas detector in the same conditions, all these flowers read with different percentages of VOC, not VSC.[153] Such simple experiments show that the Halimeter seems to confuse VSC with other odorants, and may not be selective enough for halitosis. The OralChroma gives more comprehensive VSC level readings than the Halimeter,[41] but it shares the VSC exclusivity limitation, and therefore cannot fully determine the actual level of breath odor due to potential minor contributions from non-VSC gases.

New gas detectors capable of detecting sulfur and nitrogen containing gases, as well as VOCs should be developed for use in halitosis detection. There are industrial, portable gas detectors that are capable of detect more than 4 gas groups including VSC, NH_3, or VOC that could potentially be utilized at one reading. A sensor system for monitoring the simple gases H_2, CO, H_2S, NH_3, VOC and ethanol,[154] and breath test kits including instruments to detect breath H_2 and methane are available.[155]

Perturbation on threshold of halitosis

There is no consensus regarding what VSC reading corresponds to clinically present halitosis (see Table 3).

VSC thresholds should be revisited to improve clinical utility.[163] According to the

new concepts described in this paper, there is no need to establish any precise VSC level which constitutes a halitosis diagnosis. Any patient complaining of halitosis is at least Type 5, even if objective halitosis is not diagnosed. Treatment should be targeted reducing patholgic halitosis to physiologic halitosis. Setting the goal at zero odor is unrealistic and arguably impossible.

Table 3. Variation in the "halitosis threshold" reported in the literature

Halitosis threshold (VSC ppb)	**Reference**
75	[156]
100 (2*)	[7,31,157,158]
110	[12]
125	[40]
150	[14,35,94,159]
250 (3*)	[81,160,161]
Total: VSC 250	[81]
H_2S, CH_3SH, $(CH_3)_2S$: 150, 50, 20 respectively (3*)	

Besides these data, some describe ranges of VSC readings, e.g.: 0-40, healthy; 41-60, physiologic; 61-80, slight; 81-110, moderate; 111-140, severe; over 141, very strong.[162]
** organoleptic score*

There are three reasons to restrict the use of halitometers. Firstly, since baseline mouth air VSC concentrations fluctuate throughout day,[151] halitometric reading at any particular time may not be representative. For this reason, multiple halitometric examinations carried out at different times throughout the day may be more representative compared to a single occasion in the dental clinic. Or cysteine challenge,[164] should be applied to decide optimal VSC level of that individual.

Secondly, popular, portable halitometers are simply sulfide detectors, capable of detecting only VSC. However, nonsulfurous gases are also present in the mouth or breath, albeit in a lesser concentration than VSC. Halitometers are poor at distinguishing one odor from another. E.g., in TMAU, the breath could be malodorous due to the presence of TMA, and VSC levels may be under the normal range in such patients. Thus, examiners may misdiagnose some objective halitosis cases as if subjective halitosis by relying entirely on the specifity of halitometers.

Thirdly, there is no scientifically accepted quantitative threshold between physiologic odor and pathologic halitosis.

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