Breath odor or halitosis denotes any type of disagreeable scent felt on a person’s breath during exhalation and speech. These odors have many different causes and may originate from various locations such as the oral cavity, nasal cavity, upper respiratory tract, and lungs.

According to research performed in multidisciplinary breath clinics (involving professionals from various fields: dentistry, E.N.T., internal medicine, and psychology) in various centers, some 90% of breath odors originate from within the oral cavity itself (Table 2.1). This condition in which the malodor originates from the mouth is commonly known as oral malodor (also termed: Fetor oris or Feotor ex ora).

The potential loci for malodor production within the oral cavity include the posterior portion of tongue’s dorsum, subgingival areas (e.g., periodontal pockets and interdental spaces), faulty restorations (e.g., leaking crowns and bridges), dental implants, dentures, and abscesses. Furthermore, transient oral dryness brought about by a temporal reduction in saliva flow plays an important part in promoting this condition.

Breath odors from the mouth are most commonly measured directly by human odor judges, or indirectly, based on the levels of volatile sulfide compounds (VSC) within the oral cavity (for further details, see Chap. 8).

**Oral Malodor and the Tongue**

The data presented in Table 2.1 clearly demonstrate that the tongue is by far the most common source for malodor production within the oral cavity. The posterior portion of the tongue’s dorsum, where most malodor originates, is often covered by a layer of debris comprising cellular (bacteria, desquamated epithelial cell, white blood cells) and noncellular components (especially proteins from saliva, postnasal, and gingival secretions). This layer termed “tongue coating” may vary in size, thickness, and color among individuals depending on oral activity (e.g., eating, drinking, smoking), oral hygiene, and oral health-related parameters (e.g., presence of periodontal disease); (Yaegaki and Sanada 1992a, b) (Fig. 2.1).

Over the last four decades, various measuring techniques have been suggested for tongue coating evaluation and quantification, taking into account various parameters such as coating thickness, coating area, and discoloration. Some of these methods are summarized in Table 2.2.
<table>
<thead>
<tr>
<th>Reference</th>
<th>Patients population</th>
<th>Confirmed breath odor problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quirynen et al. (2009)</td>
<td>$N = 2,000$ (1,078 F)</td>
<td>$N = 1,687$ (84.3%)</td>
</tr>
<tr>
<td>Age 2–90 year (39.2 ± 14.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Oral causes</td>
<td>Non oral causes</td>
</tr>
<tr>
<td></td>
<td>$N = 1,515$ (89.8%)</td>
<td>$N = 97$ (5.7%)</td>
</tr>
<tr>
<td></td>
<td>Tongue coating (TC)</td>
<td>ENT tonsilitis</td>
</tr>
<tr>
<td></td>
<td>$N = 868$ (51.4%)</td>
<td>Rhinitis</td>
</tr>
<tr>
<td></td>
<td>Gingivitis (G)</td>
<td>Sinusitis</td>
</tr>
<tr>
<td></td>
<td>$N = 75$ (4.4%)</td>
<td>$N = 4$ (0.2%)</td>
</tr>
<tr>
<td></td>
<td>Periodontitis (P)</td>
<td>Nose obstruction</td>
</tr>
<tr>
<td></td>
<td>$N = 148$ (8.7%)</td>
<td>$N = 8$ (0.4%)</td>
</tr>
<tr>
<td></td>
<td>Combination (TC/G/P)</td>
<td>Extra oral: GI tract</td>
</tr>
<tr>
<td></td>
<td>$N = 363$ (21.5%)</td>
<td>$N = 26$ (1.5%)</td>
</tr>
<tr>
<td></td>
<td>Xerostomia</td>
<td>TMAU</td>
</tr>
<tr>
<td></td>
<td>$N = 50$ (2.9%)</td>
<td>$N = 1$ (0.05%)</td>
</tr>
<tr>
<td></td>
<td>Dental</td>
<td>Systemic</td>
</tr>
<tr>
<td></td>
<td>$N = 7$ (0.4%)</td>
<td>$N = 5$ (0.2%)</td>
</tr>
<tr>
<td></td>
<td>Candida</td>
<td>Medication</td>
</tr>
<tr>
<td></td>
<td>$N = 4$ (0.2%)</td>
<td>$N = 2$ (0.1%)</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td>Hormonal</td>
</tr>
<tr>
<td></td>
<td>$N = 15$ (0.8%)</td>
<td>$N = 2$ (0.1%)</td>
</tr>
<tr>
<td></td>
<td>Oral-non oral combination</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$N = 75$ (4.3%)</td>
<td>ENT + Oral:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GI + Oral:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$N = 42$ (2.4%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$N = 33$ (1.9%)</td>
</tr>
</tbody>
</table>

| Delanghe et al. (1996)     | $N = 260$ (135 F)   | $N = 246$ (94.6%)              |
| Age 2–77 year (36 ± 13.5) |                     |                                |
|                            | Oral causes        | Non oral causes                |
|                            | $N = 225$ (91.4%)   | $N = 21$ (8.5%)                |
|                            | Tongue coating     | ENT:                           |
|                            | $N = 92$ (37.3%)    | Chr. tonsilitis               |
|                            | Gingivitis         | $N = 15$ (6%)                 |
|                            | $N = 70$ (28.4%)    | Chr. sinusitis                 |
|                            | Periodontitis      | $N = 4$ (1.6%)                 |
|                            | $N = 63$ (25.6%)    | Foreign bodies                 |
|                            |                     | $N = 1$ (0.4%)                 |
|                            |                     | Rhinitis                       |
|                            |                     | $N = 1$ (0.4%)                 |
Table 2.1 (continued)

<table>
<thead>
<tr>
<th>Reference</th>
<th>Patients population</th>
<th>Confirmed breath odor problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seemann et al. (2006)</td>
<td>N = 407 (204 F) Age 6–76 year (41.5 ± 13.8)</td>
<td>N = 293 (72%)</td>
</tr>
<tr>
<td></td>
<td>Oral causes</td>
<td>Non oral causes</td>
</tr>
<tr>
<td></td>
<td>N = 272 (92.7%)</td>
<td>N = 22 (7.3%)</td>
</tr>
<tr>
<td></td>
<td>Tongue coating (“physiologic”)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 175 (59.7%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>“Pathologic”</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 97 (33%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Periodontitis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 80 (27.3%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gingival hyperplasia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 10 (3.4%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Faulty restorations</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 6 (2%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ENT: Chr. tonsillitis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 15 (5.1%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chr. sinusitis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 2 (0.6%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Foreign bodies</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 2 (0.6%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Systemic: diabetes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 2 (0.6%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Smokers breath</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N = 3 (1%)</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 2.1 Typical photo of a mild tongue coating covering the posterior third of the tongue dorsum
Table 2.2  Tongue coating measurements

<table>
<thead>
<tr>
<th>Reference</th>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gross et al. (1975)</td>
<td>0</td>
<td>No coating</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>Slight coating</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Moderate coating</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>Heavy coating</td>
</tr>
<tr>
<td>Yaegaki and Sanada (1992a)</td>
<td></td>
<td>Scrupling off and weighing the tongue coating.</td>
</tr>
<tr>
<td>Miyazaki et al. (1995)</td>
<td>0</td>
<td>None visible</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>&lt;1/3 tongue dorsum surface covered</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>&lt;2/3 tongue dorsum surface covered</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>&gt;2/3 tongue dorsum surface covered</td>
</tr>
<tr>
<td>Mantilla Gomez (2001)</td>
<td>Discoloration</td>
<td>Pink</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>Pink</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>White</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Yellow/light brown</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>Brown</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>Black</td>
</tr>
<tr>
<td></td>
<td>Thickness</td>
<td>No coating</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>No coating</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>Light-thin coating</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Heavy-thick coating</td>
</tr>
<tr>
<td>Oho et al. (2001)</td>
<td>Area</td>
<td>Area score × thickness score = tongue coating (range 0–6).</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>No tongue coating</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>&lt;1/2 tongue dorsum surface covered</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1/2–3/2 tongue dorsum surface covered</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>&gt;3/2 tongue dorsum surface covered</td>
</tr>
<tr>
<td></td>
<td>Thickness</td>
<td>No tongue coating</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>No tongue coating</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>Thin tongue coating (papillae visible)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Thick tongue coating (papillae invisible)</td>
</tr>
<tr>
<td>Winkel et al. (2003)</td>
<td>(Six areas grid)</td>
<td>Tongue dorsum is divided into six areas (i.e. three posterior and three anterior)</td>
</tr>
<tr>
<td></td>
<td>Coating</td>
<td>No coating</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>No coating</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>Light coating</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Severe coating</td>
</tr>
<tr>
<td></td>
<td>Discoloration</td>
<td>No discoloration</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>No discoloration</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>Light discoloration</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Severe discoloration</td>
</tr>
<tr>
<td></td>
<td>Score is calculated by adding all six scores (range 0–12)</td>
<td></td>
</tr>
<tr>
<td>Kim et al. (2009)</td>
<td>Tongue coating area</td>
<td>Calculated from digital images obtained by the digital tongue imaging system (DTIS)</td>
</tr>
</tbody>
</table>
Large epidemiological studies on the prevalence of oral malodor and its related parameters in the general population have been conducted in Japan (Miyazaki et al. 1995), China (Liu et al. 2006), and Switzerland (Bornstein et al. 2009). Significant associations have been reported comparing oral malodor with the level of tongue coating (Table 2.3). Furthermore, the level of malodor-related compounds (e.g., sulfide-containing compounds) produced on the posterior portion of the tongue dorsum were highly correlated with the overall mouth odor as measured by a human odor judge \((r=0.77, p<0.01)\), as well as volatile sulfide levels \((r=0.63, p<0.01)\) (Morita et al. 2001).

Various studies comparing the tongue parameters of subjects with and without oral malodor showed that subjects with oral malodor had significantly greater tongue coating as compared with the no malodor controls (Haraszthy et al. 2007; Oho et al. 2001; Washio et al. 2005). Furthermore, mechanical removal of tongue coating results in a substantial decrease in oral malodor and its components (Yaegaki and Sanada 1992a). Research done by Tonzetich and Ng (Tonzetich and Ng 1976) showed that tongue brushing was very effective in reducing over 70% of the volatile sulfides as compared with about 30% reduction that resulted from tooth brushing.

**Table 2.3  Correlations among malodor related parameters**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Malodor related parameters</th>
<th>Correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miyazaki et al. (1995) ((n = 2,672))</td>
<td>Tongue coating(^a) vs. sulfide monitor</td>
<td>Spearman correlation</td>
</tr>
<tr>
<td></td>
<td>Periodontal condition(^b) vs. sulfide monitor</td>
<td>(r = 0.44^* - 0.57^*)</td>
</tr>
<tr>
<td></td>
<td>Plaque index(^c) vs. sulfide monitor</td>
<td>(r = 0.13^* - 0.31^<em>) ((^</em> p &lt; 0.001))</td>
</tr>
<tr>
<td>Liu et al. (2006) ((n = 2,000))</td>
<td>Tongue coating(^1) vs. odor judge</td>
<td>Pearson correlation</td>
</tr>
<tr>
<td></td>
<td>Periodontal condition(^4) vs. odor judge: Calculus</td>
<td>(r = 0.15^* - 0.24^*)</td>
</tr>
<tr>
<td></td>
<td>Pocket depth</td>
<td>(r = 0.04^NS - 0.21^*)</td>
</tr>
<tr>
<td></td>
<td>Bleeding index</td>
<td>(r = 0.02^NS - 0.25^*)</td>
</tr>
<tr>
<td></td>
<td>Plaque index(^c) vs. sulfide monitor</td>
<td>(r = 0.08^NS - 0.21^*)</td>
</tr>
<tr>
<td></td>
<td>Tongue coating(^1) vs. odor judge: Calculus</td>
<td>(r = 0.20^* - 0.31^*)</td>
</tr>
<tr>
<td></td>
<td>Pocket depth</td>
<td>(r = 0.20^* - 0.29^*)</td>
</tr>
<tr>
<td></td>
<td>Bleeding index</td>
<td>(r = 0.17^* - 0.31^*)</td>
</tr>
<tr>
<td></td>
<td>Plaque index(^c) vs. odor judge</td>
<td>(r = 0.12^* - 0.22^<em>) ((^NS\text{-non significant, }^</em> p &lt; 0.01))</td>
</tr>
<tr>
<td>Bornstein et al. (2009) ((n = 419))</td>
<td>Tongue coating(^c) vs. sulfide monitor</td>
<td>Linear regression</td>
</tr>
<tr>
<td></td>
<td>Tongue coating(^c) vs. odor judge</td>
<td>4.29</td>
</tr>
<tr>
<td></td>
<td>Periodontal condition(^4) vs. odor judge</td>
<td>2.35</td>
</tr>
</tbody>
</table>

\(^a\)Tongue coating area: 0 = none, 1 = less than 1/3, 2 = less than 2/3, 3 = more than 2/3

\(^b\)According to WHO guidelines (CPITN)

\(^c\)Silness and Löe (1964)

\(^d\)Periodontal screening index (PSI)

\(^0\) = no coating, 1 = light coating (10%), 2 = moderate (10–50%), 3 = severe (>50%)
Tongue coating samples can be obtained by scraping the posterior portion of the tongue’s dorsum using wooden spatula, plastic spoons, tooth brushes, swabs, or gauze pads (Grapp 1933). These samples very often release a putrid malodor very similar in character to oral malodor. Sampling the tongue coating using a plastic spoon (i.e., “spoon test”; (Rosenberg 1996) and scoring the malodor emanating from the spoon yielded highly significant associations with the oral malodor scores (i.e., odor judge) as well as other oral malodor-related parameters (Table 2.4).

### Table 2.4 Correlations between tongue malodor scores and malodor related parameters

<table>
<thead>
<tr>
<th>Reference</th>
<th>Malodor related parameters</th>
<th>Correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kozlovsky et al. (1994)</td>
<td></td>
<td>Pearson correlation</td>
</tr>
<tr>
<td>(n = 52)</td>
<td>Tongue malodor&lt;sup&gt;a&lt;/sup&gt; vs. oral malodor&lt;sup&gt;b&lt;/sup&gt;</td>
<td>r = 0.73, p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Tongue malodor vs. sulfide monitor&lt;sup&gt;c&lt;/sup&gt;</td>
<td>r = 0.38, p = 0.003</td>
</tr>
<tr>
<td></td>
<td>Tongue malodor vs. pocket depth&lt;sup&gt;d&lt;/sup&gt;</td>
<td>r = 0.47, p = 0.005</td>
</tr>
<tr>
<td></td>
<td>Tongue malodor vs. GI&lt;sup&gt;e&lt;/sup&gt;</td>
<td>r = 0.47, p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Tongue malodor vs. PI&lt;sup&gt;f&lt;/sup&gt;</td>
<td>r = 0.38, p = 0.003</td>
</tr>
<tr>
<td>Bosy et al. (1994)</td>
<td></td>
<td>Pearson correlation</td>
</tr>
<tr>
<td>(n = 127)</td>
<td>Tongue malodor&lt;sup&gt;a&lt;/sup&gt; vs. oral malodor&lt;sup&gt;b&lt;/sup&gt;</td>
<td>r = 0.55, p &lt; 0.01</td>
</tr>
<tr>
<td></td>
<td>Tongue malodor vs. sulfide monitor&lt;sup&gt;c&lt;/sup&gt;</td>
<td>r = 0.40, p &lt; 0.01</td>
</tr>
<tr>
<td></td>
<td>Tongue malodor vs. GI&lt;sup&gt;e&lt;/sup&gt;</td>
<td>r = 0.23, p &lt; 0.01</td>
</tr>
<tr>
<td>Greenstein et al. (1997)</td>
<td></td>
<td>Pearson correlation</td>
</tr>
<tr>
<td>(n = 123)</td>
<td>Tongue malodor&lt;sup&gt;a&lt;/sup&gt; vs. oral malodor&lt;sup&gt;b&lt;/sup&gt;</td>
<td>r = 0.40, p &lt; 0.001</td>
</tr>
<tr>
<td>Sterer et al. (2002)</td>
<td>Tongue malodor&lt;sup&gt;a&lt;/sup&gt; vs. oral malodor&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Spearman correlation</td>
</tr>
<tr>
<td>(n = 64)</td>
<td>Odor judge 1</td>
<td>r = 0.63, p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Odor judge 2</td>
<td>r = 0.69, p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Tongue malodor vs. sulfide monitor&lt;sup&gt;c&lt;/sup&gt;</td>
<td>r = 0.26, p = 0.036</td>
</tr>
<tr>
<td></td>
<td>Odor judge 1</td>
<td>r = 0.38, p = 0.002</td>
</tr>
<tr>
<td></td>
<td>Odor judge 2</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Tongue coating malodor scored by an odor judge (0–5)
<sup>b</sup>Whole mouth malodor scored by an odor judge (0–5)
<sup>c</sup>Whole mouth ppb sulfide equivalents, Halimeter™
<sup>d</sup>Mean probing depth
<sup>e</sup>Gingival index (Löe and Silness 1963)
<sup>f</sup>Plaque index (Silness and Löe 1964)

Tongue coating samples can be obtained by scraping the posterior portion of the tongue’s dorsum using wooden spatula, plastic spoons, tooth brushes, swabs, or gauze pads (Grapp 1933). These samples very often release a putrid malodor very similar in character to oral malodor. Sampling the tongue coating using a plastic spoon (i.e., “spoon test”; (Rosenberg 1996) and scoring the malodor emanating from the spoon yielded highly significant associations with the oral malodor scores (i.e., odor judge) as well as other oral malodor-related parameters (Table 2.4).

### Oral Malodor, Gingival Health, and Periodontal Disease

The relationships between oral malodor and gingival or periodontal health are not as straightforward as in the case of the tongue and are the cause of controversy in the literature. Conflicting data from various studies best demonstrate the complexity of this issue (Table 2.5).
Table 2.5 Oral malodor parameters and periodontal disease

<table>
<thead>
<tr>
<th>Reference</th>
<th>Parameters/Criteria</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kostelc et al. (1984)</td>
<td>Measurement of malodor related compounds by gas chromatography in experimental gingivitis ($n = 5$) and controls ($n = 5$)</td>
<td>Significant increase in malodor related compounds in experimental gingivitis</td>
</tr>
<tr>
<td>Rosenberg et al. (1991)</td>
<td>Odor judge vs. pocket depth (No. &gt;5 mm)</td>
<td>Pearson correlation</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. gingival index</td>
<td>$r = 0.184, p = 0.047$</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. gingoal index</td>
<td>$r = 0.107, NS$</td>
</tr>
<tr>
<td></td>
<td>VSC vs. pocket depth (No. &gt;5 mm)</td>
<td>$r = 0.280, p = 0.002$</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. gingival index</td>
<td>$r = 0.099, NS$</td>
</tr>
<tr>
<td></td>
<td>VSC vs. gingival index</td>
<td>$r = 0.081, NS$</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. plaque index</td>
<td>$r = 0.353, p = 0.0001$</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. plaque index</td>
<td>$r = 0.359, p = 0.0001$</td>
</tr>
<tr>
<td></td>
<td>VSC vs. plaque index</td>
<td>$r = 0.373, p = 0.0001$</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. floss odor</td>
<td>$r = 0.381, p = 0.0001$</td>
</tr>
<tr>
<td></td>
<td>VSC vs. floss odor</td>
<td>$r = 0.422, p = 0.0001$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$r = 0.208, p = 0.026$</td>
</tr>
<tr>
<td>Yaegaki and Sanada (1992a)</td>
<td>Probing depth ≥4 mm considered periodontal disease</td>
<td>VSC is significantly higher in periodontal subjects</td>
</tr>
<tr>
<td></td>
<td>Bleeding on probing (% BOP)</td>
<td>VSC is associated with bleeding on probing</td>
</tr>
<tr>
<td>Bosy et al. (1994)</td>
<td>Odor judge vs. pocket depth (No. &gt;5 mm)</td>
<td>Pearson correlation</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. gingival index</td>
<td>$r = 0.11, NS$</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. plaque index</td>
<td>$r = 0.15, NS$</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. floss odor</td>
<td>$r = 0.12, NS$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$r = 0.23, p &lt; 0.01$</td>
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<tr>
<td>De Boever et al. (1994)</td>
<td>Malodor complaint</td>
<td>Differences between groups (ANOVA)</td>
</tr>
<tr>
<td></td>
<td>No complaint</td>
<td>$p &lt; 0.005$</td>
</tr>
<tr>
<td></td>
<td>Odor judge vs. VSC</td>
<td>$p &lt; 0.05$</td>
</tr>
<tr>
<td></td>
<td>Bleeding on probing (% BOP)</td>
<td>$p &lt; 0.005$</td>
</tr>
<tr>
<td>Kozlovsky et al. (1994)</td>
<td>Odor judge vs. mean pocket depth</td>
<td>Pearson correlation</td>
</tr>
<tr>
<td></td>
<td>VSC vs. mean pocket depth</td>
<td>$r = 0.581, p &lt; 0.001$</td>
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<tr>
<td></td>
<td>Odor judge vs. gingival index</td>
<td>$r = 0.305, p = 0.050$</td>
</tr>
<tr>
<td></td>
<td>VSC vs. gingival index</td>
<td>$r = 0.536, p &lt; 0.001$</td>
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<tr>
<td></td>
<td>Odor judge vs. plaque index</td>
<td>$r = 0.298, p = 0.017$</td>
</tr>
<tr>
<td></td>
<td>VSC vs. plaque index</td>
<td>$r = 0.383, p = 0.003$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$r = 0.188, p = 0.093$</td>
</tr>
<tr>
<td>Soder et al. (2000)</td>
<td>Foetor ex ore (severe malodor)</td>
<td>Subject with severe malodor ($n = 41$) had significantly higher probing depth and gingival index scores ($p &lt; 0.001$)</td>
</tr>
<tr>
<td></td>
<td>Probing depth scores (% of teeth with probing depth &gt;5 mm)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gingival index scores (redness swelling and bleeding)</td>
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</tbody>
</table>

(continued)
Many earlier studies addressing this topic suggested a relationship between oral malodor and periodontal disease. Sulser and coworkers (Sulser et al. 1939) claimed that gingivitis and pyorrhea (i.e., periodontitis) are among the main conditions affecting breath odor concentration. Other researchers (Kostelc et al. 1984) found a significant increase in malodor-related compounds in experimental gingivitis. Studies that compared malodor-related parameters in subjects with or without periodontal disease showed that oral sulfide levels were significantly higher in subjects with periodontal pocket depths of 4 mm or more (Yaegaki and Sanada 1992a, b), and that subjects with periodontal disease (pocket depth >3 mm) had significantly higher malodor ratings as scored by an odor judge (Figueiredo et al. 2002). In another study (Soder et al. 2000), subjects with severe oral malodor (Foetor ex ore) had a significantly higher percentage of teeth with periodontal pocket depths of at least 5 mm as well as higher gingival index scores (redness, swelling and bleeding). Furthermore, large studies done in Japan

Table 2.5 (continued)

<table>
<thead>
<tr>
<th>Reference</th>
<th>Parameters/Criteria</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morita and Wang</td>
<td>Odor judge\textsuperscript{a} vs. pocket depth (%(\geq6) mm)</td>
<td>\textit{Pearson correlation}</td>
</tr>
<tr>
<td>(2001) ((n = 81))</td>
<td>VSC\textsuperscript{b} vs. pocket depth (%(\geq6) mm)</td>
<td>(r = 0.371, p = 0.001)</td>
</tr>
<tr>
<td></td>
<td>Odor judge\textsuperscript{a} vs. bleeding on probing (%)</td>
<td>(r = 0.411, p &lt; 0.001)</td>
</tr>
<tr>
<td></td>
<td>VSC\textsuperscript{b} vs. bleeding on probing (%)</td>
<td>(r = 0.489, p &lt; 0.001)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(r = 0.472, p &lt; 0.001)</td>
</tr>
<tr>
<td>Figueiredo et al.</td>
<td>Probing (&gt;3) mm ((n = 21))</td>
<td>Differences between groups (ANOVA)</td>
</tr>
<tr>
<td>(2002)</td>
<td>Probing (\leq3) mm ((n = 20))</td>
<td>(p = 0.001)</td>
</tr>
<tr>
<td></td>
<td>Odor judge\textsuperscript{f}</td>
<td>(p = 0.02)</td>
</tr>
<tr>
<td></td>
<td>VSC\textsuperscript{g}</td>
<td>(p = 0.005)</td>
</tr>
<tr>
<td></td>
<td>Gingival index\textsuperscript{c}</td>
<td>(p = 0.006)</td>
</tr>
<tr>
<td></td>
<td>Plaque index\textsuperscript{d}</td>
<td></td>
</tr>
<tr>
<td>Stamou et al.</td>
<td>Odor judge\textsuperscript{e} vs. probing depth</td>
<td>\textit{Pearson correlation}</td>
</tr>
<tr>
<td>(2005) ((n = 71))</td>
<td>Odor judge\textsuperscript{e} vs. plaque index\textsuperscript{d}</td>
<td>(r = \text{-0.054}, \text{NS})</td>
</tr>
<tr>
<td></td>
<td>Odor judge\textsuperscript{e} vs. gingival index\textsuperscript{c}</td>
<td>(r = 0.185, \text{NS})</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(r = 0.111, \text{NS})</td>
</tr>
<tr>
<td>Tsai et al. (2008)</td>
<td>Odor judge\textsuperscript{e} vs. pocket depth (%(&gt;5) mm)</td>
<td>\textit{Pearson correlation}</td>
</tr>
<tr>
<td>((n = 72))</td>
<td>Odor judge\textsuperscript{e} vs. bleeding on probing (%)</td>
<td>(r = 0.22, \text{NS})</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(r = 0.54, p &lt; 0.001)</td>
</tr>
</tbody>
</table>

\(\text{NS}\) non significant

\textsuperscript{a}Odor judge scores on a scale of 0–5
\textsuperscript{b}Volatile sulfide compounds measured by sulfide monitor (model no. 1170)
\textsuperscript{c}Gingival index (GI; Löe and Silness 1963)
\textsuperscript{d}Plaque index (PI; Silness and Löe 1964)
\textsuperscript{e}Volatile sulfide compounds (VSC) measured by gas chromatography
\textsuperscript{f}Odor judge scores on a scale of 0–4
\textsuperscript{g}Volatile sulfide compounds measured by Halimeter
Oral Malodor, Gingival Health, and Periodontal Disease (Miyazaki et al. 1995), China (Liu et al., 2006), and Switzerland (Bornstein et al. 2009) also found significant correlations comparing oral malodor and periodontal status (Table 2.2).

In contrast, a study by Bosy and coworkers (Bosy et al. 1994) found no association between oral malodor and periodontal disease (pocket depth ≥5 mm), suggesting that these may be two independent conditions. Another more recent study by Tsai and coworkers (Tsai et al. 2008) also failed to show an association between malodor levels and the percentage of teeth with periodontal involvement (pocket depth ≥5 mm). However, the latter did find an association between malodor levels and the percentage of teeth with bleeding on probing.

To complicate matters further, some studies (Yaegaki and Sanada 1992a, b) reported that subjects with periodontal disease (probing depth ≥4 mm) had significantly more tongue coating (four times more wet weight) than the control non-periodontal subjects. These studies further showed that the tongue coating was responsible for production of 60% of the malodor-related volatile sulfide compounds and that these compounds were associated with the percentage of sites exhibiting bleeding on probing.

These data, taken together, imply that with respect to oral malodor production, periodontal disease is less dominant than tongue coating. It is also apparent that the active inflammation process is the main link between gingival and periodontal disease, and malodor production. Typically represented by one of its chief signs (i.e., bleeding on probing or papillary bleeding), active gingivitis or periodontitis, accompanied by bacterial activity, and increased flow of crevicular fluid and blood cells is more relevant to the malodor production process than the presence or depth of periodontal pockets. The inflammatory process may be further exacerbated by the malodorous compounds, which are for the most part toxic and increase tissue permeability and damage (Ng and Tonzetich 1984) (Fig. 2.2).

Fig. 2.2 Gingivitis; swollen bleeding gums showing signs of inflammation (kindly provided by Dr. M. Perez-Davidi)
Oral Malodor and Dental Restorations

As a rule, any appliance, fixed or removable, within the oral cavity, which hinders the common practice of oral hygiene and facilitates plaque accumulation, has the potential to increase oral malodor production.

Dental crowns and bridges are the most common examples of this principle. Dental bridges in particular form an obstacle to maintaining good interdental hygiene by preventing regular flossing. This is also true for orthodontic appliances and splints. Furthermore, faulty restorations, particularly if ill fitted or decemented, may even intensify the problem by allowing bacterial proliferation and accumulation in the inner gaps between the prepared tooth and the restoration, thus forming a reservoir of anaerobic bacteria.

Another such reservoir has been demonstrated in the inner space of the implant-abutment interface (IAI) of osseointegrated dental implants. Research showed that when this interface is situated over 2 mm in depth with respect to the surrounding soft tissue, the inner compartment of the implant harbors significantly more malodor producing bacteria as compared to implants with shallower transmucosal depth (Sterer et al. 2008).

Acrylic appliances such as dentures and obturators are known for their plaque accumulating properties. Their porous nature and increased tendency to adsorb salivary proteins, resulting in bacterial adhesion, turns the acrylic appliance into a bacterial reservoir. Although little research has been carried out on the composition of denture biofilm, in a study performed by Goldberg and coworkers (Goldberg et al. 1997), certain types of malodor-producing bacteria, not normally considered important members of the oral microbiota (i.e., Enterobacteriaceae), were shown to be dramatically increased in the case of denture wearers. Furthermore, when cultivated in the lab, these bacteria produce malodor similar to denture malodor.

Apart from bacterial accumulation and denture contamination, it seems that behavioral aspects also play a part in denture-related malodor. Research done on denture-wearing habits and malodor showed that malodor-related compounds were significantly increased in subjects who did not remove their dentures during the night (Nalcaci and Baran 2008).

Oral Malodor and Oral Dryness

Saliva has an important role in maintaining oral health and many oral functions (e.g., eating, talking). However, comprising 98% water, it also provides suitable moist environment leading to an abundance of microorganisms within the oral cavity.

This mouthful of bacteria produces many by-products and waste as a result of its activity, many of which (described further in Chap. 3) are foul smelling. Some of these bacterial by-products are more volatile than others. However, when there is a decrease in saliva flow and the oral mucosa becomes dry, many more of these compounds and other less volatile compounds can escape the oral surfaces, and become detected on the breath. This and other possible mechanisms (e.g., lack of salivary antibacterial and washing effect, as well as salivary stagnation and degradation of salivary glycoproteins) may explain why
decreasing saliva flow increases the severity of malodor in individual subjects. In a study comparing saliva flow and malodor-related compounds in 147 subjects (Koshimune et al. 2003), lower levels of resting saliva (e.g., without stimulating saliva flow by chewing) were associated with higher levels of malodor-related compounds.

There are some physiological conditions that affect saliva flow. For example, during sleep saliva flow is brought to a halt (Dawes 1972). That is one of the reasons that sleep, especially when accompanied by mouth breathing, may promote oral malodor production.

Normally, saliva flow decreases between meals. Dehydration due to insufficient fluid intake and prolonged fasting may also result in reduced saliva flow.

Another condition that might affect saliva flow is stress. Research by Queiroz and coworkers (Queiroz et al. 2002) showed that stress can cause salivary flow reduction and concomitant increase in malodor-related compounds. Anxiety was also shown to elevate these compounds (Calil and Marcondes 2006).

Many widely used medications such as antihypertensive and antidepressants drugs are known to have reducing effect on saliva flow as an undesirable side effect. The use of such drugs, as well as increased consumption of coffee and alcohol may cause an increase in malodor production as a result of salivary flow decrease (Tschoppe et al. 2010).

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ISBN: 978-3-642-19311-8