The ecology of *Staphylococcus* species in the oral cavity

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Whilst the diversity of organisms present in the oral cavity is well accepted, there remains considerable controversy as to whether *Staphylococcus* spp. play a role in the ecology of the normal oral flora. Surprisingly little detailed work has been performed on the quantitative and qualitative aspects of colonisation or infection either by coagulase-negative staphylococci (CNS) or *S. aureus*. The latter is especially interesting in the light of present difficulties in eradicating carriage of methicillin-resistant *S. aureus* (MRSA) from the oropharynx in affected individuals. This paper reviews the current knowledge of staphylococcal colonisation and infection of the oral cavity in health and disease. *S. aureus* has been isolated from a wide range of infective oral conditions, such as angular cheilitis and parotitis. More recently, a clinical condition classified as staphylococcal mucositis has emerged as a clinical problem in many debilitated elderly patients and those with oral Crohn’s disease. Higher carriage rates of both CNS or *S. aureus*, or both, in patients prone to joint infections raises the interesting possibility of the oral cavity serving as a potential source for bacteremic spread to compromised joint spaces. In conclusion, there is a surprising paucity of knowledge regarding the role of oral staphylococci in both health and disease. Further work in this area may lead to benefits, such as improved decolonisation regimens for eradication of MRSA and acknowledgment of the mouth as a source of bacteraemic staphylococci.

Introduction

Despite the extensive literature on *Staphylococcus aureus* and coagulase-negative staphylococci (CNS), relatively little attention has been paid to the oral cavity as a reservoir for these organisms. The oral flora contains >300 known species of bacteria in addition to numerous non-cultivable organisms which are being discovered as a result of molecular biological techniques [1]. Whilst the importance of staphylococci as medical pathogens has been recognised for many years, the presence of *Staphylococcus* species as a component of the resident oral flora is controversial but, surprisingly, there have been relatively few detailed studies of the distribution of staphylococci in the mouth. Some infections in the circum-oral region are caused, at least in part, by *S. aureus*. These include angular cheilitis [2], some endodontic infections [3–5], osteomyelitis of the jaw [6], parotitis [7,8] and, more recently recognised, a form of oral mucositis in elderly, highly dependent patients receiving parenteral nutrition [9]. Interestingly, there is now a growing body of evidence to suggest that staphylococci can be isolated frequently from the oral cavity of particular patient groups such as children [10], the elderly [9] and some groups with systemic disease such as the terminally ill [11], those with rheumatoid arthritis [12] and patients with haematological malignancies [13]. Of further concern is the observation that the oropharynx is frequently colonised with strains of methicillin-resistant *S. aureus* (MRSA) which may prove difficult to eradicate [14]. Therefore, it is apparent that the oral cavity may represent a hitherto poorly recognised reservoir of staphylococci, some of which may, under appropriate conditions, cause local or systemic infection. There is also the potential for dissemination of oral staphylococcal strains to re-colonise other body sites or as a source of cross-infection to other patients or staff. The aim of this review is to examine the current knowledge of the ecology of staphylococcal species in the oral cavity and their impact on systemic health.

The normal oral flora

The normal oral flora comprises a diverse group of micro-organisms, including bacteria, fungi, protozoa...
and possibly even viruses [15]. More than 300 species inhabit the oral cavity [1], of which about 30 are found routinely and account for the majority of the cultivable strains. These factors, together with the fact that the oral cavity has a wide range of sites with different environmental conditions, make the study of oral microbiology complex and difficult.

Staphylococci in the healthy oral cavity

There are several early qualitative reports of *S. aureus* isolated from the healthy oral cavity but detailed information on the intra-oral distribution of staphylococci is lacking. Most studies sample the oral cavity by use of swabs, rinses or plaque scrapings, although one group [16] found staphylococci in plaque from the fissures of teeth. Percival and colleagues [17] reported the isolation of staphylococci, albeit with low mean percentage counts, from *c.* 12% of supragingival plaque samples in 79 healthy individuals with ages ranging from 27 to 84 years. There were no clear age-related trends in the isolation frequency or proportions of staphylococci in this study. Another study [10] investigating oral staphylococcal carriage in 307 children aged <1–5 years attending a paedodontic department found that 84% carried staphylococcal species, 33% of which were *S. aureus* (5% of the *S. aureus* isolates were MRSA). Of interest was the fact that 19% of the *S. aureus* isolates produced exfoliative toxin and 40% produced enterotoxin [10]. A more recent study [13] found that 64% of healthy children carried *S. aureus* in the oral cavity.

In studies of student populations, oral carriage rates of *S. aureus* have ranged from 17% [18] to 48% [19]. More recently, workers have revealed that between 94% and 100% of healthy adults had oral colonisation with *Staphylococcus* spp. [17, 20, 21] and oral carriage of *S. aureus* ranged from 24% to 36% [20, 21]. The presence of prosthetic devices within the oral cavity, such as acrylic dentures, may encourage carriage of staphylococci [22]. In studies of denture-wearing patients, carriage rates of *S. aureus* varied from 23% to 48% [23, 24].

Staphylococci have been isolated from supragingival plaque (see Table 1 for details) [16, 25, 26] and from subgingival plaque [27–34]. Relatively few plaque samples have been collected from non-diseased sites and, therefore, no definitive interpretation of the available qualitative and quantitative data for staphylococci in health is possible. The species of staphylococci most frequently reported from oral samples are *S. epidermidis* and *S. aureus*, but *S. haemolyticus*, *S. hominis*, *S. warneri*, *S. capitis*, *S. saprophyticus*, *S. xylosus* and *S. simulans* have also been reported [32, 35].

Most investigators employ selective agar (mannitol salt agar, MSA) to identify the presence of *S. aureus* from oral specimens. Very few studies report actual numbers (cfu/ml) of staphylococci in healthy or compromised patients, which makes comparison between different studies difficult. Furthermore, few longitudinal studies have been performed, so that there is little definitive evidence to confirm or refute the general impression that *Staphylococcus* spp. may be transitory members of the oral microflora.

Staphylococci in oral disease

Despite its pathogenic potential, *S. aureus* is only infrequently associated with acute dento-alveolar infections [36, 37]. Other oral infections from which *S. aureus* has been cultured include infected jaw cysts [38], oral mucosal lesions [23, 39] and denture-induced

<table>
<thead>
<tr>
<th>Patient group</th>
<th>Mean age of patient (years)</th>
<th>Number of patients sampled</th>
<th>Number of sites sampled</th>
<th>S. aureus isolation rate <em>n</em> (% isolation frequency)</th>
<th>CNS isolation rate <em>n</em> (% isolation frequency)</th>
<th>Ref. no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tooth fissures</td>
<td>NR</td>
<td>10</td>
<td>1</td>
<td>1 (10%) (80%)</td>
<td>8 (80%)</td>
<td>16</td>
</tr>
<tr>
<td>Healthy dentate adults</td>
<td>27</td>
<td>30</td>
<td>1</td>
<td>2 (7%) (10%)</td>
<td>3 (10%)</td>
<td>17</td>
</tr>
<tr>
<td>Healthy dentate adults</td>
<td>50</td>
<td>23</td>
<td>1</td>
<td>1 (6%) (4%)</td>
<td>1 (4%)</td>
<td>17</td>
</tr>
<tr>
<td>Healthy dentate adults</td>
<td>73</td>
<td>16</td>
<td>1</td>
<td>1 (6%) (2%)</td>
<td>2 (12%)</td>
<td>17</td>
</tr>
<tr>
<td>Healthy dentate adults</td>
<td>84</td>
<td>10</td>
<td>1</td>
<td>0</td>
<td>1 (10%)</td>
<td>17</td>
</tr>
<tr>
<td>Gingivitis</td>
<td>39</td>
<td>18</td>
<td>1</td>
<td>9 (50%) (NR)</td>
<td>11 (52%) (NR)</td>
<td>32</td>
</tr>
<tr>
<td>Periodontal disease</td>
<td>32–66</td>
<td>21</td>
<td>1</td>
<td>11 (52%) (NR)</td>
<td>11 (52%) (NR)</td>
<td>27</td>
</tr>
<tr>
<td>Advanced adult</td>
<td>40–49</td>
<td>72</td>
<td>1</td>
<td>0</td>
<td>16 (22%)</td>
<td>28</td>
</tr>
<tr>
<td>Periodontal disease</td>
<td>50</td>
<td>506</td>
<td>3</td>
<td>253 (50%) (NR)</td>
<td>289 (54%) (NR)</td>
<td>33</td>
</tr>
<tr>
<td>Adult periodontal disease</td>
<td>45</td>
<td>535</td>
<td>2</td>
<td>43 (8%) (289)</td>
<td>289 (54%) (NR)</td>
<td>33</td>
</tr>
<tr>
<td>Early onset periodontal disease</td>
<td>35</td>
<td>108</td>
<td>3</td>
<td>49 (45%) (NR)</td>
<td>49 (45%) (NR)</td>
<td>32</td>
</tr>
<tr>
<td>Localised juvenile periodontitis</td>
<td>18</td>
<td>13</td>
<td>3</td>
<td>4 (31%) (NR)</td>
<td>4 (31%) (NR)</td>
<td>32</td>
</tr>
<tr>
<td>Failing dental implants</td>
<td>58</td>
<td>13</td>
<td>3</td>
<td>9 (60%) (NR)</td>
<td>9 (60%) (NR)</td>
<td>32</td>
</tr>
</tbody>
</table>

NR, not recorded.

*Staphylococci isolated but S. aureus isolation rate not specified.*

Table 1. Summary of studies in which *Staphylococcus* spp. have been isolated from dental plaque.
942  A. J. SMITH, M. S. JACKSON AND J. BAGG

stomatitis [22, 40]. More usually, S. aureus infection is associated with angular cheilitis. Workers have found an isolation rate of 63% for S. aureus from this condition — usually in the presence of other microorganisms such as Candida albicans or Streptococcus pyogenes [41]. Angular cheilitis is thought to be due to a combination of ill-fitting dentures, nutritional deficiencies and infection by S. aureus or C. albicans, or both. Whilst there are few studies on the presence of Staphylococcus spp. in the healthy oral cavity, more has been reported from patients demonstrating ill health. In a study of 110 patients attending a dental hospital with a range of oral diseases [42], there was an observed prevalence of S. aureus in saliva of 21% and from gingival swabs of 11%. Salivary carriage of S. aureus in a cohort of patients with reduced salivary flow rates attending an oral medicine clinic was found in 41% of patients with a range of concentrations from 3.7 × 10³ to 5.2 × 10³ cfu/ml [43].

Oral mucosal infection with S. aureus has recently been incriminated in a severe form of mucositis reported in some groups with systemic disease such as patients with oral Crohn’s disease [44] and geriatric patients [9]. The case for S. aureus in the aetiology of oral mucositis is complicated by the complexity of the normal oral flora and by healthy carriage of S. aureus in some patient groups. However, both clinical and microbiological data lend support to this hypothesis. The clinical presentation of staphylococcal mucositis commonly takes the form of oral discomfort and panoral mucosal erythema which may progress to marked crusting and bleeding of the oral mucosa [9, 11, 44]. Treatment of this condition with appropriate anti-staphylococcal agents leads to marked clinical improvement [9, 44]. The reason for the breakdown in colonisation of the oral mucosa is unclear, although in some groups of patients, notably elderly patients receiving parenteral nutrition and patients with advanced malignant disease, reduced salivary flow rate is an important risk factor. Saliva, in addition to its mechanical cleaning effects, also contains many antimicrobial factors, such as lysozyme and secretory IgA. An alternative hypothesis for the clinical presentation of staphylococcal mucositis includes colonisation by toxin-producing strains of S. aureus. In one study, three of five patients with mucositis were colonised by toxic-shock syndrome toxin (TSST)-1-producing strains, suggesting that heavy colonisation of the oral cavity with toxin-producing strains may cause local mucosal damage [9].

In a study of children with haematological malignancies [13], only 6 (5%) of the 120 children with haematological malignancy carried oral S. aureus compared with 64% of 25 healthy children. It seems likely that the lower levels of S. aureus from children with malignancies are a result of frequent antibiotic treatment. In the same study, a significantly smaller proportion of the children with malignant disease (37%) carried CNS compared with the healthy control children (64%), but levels of carriage are higher than previously reported (Tables 2 and 3).

Rams and colleagues [32] discovered that c. 50% of periodontal lesions harboured staphylococci. Only low proportions (<1%) of staphylococci were present in the cultivable subgingival flora in 95% of the patients studied. No significant differences were detected in the occurrence of S. aureus between patients with gingip-

Table 2. Summary of studies in which Staphylococcus spp. have been isolated from saliva or oral swabs in patients with systemic or oral disease

<table>
<thead>
<tr>
<th>Patient group</th>
<th>Mean age of patient (years)</th>
<th>Number of patients sampled</th>
<th>Number of sites sampled</th>
<th>Specimen(s) collected</th>
<th>S. aureus isolation rate (%)</th>
<th>CNS isolation rate (%)</th>
<th>Ref. no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children with neoplasia</td>
<td>8</td>
<td>125</td>
<td>1</td>
<td>Oral swab</td>
<td>6 (3%)</td>
<td>46 (37%)</td>
<td>13</td>
</tr>
<tr>
<td>Adults with neoplasia</td>
<td>69</td>
<td>197</td>
<td>1</td>
<td>Saliva</td>
<td>50 (25%)</td>
<td>NR</td>
<td>11</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>59</td>
<td>111</td>
<td>4</td>
<td>Saliva and oral swab</td>
<td>16 (14%)</td>
<td>0</td>
<td>63</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>60</td>
<td>25</td>
<td>2</td>
<td>Saliva and oral swab</td>
<td>14 (56%)</td>
<td>10 (40%)</td>
<td>20</td>
</tr>
<tr>
<td>Geriatric unit</td>
<td>70</td>
<td>107</td>
<td>4</td>
<td>Oral swab</td>
<td>20 (10%)</td>
<td>NR</td>
<td>71</td>
</tr>
<tr>
<td>Chronically ill and elderly clinic</td>
<td>NR</td>
<td>40</td>
<td>4</td>
<td>Saliva and oral swabs</td>
<td>12 (36%)</td>
<td>NR</td>
<td>42</td>
</tr>
<tr>
<td>Xenostomia (out-patient clinic)</td>
<td>NR</td>
<td>75</td>
<td>1</td>
<td>Saliva</td>
<td>31 (41%)</td>
<td>NR</td>
<td>43</td>
</tr>
<tr>
<td>Mucositis</td>
<td>68 – 87</td>
<td>5</td>
<td>4</td>
<td>Oral swabs</td>
<td>5 (100%)</td>
<td>NR</td>
<td>9</td>
</tr>
<tr>
<td>Dentate patients with mucosal oral disease</td>
<td>0 – 90</td>
<td>155</td>
<td>1</td>
<td>Curettings and swabs</td>
<td>30 (19%)</td>
<td>NR</td>
<td>23</td>
</tr>
<tr>
<td>Oropharyngeal granulomatosis and Crohn’s disease</td>
<td>NR</td>
<td>450</td>
<td>1</td>
<td>Oral rinse</td>
<td>4 (0.8%)</td>
<td>NR</td>
<td>44</td>
</tr>
<tr>
<td>Denture stomatitis</td>
<td>0 – 90</td>
<td>116</td>
<td>1</td>
<td>Curettings and swabs</td>
<td>27 (23%)</td>
<td>NR</td>
<td>23</td>
</tr>
<tr>
<td>Patients with oral abscess/osteomyelitis</td>
<td>NR</td>
<td>10</td>
<td>1</td>
<td>Oral swab</td>
<td>3 (36%)</td>
<td>7 (70%)</td>
<td>21</td>
</tr>
<tr>
<td>Dental out-patients</td>
<td>NR</td>
<td>80</td>
<td>4</td>
<td>Saliva and oral swabs</td>
<td>40 (50%)</td>
<td>NR</td>
<td>42</td>
</tr>
<tr>
<td>Surgical in-patients</td>
<td>NR</td>
<td>40</td>
<td>4</td>
<td>Saliva and oral swabs</td>
<td>13 (33%)</td>
<td>NR</td>
<td>42</td>
</tr>
</tbody>
</table>

NR, not recorded.
Table 3. Summary of studies in which Staphylococcus spp. have been isolated from saliva or oral swabs in healthy patients

<table>
<thead>
<tr>
<th>Patient group</th>
<th>Mean age of patient (years)</th>
<th>Number of patients sampled</th>
<th>Number of sites sampled</th>
<th>Specimen(s) collected</th>
<th>St. aureus isolation rate n (% isolation frequency)</th>
<th>CNS isolation rate n (% isolation frequency)</th>
<th>Ref. no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy children</td>
<td>11</td>
<td>25</td>
<td>1</td>
<td>Oral rinse and oral swab</td>
<td>16 (64%)</td>
<td>20 (80%)</td>
<td>13</td>
</tr>
<tr>
<td>Healthy children</td>
<td>0–5</td>
<td>307</td>
<td>1</td>
<td>Tongue swab</td>
<td>100 (33%)</td>
<td>157 (51%)</td>
<td>10</td>
</tr>
<tr>
<td>Healthy children</td>
<td>7–8</td>
<td>539</td>
<td>1</td>
<td>Oral rinse</td>
<td>200 (37%)</td>
<td>NR</td>
<td>72</td>
</tr>
<tr>
<td>Students</td>
<td>NR</td>
<td>100</td>
<td>2</td>
<td>Oral swab</td>
<td>27 (27%)</td>
<td>69 (69%)</td>
<td>18</td>
</tr>
<tr>
<td>Dental students</td>
<td>NR</td>
<td>70</td>
<td>2</td>
<td>Saliva</td>
<td>32 (46%)</td>
<td>NR</td>
<td>19</td>
</tr>
<tr>
<td>Healthy dentate adults</td>
<td>27</td>
<td>30</td>
<td>1</td>
<td>Saliva</td>
<td>9 (30%)</td>
<td>9 (30%)</td>
<td>17</td>
</tr>
<tr>
<td>Healthy adults</td>
<td>32</td>
<td>50</td>
<td>2</td>
<td>Saliva and oral swab</td>
<td>12 (24%)</td>
<td>35 (70%)</td>
<td>20</td>
</tr>
<tr>
<td>Healthy dentate adults</td>
<td>56</td>
<td>23</td>
<td>1</td>
<td>Saliva</td>
<td>7 (30%)</td>
<td>17 (70%)</td>
<td>17</td>
</tr>
<tr>
<td>Healthy Adults</td>
<td>56</td>
<td>83</td>
<td>4</td>
<td>Saliva and oral swab</td>
<td>3 (4%)</td>
<td>NR</td>
<td>63</td>
</tr>
<tr>
<td>Healthy adults</td>
<td>NR</td>
<td>22</td>
<td>1</td>
<td>Oral swab</td>
<td>8 (36%)</td>
<td>14 (64%)</td>
<td>21</td>
</tr>
<tr>
<td>Healthy dentate older, wearing adults</td>
<td>65</td>
<td>29</td>
<td>2</td>
<td>Oral swab</td>
<td>14 (48%)</td>
<td>3 (10%)</td>
<td>24</td>
</tr>
<tr>
<td>Healthy elderly</td>
<td>83</td>
<td>27</td>
<td>4</td>
<td>Oral swabs</td>
<td>11 (41%)</td>
<td>NR</td>
<td>9</td>
</tr>
<tr>
<td>Healthy elderly</td>
<td>82</td>
<td>25</td>
<td>2</td>
<td>Saliva and oral swab</td>
<td>9 (36%)</td>
<td>16 (64%)</td>
<td>20</td>
</tr>
<tr>
<td>Healthy dentate elderly</td>
<td>73</td>
<td>16</td>
<td>1</td>
<td>Saliva</td>
<td>7 (44%)</td>
<td>7 (44%)</td>
<td>17</td>
</tr>
<tr>
<td>Healthy dentate elderly</td>
<td>84</td>
<td>10</td>
<td>1</td>
<td>Saliva</td>
<td>3 (30%)</td>
<td>4 (40%)</td>
<td>17</td>
</tr>
</tbody>
</table>

NR, not recorded.

vitis or other more destructive forms of periodontal disease. Some workers [45] have reported elevated proportions of S. sanguis in actively destructive periodontal disease that was non-responsive to treatment. Other workers have isolated staphylococci from periodontitis in healthy persons [46–48], those with diabetes mellitus [47] and acute periodontal abscesses in immunocompromised patients [49]. In early studies [50, 51], cellular components of St. aureus were found in the subepithelial connective tissues of 47% of specimens of inflamed gingival tissue, but not in healthy gingival tissue. However, in cases of failing dental implants, a significant proportion (69%) of the affected sites had high proportions of Staphylococcus spp. (15–100% of total flora) in the associated dental plaque [52]. This association has not been confirmed in other studies [53].

Oral staphylococci as a source of systemic infection

Classically, the mouth is recognised as a source of bacteremia in infective endocarditis, but in studies of organisms isolated from bacteremia associated with dental procedures [54, 55] St. aureus is infrequently detected. Nevertheless, there is an increasing number of reports suggesting that staphylococci from an oral source may cause infection at distant sites [56, 57]. For example, St. lugdunensis endocarditis has been reported after a tooth extraction [56]. These bacteremic episodes may assume greater significance in medically compromised patients. Oral infections have been associated with septicemias among those with hematological malignancy [35, 58]. CNS are frequently isolated from blood cultures in patients receiving treatment for malignant disease [35]. This is commonly a result of colonisation of venous access devices, but in some cases no such source of infection is present. The mouth has not been generally considered to play a role in such infections, but many patients receiving chemotherapeutic drug regimens develop severe oral ulceration (mucositis) as a result of the effect of these drugs on the oral mucosa [59]. Such ulceration provides a portal of entry for organisms in the oral flora, and has already been identified in relation to septicemia caused by oral streptococci in children receiving treatment for leukaemia. This has been demonstrated more recently by a St. epidermidis and Strep. oralis bacteremia in a bone marrow transplant patient [58]. Identical isolates, as analysed by pulsed-field gel electrophoresis (PFGE), of St. epidermidis were found in the mouth and bloodstream, but not at the site of venous access. In many patients with serious staphylococcal infections the original source of the organism is unknown, but the mouth has not usually been sought as a primary focus, as staphylococci are not considered part of the normal oral flora. There has been a long-standing debate over the role of oral micro-organisms in the aetiology of late prosthetic joint infections [60] and the possible need for antibiotic prophylaxis in such patients during dental treatment. Staphylococci are the most important cause of prosthetic joint infections. St. aureus is a significant pathogen in acute septic arthritis, affecting both native and prosthetic joints, whereas St. epidermidis is responsible for a large percentage of late or chronic infections [61]. Such joints are frequently placed in patients with rheumatoid arthritis and the possibility of a bacteremia from an oral source resulting in a prosthetic joint infection should be of considerable interest. At present, the source of staphylococcal joint

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**Staphylococcal Species in the Oral Cavity**

943

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MRSA in the oral cavity

The prevention of horizontal transmission of MRSA has become increasingly important as the prevalence of this pathogen increases. Oral carriage of MRSA may serve as a reservoir for re-colonisation of other body sites or for cross-infection to other patients or healthcare workers. At least two cases have been reported of cross-infection from a general dental practitioner to patients [66]. The practitioner had probably been colonised whilst a patient in hospital. Nursing homes are another important source of colonisation and infection and two cases of acute parotitis caused by MRSA in elderly patients have been described [67].

Attempts are frequently made to eradicate carriage of MRSA from either patients or medical staff colonised by this organism. However, clinical experience has shown that oropharyngeal carriage of MRSA can be difficult to eradicate [14]. Therefore, it is important that consideration be given to the oral cavity if eradication of colonisation by MRSA is clinically appropriate. Mupirocin is rarely effective alone in clearing oropharyngeal colonisation. Successful eradication of throat carriage of MRSA in a health-care worker has been achieved by the use of rifampicin and fusidic acid, in addition to topical mupirocin [68]. However, eradication of throat carriage of MRSA has been achieved with use of topical chlorhexidine (0.2%) in addition to normal control measures of patient isolation, nasal mupirocin and chlorhexidine body washes [69]. Within the oral cavity MRSA may preferably colonise denture surfaces. One group of workers [24] found 10% of unslected denture-wearing patients carrying MRSA on their dentures which proved difficult to eradicate with conventional denture-cleaning agents. In a subsequent study, eradication of the long-term carriage of MRSA from denture-wearing patients was successful only after heat sterilising or remaking the dentures that had become persistently colonised by MRSA [70]. More recently, 19% of an elderly institutionalised veteran population were shown to be colonised by MRSA in the oral cavity, compared with a prevalence of 20% in the nares. Interestingly, 4% of subjects were culture positive for oral MRSA without evidence of nasal carriage [71].

Carriage of MRSA is not restricted to the chronically ill or institutionalised patients. One study [10] showed that a small proportion of children (5 from 307) carried MRSA, a larger proportion than a more recent study (4 from 539) [72]. Another group demonstrated that MRSA clones may colonise the oral cavity of healthy children for relatively long periods of time (5 years), challenging the hypothesis that staphylococci are transient members of the oral flora [73]. Surprisingly little work has been performed on factors affecting oropharyngeal colonisation by MRSA.

Conclusion

Our current knowledge of the role of staphylococci in the ecology of the oral flora in health and disease is incomplete. The majority of workers report the presence or absence of staphylococci using selective agar (MSA), which makes it difficult to compare isolation rates between different studies and different patient groups. Much of the data relates to mucosal swabs and salivary rinses with relatively little information linked specifically to plaque samples that have been both carefully collected and subsequently investigated by appropriate laboratory techniques. There is increasing interest in the oral ecology of opportunistic micro-organisms, which may include MRSA. As micro-organisms growing in a biofilm such as plaque tend to be less susceptible to a range of antimicrobial agents, this could have important implications in diseases caused by these organisms, with regard to the creation of reservoirs of infection, re-infection and treatment failure due to antimicrobial resistance.

Future work in this area may provide clues for more successful eradication of carriage of MRSA strains. Advances in molecular microbiology may provide some interesting insights into the source of bacteraemic staphylococcal infection and these may challenge current concepts of antibiotic prophylaxis in groups of at-risk patients undergoing dental treatment.
The results of this review clearly demonstrate that staphylococcal species can be frequently isolated in the oral flora of children and adults in both health and disease. Further longitudinal studies are required to establish whether this is a transient presence. This also implies that the oral cavity should be considered a potential source of infection for distant sites when other more obvious foci have been eliminated.

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