REVIEW ARTICLE

Plaque-related infections

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A wide range of infections has been described in the human mouth, but by far the most common are the localised, endogenous and polymicrobial infections such as dental caries, various periodontal infections and dento-alveolar abscess. Since much of the recent research in oral microbiology has concentrated on these infections, it is appropriate that this review should be limited to plaque-related diseases. There is a vast literature dealing with the ecology of dental plaque which is outside the scope of this article but interested readers are directed to the review by Marsh and Martin (1984).

Microbiology of dental caries

Introduction

Dental caries is ubiquitous, and while its prevalence is falling in most developed countries, the opposite is true in the underdeveloped countries, where recent changes in diet, especially with respect to carbohydrate content, have occurred. Caries can be described as a chronic infection in which the causative agents are members of the normal commensal flora. Lesions result from the demineralisation of enamel by acids produced by plaque micro-organisms as they metabolise dietary carbohydrates. The earliest clinical appearance of enamel caries is a well-demarcated chalky-white lesion in which the surface continuity of enamel is still intact. This so called “white spot” lesion can heal by remineralisation, but if the environment is unsuited to healing the lesion progresses with the surface becoming roughened and, in time, a cavity is formed. If the lesion is untreated, micro-organisms extend the disease into the dentine and often finally destroy the dental pulp (fig. 1).

The three main factors involved in dental caries are the host, the microflora in the form of supragingival plaque, and the diet. These complex factors can interact in numerous different ways, but only certain patterns of inter-relationships can result in the initiation and progression of carious

lesions (see fig. 2). It is important to realise that the ways in which these factors interact are of prime importance in determining whether an early carious lesion will develop and subsequently heal or progress. Several epidemiological studies have clearly demonstrated a direct relationship between dental caries and the intake of carbohydrate (Rugg-Gunn, 1983). In this review it is possible to give only a brief account of the microbiological factors.

Microbiology

Dental caries does not occur *in vivo* in the absence of micro-organisms in the form of dental plaque. Over the years there has been debate about whether one or more specific bacteria are principally involved in the initiation of caries or if the disease is caused by a non-specific mixture of bacteria. At present, there are several opinions; at one end of the spectrum it is thought that *Streptococcus mutans* is of prime importance in the initiation of caries in most if not all enamel lesions, while at the other extreme the same organism is thought to be no more important than other supragingival plaque bacteria. The evidence advanced to support, or disprove, these opinions is incomplete and it is likely that both may be correct in specific circumstances. Given the extreme variation found in the microbial composition of supragingival plaque collected from the same site in the same mouth at different times, it seems unlikely that the initiation and progression of all carious lesions is associated with identical or even similar plaque. However, some bacteria (*S. mutans*, *Lactobacillus* spp. and *Actinomyces* spp.) seem to be more important than others.

Streptococcus mutans. While there has been a substantial volume of research into the role of *S. mutans* in dental caries during the past 10–15 years, surprisingly few other bacterial species have been investigated to the same depth. This fact may partly explain the apparently overwhelming evidence supporting its prime importance in dental caries.

S. mutans, can be considered as a group name to comprise six different species, (*S. mutans*, *S.*

Fig. 1. Diagram of a human molar tooth with supporting tissues both in health and when affected by dental caries, chronic periodontitis and dento-alveolar abscess.

Fig. 2. The four circles represent the interplay of the aetiological factors in dental caries. All four factors must be acting simultaneously for caries to occur.

sobrinus, *S. cricetus*, *S. ferus*, *S. rattus* and *S. macacae*) and eight serotypes (a–h) (Beighton *et al.*, 1984; Schleifer *et al.*, 1984). *S. mutans* (serotypes c/e/f) and *S. sobrinus* (serotypes d/g) are the species most commonly found in man; serotype c strains are the most frequently isolated.

The evidence linking *S. mutans* with the initiation of dental caries has been reviewed by a number of workers (Hamada and Slade, 1980; Loesche, 1982; Emilson and Krasse, 1985). The main points in the argument are as follows: (i) *S. mutans* is the most

effective oral streptococcus in producing caries in experimental animals, e.g., rats, hamsters and non-human primates. (ii) Significant correlations have been described in human association studies between the presence of *S. mutans* in saliva and dental plaque, and the incidence of dental caries. (iii) *S. mutans* can often be isolated from the tooth surface before the development of a carious lesion. (iv) Immunisation of experimental animals against *S. mutans* antigens significantly reduces the incidence of dental caries. (v) *S. mutans* tends to produce optimal growth at a lower pH than other plaque bacteria (with the exception of lactobacilli) and also attains the critical pH (c. 5.5) for enamel demineralisation more rapidly. (vi) Sugars are rapidly metabolised to lactic and other organic acids. (vii) Extracellular polysaccharide is produced from sucrose which may help to cement members of the plaque microflora to one another and to the tooth surface. (viii) *S. mutans* produces intracellular polysaccharide which is available for use when the supply of dietary carbohydrate ceases.

As it is unlikely that all strains of *S. mutans* possess every property described above it is possible that some strains are much more pathogenic than others. However, there is little evidence to suggest that caries is a truly infectious disease, with pathogenic strains being transmitted *via* droplets or kissing.

Although several cross-sectional and longitudinal studies have supported the role of *S. mutans* in the initiation of caries, the longitudinal reports tend to show that the increase in numbers of *S. mutans*

occurred either at the time of diagnosis or at some later stage (Hardie *et al.*, 1977; Loesche and Straffon, 1979; Loesche *et al.*, 1984). However, there are many problems associated with such studies, e.g. difficulties in diagnosing approximal "white spot" lesions at specific times, problems in obtaining plaque samples from the surface of the developing lesion in fissures or between the teeth free of surrounding plaque, technical problems in the microbiological analysis and enumeration of the plaque microflora, as well as in analysing the resulting data.

Lactobacilli. The most commonly isolated species from oral samples appear to be *L. casei* and *L. fermentum* although few in-depth epidemiological studies have been performed. For many years, lactobacilli were believed to be the causative agents of dental caries. The main evidence supporting this relationship was as follows; (i) high numbers of lactobacilli were present in most carious lesions; (ii) some strains produced caries in gnotobiotic rats; (iii) lactobacilli generally were able to initiate and maintain growth at low pH levels, producing acid in conditions below pH 5.0; and (iv) some strains synthesised both extra- and intra-cellular polysaccharides from sucrose (van Houte, 1980; Harper and Loesche, 1984; Edwardsson, 1986). At present, the general opinion supports the concept that lactobacilli are not usually involved in the initiation of dental caries, but more in the progression of the lesion deep into enamel and dentine. However, the role of lactobacilli in the initiation of dental caries, in at least some cases, cannot be totally discounted.

Actinomyces. *Actinomyces viscosus* has been associated with the development of caries in root dentine which has become exposed to the oral environment due to age or disease-related recession of the gum margin. The lesions are different from enamel caries, in that the calcified tissues are softened without obvious cavitation. They tend to form on the buccal and lingual surfaces close to the gingival margin and slowly progress laterally around the neck of the tooth. Few detailed in-vivo microbiological studies of root surface caries have been reported, with the majority being cross-sectional rather than the more appropriate longitudinal type of study. Evidence for the involvement of *A. viscosus* in root surface caries (Nyvad and Fejerskov, 1982) is based on (i) association studies *in vivo*, and (ii) experimental work with pure cultures *in vitro* as well as in gnotobiotic rodents. Other factors such as the role of diet, and salivary flow rate and constituents, have received little or no detailed study in this disease. Therefore, at

present, the role of *A. viscosus* in root surface caries is uncertain.

Plaque metabolism

Saliva is the main source of nutrition for oral micro-organisms, and although the carbohydrate content of saliva is normally low, levels can increase 1000-fold following a meal. To avoid possible toxic effects and to gain maximum benefit from these high levels of carbohydrate, oral bacteria have developed a number of regulatory mechanisms acting at three main levels; (i) transport of sugar; (ii) the glycolytic pathway; and (iii) the conversion of pyruvate into metabolic end products (Carlsson, 1984).

In most oral bacteria, glucose is metabolised via the Embden-Myerhof pathway, with the production of two pyruvate molecules from each molecule of glucose. The pyruvate can be converted into ethanol, acetate and formate. When excess sugar is present many bacteria (particularly streptococci and lactobacilli) prevent the accumulation of toxic intermediate metabolites in the cell by increasing both their glycolytic rate and drainage of products from the cell, with pyruvate being converted into lactate molecules (Carlsson, 1986). Overall a rapid fall in plaque pH occurs followed by a slow return to the original value in about 1 h. In a low sugar environment, acetic acid predominates and lactate is low, but when plaque is exposed to carbohydrate, a significant increase in lactate occurs often accompanied by a fall in acetate. In addition, small amounts of butyric, formic, propionic and succinic acids may be present (Geddes *et al.*, 1984). Since lactate is both the strongest and major acid produced during plaque metabolism of carbohydrate, it seems likely that it is the most important in enamel demineralisation. However, this is not certain and there is some evidence that at low pH values acetate can attack enamel crystals preferentially (Featherstone and Rodgers, 1981).

Management

In the past, the general approach in the treatment of dental caries was to remove diseased tissue and replace it with an inert restoration. This form of management made no attempt to cure the disease, and the patient often returned 12 months later requiring further fillings due to new or recurrent caries. The modern philosophy in dental caries management highlights the importance of accurate diagnosis, minimal cavity preparation techniques, and active prevention. Microbiological tests have

been recommended (Krasse, 1985) in the clinical evaluation of the caries status of a patient.

Microbiological tests

Mixed saliva samples are used to enumerate the numbers of *S. mutans* and *Lactobacillus* spp. in the mouths of patients. Briefly, a paraffin-wax-stimulated sample of mixed saliva is collected and sent to the laboratory, where it is vortex-mixed, diluted, and cultured on selective media for *S. mutans* (Mitis Salivarius Bacitracin Agar) and *Lactobacillus* spp. (Rogosa SL Agar). The number of typical colonies at a suitable dilution is recorded and the count per ml of saliva is calculated. The salivary counts can be recorded as high and low as follows: High value $> 10^6$ *S. mutans*; $> 10^5$ *Lactobacillus* spp. Low value $< 10^5$ *S. mutans*; $< 10^3$ *Lactobacillus* spp. Generally salivary counts of *S. mutans* and *Lactobacillus* spp. correlate well with plaque counts in the same patient.

Whilst there is good correlation between caries prevalence and high counts of lactobacilli and *S. mutans* when large groups are studied (Klock and Krasse, 1987; Zickert *et al.*, 1982), these tests are accurate in only about 45% of cases when the results for individuals are studied. The presence of high salivary levels of *S. mutans* or lactobacilli does not necessarily mean that the patient has a high incidence or risk of developing dental caries because other factors—such as diet, buffering capacity of saliva, fluoride content of enamel, and level of oral hygiene—may combine to produce a protective effect thus tipping the host-parasite balance. The main use of microbiology in caries assessment is to identify patients who have abnormally high oral counts of potential pathogens, so that this fact can be taken into account when integrating the factors which may contribute to the carious process, and deciding on treatment, e.g., the use of chlorhexidine gel to reduce a high salivary *S. mutans* count in a patient with recurrent caries. Furthermore, the tests can be used to assess the efficacy of preventative techniques, e.g., dietary and oral hygiene advice, and the use of antimicrobial agents such as chlorhexidine (Karjalainen *et al.*, 1987).

Immunisation procedures

It is well established that immunisation with either cell-wall associated antigens or glucosyltransferases from *S. mutans* is effective in reducing experimental dental caries in rats and monkeys (Hamada *et al.*, 1986). It is not entirely clear how the cell wall vaccine produces its protective effect,

although the following mechanisms have been suggested: (i) inhibition of the microbial colonisation of enamel by secretory IgA; (ii) interference with bacterial metabolism; and (iii) enhancement of phagocytic activity in the gingival crevice area due to the opsonisation of *S. mutans* with IgG or IgA (Cohen *et al.*, 1983). However, the evidence supporting any of these suggestions is far from complete (Sims, 1985). To date the vaccine has not been tested in man, due mainly to fears of possible side-effects. Generally, the antibodies which develop after immunisation with *S. mutans* antigens tend to cross-react with heart tissue *in vitro*. While the significance of such cross-reactivity is unknown, the possibility of resultant heart damage must be seriously considered.

Considering also that the incidence of dental caries is falling in the West, due in part to water fluoridation and use of sugar substitutes and fissure sealants, vaccination may be deemed unnecessary. However, it can be argued that not all Western countries are experiencing a decrease in caries rate and that on a world-wide basis, a vast increase in caries may occur, especially in poor countries with little or no organised dental services. In this situation a safe and successful vaccine could be valuable in controlling the disease in a population. Also, prevention of disease in special high risk groups, e.g., mentally or physically handicapped children, could be achieved (Russell and Johnson, 1987).

Microbiology of periodontal disease

Periodontal disease occurs in all parts of the world and few individuals live out their natural life span without becoming affected. However, in the majority of individuals, the common chronic inflammatory diseases which involve the gingival and periodontal tissues progress slowly and can be controlled, if not cured, by mechanical cleansing techniques and by encouraging good oral hygiene. A small but significant number of patients experience more rapidly destructive disease, which requires assessment and treatment by periodontologists. Brief descriptions of the main types of periodontal disease which are believed to have a bacterial aetiology are shown in table I. Both soft tissues (epithelium, and connective tissue) and hard tissues (alveolar bone) may be damaged as a result of periodontal infection. The important factors involved in the pathogenesis of these infections are the periodontal tissues themselves, the non-specific and specific host defence mechanisms, and subgingival plaque bacteria.

Table I. Different types of periodontal disease

Disease	Clinical presentation	Sequelae if untreated	Treatment
Acute ulcerative gingivitis	Inflamed gums which bleed easily, and have irregular ulcers on their margins between teeth	Chronic infection with acute exacerbation: both soft tissue and bone destruction	Metronidazole 200 mg, 3 times daily for 3 days
Chronic gingivitis	Inflamed gums which bleed easily	Progresses over months or years to chronic periodontitis	Mechanical removal of plaque and calculus
Chronic periodontitis	Inflamed gums which bleed easily and sub-gingival pockets of more than 4 mm in depth	Either slow destruction of supporting tissues throughout life, or less commonly more rapid damage, with tooth loss by 30–40 years of age	Mechanical removal of plaque and calculus plus metronidazole, or tetracycline or chlorhexidine, in selected patients
Localised juvenile periodontitis	Little sign of obvious disease, but on probing or X-rays, substantial loss of alveolar bone noted	Early loss of teeth	Mechanical removal of plaque plus tetracycline 250 mg 3 times daily for 4 weeks

While there is little doubt that dental plaque bacteria are essential in the aetiology of acute ulcerative gingivitis (AUG), chronic gingivitis and periodontitis, and juvenile periodontitis, it is not clear, at present, if tissue destruction is related to a non-specific mixture of micro-organisms, or if a single species or specific synergic complexes of a few different species, are implicated. To date no exogenous pathogens have been described nor have Koch's postulates been satisfied. However, it is always difficult to obtain conclusive evidence of cause and effect in endogenous infections and it is known that only some plaques are periodontopathic and are colonised, and perhaps dominated, by one or more specific microbial species. The problem is complicated by the fact that 300 or more different species have been described in subgingival plaque, many of which are poorly characterised. However, since local and immune factors probably play important roles in these infections, the isolation of periodontopathogens cannot be assumed to indicate tissue destruction in all sites in all individuals.

Before discussing the micro-organisms involved, it is necessary to describe briefly the considerable difficulties of relating micro-organisms to periodontal diseases. There are problems in obtaining appropriate samples and laboratory difficulties in isolating, identifying and enumerating the complex microflora of sub-gingival plaque. If specific pathogens are involved, it is likely that they will be present in high concentration at the site of infection which is often deep between the teeth at the base of periodontal pockets (fig. 1). However, it is unlikely that the present sampling procedures either reach these sites or prevent the "pathogens" from

becoming grossly contaminated with the "non-pathogenic" more superficially placed plaque bacteria.

Furthermore, destruction of periodontal tissues is often episodic, with relatively brief periods of activity being followed by longer periods of quiescence (Socransky *et al.*, 1984). At present, clinical and radiological examination are unable to diagnose active disease, or to predict which patients are likely to experience severe progressive periodontitis. Tissue destruction can be recorded with certainty only in a retrospective manner. Therefore, it is extremely difficult to relate any given subgingival plaque sample to a period of disease activity.

Much of the current research in periodontology is directed towards developing laboratory tests which will indicate or predict disease activity and so allow high risk patients to be identified early and ensure that subsequent specialised treatment is effective.

Localised juvenile periodontitis and acute ulcerative gingivitis

The best evidence for microbial specificity is found in two relatively uncommon forms of periodontal disease—localised juvenile periodontitis (LJP) and acute ulcerative gingivitis (AUG).

Much of the literature which supports the specificity of *Actinobacillus actinomycetemcomitans* for LJP has been reviewed by Zambon (1985). The evidence includes association studies, immunological investigations, evidence of tissue invasion, production of a range of exoproducts (table II), and successful treatment with tetracycline resulting in

Table II. Potentially pathogenic factors produced by *B. gingivalis* and *Actinobac. actinomycetemcomitans*

	<i>B. gingivalis</i>	<i>Actinobac. actinomycetemcomitans</i>
Degradation of IgA and IgG	+	—
Collagenase	+	+
Hyaluronidase	+	—
Endotoxin	+	+
Fibroblast growth inhibitors	+	+
Leucotoxin	—	+

the elimination of *Actinobac. actinomycetemcomitans* from disease sites (Slots and Genco, 1984).

Although there is a strong association between the clear-cut clinical diagnosis of AUG and the microscopical demonstration of the fusospirochaetal complex, there are surprisingly few culture studies of this condition. Loesche *et al.* (1982) found that *Treponema* spp. (32%), *Bacteroides intermedius* (24%), *Fusobacterium* spp. (3%) and *Selenomonas* spp. (6%) were regularly present. The main evidence of specificity in AUG is as follows: (i) microscopical association studies; (ii) the ability of the fusospirochaetal complex to cause tissue destruction in other sites, e.g., Vincent's angina; (iii) animal studies (Mikx *et al.*, 1984); and (iv) the fact that successful treatment with metronidazole is related to the rapid elimination of the complex from the disease sites with associated obvious clinical improvement. Factors which predispose to AUG include heavy smoking, stress and poor oral hygiene (Johnson and Engel, 1986). In parts of Africa and other third world countries, malnourished children develop a form of AUG which spreads rapidly causing extensive necrosis to the soft tissues of the face. The condition is called Noma (Cancrum Oris) and is commonly associated with a recent history of viral infection, e.g., measles (Guillozet, 1981).

Chronic periodontitis

The evidence of microbial specificity in chronic periodontitis is based on three main laboratory approaches: (i) use of dark field microscopy of plaque to estimate the percentage of different morphological types, especially spirochaetes and other motile organisms; (ii) screening plaque for the presence of selected periodontopathic micro-organisms by conventional culture techniques as well as with DNA probes; and (iii) detailed microbiological studies which attempt to isolate,

identify and enumerate all the microbial species in plaque samples.

A significant difference in the percentage of spirochaetes is found in healthy compared to diseased sites affected by chronic periodontitis (Lindhe *et al.*, 1980; Evian *et al.*, 1982). The results of a longitudinal study by Listgarten and Levin (1981) showed marked differences in the percentage of spirochaetes and other micro-organisms between sites which experienced no loss of attachment and those sites with loss of 2 mm or more. As a result, the concept that the percentage of spirochaetes in a plaque sample could be used as marker or predictor of active-disease sites emerged. Over the years several further studies have been performed (Armitage *et al.*, 1982; Listgarten and Schifter, 1982; Africa *et al.*, 1985) but the early promise of this technique has not been sustained, and the evidence for spirochaetal specificity is conflicting and confused (Claffey *et al.*, 1985; MacFarlane *et al.*, 1988).

The role of black pigmented *Bacteroides* spp., especially *B. gingivalis* and to a lesser extent *B. intermedius*, has also been investigated (Winkelhoff *et al.*, 1988). The evidence for the involvement of *B. gingivalis* in chronic periodontitis (Moore, 1987) comprises: (i) association studies; (ii) the production of a wide range of potentially damaging factors *in vitro* (table II) and (iii) experimental animal infections which have resulted in soft tissue destruction (van Steenberg *et al.*, 1982), and bone resorption (Roeterink *et al.*, 1985). However, there is no definitive proof that *B. gingivalis* produces measurable periodontal destruction *in vivo*. Finally there is some evidence that the presence of *Actinobac. actinomycetemcomitans* in the subgingival plaque of adult patients with "refractory" chronic periodontitis may be related to disease activity, and that the elimination of this organism by treatment with tetracycline results in marked clinical improvement (Slots *et al.*, 1986).

Detailed microbiological studies have been performed (Moore *et al.*, 1985), and have produced a lengthy list of micro-organisms, which were isolated more commonly and in higher numbers from 'diseased' compared with 'healthy' sites. They include: two treponemal species, various *Eubacterium* spp. (e.g., *E. nodatum*), *L. minutus* and *Peptostreptococcus micros*, in addition to *B. gingivalis* and *F. nucleatum*.

In conclusion, it is not clear whether the species highlighted as possible pathogens to date are indeed causative, or have become selected because of the favourable environment present in periodontal pockets.

Dento-alveolar infections

Development

A dento-alveolar abscess is a pyogenic infection which is associated with a tooth and its surrounding tissues. The clinical presentation is variable and is related to the interaction of several factors, such as the virulence of the causative micro-organisms, the state of the local and systemic defence mechanisms of the host, and the site of the lesion. In the usual sequence of the development and spread of a dento-alveolar abscess, micro-organisms present in an untreated carious lesion in enamel extend into dentine and spread to the pulp *via* the dentinal tubules (fig. 1). The pulp becomes acutely inflamed and subsequently undergoes necrosis. Micro-organisms can enter the pulp by other means including tooth fracture or traumatic exposure of the pulp during dental treatment. Infection and death of the pulp result in the pulp chamber and root canal becoming colonised by micro-organisms, which have a potential to produce a wide range of irritant substances including enzymes, acids and toxins. Bacteria and their products may subsequently leak from the root canal into the periapical tissues thus stimulating abscess formation. Pus once formed may remain localised at the root apex with the formation of a chronic abscess which can develop into a focal osteomyelitis. Conversely pus may spread through the cortical bone of the jaws into

the superficial soft tissues to form a localised abscess, or break through the overlying oral mucosa or skin producing a sinus, or extend through the soft tissue to produce cellulitis.

Microbiology

Pus from a dento-alveolar infection may yield a single isolate, a mixture of two to three different bacterial species or a complex mixture of micro-organisms involving perhaps eight to ten different species. A single isolate is unusual and a mixture of three to four different species is much more common. Well controlled studies have shown both qualitatively and quantitatively that strict anaerobes are usually the predominant organisms present in these infections. That the viridans streptococci are less common than one would assume from many earlier reports is probably related to contamination of pus samples during sampling, inadequate transportation to the laboratory and poor laboratory technique. Whilst almost every member of the oral flora has been isolated from dento-alveolar abscesses at one time or another (table III), it is clear that strict anaerobes predominate, especially *Bacteroides* spp. and anaerobic cocci, although facultative bacteria, e.g., *S. milleri*, are also found both in pure and mixed culture (Heimdahl *et al.*, 1985; Lewis *et al.*, 1986a).

Until recently, published reports tended to

Table III. Identity of 166 bacterial strains isolated from 50 acute dento-alveolar abscesses (Lewis *et al.*, 1986a)

Facultative organisms	Number of isolates	Strict anaerobes	Number of isolates
<i>S. milleri</i>	25	<i>Peptostreptococcus</i> spp.	14
<i>S. mitior</i>	3	<i>Peptococcus</i> spp.	32
<i>S. sanguis</i>	3	<i>S. intermedius</i>	3
<i>S. mutans</i>	1	<i>S. constellatus</i>	1
<i>L. fermentum</i>	2	<i>Propionibacterium acnes</i>	1
<i>L. salivarius</i>	1	<i>Eubacterium lentum</i>	1
<i>Actinomyces</i> spp.	3		
<i>Arachnia propionica</i>	1		
		<i>Veillonella parvula</i>	3
<i>Haemophilus parainfluenzae</i>	2	<i>B. oralis</i>	20
<i>Capnocytophaga ochracea</i>	1	<i>B. gingivalis</i>	14
<i>Eikenella corrodens</i>	1	<i>B. melaninogenicus</i>	12
		<i>B. intermedius</i>	5
		Non-pigmented <i>Bacteroides</i> spp.	10
		<i>F. nucleatum</i>	6
		<i>F. mortiferum</i>	1
Total	43	Total	123

support the idea that most oral bacteria, with the exception of some species of black-pigmented *Bacteroides* spp., had low pathogenicity when tested in various experimental animal models (MacDonald *et al.*, 1963; Sundqvist *et al.*, 1979; Pancholi *et al.*, 1985). However, there is evidence that other oral species are pathogenic both in pure and mixed cultures when tested in animals, e.g., *F. nucleatum*, *Peptococcus* spp., *Peptostreptococcus* spp. and *S. milleri* (Brook and Walker, 1983, 1984; Lewis *et al.*, 1988). Generally, pure cultures of *Bacteroides* spp. and *F. nucleatum* produce more severe abscesses, are isolated in greater numbers from pus, and appear to protect gram-positive cocci when present in mixed cultures (Lewis *et al.*, 1988). *In vitro*, a number of virulence factors have been described for *B. gingivalis* including the possession of a capsule and other factors which may inhibit phagocytosis (Brook, 1987; Rotstein *et al.*, 1985), and IgA proteases. These and other potential virulence factors in *Bacteroides* spp. have been reviewed by Slots and Genco (1984).

Ludwig's angina and necrotising fasciitis

In a small number of cases, dento-alveolar infection can spread rapidly via fascial planes with severe consequences. Cases of necrotising fasciitis of the neck and chest wall have been reported which required high doses of ampicillin, metronidazole and cefotaxime together with extensive debridement involving mastectomy, to control the infection (McAndrew *et al.*, 1987). Ludwig's angina is another life-threatening infection which is characterised by a bilateral swelling of the sublingual and submandibular spaces which raises the floor of the mouth and tongue and distends the tissues at the front of the neck (Patterson *et al.*, 1982). As a result, the airway becomes obstructed due either to oedema of the glottis or to a swollen tongue blocking the nasopharynx. In about 90% of cases of Ludwig's angina the primary source of infection is of dental or post-extraction origin.

In the past *S. pyogenes* was associated with Ludwig's angina but more recently *Bacteroides* spp., *Fusobacterium* spp. and anaerobic cocci have been isolated more commonly. Pus from these infections usually contains a mixture of micro-organisms, occasionally with coliforms such as *Escherichia coli*.

Staphylococcal submandibular lymphadenitis in children

This disease is of endogenous origin and is caused by *Staphylococcus aureus*. By bacteriophage typing

of staphylococcal isolates from the nose and abscess in the same patient, it is known that the anterior nares is usually the source of the causative organism (Brook and Winter, 1972). It is believed that *S. aureus* spreads from the anterior nares to the submandibular lymph nodes *via* interconnecting lymphatics and, for reasons unknown, then proliferates to produce an abscess. The infection is uncommon and usually limited to children 2–5 years old. Pain is not a marked feature, nor is severe constitutional upset. The total absence of any dental focus of infection is common, and the causative organism often produces β -lactamase (Stenhouse *et al.*, 1978).

Cervico-facial actinomycosis

Actinomycosis of the cervico-facial region accounts for well over half the recorded cases. Actinomycosis can present in an acute, subacute or chronic form and most cases of cervico-facial actinomycosis probably start as acute swellings which are indistinguishable on clinical grounds from acute dentoalveolar abscesses. The chronic form, with multiple sinuses, usually follows acute infection which has received either no therapy or has been inadequately treated. Cases have also been reported in the maxillary antrum, salivary glands, and tongue as well as in localised intra-oral situations such as periodontal abscess, pulp infection, apical granuloma and odontogenic cyst (Bronner and Bronner, 1971). Direct involvement of bone causing actinomycotic osteomyelitis is rare. Pain is a variable feature and, in some patients with acute infections, the abscess is painless.

A. israeli is the causative organism although there are reports in which other *Actinomyces* spp. (e.g., *A. naeslundii*) have been isolated from patients presenting with the typical signs and symptoms of the disease. Occasionally *Actinobac. actinomycetemcomitans* has been isolated in mixed culture with *A. israeli*.

Treatment

Treatment of dento-alveolar infection is based on drainage of pus, removal of the source of infection (usually a tooth) and, where necessary, treatment with antibacterial drugs. Indications for the use of antimicrobial agents include spread of infection into the soft tissues, patients with a compromised immune system, and when the infected tooth is to be retained and treated by root canal therapy. A conventional 5-day course of penicillin V (250 mg, three times per day) or short-

course, high-dose amoxycillin (two 3-g sachets taken 8 h apart) are suitable regimens (Lewis *et al.*, 1986b). However, when penicillin therapy is inappropriate, erythromycin or metronidazole are effective. Flucloxacillin should be used in cases of staphylococcal lymphadenitis, and penicillin or tetracycline for cases of actinomycosis (the latter is preferable if *Actinobac. actinomycetemcomitans* is present).

Conclusion

Plaque-related infections are common in the community and range from mild inflammation of

the gingivae to aggressive life-threatening infections related to dento-alveolar abscess. Because many of the species associated with these diseases are difficult to characterise, especially using only Gram's stain, colonial morphology and a few rapid tests, it is understandable why collective terms such as "oro-pharyngeal commensals" or "commensal flora only" tend to be used in medical microbiology departments. However, as knowledge about the micro-organisms associated with oral and dental infections increases, it is clear that much of the information, especially in relation to host parasite interactions, is of relevance to medical microbiologists.

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