Periodontitis is a chronic inflammatory disease of the periodontium that involves the gingival tissues and bone around the tooth roots. The progression of periodontitis is associated with the presence of a subgingival plaque biofilm and subsequent clinical attachment loss and pocket formation (Figure 1) due to destruction of the periodontal ligament attachment and associated alveolar bone.

Traditionally, the treatment of periodontitis has involved a number of different therapeutic strategies aimed principally at eradication of periodontal pathogens. These strategies have involved both surgical and non-surgical approaches to management depending on the severity of disease. Both surgical and non-surgical debridement results in clinical attachment gains and reduction in pocket depths. In shallow to moderate pockets, a non-surgical approach can be as effective as a surgical approach, with deeper pockets (>6mm) responding better following surgical therapy with greater probing depth reduction and clinical attachment gains.1

Our current understanding of periodontal disease recognizes that while periodontal pathogens are necessary to initiate disease, the extent and severity of tissue destruction is also related to the host response-microbial interactions. This has lead to new research looking specifically into how we may modulate the host response to ultimately reduce tissue destruction and disease progression.

**Systemic antibiotics**

Systemic antibiotics enter the gingival sulcus by transudation through the crevicular and junctional epithelium from the serum. However, the concentration of the antibiotic can often be so low as to be ineffective unless the bacterial biofilm is also disrupted through mechanical debridement. This highlights the need to undertake effective mechanical treatment (scaling, root planning) in conjunction with the use of systemic antibiotic therapy for the management of periodontitis. Thus, systemic antibiotics should not really be provided in isolation for the management of chronic periodontitis.

A number of different systemic antibiotic classes have been recommend as adjuncts to mechanical debridement of periodontal pockets (Table 1).

One of the more effective antibiotic regimes is the use of a combination therapy of Amoxycilin and Metronidizole as proposed by Winkel etal.2 The recommended dose being 250 mg of each three times daily for 8 days. If the patient has sensitivity to penicillin, then a combination of Clindamycin and Metronidizole can also be very effective.3

Recent data also supports the use of Azithromycin in the management of periodontitis in combination with mechanical debridement. This particular antibiotic has the advantage of being therapeutic at a low dose. Azithromycin has also been shown to reduce bleeding on probing and increase the rate of wound healing.4,5

**Local antibiotic therapy**

Problems associated with the use of systemic antibiotics in the management of periodontitis include: risk of antibiotic sensitivity; increasing bacterial resistance to antibiotic medications; gastrointestinal side effects; and low therapeutic dose reaching the target “organ” i.e. the periodontal pocket. This has lead to interest in delivering local antibiotic treatment, directly into the periodontal pocket. One of the first attempts in this regard was the use of Actisite™, which consisted of fine fiber willed with tetracycline. These fibers were inserted into the pockets for around
10 days. While Actisite™ was found to be effective, it was also labour intensive, which ultimately led to the search for a resorbable delivery system.

Atridox™ (Atrix Laboratories) was a flowable gel containing Doxycycline that could be “injected” into the periodontal pocket would it flow and adapt around the root, setting and then allowing the release of the Doxycycline to the surrounding tissues. The other big advantage was that Atridox was resorbable. One of the disadvantages of this material is that it sets quite hard and can result in irritation and inflammation in the periodontal pocket.

Aristin™ (OraPharma) is minocycline incorporated into tiny bioabsorable polymer microspheres. When delivered into the periodontal pocket, it maintains a therapeutic concentration within the pocket for up to 14 days.

A recent meta-analysis of over 50 published papers on the subject of local delivery of antibiotics suggested that while overall treatment results were favorable, the differences were minimal when compared to scaling and root planning alone. Such findings as well as difficulty in access to these products due to limited distribution and availability have meant that their use has not become widespread among clinicians.

<table>
<thead>
<tr>
<th>Table 1. Systemic antibiotic classes</th>
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<tr>
<td>Penicillin</td>
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<tr>
<td>Tetracycline</td>
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<td>Quinolone</td>
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<tr>
<td>Lincomycin</td>
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<tr>
<td>Nitroimidazole</td>
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<td>Macrolide</td>
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Antiseptics

The use of chemical agents with anti-plaque action as adjuncts to periodontal therapy appears to be of limited value. This is due principally to the inability of such mouthwashes to penetrate into the periodontal pocket. While oral antiseptics such as Phenolic compounds (Listerine™), Chorhexidine gluconate, Triclosan and cetylpyridium chloride can be useful in reducing supragingival biofilm; and provision of a clean root surface, scaling and root planning can initiate a healing response which can result in new clinical attachment and pocket depth reduction. Future work is directed at how we may enhance this process though adjunctive therapies such as those outlined here. It is clear that modulation of the host response and in particular the negative effects of inflammation shows considerable promise in enhancing the results of non-surgical therapy.

Future directions

At present, scaling and root planing remain central to the non-surgical management of periodontitis. Through the removal of pathogenic bacteria and mineralized deposit; disruption of the biofilm; and provision of a clean root surface, scaling and root planning can initiate a healing response which can result in new clinical attachment and pocket depth reduction. Future work is directed at how we may enhance this process though adjunctive therapies such as those outlined here. It is clear that modulation of the host response and in particular the negative effects of inflammation shows considerable promise in enhancing the results of non-surgical therapy.

About the author

Dr Michael Danesh-Meyer is a specialist periodontist in private practice in Auckland, New Zealand. He was a Clinical Assistant Professor in Periodontology and Associate Scientist in the Laboratory for Applied Periodontal and Craniofacial Regeneration at Temple University, School of Dentistry in Philadelphia, USA. He has been involved in pre-clinical and clinical research involving Guided Tissue Regeneration/Guided Bone Regeneration and dental implants since 1991, has authored numerous scientific articles and lectures both nationally and internationally on topics related to implant dentistry and tissue regeneration therapy. He established the Institute of Dental Implants & Periodontics and Auckland Clinical Training Centre in 2000 and is Director of Dental Education Continuum and DentalMentor.org.

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