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EDITORIAL

Infatuated by *Enterococci*

In a paper published in 1964, Engström[1] outlined the presence of *Enterococci* in the oral cavity with special emphasis on the root canal system. He reported that it was not unusual to find *Enterococci* in previously root-filled teeth, even if no periapical lesion could be diagnosed. He also showed that there was an association between findings of *Enterococci* in root canal samples and their presence in other areas of the oral cavity, such as inter-proximal spaces and tonsils.

Since then there has been little interest in *Enterococci* until 24 years later, when there were two successive reports on significant presence of *Enterococci* in the root canals of teeth with failed endodontic treatments.[2,3] Subsequently, there has been 8 years of endodontic research nearly totally focused on *Enterococci* and published in a frequency exceeding the number of published "leakage studies." This frenzy has to be reconsidered. To some degree these reports of *Enterococci* in endodontics have had an intellectual effect on endodontology similar to the changes experienced in the middle of the 1960s foreboded by the well known "To culture or not to culture?"[4,5] This resulted in a "walk in the desert" for clinical endodontology until the importance of microorganisms for the development of periapical disease was re-established in 1976.[6]

After several studies of refractory endodontic cases using classic culturing techniques, molecular techniques were applied in 2001 by Rolph and coworkers.[7] Although this study was unsuccessful in identifying *Enterococci* in refractory endodontic cases, numerous reports using molecular techniques in recent years have found high presence of *Enterococci* in root canals of teeth with failed endodontic treatment.

Enterococci are very resistant to antimicrobial regiments applied in living tissues. Therefore, much research effort has been directed toward this specific area of antisepsis, often ignoring chemical interactions and tissue tolerance, however.

In addition to the pure observation of *Enterococci* associated with failed endodontic treatment cases, there is no clear evidence that *Enterococci* are solely or partially responsible for endodontic infections resulting in treatment failure. As demonstrated earlier, findings of *Enterococci* in root canal systems are closely related to periodontal and oral presence of the bacterium.[1]

A root-filled and restored tooth offers rich opportunities for periodontal bacteria to contaminate, at the least, the coronal part of the root canal and the pulp chamber area as no known restoration provides a permanent hydraulic seal. This area of the tooth is also known to be difficult to disinfect in preparation for conventional bacteriological culturing. There is no known, highly efficient method to eliminate DNA remnants from this area, even if conventional disinfection methods effectively kill bacteria. Presently, strong solutions of NaOCl appear to be the most promising method, but it is rarely applied.[8]

Despite the lack of even a minor evidence that *Enterococci* are responsible for refractory periapical inflammation, the endodontic community has accepted the causation without even asking for any sign of a modern version of Koch's postulates.[9] In two recent studies by independent groups it was demonstrated that the presence of *Enterococci* in root-filled teeth are as common in teeth with or without a periapical lesion.[10,11] It may just be an opportunistic bystander to other polymicrobial pathogens. These findings may set us back to square one after 8 years of work.

To move us off the center, there are now three important facts to be established before moving on with our focused attempts to kill every single *Enterococcus* in root canals.

First, we have to be less gullible when new findings are presented and ask many more probing questions.

Second, we need to establish and agree on the best methods of sampling for molecular techniques and culturing methods. Presently, each laboratory uses its own procedures, which are often mostly partially or totally inadequate, resulting in questionable results. It is true that molecular techniques are superior for sampling of

low numbers of bacteria and identifying very fastidious species. However, if we are to move into more significant research on endodontic diseases using molecular techniques, there needs to be well established safeguards as it is easy to err.

Third, we need to establish the true role of *Enterococci* in endodontic infections. Are *Enterococci* solely responsible for the periapical immune response we recognize as a periradicular lesion, or is a cohort of various species needed?

Or, do they play any role at all?

I hope I am wrong in my doubt as I hate to have lost 8 years – especially at my age.

Larz S.W. Spångberg, DDS, PhD
Section Editor, Endodontology
Department of Endodontology
University of Connecticut Health Center
Farmington, CT

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