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Institutionen för Odontologi

Origin of intraradicular infection with *Enterococcus faecalis* in endodontically treated teeth

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ABSTRACT

The reported prevalence of *Enterococcus faecalis*, both a commensal of the gastro-intestinal tract and a common nosocomial pathogen, ranges from 24% to 77% in post-treatment root canal infections. To date it has not been possible to explain this prevalence, since its origin remains unknown. Its exceptional array of intrinsic and easily acquired traits, including resistance to a multitude of antibiotics, enables an adaptation to a wide variety of different environmental settings and poses a challenge in treatments.

The aim of the research was to elucidate the origin of *E. faecalis* in root canal infections to enable a means of preventing costly and time-consuming treatment failures. The potential for acquisition from the endogenous flora, a nosocomial transmission from contaminated surfaces during a root canal procedure and a food-borne route of infection were evaluated by measuring its occurrence on environmental surfaces, comparing genetic relatedness, distribution of putative virulence factors and antibiotic resistance between isolates from different sources.

DNA fingerprinting by PFGE concluded that *E. faecalis* retrieved from eight (16%) secondary root canal infections in 50 consecutively treated patients were genetically unrelated to those recovered from the patients' own intestinal tract. *E. faecalis* could not be retrieved from any of the saliva samples pertaining to the patients with the microorganism in the root canal sample, validating its transient presence in the oral cavity. Analysis of a total of 320 collected samples from 10 high-touch surfaces in six general dentistry clinics and two specialist clinics displayed a very low occurrence (0.9%) of *E. faecalis* on surfaces despite clear deficiencies in decontamination procedures. Determination of the distribution of putative virulence genes and susceptibility to clinically relevant antibiotics amongst strains isolated from root canals, foods, stool and blood culture samples by PCR and the agar dilution method, respectively, detected an association between endodontic isolates and isolates from food and stool based on a common gene pattern, consisting of *gel*E, *efa*A and *gel*E. The linkage could be corroborated by MLST analysis, demonstrating that 66.7% of the root canal isolates, 42.1% of the food strains, 34.5% of the stool isolates but only 10% of the blood isolates shared genetic lineages. Correlation of detected virulence determinants to MLST data revealed distinctive features of the resulting major genetic lineages. All isolates in CC25 were impaired to express gelatinase and all strains in CC6 lacked the gene *ace* but were enriched with antibiotic resistance and the ability to express cytolysin.

In conclusion, *E. faecalis* in root canal infections is most likely not derived from the endogenous flora or nosocomially transmitted but instead food-borne. It presumably gains access to the treated canals via micro-leakage, which stresses the need for better ways of sealing endodontically treated teeth.

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