

Taurolidine's Effect on Species Associated to Periodontitis

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ABOUT THE STUDY

According to the most recent statistics, periodontitis affected 42% of non-institutionalized Americans. The tissues that support the teeth are destroyed by the condition known as periodontitis. The host's inflammatory reaction to subgingival bacteria that results in a pathogenic microbiota plays a key role in the pathophysiology of this illness. The primary virulence components of Porphyromonas gingivalis, known as gingipains, are thought to represent a critical stone pathogen in this shift. *Tannerella forsythia* and Aggregatibacter actinomycetemcomitans, which produce a leukotoxin, are additional bacteria involved in the aetiology of periodontitis. actinomycetemcomitans strains exhibit varying levels of leukotoxin production; those with the highest levels (JP2-clone) had a deletion in the promotor region.

Periodontal infections are frequently treated with antibiotics. Better clinical outcomes are seen when chlorhexidine digluconate is used in addition. In severe and advanced cases of periodontitis, supplementary systemic antibiotic usage seems to be advantageous. However, it is still unknown whether using antibiotics will have any long-term clinical benefits. When used as a topical antibiotic, the tetracycline derivative minocycline in microspheres has demonstrated more therapeutic advantages than nonsurgical periodontal treatment alone.

Over half a million fatalities a year are linked to diseases brought on by microorganisms that are resistant to antibiotics, making the development of antimicrobial resistance a global issue. This depends on the antibiotic that was used; for example, when quinolones were used, the link was strong, yet when beta-lactams were employed, it was fairly weak. Additionally, a significant number of studies have documented the emergence of resistance to widely used antiseptics, and in some cases, cross-resistance with antibiotics. The inevitable result of bacterial genetic evolution is the rising incidence and spread of antibiotic resistant microorganisms. The likelihood of developing resistance to an antimicrobial agent increases with its frequency of usage.

Taurolidine is one possible substitute for an antibacterial agent. Taurolidine, an antibiotic that is a derivative of the amino acid taurine, has been shown to be both safe and efficient in preventing central venous catheter infection. Studies conducted in the lab show that oral microbes can still exhibit antibiotic activity when arranged as a biofilm. Dental applications have been proposed for a while now. A 2% taurolidine solution rinse reduced dental biofilm formation by nearly 50%. We examined the potential of taurolidine in vitro in different experiments. With the exception of Candida albicans, all taurolidine Minimum Inhibitory Concentrations (MICs) were less than 1 mg/ml. Taurolidine at 1% clearly prevented the growth of 12species biofilms. However, the impact on a biofilm that had already formed was as minimal as that for minocycline. Following application of 3% taurolidine gel for 60 minutes, the reduction in bacterial counts in biofilms was 0.87 log10 cfu, or 86.5%, in an ex-vivo model using subgingival in addition to supragingival biofilm samples from periodontitis patients.

Bacterial resistance to antimicrobial drugs can have both innate and acquired resistance mechanisms, which can have different sources. Microorganisms naturally possess an intrinsic resistance mechanism. The bacterium has undergone genetic change as part of the acquired resistance mechanism. A mutation or the consumption of alien resistance genes can cause it. In addition to the microorganism's genetic potential, the current selection pressure of an antibiotic is crucial. For instance, bacteria that are exposed to higher concentrations of antimicrobial drugs may experience mutations as a result. Within a few hours, gene transfer can take place, such as when transposons or plasmids pick up extra-chromosomal gene elements. Target modification, impermeability, enzymatic modification or destruction, and efflux are the foundations of bacterial antimicrobial resistance mechanisms.

Gingipains' activity is inhibited by taurolidine's interaction with the LPS of periodontopathogens. Further research is required to assess taurolidine's potential utility in the treatment of periodontal and peri-implant infections because resistance to it develops much less frequently than it is to minocycline.

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