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Zinc chloride inhibits lysine decarboxylase production from Eikenella corrodens in-vitro and its therapeutic implications**Martin Levine***University of Oklahoma Health Sciences Center, United States*

Objectives: Dentifrices containing zinc reduce gingival inflammation and bleeding better than control dentifrices (no zinc). How zinc might work is not understood. We have shown that lysine decarboxylase (LdcE), an enzyme from *Eikenella corrodens*, converts lysine to cadaverine in dental biofilms. The lack of lysine impairs the dentally attached cell barrier to biofilm, causing biofilm products to leak into junctional epithelium and stimulate inflammation. In year-old beagle dogs, immunization with LdcE, induces antibodies that inhibit LdcE activity and retard gingivitis development. We therefore examined whether a zinc-mediated loss of LdcE activity could explain the beneficial effect of zinc dentifrices.

Methods: We grew *E. corrodens* in modified tryptic soy broth with or without zinc chloride, and extracted LdcE from the cell surface using a Potter Elvehjem homogenizer.

Results: Up to 0.96 mM zinc chloride in the bacterial growth medium did not change cell yield, but reduced the extracted protein content by 41% ($R^2 = 0.27$, $p < 0.05$) and LdcE activity/mg extracted protein by 85% ($R^2 = 0.90$, $p < 0.001$). In extracts from cells grown without zinc, 78 times this zinc chloride concentration (73 mM) was required to reduce LdcE activity by 75%.

Conclusions: Zinc ions inhibit the production of protein with LdcE activity at *E. corrodens* cell surfaces. The zinc ions may attach to cysteine residues that are unique to the N-terminal region of LdcE by interfering with the non-covalent polypeptide assembly that produces enzyme activity.

Clinical significance: Zinc ion-mediated inhibition of LdcE assembly may provide a rationale for the improved control of gingival inflammation by zinc dentifrices.

Biography

Martin Levine was graduated from the University in Glasgow with bachelor degrees in Dentistry (BDS) and Biochemistry (BSc) and completed a PhD combining biochemistry with dentistry 1973. He came to US on a travelling Research Fellowship to work with Dr P.A Keller at the Oral Biology Dept, University of Washington. After another 2 years at SUNY Buffalo, he took up his present position. His interests have always been to relate biochemistry to dentistry. In 2011, he completed a textbook, Topics in dental biochemistry to fill a gap in the dental student curriculum and raise awareness of this discipline. His current studies are directed at developing better methods of dental biofilm (plaque) control and better diagnosis and therapy for young children who develop severe caries.