Effect of HEMA on human gingival fibroblasts / Streptococcus mitis adhesion mediated by PKCα/ integrin β 1 intracellular signaling

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Human gingival fibroblasts are the major constituents of periodontal connective tissue, exposed to leachable form of dental restorative materials, such as HEMA, undergoing biological effects as reduction of proliferation, occurrence of apoptosis and inflammation. Integrin β 1 is a protein involved not only in the regulation of cell migration, proliferation, survival, apoptosis and differentiation but also in the adhesion eukaryotic/prokaryotic cells (Engels-Deutsch et al., 2011). Since previous reported evidences suggest PKC α as the main PKC expressed and activated by HEMA in human gingival fibroblasts (Cataldi et al., 2012) the molecular mechanisms mediated by PKC α through integrin β 1 driving the response to HEMA of HGF/Streptococcus mitis co-culture in terms of proliferation, adhesion and apoptosis have been investigated. HEMA treatment increases the adhesion between S. mitis and HGF, mediated by PKC α / integrin β 1 signalling system, improved by the presence of saliva, and reduces the expression of MMP2, involved in the remodelling of the extracellular matrix, which seems to control apoptosis occurrence, mainly reduced when saliva is added to the co- culture. These results, shedding more light on the biological and molecular events occurring in vitro in a co-culture model, which mimics the environment of the oral cavity, upon HEMA treatment, allow to confirm the key role played by oral bacteria and saliva to prevent inflammatory and toxic processes which can occur in vivo in human gingival fibroblasts upon the release of dental material monomers.

References

Engels-Deutsch et al (2011) Streptococcus mutans antigen I/II binds to $\alpha5\beta1$ integrins via it's a-domain and increases $\beta1$ integrins expression on periodontal ligament fibroblast cells Arch Oral Biol 56,23-28.

Cataldi et al. (2012) Protein Kinase C alpha mediated stress response to HEMA in human gingival fibroblasts. J Biomat Res Part A in press.

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