THE ROLE OF NFΚB REGULATES NLRP3 AND IL-1B TO GENERATE INFLAMMATION RESPONSE INDUCED BY RESIN MONOMER HEMAIN PULP COMPLEX

Abstract

Introduction:The cytotoxicity of dental monomers has been widely investigated, but the underlying mechanisms have not been elucidated. We studied the molecular mechanisms involved NFκB, NLRP3 and IL-1β which induced by HEMA. In odontoblas cells of rat, HEMA caused a severe damage that induced inate immunity response which lead to activate NFκB, NLRP3 and IL-1B. NLRP3, a cytosolic intracellular pattern recognice receptor (PRR) regulates a highly pro inflammatory cytokines IL-1β.We found an increase of reactive oxygen species (ROS) and NFκB activation after HEMA exposure. However, it is still unclear how HEMA could drive innate immun response to maintain homeostatic dentin pulp complex.

Purpose: to identify molecular mechanism of activation NFκB, NLRP3 and IL-1β in odontoblast pulp cells that induced by resin monomer HEMA to regulate innate immunity.***Methods*** *:* The study design was randomized post test only control groups. Sprague Dawley (SPD) rats are divided into 4 groups. 1 control group and 3 experimental groups.Tooth cavity was applied with HEMA liquid (Sigma Aldrich) with concentration 0,016µg/ml, then filled with glass ionomer cements (Fuji IX). Teeth were extrated after 24, 48 and 72 hours. The teeth were decalsificated using EDTA for 8 weeks. Immunohistochemistry staining were applied and using antibody anti NLRP3, NFκB and IL-1β to investigate the expression in odontoblast pulp cells. The sample were analyzed statistically by Anova and Tukey HSD**.** ***Result*** *:* HEMA induced increasingly expressions of NLRP3, NFκB and IL-1β in odontoblast pulp cells at 24, 48 and 72 hours.

Conclusion : HEMA regulatesNFκB, NLRP3 and IL-1β and leads to activate innate immunity response in odontoblast pulp cells

Keywords *:* HEMA, innate immunity, NLRP3, NFκB, odontoblast pulp cells