Altern Integr Med 2016, 5:4(Suppl)
http://dx.doi.org/10.4172/2327-5162.C1.021

3D FEA of cemented glass fiber and cast posts with various dental cements in a maxillary central incisor

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This study aimed to analyze and compare the stability of two dental posts cemented with four different luting agents by examining their shear stress transfer through the FEM. 83-dimensional finite element models of a maxillary central incisor restored with glass fiber and Ni-Cr alloy caste dental posts. Each dental post was luted with zinc phosphate, panavia resin, super bond C&B resin and glass ionomer materials. Finite element models were constructed and oblique loading of 100N was applied. The distribution of shear stress was investigated at posts and cement/dentine interfaces using ABAQUS/CAE software. The peak shear stress for glass fiber post models minimized approximately three to four times of those for Ni-Cr alloy cast post models. There was negligible difference in peak of shear stress when various cements were compared irrespective of post materials. The shear stress had same trend for all cement materials. This study found that the glass fiber dental post reduced the shear stress concentration at interfacial of post and cement/dentine compared to Ni-Cr alloy cast dental post.

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Activation of Nrf2 signaling pathway by glyceollins is independent of p53 in human colon cancer model

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Glyceollins, soybean-derived phytoalexins are reported to induce antioxidant and phase two detoxifying enzymes through nuclear factor (erythroid-derived 2)-like 2 (Nrf2)-mediated signaling pathway and thus protect normal cells from xenobiotic or cellular stress stimuli. Serendipitously, we observed that low doses of glyceollins promoted the proliferation of colorectal cancer cells carrying normal *p53* gene while the compounds inhibited the growth of *p53*-negative colon cancer cells in a dose-dependent manner. As *p53* is known to activate its downstream gene *p21* that interferes with Kelch-like ECH-associated protein 1 (Keap1)/Nrf2 heterodimer formation, we hypothesized that glyceollins at low doses stimulate Nrf2 signaling pathway by up-regulating *p53*-mediated *p21* expression, leading to proliferation of *p53*-positive colon carcinoma cells. When treated with glyceollins, the growth of wt *p53* HCT116 cells was increased by glyceollins in the range of 1-20 μg/mL while the growth of HT29 and Caco-2 cells carrying mutated *p53* was unaffected or gradually decreased by glyceollins treatment. Glyceollins led to significantly increased expressions of *p53* and *p21* in wt *p53* HCT116 cells, but did not affect the gene expression in *p53*-knockout HCT116 cells. However, the expression of Nrf2 and its downstream genes was significantly enhanced both in *p53*-mutant cells as well as *p53*-wild type cells, suggesting that glyceollins promote Nrf2 signaling pathway in a *p53*-independent manner. In contrast, sulforaphane and tert-butylhydroquinone, well-known activators of Nrf2 signaling pathway did not activate Nrf2 signaling pathway in p53 wild type HCT116 cells although they stimulated Nrf2 signaling pathway in p53-mutant cells. In conclusion, the activation mechanism of Nrf2 signaling pathway by glyceollins is likely to be different from common phase two enzyme inducers and not suppressed by *p53*.

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