Abstract

Interaction between Wnt/β-catenin pathway, dental materials and dentine formation.

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The Wnt/β-catenin pathway participates in various physiological processes. The bind of Wnt ligand to Frizzled and LRP5/6 receptors promotes the signal transduction, ensuring an elevate concentration of β-catenin in the cytosol that migrates to the nucleus, interacting with transcription factors. Its abnormal regulation leads to early dysfunctional events. The degradation of β-catenin is regulated by GSK3β and CK1α interactions, and several proteins modulate Wnt/β-catenin pathway such as lithium, Dkk1 and indirubin isomers. The aim of this study is to review the scientific knowledge in order to highlight the role of Wnt/β-catenin pathway in response to dental biomaterials for reparative dentine formation following tooth damage, by triggering the natural process of dentinogenesis. Wnt activation via GSK-3 inhibitor drugs increases dentine secretion, although Wnt inhibition does not impair dentine secretion. Moreover, the interaction of the Wnt/β-catenin with dental materials and the effect on pulp was detected. Recently, odontogenic/osteogenic gene expression in human dental pulp stem cells (hDPSCs) cultured with various concentrations of Mineral Trioxide Aggregate (MTA) was evaluated and differentiation of hDPSCs induced by MTA extract and mediated by pathway was observed. Furthermore, pulp injury caused by resinous monomers with or without the presence of GSK-3 inhibitor Lithium was assessed, indicating the activation of Wnt signaling after exposure to TEGDMA and a cumulative effect with the co-treatment with Lithium. In conclusion, the development of a concept of biological repair based on the role of the Wnt/β-catenin pathway in dentin formation seems to offer a new translational approach into development of future treatments.