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Exosome-derived lncRNA-Ankrd26 promotes dental pulp restoration by regulating miR-150-TLR4 signaling

Lin Li [1], Jianping Ge [1]

Affiliations

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Abstract

At present, retaining the biological function of dental pulp is an urgent requirement in the treatment of pulp disease; it has been recognized that application of dental pulp stem cells (DPSCs) in regenerating dental pulp and dentin complexes is expected to become a safe and effective treatment of pulp disease; meanwhile the role of DPSC-derived exosomes in dental pulp regeneration and repair is gaining attention. However, the underlying mechanism of DPSCs in dental pulp regeneration and repair is still unclear. In the present study, a variety of *in vitro* biological experiments and an animal model, as well as next-generation sequencing and bioinformatics analysis, demonstrated that DPSCs promoted migration and osteoblastic differentiation of mesenchymal stem cells (MSCs) via exosomes; this was induced by DPSC-derived exosomal long non-coding (lnc)RNA-ankyrin repeat domain (Ankrd)26. Mechanistically, the effect of exosomal lncRNA-Ankrd26 on migration and osteoblastic differentiation of MSCs was dependent on microRNA (miR)-150/Toll-like receptor (TLR)4 signaling; this was regulated by lncRNA-Ankrd26. The present study demonstrated that exosomes-derived lncRNA-Ankrd26 from DPSCs promoted dental pulp restoration via regulating miR-150-TLR4 signaling in MSCs; these findings help to understand the mechanism of dental pulp repair, identify therapeutic targets in the development of pulpitis and develop clinical treatments.

Keywords: Toll-like receptor 4 signaling; dental pulp stem cells; exosomes; injury-repair; long non-coding RNA-Ankyrin repeat domain 26; mesenchymal stem cells; microRNA-150.

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