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Perspective

Roles and Differentiations of Mesenchymal Stem Cells in Dental Therapy

Ye Zhang*

Department of Anatomy, University of Turku, Turku, Finland

DESCRIPTION

Mammalian teeth have been found to contain Mesenchymal Stem Cells (MSC). All MSCs that have been isolated from dental pulp, periodontal ligament, dental follicle, apical papilla, and even gingiva are now referred to as Dental-Derived MSCs (DMSCs). Similar to MSCs produced from bone marrow, these DMSCs are multipotent and capable of differentiating into cells with odontoblast, cementoblast, osteoblast, chondrocyte, myocyte, epithelial, neuronal, hepatocyte, and adipocyte properties. Additionally, DMSCs have strong immunomodulatory capabilities that let them control the local immunological microenvironment. Due to these characteristics, DMSCs offer a promising method for healing wounds, regenerating tissue, and treating a variety of diseases. This describes the most recent developments in the uses of DMSCs and illuminates how these developments are paving the way for DMSC-based therapeutics.

Odontoblasts are highly specialised cells that contribute to dentinogenesis, also known as the creation of dentin, which is the deposition and mineralization of the dentin matrix. The creation and renewal of the dentin-pulp complex are among the tasks performed by odontoblasts. Two DMSC subpopulations, DPSCs and SHEDs, serve as the main sources of odontoblasts during the production of tertiary dentin following postnatal injury because the previous odontoblast cannot create reparative dentin. By cultivating DMSCs in an odontogenic media containing dexamethasone, -glycerophosphate, and ascorbic acid, the typical procedure for odontogenic differentiation can be induced in a test tube. Alkaline Phosphatase (ALP), Collagen type 1 (COL1), Osteopontin (OPN), Osteocalcin (OCN), Dentin Matrix acid Phosphoprotein 1 (DMP1), Matrix Extracellular Phosphoglycoprotein (MEPE), and Dentin Sialo Phosphoprotein (DSPP) are just a few of the osteoblast-associated markers that DMSCs express under these culture conditions. These markers are also frequently employed in numerous investigations as differentiation markers specific to odontoblasts. In addition to the aforementioned markers, single-cell RNA sequencing (scRNA-seq), a groundbreaking technique developed recently that enables researchers to examine transcriptional profiles at the level of a single cell with unprecedented resolution, has revealed previously unknown odontoblast markers, such as NOTUM and SALL1. It's interesting to note that scRNA-seq also revealed previously unknown roles for some established odontoblast indicators, such as DSPP's function in amelogenesis.

Different induction circumstances that aim to enhance or hinder odontogenic differentiation have been studied thus far. Human DPSCs and human PDLSCs were differentiated into odontogenic cells *in vitro* using Allogeneic Fibrin Clot (AFC) serum, which contained enough cytokines and growth factors to do so. Additionally, it has been demonstrated that Sapindus mukorossi seed oil can induce osteogenic or odontogenic differentiation and matrix vesicle release of DPSCs when odontogenic induction is present. Additionally, a few other signalling molecules take role in the process of odontogenic differentiation. It has been demonstrated that Copine 7 (CPNE7), a diffusible signalling molecule, induces non-dental MSCs to develop into odontoblasts *in vitro* through regulating DSPP expression. Additionally, it has been shown that CPNE7 encourages the growth of tissues that resemble dentin *in vivo*. Endothelin-1 (ET-1) has a pro-odontogenic impact that is comparable and can help DPSCs differentiate into odontoblasts. The biggest known subgroup of Receptor Tyrosine Kinases (RTKs), Ephrin receptors (Eph), played a crucial role in determining cell destiny. EphrinB2 (ligand) and its corresponding receptors EphB2 and EphB4 were increased in DPSCs following odontogenic induction at both the gene and protein levels, and EphrinB2 signalling demonstrated improved effects on odontogenic/osteogenic differentiation of human DPSCs. Pentraxin-3 and lysine demethylase are additional regulating molecules. Mechanistically, KDM6B was drawn to the BMP2 promoters, removing the epigenetic mark H3K27me3, activating BMP2 transcription, and ultimately causing DMSCs to differentiate into odontogenic cells.

Correspondence to: Ye Zhang, Department of Anatomy, University of Turku, Turku, Finland, Email: zhang.y@gmail.com

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