

Dental pulp stem cell

Dental pulp stem cells (DPSCs) are a heterogeneous population of [cells](#) isolated from the human permanent third molar pulp. These [stem cells](#) have similar characteristics as MSCs with the major difference being that DPSCs can differentiate into dentin-forming odontoblast-like cells.

There is only one [skull base chordoma cell line](#), UM-chor1, freely available to researchers. The established TSK-CHO1 cells were neoplastic, exhibited pleomorphic features, and secreted brachyury, as revealed by immunocytochemical staining or ELISA of conditioned medium (CM). Cells also secreted SOX9, which enhanced brachyury production. The CM of TSK-CHO1 cells promoted the production of [hyaluronic acid](#) and type II collagen during the differentiation of human [dental pulp stem cells](#) (DPSCs) into fibrocartilage cells. Culture of DPSC pellets in a growth medium supplemented with 10% CM of TSK-CHO1 cells for 2 weeks resulted in the induction of fibrocartilage tissue under normoxic conditions. Brachyury produced by TSK-CHO1 cells promoted the production of collagen type II, peculiar to cartilage, in a dose-dependent manner. The newly established skull base chordoma cell line, TSK-CHO1, is expected to be used for elucidating the pathogenesis of skull base chordoma and for investigating the mechanism underlying the production of [fibrocartilage](#) ¹⁾.

A study aimed to determine whether dental pulp [stem cell](#)-derived [exosomes](#) (DPSC-Exos) exert protective effects against [cerebral ischemia-reperfusion injury](#) and explore its underlying mechanism.

Materials and methods: [Exosomes](#) were isolated from the culture medium of human DPSC. Adult male [C57BL/6 mice](#) were subjected to 2 hours transient [middle cerebral artery occlusion](#) (tMCAO) injury followed by 2 hours [reperfusion](#), after which singular injection of DPSC-Exos via tail vein was administrated. [Brain edema](#), [cerebral infarction](#) and neurological impairment were measured on day 7 after [exosomes](#) injection. Then, oxygen-glucose deprivation-reperfusion (OGD/R) induced BV2 cells were studied to analyze the therapeutic effects of DPSC-Exos on I/R injury in vitro. Protein levels of TLR4, MyD88, NF- κ B p65, HMGB1, IL-6, IL-1 β , and TNF- α were determined by western blot or enzyme-linked immunosorbent assay. The cytoplasmic translocation of HMGB1 was detected by immunofluorescence staining.

Results: DPSC-Exos alleviated brain edema, [cerebral infarction](#) and neurological impairment in I/R mice. DPSC-Exos inhibited the I/R-mediated expression of TLR4, MyD88, and NF- κ B significantly. DPSC-Exos also reduced the protein expression of IL-6, IL-1 β , and TNF- α compared with those of the control both in vitro and in vivo. Meanwhile, DPSC-Exos markedly decreased the HMGB1 cytoplasmic translocation induced by I/R damage.

Conclusions: DPSC-Exos can ameliorate I/R-induced cerebral injury in mice. Its anti-inflammatory mechanism might be related to the inhibition of the [HMGB1/TLR4/MYD88/NF- \$\kappa\$ B pathway](#) ²⁾.

Research aimed to evaluate the effects of dental pulp-derived stem cells conditioned medium loaded in collagen hydrogel in SCI. After culturing of Stem cells from human exfoliated deciduous teeth (SHEDs), SHED-conditioned medium (SHED-CM) was harvested and concentrated. Collagen hydrogel containing SHED-CM was prepared. The rats were divided into five groups receiving laminectomy,

compressive SCI with or without intraspinal injection of biomaterials (SHED-CM and collagen hydrogel with or without SHED-CM). After 6 weeks, histological parameters were estimated using stereological methods. The total volume of preserved white matter and gray matter ($p < 0.05$), as well as the total number of neurons and oligodendrocytes in the rats, received SHED-CM loaded in collagen hydrogel were significantly higher, and also lesion volume and lesion length were significantly lower ($p < 0.05$) compared to those of the other injured groups. In conclusion, intraspinal administration of SHED-CM loaded in collagen hydrogel leads to neuroprotection, proposing a cell-free therapeutic approach in SCI ³⁾.

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