Objective: To elucidate the regulation of deciduous dental pulp stem cells (DDPSCs) through alpha 7 nicotinic acetylcholine receptor (+7 nAChR) on osteoclast differentiation during physiological root resorption.

Methods: In this study, DDPSCs and permanent dental pulp stem cells (DDPSCs) were derived from deciduous teeth and normal permanent teeth at different stages of resorption, confirming their differentiative abilities and origin. The ability to induce osteoclast differentiation from different stages were compared. The expressions of RANKL, OPG, +7 nAChR, SLURP-1 as well as NF-κB p65 subunit were further detected by Real-Time-PCR and Western blot. Mechanical stress was applied with the DDPSCs from 5 group. The expression levels of SLURP-1 and +7 nAChR in the 5 group of DDPSCs were detected through Real-Time PCR and Western blot.

Results: In the middle stage of root resorption, DDPSCs exhibited an increase in the ability to induce osteoclast differentiation. Activation of the alpha 7 nicotinic acetylcholine receptor (+7 nAChR) by secretory mammalian Ly6-urokinase-type plasminogen activator receptor-associated protein 1 (SLURP-1) caused a significant increase in the expression levels of NF-κB, receptor activator of nuclear factor-kappa B ligand (RANKL), and the ratio of RANKL/osteoprotegerin (OPG). These effects were inhibited by alpha-bungarotoxin (a-BTX). Furthermore, the expression levels of RANKL/OPG were significantly reduced following inhibition of NF-κB. High-strength, dynamic positive pressure increased the expression of SLURP-1 and +7 nAChR in DDPSCs in the stable stage.

Conclusions: Mechanical stress stimulated the expression of SLURP-1 and +7 nAChR in DDPSCs. Additionally, SLURP-1 activated +7 nAChR, thereby upregulating the expression of NF-κB and enhancing its activity, thus regulating RANKL/OPG expression and affecting the ability of DDPSCs to influence osteoclastogenesis, which likely enhances root resorption and leads to the physiological loss of deciduous teeth.