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Powerful preclinical mouse model to study live mucin and mucous cell differentiation

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Mucous cells are specialized cells that produce gelling mucins responsible for the mucus gel formation. Modification of mucous cell density and gelling mucin production are established hallmarks of many mucosal diseases including solid tumors (breast cancer, tumors from the digestive, respiratory and reproductive tract), otitis, rhinosinusitis, dry eye, cystic fibrosis, pulmonary fibrosis and asthma. A genetically engineered Muc5b-GFP tagged reporter mouse line at the peptide level was obtained by homologous recombination. Embryonic lung explants allowed to show that interleukine (IL) 13 stimulates Muc5b production. Live Muc5b was easily monitored by probe-based confocal laser endomicroscopy (pCLE) in the nasal cavity, trachea, eye conjunctiva and vagina. As proof of concept that the mouse strain was a valuable preclinical model, we first showed that Muc5b production greatly varied during estrous cycle. We next demonstrated that the decrease in conjunctival goblet cell density monitored by pCLE in living mice with chemically-induced dry eye was reversed by topical application of IL13. The transgenic mouse is unique and suitable for preclinical drug development and suited for pharmacological studies to study the effect of compounds on mucosal homeostasis in living animals.

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Adipose-derived stem cells induce autophagic activation and inhibit catabolic response to pro-inflammatory cytokines in rat chondrocytes

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Aim: Adipose-derived stem cells (ADSCs) have been demonstrated to have an anti-apoptosis effect on chondrocytes. However, their effect on autophagic activation remains unclear. We sought to explore whether ADSCs can activate autophagy and inhibit IL-1β- and lipopolysacccharide (LPS)- induced catabolism in chondrocytes.

Methods: ADSCs and chondrocytes were collected from SD rats. The biologic characteristics of ADSCs were analyzed by flow cytometric analysis, Oil red O and alizarin red staining. Autophagic level and autophagic flux were revealed by western blotting for LC3-II and SQSTM1/P62, MDC (monodansylcadaverine) staining and mRFP-GFP-LC3 analysis. The mTOR pathway was investigated by western blotting for p-mTOR. The mRNA level of matrix metalloproteinases (MMPs) and thrombospondin motifs (ADAMTSs) was detected by real-time PCR.

Results: The typical surface markers and differentiation potentials of ADSCs were proved. ADSCs enhanced the expression of LC3-II/LC3-I and reduced SQSTM1 levels in IL-1β-induced chondrocytes after 24 and 48 hours co-culturing and in LPS-induced chondrocytes after 48 hours co-culturing respectively. mRFP-GFP-LC3 analysis suggested that autophagosomes and autolysosomes were formed earlier in IL-1β-treated chondrocytes than in LPS-treated chondrocytes. Bafilomycin A1 treatment further increased the LC3-II/LC3-I level in chondrocytes in co-culture with ADSCs. The mTOR pathway was inhibited in the chondrocytes in co-culture with ADSCs. Finally, ADSCs inhibited the increase of MMPs and ADAMTSs in chondrocytes induced by IL-1β and LPS.

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