Managing Vocal Fold Fibrosis

Macrophage phenotype, which is regulated by peroxisome proliferator-activated receptor-γ (PPARγ), contributes to fibrosis following vocal fold injury. Using a rat model, Kaba et al (Am J Pathol 2022, 771–782) studied the effect of the PPARγ agonist pioglitazone on vocal cord fibrosis in response to injury. Administration of pioglitazone inhibited accumulation of inflammatory macrophages and improved tissue repair. Targeting inflammatory macrophages and PPARγ activation may help manage vocal fold fibrosis.

Quantifying Fibrosis in Tissue Sections

The amount of fibrosis generally correlates with disease extension; however, its quantification in tissue sections remains variable. Facchin and Certain et al (Am J Pathol 2022, 783–793) studied the use of a semi-automatic, free, and open-source machine learning-based software, FIBER-ML, to quantify fibrosis in diverse tissue sections. Fibrosis was successfully quantified in stained tissue sections from rat cardiomyopathy and mouse nephropathy models. FIBER-ML is a fast, simple, reproducible, and consistent approach to quantify fibrosis in various tissue sections.

Understanding Rapidly Destructive Coxopathy

Inflammasome signaling has been implicated in the progression of rapidly destructive coxopathy (RDC). Using synovial tissue from RDC patients and in vitro co-culture models, Yokota et al (Am J Pathol 2022, 794–804) studied the underlying mechanisms. Activation of inflammasome signaling in the synovium increased local inflammation and osteoclastogenesis and caused rapid bone destruction in RDC. Targeting inflammasome signaling may help manage RDC.

Characterizing Crmp1-Deficient Mice

Though Crmp1 is highly expressed in cochlear hair cells, the expression and localization of the collapsin response mediator protein 1 (CRMP1) protein in the inner ear are unclear. Using Crmp1 null mice, Li et al (Am J Pathol 2022, 805–812) studied the expression and localization of Crmp1 protein in hair cells. Crmp1 is highly expressed in murine cochlear hair cells. Adult Crmp1 null mice show hair loss at the basal cochlear region. Crmp1 deficiency leads to progressive high-frequency hearing loss. Crmp1 is required for high-frequency auditory perception in adult mice.

Inhibiting Prostate Cancer Progression

Cyclin-dependent kinase (CDK) 8 and CDK19 contribute prostate cancer (PCa) progression. Using cultured prostate cancer cells, Offermann and Joerg et al (Am J Pathol 2022, 813–823) studied the underlying mechanisms as well as the anti-tumor effects of CDK8/CDK19 inhibitors. Inhibition of CDK8/CDK19 sensitized prostate cancer cells to androgen blockade, decreased cell migration and invasion via increased adhesion in a collagen I-dependent manner, and highlighted the significance of CDK8/CDK19 kinase function in phosphorylation of downstream signaling molecules. Blocking CDK8/CDK19 may help manage prostate cancer progression.