This Month in *AJP*

**Targeting Lung Fibrosis**
The expression of the E3 ubiquitin ligase WSB1 is associated with hypoxia; however, its role in lung fibrosis is unclear. Using a mouse model of bleomycin (BLM)-induced lung injury and fibrosis and human embryonic lung fibroblasts, Chong and Zou et al (*Am J Pathol, AJPA-D-23-00727*) studied its roles. BLM injury resulted in increased WSB1 expression, which correlated with the progression of lung fibrosis. BLM-induced lung fibrosis was ameliorated by conditional deletion of *Wsb1* in adult mice. Caveolin2 was identified as a downstream target of WSB1 and a functional mediator in lung injury repair and fibrosis. WSB1-Cav2 pathway may be targeted to treat pulmonary fibrosis.

**Managing Psoriasis**
The mechanisms underlying IL-6 up-regulation of the expression of transcription factor LIM-domain only protein 4 (LMO4) in psoriatic lesions are unclear. Using human immortalized keratinocyte (HaCaT) cells, clinical biopsy specimens, and an animal model of imiquimod-induced psoriasis, Tu et al (*Am J Pathol, AJPA-D-23-00561*) studied these mechanisms. Psoriatic epidermis displayed abnormal expression of IL-6 and LMO4. IL-6 upregulates LMO4 expression by activating the MEK/ERK/NF-κB signaling pathway and promotes keratinocyte proliferation and differentiation. Modulating IL-6 may help manage psoriasis.

**Understanding Uremic Sarcopenia**
The mechanisms underlying regeneration defects of uremia-induced muscle injury are unclear. Using a mouse chronic kidney disease (CKD) model and C2C12 myoblast cells, Higashihara and Odawara et al (*Am J Pathol, AJPA-D-23-00293*) studied these mechanisms. Skeletal muscle regeneration deteriorated in CKD mice, which was relieved by treatment with L-ascorbic acid. Transcriptome analysis identified gene expression patterns associated with uremic toxin–induced aberrant myocyte differentiation. Uremic microenvironment impaired the expression of myomixer and impeded myofusion. Defective myocyte fusion and delayed muscle damage recovery may lead to muscle loss and weakness, forming uremic sarcopenia.

**Offering Cytoprotection in Diabetic Enteric Neuropathy**
The role of 5-hydroxytryptamine 4 receptor (5-HT₄R) in diabetic enteric neuropathy is unclear. Using a mouse model of diabetes, Cheng and Kou et al (*Am J Pathol, AJPA-D-23-00433*) studied this role. Diabetes resulted in neural loss in the colon of mice, which was reversed by treatment with 5-HT₄R agonist. RIPK3 and its downstream target mixed lineage kinase domain-like protein (MLKL) were significantly up-regulated in the colon of diabetic mice and were suppressed by

**Unraveling Cellular Functions of Ninjurin 2**
The adhesion molecule, nerve injury-induced protein 2 (NINJ2), is involved in nerve injury and regeneration; however, its other cellular roles remain unclear. Zhang et al (Am J Pathol, AJPA-D-23-00817) generated a Ninj2-deficient mouse model to understand these roles. Ninj2-deficient mice have a shorter life span and are susceptible to spontaneous tumors, systemic inflammation, and metabolic defects. Ninj2-deficiency led to defects in glucose and lipid metabolism and promoted pyroptosis by activating the NLRP3 inflammasome. Ninj2 plays a critical role in tumorigenesis, longevity, cell death, inflammatory response, and metabolism via pyroptosis.