

**Q1 This Month in *AJP*****Managing Alveolar Capillary Dysplasia**

Q2 The mechanisms underlying alveolar capillary dysplasia (ACD) are unclear. Using patient groups with ACD, pulmonary arterial hypertension (PAH), and healthy controls, Kamp and Neubert et al (*Am J Pathol* 2022, XX–XX) studied the microvascular morphology in ACD and the underlying molecular background. Nucleic acid and protein level differences were found in ACD and PAH groups. TEK receptor tyrosine kinase (TIE2)–positive macrophages were markedly increased in ACD, which was characterized by dysfunctional capillaries and a high incidence of intussusceptive angiogenesis. Using anti-angiogenic agents or targeting TIE2 may help manage ACD.

Understanding Neonatal Entesis Healing

The healing capacity of entesis, a hypocellular and avascular tissue, is understudied. To study the efficacy of early-age entesis healing, Vinestock and Felsenthal et al (*Am J Pathol* 2022, XX–XX) established an injury model to the Achilles enteses in neonatal mice. Surprisingly, local extracellular matrix (ECM) secretion by resident cells formed an acellular ECM deposit without inflammation, restoring gait. Neonatal entesis healing may be more functional compared to adult healing.

Reducing Opioid-Induced Gastric Dysfunction

Our understanding of the opioid-induced gastric dysfunction is currently limited. Using a mouse model, Ghosh et al (*Am J Pathol* 2022, XX–XX) studied this role. Opioid use increases the levels of inflammatory cytokines, leading to excess acidity, delayed gastric emptying, and gastric

damage. Morphine-mediated gastric damage results from accrual of acid in the stomach due to increased gastric acid secretion and delayed gastric emptying, which can be reduced by treatment with a proton pump inhibitor. The use of proton pump inhibitors may help manage morphine-associated gastric pathophysiology and palliative tolerance.

Understanding Late-Onset Pompe Disease

Mechanisms leading to the neuromuscular disorder late-onset Pompe disease (LOPD) are unclear. Using LOPD muscle biopsies, Carrasco-Rozas et al (*Am J Pathol* 2022, XX–XX) studied these mechanisms. Atrogene BNIP3 was identified as a potential mediator of muscle fiber atrophy in LOPD muscle biopsies. BNIP3 expression is modulated by the AKT-mTOR pathway. Targeting BNIP3 may help reduce muscle fiber atrophy in LOPD.

Studying Nucleophosmin–Anaplastic Lymphoma Kinase Positive T-Cell Lymphoma

The role of the transcription factor hairy and enhancer of split homolog-1 (HES1) in nucleophosmin–anaplastic lymphoma kinase (NPM-ALK) positive T-cell lymphoma (TCL) progression is unclear. Using patient-derived cultured cells and mouse xenograft studies, Zhang et al (*Am J Pathol* 2022, XX–XX) studied this role. *HES1* is up-regulated in ALK⁺ TCL that is conferred by NPM-ALK–induced STAT3 activation leading to concerted repression of tumor suppressor *TXNIP*. Understanding oncogenic mechanisms exerting lymphoma growth may help identify markers associated with disease severity and might represent therapeutic targets.