Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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This Month in *AJP*

Understanding Taste Loss in COVID-19

COVID-19 patients often complain about loss of taste; however, it is unclear if SARS-CoV-2 can directly infect taste receptor cells. Using biopsied fungiform papillae (FP) from a symptomatic SARS-CoV-2^{+} patient who reported of taste changes and circumvallate papilla from COVID-19 patients who consented to autopsy, Doyle et al (**Am J Pathol 2021, 1511−1519**) studied this possibility. *In situ* hybridization probe and an antibody specific to angiotensin-converting enzyme- 2 (ACE2)—the cell surface receptor that facilitates viral entry—confirmed the co-localization of replicating SARS-CoV-2 and ACE2 in type II cells in the taste buds. Viral infection perturbed patient's FP taste stem cell layer that persisted for 6 weeks after symptom onset. Direct infection of taste papillae by SARS-CoV-2 may result in prolonged taste changes.

Reducing Lung Injury

Recombinant antithrombin (rAT) protects the endothelial glycocalyx from septic vasculitis; however, the underlying mechanisms are unclear. Using lipopolysaccharide (LPS)-induced acute respiratory distress syndrome mouse model, Okamoto, Muraki, and Okada et al (**Am J Pathol, 1526−1536**) studied the effect of rAT treatment on vascular endothelial injury. Treatment with rAT enhanced survival after LPS administration, improved outcomes of LPS-induced sepsis, and up-regulated the expression of genes involved in DNA repair. rAT may limit LPS-induced lung injury by protecting the vascular glycocalyx.

Preventing Epithelial Barrier Impairment

Impairment of epithelial barrier in the gut may result in conditions such as inflammatory bowel disease (IBD). Using mouse models, Castro-Martinez, Candelario-Martinez, and Encarnacion-Garcia et al (**Am J Pathol, 1537−1549**) studied the mechanisms regulating the maintenance of the epithelial barrier. In chemical-induced colitis model, rictor/mTORC2 signaling was reduced in colonocytes of colitic mice and led to decreased Akt activity. The intestinal epithelial cells (IECs) at the crypt surface underwent apoptosis/anoikis, which disturbed the intestinal barrier function. Rictor/mTORC2/Akt signaling was also found to prevent apoptosis/anoikis of surface colonocytes in mice in which *Rictor* was genetically ablated in IEC. Rictor/mTORC2/Akt signaling may be targeted to manage IBD.

Linking Liver Fibrosis and Hepatocellular Carcinoma

The link between liver fibrosis and pathogenesis of hepatocellular cancer (HCC) is unclear. Using mouse liver cell cultures and mouse models of liver cancer, Baglieri et al (**Am J Pathol, 1564−1579**) studied this relationship. Studies were performed using Col$^{r/r}$ mice that are susceptible to fibrosis and produce a collagenase resistant type I collagen and wild type control mice. Col$^{r/r}$ mice showed increased liver fibrosis but decreased HCC in the experimental models. Increased liver type I collagen may not contribute to increased experimental HCC.

Understanding Male Fertility

Both the mutations in autoimmune regulator (*AIRE*) in humans and the loss of *Aire* in mice are linked to infertility. Using male *Aire*-deficient ($Aire^{-/-}$) mice, Warren et al (**Am J Pathol, 1592−1609**) studied the immune targets contributing to infertility. $Aire^{-/-}$ or wild-type (WT) males were paired with WT females. $Aire^{-/-}$ males had hugely reduced mating frequency and fertility, exhibited defects in spermatogenesis, and showed disrupted testicular autoimmunity. Aire is critical in maintaining male fertility and may prevent autoimmunity against multiple reproductive targets.

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