



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.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.



This Month in *AJP*

Molecularly Profiling COVID-19 Patients

Our understanding of COVID-19 pathophysiology is currently limited. Using rapid autopsy tissues from infected and uninfected individuals, Pujadas and Beaumont et al (*Am J Pathol* 2021, 2064–2071) studied the underlying mechanisms. Comparison of COVID-19 tissues with control tissues revealed four main regulatory pathways: blood vessel development, cytokine production, cell activation, and structure and degradation. Effectors within the identified pathways may be targeted to manage COVID-19.

Reversing Aging Lacrimal Dysfunction

Aging affects the lacrimal glands and the microbiota; however, the underlying mechanisms are unclear. Using a mouse model, Jiao and Pei et al (*Am J Pathol* 2021, 2091–2116) studied these mechanisms. Mice were divided into three groups: young, old, and fecal microbiota transplant–treated old groups. Reconstitution of old mice with the microbiome of young mice over time shifted the microbial communities to young donors and significantly reduced the chronic inflammation, lipid deposition, and abnormal neural response of the aging lacrimal glands. Microbiome-based intervention may help reverse age-related dysfunction of the lacrimal glands.

Understanding Endometriosis

The role of casein kinase 1 α (CK1 α)—a widely expressed protein in the endometrium that regulates autophagy—in endometriosis is unclear. Using patient and control clinical samples and cultured cells, Zhou et al (*Am J Pathol* 2021, 2195–2202) studied this role. CK1 α , phosphatase and tensin homolog (PTEN), and autophagy-related 7 (Atg7) were expressed at lower levels in the ectopic endometrium and a positive correlation was observed between CK1 α and PTEN, CK1 α and Atg7, and PTEN and Atg7. CK1 α , PTEN, and

autophagy-related markers were repressed in the endometrial stromal cells. CK1 α regulates PTEN/Atg7-mediated autophagy in human endometrial stromal cells.

Managing Bladder Cancer

The role of the arginine derivative nitric oxide (NO) in bladder cancer invasion is unclear. Using patient samples, cultured cells, and a zebrafish model, Sahu et al (*Am J Pathol* 2021, 2203–2218) studied this role. A stage-associated increase was observed in the expression of NO-generating enzymes, endothelial NOS (eNOS) and inducible NOS (iNOS), in human bladder cancer. Reducing NOS activity decreased cancer cell invasion; whereas, increasing NOS activity enhanced invasion. *In vivo*, reducing NOS activity decreased bladder cancer cell metastasis. The invasive tips of bladder cancer cells, invadopodia, were enriched in NOS proteins as well as mTORC2 activity, which in turn regulated invadopodia formation, eNOS and iNOS expression, and cyclicGMP production in the invadopodia. Blocking NO may help manage bladder cancer.

Attenuating the Growth of Abdominal Aortic Aneurysm

The role of the cytokine B cell activating factor (BAFF) in aortic aneurysms is unclear. By performing prevention and intervention studies in a mouse model of abdominal aortic aneurysm (AAA), Spinosa et al (*Am J Pathol* 2021, 2231–2244) studied this role. The formation of AAA was attenuated by injecting BAFF antagonists before and after the induction of AAA in prevention and intervention studies, respectively. In the intervention group, BAFF antagonism enhanced resolution of inflammation in AAA. BAFF antagonism may deplete mature B cells, promote resolution of inflammation in aorta, and attenuate the growth of AAA.