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Modeling Inflammatory Preterm Birth

Over twenty-five percent of preterm births (PTBs) result from inflammation or infection. Zierden et al (**Am J Pathol 2020, 295–306**) generated a new mouse model of inflammation-related PTB. The common lipopolysaccharide (LPS)-preterm birth model that used a single proximal uterine injection (SPI) was improved with a novel double distal injection (DDI). The total LPS dose was divided into two injections given distally into each uterine horn. Compared to the SPI method, the DDI method enhanced uterine fluid distribution and PTB induction rates at a specific LPS dose, and increased the expression of genes related to myometrial contractility more significantly. The DDI mouse model may help test potential therapeutics against inflammation-induced PTB.

Diagnosing Rickettsial Infection

Mediterranean spotted fever results in high mortality due to a lack of availability of diagnostic tests. Using quantitative proteomics, Zhao et al (**Am J Pathol 2020, 307–322**) analyzed the proteins expressed during *Rickettsia* infection to identify potential biomarkers. Analysis of secreted proteins from primary human umbilical vein endothelial cells infected with *Rickettsia conorii*, and proteins present in the plasma and serum derived from mice and humans infected with *R. conorii* independently identified one rickettsial putative amidase, RC0497, to be differentially expressed. RC0497 may serve as a biomarker for diagnosing *Rickettsial* infection.

Targeting Urothelial Carcinoma *in Situ*

There is an urgent need for effective bladder-preserving therapies for the treatment of urothelial carcinoma *in situ* (CIS). Using laser-capture microdissection and small exome sequencing panel, Garczyk et al (**Am J Pathol 2020, 323–332**) identified therapeutically actionable genomic alterations (GAs) in a cohort of fresh-frozen CIS. The majority of CIS cases displayed at least one potentially actionable GA. These findings may help guide mechanistic studies of urothelial CIS pathogenesis in the future.

Preventing Liver Damage during Regeneration

Liver sinusoidal endothelial cells (LSECs) express hepatocyte growth factor (Hgf), which is critical for prenatal development, metabolic homeostasis, and liver regeneration. Zhang and et al (**Am J Pathol 2020, 358–371**) studied the role of LSEC-derived Hgf in physiologic homeostasis and liver regeneration. Hgf was deleted in mouse LSECs (LSEC-KO) during embryonic phase. Though the liver developed normally, liver regeneration was compromised after partial hepatectomy in LSEC-KO mice. LSEC-derived Hgf may prevent liver damage during regeneration.

Collagen VI Helps Maintain Lung Structure

Despite its widespread presence, the role of collagen VI (COL6) in the lung is unclear. Using established *Col6a1*$^{-/-}$ mice, Mereness et al (**Am J Pathol 2020, 426–441**) studied lung morphology. *Col6a1*$^{-/-}$ mice exhibited altered airway architecture and physiology as well as abnormal behavior of airway epithelial cells. Decreased COL6 expression may impact development or homeostasis, contributing to chronic lung disease.

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