Innate Immunity Mediated by Phagocytosis of Infected Cells

Birkle and Brown (e00476-20) propose and review evidence for a novel mode of innate immunity whereby live, infected host cells induce phagocytes to phagocytose the infected cell, thereby potentially reducing infection. They discuss evidence that live host cells, infected by virus, bacteria, or other intracellular pathogens, release signals that recruit phagocytes to phagocytose the infected cell. Digestion of the infected cell within the phagocyte can curtail host infection but may also result in cross-presentation of pathogen antigens, resulting in innate and adaptive immunity.

Broad Spectrum Human Monoclonal Antibodies for Pneumococcal Disease

Streptococcus pneumoniae remains a leading cause of bacterial pneumonia despite the widespread use of vaccines. Rising infections due to nonvaccine serotypes along with antibiotic resistance among these serotypes have contributed to high disease incidence. An unexplored tool for disease prevention and treatment is the use of human monoclonal antibodies (MAbs) targeting conserved pneumococcal proteins. Huang et al. (e00747-20) isolated the first human MAbs against the pneumococcal histidine triad protein (PhtD), and one of which was shown to protect against pneumococcal disease from two diverse serotypes. The results identify new human MAbs for pneumococcal disease prevention and treatment.

Purinergic P2X7 Receptor Mediates the Elimination of Trichinella spiralis by Activating NF-κB/NLRP3/IL-1β Pathway in Macrophages

Trichinellosis is one of most neglected foodborne zoonoses worldwide. During Trichinella spiralis infection, the intestinal immune response plays a vital role in the host’s resistance. Guan et al. (e00683-20) find that T. spiralis infection upregulated expression of P2X7R and activation of NLRP3 in macrophages during the enteral stage of murine trichinellosis. Moreover, P2X7R mediated the capacity of macrophages to kill the parasite by activating NF-κB/NLRP3/IL-1β pathway. Their findings contribute to the understanding of the intestinal immune mechanism of T. spiralis infection but also offer new insight into the identification of innate resistance during the enteral stage of trichinellosis.

Microbes and Alarmins in the Amniotic Cavity Trigger Distinct Immune Responses in the Fetal Membranes

Microbial and sterile inflammation in the amniotic cavity is causally linked to preterm labor and birth, the leading cause of neonatal morbidity and mortality worldwide. However, the effects of such inflammatory processes on the membranes surrounding the fetus are poorly understood. Motomura et al. (e00819-20) use RNA sequencing to reveal that intra-amniotic microbes induced a severe host defense response in the fetal membranes, including neutrophil function. Notably, sterile intra-amniotic inflammation was milder than intra-amniotic infection, involving the upregulation of alarmins and inflammasome-related molecules. Such findings highlight the distinct nature of these two inflammatory conditions, shedding light on the mechanisms responsible for preterm labor and birth.