**RED ALERT**

**HIGHLIGHTED PAPERS BY JUNIOR INVESTIGATORS**

**No more submission fees for Red Journal articles!**

The ATS has suspended submission fees for articles submitted to the American Journal of Respiratory Cell and Molecular Biology in 2019. Another great reason to submit your best science related to cellular, biochemical, molecular, developmental, genetic, and immunologic studies in health and in acute and chronic disorders related to the respiratory system and sleep!

**Cystic Fibrosis Plasma Blunts the Immune Response to Bacterial Infection**

Cystic fibrosis (CF) is caused by dysfunction of the cystic fibrosis transmembrane conductance regulator and is characterized by chronic lower airway infections. New high-throughput immunogenetic platforms such as transcriptome profiling and RNA-seq can provide a holistic picture of the immune system from a systems immunology perspective. Dr. Xi Zhang (formerly a research assistant professor in the Department of Pediatrics at Ann and Robert H. Lurie Children’s Hospital of Chicago and Northwestern University, and currently an assistant professor in the Department of Pediatrics at National Jewish Health) and colleagues investigated the immune cell response in peripheral blood mononuclear cells. Their findings demonstrate that alveolar macrophage ABCG1 deficiency as a therapeutic target in mitigating lung diseases with goblet cell metaplasia. This work highlights a potential opportunity for SMAD modulation as a therapeutic target in mitigating lung diseases with goblet cell metaplasia.

See article by Feldman and colleagues on page 322.

**Alveolar Macrophage ABCG1 Deficiency Promotes Pulmonary Granulomatous Inflammation**

The etiology of sarcoidosis and the underlying mechanisms that lead to progression of the disease remain largely unknown. Previous studies have identified a deficiency of alveolar macrophage ATP binding cassette lipid transporters in both patients with pulmonary sarcoidosis and mice bearing carbon nanotube–induced granulomas. Utilizing ABC-transporter conditional knockout mice, Matthew McPeek (a Ph.D. student in the laboratory of Mary Jane Thomassen at East Carolina University) and colleagues extend previous work identifying ABCG1 pathways as effectors of pulmonary granuloma formation. They report that alveolar macrophage ABCG1 deficiency promotes the formation of carbon nanotube–induced granulomas and the expression of the inflammatory mediators CCL2 and osteopontin. In addition, ABCG1-deficient mice develop lung fibrosis and increased expression of the profibrotic mediators platelet derived growth factor-α and transforming growth factor-β. These observations correlate with progressive pulmonary lipid accumulation in these animals. Dysfunctional ATP-lipid transporter activity and pulmonary dyslipidemia are known to promote macrophage activation, inflammation, and profibrotic pathways. These findings suggest that alveolar macrophage ABCG1 deficiency may contribute to the pathogenesis and progression of granulomatous lung diseases and may represent a therapeutic target that could be integrated into a broader treatment strategy.

See article by McPeek and colleagues on page 332.

**NIH Corner**

**News from the National Heart, Lung, and Blood Institute**

**NOSI - A New Method for Communicating NIH’s Research Priorities**

The National Institutes of Health (NIH) developed and National Heart, Lung, and Blood Institute (NHLBI) adopted a new way to communicate research priorities to the extramural community. A Notice of Special Interest (NOSI) will be released to identify scientific research topics that are of high interest to the Institute. A NOSI is much like what used to be called a Program Announcement, but it is streamlined by omitting the routine administrative details about how to submit an NIH application. Instead, applicants are simply directed to an appropriate Parent Funding Opportunity Announcement (FOA) that gives instructions for how to apply. In making funding decisions, NHLBI will give special attention to meritorious applications within the scope of a NOSI. A recent example of a NOSI is given at https://grants.nih.gov/grants/guide/notice-files/NOT-HL-19-676.html.

To link your application to a particular NOSI, enter the NOSI number in the Agency Routing Identifier field (4b) of the SF 424. Also, be sure to submit the application in response to the particular FOA (or one of the FOAs) specified in the NOSI. Applicants are encouraged to contact a Program Official listed in the NOSI for queries well in advance of submission. Also, subscribe to the NIH Guide for Grants and Contracts (https://grants.nih.gov/funding/searchguide/index.html#/ ) for weekly updates and new NOSI announcements.