CHINESE-AMERICAN LUNG ASSOCIATION

CALA Happy Friday Seminar

May 6th, 2022

Time: EST 10:30 am; PST: 7:30 am; Beijing time: 10:30pm

Zoom: 849 9682 9273 (Password: 654321)

Towards Understanding the Epigenetic Regulation Of Childhood Asthma

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Bio: Dr. Ji finished her undergraduate at University of Science and Technology of China and obtained her PhD in biological sciences at Vanderbilt University studying telomeres and telomerase. She then moved to Dr. Andy Feinberg's lab at Johns Hopkins School of Medicine and did her postdoctoral training in epigenetics, molecular immunology and cancer. After this, Dr. Ji joined the research faculty at the Cincinnati Children's Hospital Medical Center and started her independent research program in asthma and environmental epigenetics. Her lab is interested in understanding the epigenetic regulation of chronic diseases such as childhood asthma and examining how epigenetic mechanisms mediate the impact of environmental exposures during critical developmental windows (e.g., infancy) on increased disease susceptibility. An integrative approach combining molecular and bioinformatic analysis of methylome, chromatin, gene expression and gene networks with immunologic and physiologic characterization of disease models is utilized to understand disease etiology and progression. Research is performed in animal models, cell culture, and human biologic specimens. In 2018, Dr. Ji joined University of California Davis as a tenure track faculty and as a member of the California National Primate Research Center, expanding her research into the non-human primate model.

Abstract: Dr. Ji's presentation at the CALA seminar series will primarily focus on the role of Tet1 protein, an epigenetic modulator, in regulating asthma pathogenesis. Initially identified from epigenome-wide association studies as a candidate gene for asthma, the role of Tet1 in asthma has been first established by Dr. Ji's group using a mouse model of allergic airway inflammation. Genes and pathways regulated by Tet1 were discovered and single-cell analysis found that alveolar type II cells and ciliated cells were the main targets of Tet1 in the lung epithelium. Several potential mechanisms through which Tet1 regulates the epigenetic landscape to alter gene expression and protect against lung inflammation will be discussed.