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Targeting Cytokine Network as an Immunotherapeutic Modality for Kras Mutant Lung Cancer

Dr. Seyed Javad Moghaddam is a tenured Associate Professor at the Department of Pulmonary Medicine, UT MD Anderson Cancer Center (UT-MDACC), Houston, Texas. He received his medical degree from Shaheed Beheshti University of Medical Sciences, Tehran, Iran. He joined Baylor College of Medicine in 2004 for an NIH T32 fellowship program in lung diseases. Later in 2007, he accepted an Instructor position in the Department of Pulmonary Medicine, UT-MDACC where he has been a faculty since then. He is also a faculty member and lecturer at UT health graduate school of biomedical sciences, as well as other training programs such as the CPRIT-CURE Training Program, and CPRIT Postdoctoral Fellowship in Cancer Prevention Program.

Dr. Moghaddam has received numerous awards including Lung Cancer Discovery Award (American Lung Association), Research Scholar Award (American Cancer Society), and Cyrus Scholar Award in Basic/Translational Research (Cyrus Family Foundation). He is the 2017 recipient of the American Thoracic Society Early Career Achievement Award in Thoracic Oncology where he currently serves as an executive committee member on its Thoracic Oncology Assembly.

Dr. Moghaddam‘s research focuses on airway inflammation and its role in lung tumorigenesis. In his laboratory, they specifically study the cell type and sex-specific roles of oncogene-driven (intrinsic) and COPD-related (extrinsic) lung inflammation with a particular emphasis on cytokine signaling in the pathogenesis and promotion of lung cancer using genetic and pharmacologic approaches for the development of preventive and therapeutic modalities. He has actively published, been well-funded, and trained several postdoctoral fellows, medical students, as well as college, and graduate students in this field.

Abstract: Worldwide, lung adenocarcinoma (LUAD) is the leading cause of cancer mortality because of a high incidence, and a low cure rate. Unfortunately, patients harboring activating mutations of Kras, the most common type of oncogenic alteration in these patients which are heavily caused by tobacco exposure, are resistant to most forms of systemic or targeted therapies and are associated with poor prognosis.

Therefore, there is an urgent and unmet need for novel and alternative approaches to prevent and treat Kras-mutant LUAD. Recent data provide evidence that the inflammatory tumor microenvironment (TME) is one of the main players in lung tumorigenesis, not just a supporting tumor compartment. Our group and others have shown that numerous cytokines released during inflammation can reprogram the lung TME and promote carcinogenesis, introducing inflammation as a vulnerability factor for K-ras mutant LUAD. Accordingly, a better understanding of the lung TME cellular context and the complex bidirectional interplay between the TME and cytokine milieu in the pathogenesis of this deadly disease is needed. Specifically, the role of various immune cells, as well as cytokines and their downstream molecular pathways that can contribute to lung tumor initiation, progression, and metastasis should be explored. This will be fundamental in tailoring rationally directed preventive strategies for high-risk former and current smokers of whom there are more than 90 million in the United States. This could also help to improve the efficacy of currently available therapeutic modalities such as chemotherapy, immune checkpoint blockade, and targeted therapies (e.g. MEK inhibitors). These rationalized strategies that are based on reformatting the lung TME through targeting cytokine networks will be covered by this presentation.