Florencia McAllister, MD
Associate Professor
Clinical Cancer Prevention

Microbes and NETs in Pancreatic Cancer

Dr. McAllister is a physician-scientist with basic science training in Host Defense and Tumor Immunology which she acquired during her postdoctoral training at the University of Pittsburgh and Johns Hopkins University where she trained in the laboratory of Dr. Steven Leach with co-mentorship from Dr. Drew Pardoll, leaders in Pancreatic Cancer and Tumor Immunology, respectively. She has completed 2 clinical fellowships at Johns Hopkins University: in Medical Oncology with focus on Gastrointestinal Medical Oncology and Clinical Pharmacology. She has been recruited to MD Anderson in 2014 and in the past 6 years she has established a translational research program focused on further understanding the fundamental microenvironmental mechanisms that influence pancreatic tumor initiation and progression with the ultimate goal of discovering novel immunopreventive and immunotherapeutic approaches for this disease. She has participated in key discoveries on T cell immunobiology, including unraveling the epithelial IL-17 signaling pathway, the characterization of IL-17-secreting immune cells in the initiation and progression of premalignant pancreatic and colorectal lesions and the key role of inflammation and bacteria -induced T cells responses in the initiation and promotion of pancreatic and colon cancer. Recently, they have published in Cell a study that implicates the gut-tumor microbial axis in modulating the tumor microenvironment, using the powerful approach of human-into-mice fecal microbial transplantation. Furthermore, she has developed a clinical platform, including a gastrointestinal cancer microbiome repository and pancreatic cancer high risk cohort, which will be very relevant to further validate the microbiome preclinical work in a clinically relevant population. Her ultimate goal is to better understand the immune and microbial microenvironment surrounding pancreatic cancer initiation, progression and responsible for therapies responses.

Abstract: Pancreatic ductal adenocarcinoma (PDAC) is resistant to most therapies in part due to an immunosuppressive tumor microenvironment. Using several immunological analytical tools as well as genetic and pharmacological approaches, we assessed for immunosuppressive mechanisms.
We determined that IL-17 mediated neutrophils chemotaxis and induction of neutrophil extracellular traps (NETs), a host defense mechanism, can result in tumor progression and resistance to immunotherapeutics in the context of cancer.