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“STING Signaling in Inflammatory Disease and Cancer.”

“Stimulator of IFN genes (STING) is one of the innate immunity sensors activated by cytosolic DNA, such as cyclic dinucleotides (CDNs) secreted by intracellular bacteria or generated by a cellular cGAMP synthase (cGAS). While transient STING function has found to be essential for protection of the host against viral infection, chronic STING activity by self-DNA leaked from the nucleus has been implicated in causing lethal auto inflammatory diseases such as severe systemic lupus erythematosus (SLE) and STING-associated vasculopathy with onset in infancy (SAVI). Our recent data indicates that STING signaling plays a role of controlling both pro- and anti-inflammatory (IL-10) cytokines expression. Loss of STING reverses colitis observed in the absence of the anti-inflammatory cytokine IL-10 in mice. In addition, the importance of STING in controlling cancer has been demonstrated by noting that extrinsic STING signaling in phagocytes, following the engulfment of cancer cells, is essential for the stimulation of efficient anti-tumor T-cell responses. In this presentation, the molecular mechanisms of how STING plays a role in anti-tumor immune responses will be illustrated to develop a novel therapeutic strategy for converting an immunologically “cold” tumor to “hot”, as well as the role of STING in manifesting inflammatory disease.”

Tuesday, October 23, 2018
12:00p-1:00p
Rosenstiel Medical Science Building
4th Floor Auditorium