세미나 초록

발표주제	Immuno-oncology moves beyond PD-1: Next generation cancer immunotherapy
발표내용	Since the discovery of escaping mechanism of tumor from negative immune regulation, the paradigm of drug discovery for anti-cancer agents has been dramatically shifted to cancer immunotherapy by stimulating patient's immune system to treat cancer. However, the response rate for immune checkpoint inhibitors was still variable and even quite low in a certain type of cancer despite their promising clinical outcome. Immunophenotype of solid tumors is classified with two statuses by tumor microenvironment regulatory pathway - T cell-inflamed phenotype ('hot' tumor) versus non-T cell-inflamed phenotype ('cold' tumor). In the clinical stage, patients with tumor-infiltrating T cell appeared an activation of spontaneous immune response and a benefit with immunotherapy, whereas cancer recurrence was detected by patients with a lack of T cell infiltration. Type I IFN-mediated immune activation is one of the essential parts in the regulation of T cell-infiltration to guide tumor from 'cold' to 'hot'. To facilitate type I IFN signaling for anti-tumor responses, STING (Stimulator of interferon genes) has emerged as an innate immune regulator for the last decade. Based on the critical role of type I IFN signaling in host immune surveillance of cancer, IFNβ production by activation of the host STING pathway induced spontaneous T cell response and tumor regression, which means STING-mediated type I IFN phenotype as a key mechanism of action for targeting non-T cell-inflamed tumor. In this presentation, we discuss the role of STING in nest generation of cancer immunotherapy and the pharmacological effort to activate STING pathway and develop small-molecule based anti-cancer agents.