Centre universitaire de santé McGill
Institut de recherche
McGill University Health Centre
Research Institute

Programme en maladies infectieuses et immunité en santé mondiale
Infectious Diseases and Immunity in Global Health Program
La recherche pour le bénéfice de la santé mondiale
Improving Global Health through Research

Séminaire / Seminar

IDIGH

idigh.ca

@IDIGHProgram

12 mai / May 12 (12:00)

Connectez-vous à / Join the webinar at: idigh.ca/webinars

Jean Tchervenkov MD

Professor of Surgery, McGill University

Director of Renal Transplantation Hepatobiliary and Transplantation Surgery, McGill University Health Centre

The Retinoic Acid Receptor-Related Orphan Receptor (RORγt)—A Potential Key Transcription Factor in Cancer Resistance

The role of the Th17 lymphocyte and its signature cytokine Interleukin-17 has been well recognised as a key mediator of chronic inflammation and autoimmune pathology such as psoriasis, rheumatoid arthritis or multiple sclerosis. In addition to autoimmunity, dysregulated IL-17 is emerging as a major pathogenic factor involved in both the early and late stages of cancer development. Inhibition of IL-17 has also been shown to suppress metastasis and improve the sensitivity to both chemotherapy and radiation therapy in preclinical cancer models. The retinoic acid receptor-related orphan receptor γ and γt (RORγ and RORγt). The master transcription factor for IL-17 is encoded in humans by the RORC gene is expressed in TH17, ILC3, gtTcells and many other innate and adaptive immune cells producing IL-17. Amplification in the RORC gene has been recently described in pancreatic cancer and is correlated with tumor stage and lymph nodes invasion. It has been found to be expressed in many other types of cancers such as breast, colon, prostate, and neuroendocrine and is associated with poor prognosis. We have found in our lab that RORC controls transcription factors NANOG, SOX2, Oct4, and cMyc production in human pancreatic cancer cells and targeting of RORC may be a novel cancer therapeutic option.