

CONFÉRENCE

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Immune Surveillance of Senescent Tumors: Role of Natural Killer Cells

Cellular senescence is an established cellular stress response, which acts primarily to limit the proliferative potential of premalignant cell. It is accepted as one of the main mechanisms by which p53 suppresses tumorigenesis. The senescence response causes a number of alterations in cellular phenotypes including the secretion of soluble factors involved in the maintenance of the senescent state, and others that regulate the immune system, angiogenesis and other processes.

The “Senescence-Associated Secretory Phenotype” (SASP) may in some contexts promote cancer but in others it is believed to have a key role in the control of tumors by the immune system. Thus, we propose that the cellular senescence program cooperates with the innate immune system to potentially limit tumour growth.

This seminar will deal with the role of natural killer (NK) cells and the NK activating receptor NKG2D in the elimination of senescent tumor cells. We propose that at the cellular level, induction of NKG2D ligands can be insufficient to trigger NKG2D-dependent elimination of the cells, and that stress-induced or senescence-specific signals can synergize with NKG2D ligand induction to promote tumor elimination.

During this seminar, we will share our recent data proposing that the p53-dependent signals provided by senescent tumor cells converge to create a “cancer associated pattern” needed to trigger immune surveillance.

Jeudi 15 novembre 2012 à 11 h 30
Pavillon Claire McNicoll, salle Z-300

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