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Invité par : Dr Manu Rangachari

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13h30
Amphithéâtre Fisher, Local TR-54, Site CHUL

«Understanding B cell-mediated pathology in myasthenia gravis (MG) and neuromyelitis optica (NMO)»

Part I. B cell depletion has been shown to affect a decline in autoantibodies and to induce clinical improvement in MG patients. However, the duration of this benefit may be limited, as disease relapses occur. We investigated the mechanisms of such relapses by exploring autoantibody production in the reemerging B cell compartment. We demonstrated that antibody-secreting plasmablasts contribute to the production of MG autoantibodies. These findings introduce potential mechanisms for understanding MG autoantibody production and disease relapse.

Part II. NMO patients harbor circulating pathogenic autoantibodies targeting the aquaporin-4 water channel (AQP4). The source of these autoantibodies remains unclear. B cells tolerance fidelity in NMO patients was assessed; we found that tolerance mechanisms are defective. We further investigated the source of the pathogenic AQP4 autoantibodies; we collected data that suggests they may originate from a pool of B cells that escaped functional counter-selection due to tolerance defects.

Note :
Prière d’avisser vos étudiants gradués et stagiaires postdoctoraux afin d’avoir la participation de tous.