

GUEST LECTURES

RTG 2413

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>> Targeting Intramural Periarterial Drainage
(IPAD) for the treatment of Alzheimer's disease <<

Hosted by: Mahsima Khoshneviszadeh

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03:00 – 04:30 pm

Zoom Lecture

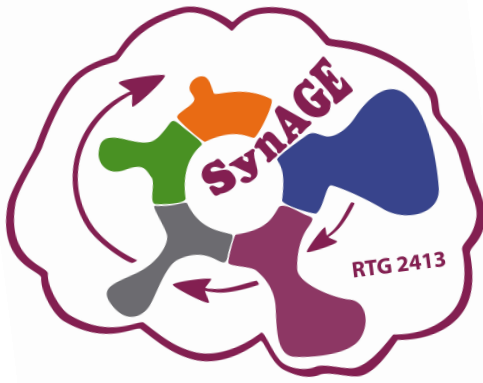
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Abstract

In most tissues of the body, interstitial fluid (ISF), soluble metabolites and inflammatory cells drain to regional lymph nodes along well-characterised lymphatic vessels. The brain has no conventional lymphatics so: How does the brain connect with the immune system? There are two extracellular fluids associated with the central nervous system (CNS): 1) cerebrospinal fluid (CSF) in the ventricles and subarachnoid spaces and 2) interstitial fluid (ISF) between the cells of the brain and spinal cord. CSF drains to regional lymph nodes along lymphatic vessels in the nasal mucosa, via the cribriform plate, and along lymphatics associated with nerve roots and the dura mater. As there are no conventional lymphatics in the brain and spinal cord, ISF and solutes, but not cells, drain to cervical lymph nodes along 100-150 nm thick basement membranes in the walls of cerebral capillaries and arteries. This route is termed the Intramural Peri-Arterial Drainage (IPAD) pathway. IPAD is almost completely separate from CSF and only 10-15% of solutes leak from IPAD pathways into the CSF. Furthermore, as IPAD pathways are not large enough to allow the direct traffic of inflammatory cells from the brain to cervical lymph nodes, this may be a major factor in “immunological privilege” of the brain with implications for Neuroimmunology and diseases such as Multiple Sclerosis. Age-related Impairment of IPAD has important implications for Neurodegenerative diseases, especially Alzheimer's disease. Soluble Amyloid- β ($A\beta$) is eliminated from the brain along IPAD pathways. With age changes in the walls of arteries, IPAD is impaired and $A\beta$ is deposited in IPAD pathways as cerebral amyloid angiopathy (CAA) with consequent effects on homeostasis in the brain. The motive force for IPAD is provided by the spontaneous contractions of vascular mural cells and targeting them promises to be a novel and efficient way of facilitating IPAD and preventing/treating Alzheimer's disease.

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