THE UNIVERSITY OF ARIZONA
COLLEGE OF MEDICINE TUCSON
Pharmacology

Presents

"Non-cell-autonomous mechanism in oligodendrocyte regeneration after white matter damage"

By

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Host: Dr. Patrick Ronaldson

Abstract: Oligodendrocyte precursor cells (OPCs) serve as progenitor cells of terminally differentiated oligodendrocytes. Mature oligodendrocytes form myelin sheaths around axons in the central nervous system, and the myelin sheath is essential in the fast impulse propagation along with the myelinated fiber. Because oligodendrocytes do not proliferate, OPCs play a critical role in increasing the number of oligodendrocytes during development or after oligodendrocyte/myelin damage. Although mechanisms of OPC differentiation have been examined and some extrinsic signaling molecules were identified as regulators of OPC differentiation into oligodendrocytes, precise mechanisms of OPC proliferation and differentiation still need to be elucidated, especially under the conditions of cerebrovascular diseases. As highlighted by the concept of the neurovascular unit, cell-cell interactions should be critical in supporting/maintaining OPC function, including oligodendrocyte generation. In my presentation, I will present some key data from my laboratory regarding the roles of endothelial cells, astrocytes, and pericytes in OPC proliferation and differentiation. Because OPC is one of the major cell types in adult white matter, understanding and dissecting OPC-related non-cell-autonomous mechanisms of oligodendrocyte regeneration may lead to novel opportunities for white matter recovery in cerebrovascular diseases.

Wednesday, September 15, 2021
11:00 am – Noon - AHSC 8403
Or by Zoom: https://arizona.zoom.us/j/84524481802
Meeting ID 845 2448 1802