Dr. Pablo Paez

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Assistant Professor of Pharmacology & Toxicology

Pablo M. Paez PhD is a Principal Investigator of the Hunter James Kelly Research Institute and Assistant Professor in Pharmacology and Toxicology in the School of Medicine and Biomedical Sciences, University at Buffalo. He graduated in Biochemistry from the University of Buenos Aires, Argentina (2000), and he obtained a PhD degree in Neurochemistry from the same university in 2005. He was a Neuroscience post-doctoral fellow of Dr. Anthony T. Campagnoni at the University of California, Los Angeles (2006-2010), where he won a Postdoctoral Fellowship Award from the National Multiple Sclerosis Society (2007) to study the regulation of oligodendrocytes development.

Dr. Paez's research program focuses on brain development, studying the development of the oligodendroglial and astroglial cell lineages in the central nervous system in normal, mutant and transgenic mice. The primary focus of the Paez laboratory is on ion channels that regulate specification, migration and differentiation of these glial cells.

Dr. Paez has recently discovered that voltage-gated Ca^{++} channels are necessary for normal myelination acting at multiple steps during oligodendrocytes development. Dr. Paez's research aims to determine if voltage-gated Ca^{++} channels plays a functional role in myelin repair. Using transgenic mice and new imaging techniques the lab is currently testing the hypothesis that voltage-gated Ca^{++} entry promotes oligodendrocyte progenitor cells survival and proliferation in the remyelinating adult brain. This work is relevant to developing means to induce remyelination in myelin degenerative diseases and for myelin repair in damaged nervous tissue.

Additionally, the Paez lab has made the novel finding of voltage-gated Ca^{++} channels function in astrocyte Ca^{++} homeostasis, and this has implications for plasticity in astrocyte development and for Ca^{++} regulation in general. Dr. Paez's lab is testing the hypothesis that voltage-gated Ca^{++} entry plays a key role in astrocyte function and glial-neuronal interactions. Astrocytes are the most abundant cell of the human brain. They perform many functions, including biochemical support of endothelial cells that form the blood brain barrier, provision of nutrients to the nervous tissue and a role in the repair and scarring process of the brain and spinal cord following traumatic injuries. Therefore, understanding the role of ion channels on astrocyte development and function could lead to novel approaches to intervene in neurodegenerative diseases and brain injuries in which inflammation and astrogliosis play a detrimental role.

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