NeuroInflammation: How to evaluate potential contributions to neurotoxicity

Inflammation is a dominant theme in contemporary biomedical research, including neuroscience. Neuroinflammation has been implicated in neurodevelopmental disorders (autism), psychological disorders (schizophrenia, depression), pharmacological addiction (amphetamine, alcohol), neurodegenerative diseases (Alzheimer’s Disease, Parkinson’s Disease), and in processes of neurotoxicity following chemical or pharmaceutical exposure. Neuroinflammation has often been considered detrimental to the nervous system; however, innate immune system responses are critical to maintain cellular and environmental homeostasis and thus, also play a beneficial role. Identifying cellular origins of inflammatory factors, regulatory signaling, and individual cell functions is critical to understanding the contribution of neuroinflammation to the initiation, progression, exacerbation of a neurological disorder. Distinguishing beneficial from detrimental effects of a neuroinflammatory response and the impact that chemical or pharmaceutical exposure can have on this tightly regulated process is critical in addressing the contribution of pro-inflammatory and anti-inflammatory signaling and the activation of neuro-immune cells to neurotoxicity.

Biography

G Jean Harry, PhD, is Head of the Neurotoxicology Group. She obtained an MS in Neuropharmacology from Virginia Commonwealth University with a research focus in drugs of abuse. Her PhD was obtained from VCU in 1981, and bridged the fields of neuropharmacology and neurotoxicology with her research conducted at NIH. Postdoctoral work was conducted in an NIH Training Program in Neuropathology followed by an NIH independent fellowship award in the Biochemistry Department, University of North Carolina. Following a position within the Developmental Disorders Center at UNC, she joined NIEHS as head of Neurotoxicology Group in 1990.

harry@niehs.nih.gov