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Preface

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Neurodegenerative disorders constitute the major theme published in the journal *Neurotoxicity Research*. What are the molecular and cellular mechanisms initiating neurodegeneration? What are the therapeutic interventions that could limit neuronal cell death? To answer these questions, this special issue collects contributions from authorities working in the areas of pathology, biomolecular mechanisms, animal models, and therapeutic approaches. The panel was assembled at Georgetown University during the 2008 Society for Neuroscience satellite meeting organized by the Neurotoxicity Society.

Three major scientific thrusts in particular have converged to produce this special issue. First, the need of discussing and understanding the central problem of similarities and differences among neurodegenerative disorders. Do they all present a similar pathological characteristic? Are they caused by an acute injury or by a chronic neuronal degeneration? A central theme emerging here is the duality of function of glial cells, which, while serving and supporting neurons in health, become the very mediators of neuronal degeneration in a disease setting. Second, the urge to find answers as to what causes neuronal cell death, and in particular what cellular and molecular signals promote neurotoxicity. Crucial experimental observations derived by exposing neurons in vitro to different types of insults are presented. Oxidative stress has been the defining and best-studied example, but many others are also emerging including pathways involving chemokines and pro-neurotrophins. Third, the necessity of presenting advantages of using animal models of human diseases to study acute (trauma) and chronic neurodegeneration (e.g., Parkinson's and Alzheimer's diseases and AIDS dementia). Transgenic mouse models of chronic diseases, whether by gene insertion or viral vector delivery, are also discussed. Is their pathology similar to that in humans? The eternal debate of the appropriateness of animal models of neurodegenerative disorders is not resolved in this issue. However, these models have made it possible to peer into the pathogenesis of neuronal death as it occurs, to develop specific hypotheses about underlying mechanisms, and to determine whether selective experimental therapies hold the potential to ameliorate neuronal cell loss in humans.

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