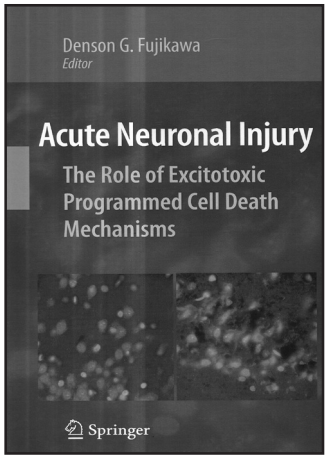
FUJIKAWA, D. G. (Ed.). *Acute neuronal injury:* the role of excitotoxic programmed cell death mechanisms. New York: Spring, 2010. 306p.



Acute Neuronal injury is a book written by Professor Denson Fujikawa (Adjunct Professor of Neurology at the David Geffen School of Medicine at UCLA, a member of the Brain Research Institute at UCLA and a Staff Neurologist at the Department of Veterans Affairs Greater Los Angeles Healthcare System) that deals with scientific information related to the molecular mechanisms triggering neuronal death in the central nervous system. Experimental evidence reinforces that stroke, brain and spinal cord trauma, coma from a low serum glucose concentration (hypoglycemia), and prolonged epileptic seizures share some mechanisms of neurodegeneration through excitotoxicity associated with excessive activation of glutamate receptors by an elevated extracellular glutamate concentration. The excitotoxic response results in an excessive influx of calcium into nerve cells. The high calcium concentration in nerve cells activates several enzymes, such as nitric oxide (NO) synthase, leading to NO production, which reacts with superoxide to produce peroxynitrite (a toxic free radical). This and other reactive oxygen species damage the plasma membrane, being responsible for degradation of cytoplasmic proteins and cleavage of nuclear DNA, resulting in nerve cell death. This high calcium concentration also interferes with mitochondrial respiration which also damages cellular membranes and nuclear DNA through imbalance of free radical production. In recent years, attention has focused on the study of apoptosis and programmed cell death pathways that are activated by the apoptotic cell death process. Both the intrinsic (mitochondrial) caspase pathway and the extrinsic (death receptor) pathways were discussed in this book. However, readers of this book can also find discussion about necrotic cell death which is not considered merely a passive process with cell swelling and lysis, but one that can also involve excitotoxic programmed cell death mechanisms, and also on developmental autophagic cell death. Therefore, I consider Acute Neuronal Injury a useful book for neuroscientists and general cell biologists interested in cell death. The book would also be helpful to clinically oriented neuroscientists, including neurologists, neurosurgeons and psychiatrists. Understanding the biochemical pathways that produce cellular death is the first step towards the development of therapeutic strategies that can effectively produce neuronal protection or restore the neuronal loss caused by aging or disease processes.

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