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Genomic mosaicism and Alzheimer's disease

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The human brain is composed of hundreds of billions of cells that are largely assumed to contain identical genomes, from one cell to the next. This view, however, is wrong, especially with respect to neuronal genomes that may be unique despite arising from a single zygote. Genomic variation can take multiple forms, ranging from aneusomies – gains/losses of chromosomes – to smaller copy number variations (CNVs) and single nucleotide variations (SNVs). In addition to sequence differences, the sum-total of these changes within a single nucleus can be detected by DNA content flow cytometry, which has revealed surprising heterogeneity of DNA content variation (DCV) amongst neurons of the human brain, producing a complex genomic mosaic of cells within the brain. Notably, these increases can be accentuated in sporadic Alzheimer's disease (SAD), the most common form of AD. Some of this increase has been attributed to CNVs in amyloid precursor protein (APP) gene but produced somatically and mosaically within neurons. Implications of genomic mosaicism for AD will discussed.

Biography

Jerold Chun is a Professor of Degenerative Diseases Program. He is Senior Vice President, Neuroscience Drug Discovery, Neuroscience and Aging Research Center. Jerold Chun's research focus on Genomic Mosaicism in the brain, Lysophospholipid Receptor Signaling and Neuroscience Drug Discovery

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