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Charnoly body as a novel biomarker in drug addiction

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Charnoly body (CB) is a pre-apoptotic biomarker of compromised mitochondrial bioenergetics, which is formed in the most vulnerable cell in response to nutritional stress, environmental toxins, or drug of abuse due to free radical overproduction and mitochondrial genome down-regulation. It is detected as a pleomorphic multi-lamellar, electron-dense, membrane stack of degenerated mitochondrial membranes in the hippocampal CA-3 and dentate gyrus neurons, hypothalamic neurons, and cerebellar Purkinje neurons in animal models of fetal alcohol syndrome, Parkinson's disease, Alzheimer's disease, vascular dementia, and chronic drug addiction. Accumulation of CB at the junction of axon hillock impairs axoplasmic flow in the synaptic terminals to cause cognitive impairments, early morbidity, and mortality in chronic drug addiction. Initially $\Delta\Psi$ collapse, down-regulation of mitochondrial -NADH-oxidoreductase, and 8-OH-2dG can be detected as CB rudiments to evaluate early symptoms of acute drug addiction as epigenetic modulators of DNA methylation and histone acetylation. During chronic phase, CB formation can be detected at the ultrastructure level. Antioxidants such as Metallothioneins, inhibit CB formation as free radical scavengers by regulating zinc-mediated transcriptional activation of genes involved in growth, proliferation, and differentiation as established in gene-manipulated human dopaminergic (SK-N-SH and SHY5Y) cells and in mouse models of multiple drug abuse. Hence novel drugs may be developed to prevent CB formation or induce charnolophagy as an efficient molecular mechanism of intracellular detoxification during acute phaseand novel CB antagonists to avert chronic drug addiction by employing CB as an early, sensitive and specific biomarker to detect, prevent and effectively treat drug addiction.

Biography

Sushil Sharma is a Professor of Pharmacology & Course Director at Saint James School of Medicine, Kralendijk, Bonaire, Dutch Caribbean.

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