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Modulation of endogenous cholinergic cytoprotection in Ischemic stroke

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The cholinergic system is essential for maintenance of cognitive, autonomic and immune homeostasis in mammals. Pre-clinical studies utilizing rodent models of ischemic stroke suggest that endogenous cholinergic tone elevated by injury, infection and/or inflammation serves as a combination therapy aiming at multiple cellular and molecular pathways with converging anti-inflammatory cytoprotective efficacies. These hardwired endogenous protective mechanisms can be augmented by cholinergic treatments including nicotinic acetylcholine receptor (nAChR) agonists, positive allosteric modulation and vagus nerve stimulation. Strategies that augment endogenous cholinergic protective mechanisms are expected to selectively target ischemic brain injury with high spatiotemporal precision. The α7 subtype of nAChRs is uniquely positioned as a promising therapeutic target in ischemic stroke because of the high anti-inflammatory cytoprotective efficacy of α7 nAChR activation and the ubiquitous expression of α7 nAChRs in mammalian neuronal, glial and immune tissues. The injury-induced endogenous α7-dependent auto-therapy may act as an important physiological function of these receptors. Selective cholinergic agents that enhance endogenous protective mechanisms may hold significant translational potential.

Speaker Biography

Victor V Uteshev has made the long-term goal of his laboratory to help develop clinically useful drug therapies that will ameliorate or even restore cognitive and autonomic functions in patients with age-, disease- and trauma-related impairments. His research concentrates on the positive effects chemicals such as nicotine may have on the brain – as in enhancing cognitive performance and resistance to brain injury, particularly in aging patients and people who have high risk for stroke and traumatic brain injury. He is focusing on compounds that are similar to nicotine but can bring mostly positive effects. In many diseases and pathological conditions, the brain doesn't create enough nicotine receptors and natural compounds that activate these receptors. In diseases and conditions such as schizophrenia, Alzheimer's and traumatic brain injury, the level of activation of nicotine receptors is deficient and cannot support normal brain function. His team's goal is to develop clinically tested drug therapies that can compensate for these deficits and improve or restore cognitive and autonomic functions in patients with age-, disease- and trauma-related impairments. He also serves as an Adjunct Associate Professor in the Department of Pharmacology of Southern Illinois University School of Medicine at Springfield, Ill., where he previously served as an Assistant Professor.

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