NUS National University of Singapore | Life Sciences Institute

Neurobiology/Ageing Programme

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"$GABA_A$ receptor subtypes and VTA dopamine neuron plasticity in drug addiction"

Biography

Esa R. Korpi, MD, PhD, is professor of pharmacology and chair of the Institute of Biomedicine, University of Helsinki, Finland. He got his MD and PhD degrees at the University of Tampere (both 1980), did his postdoc period as a Fogarty visiting fellow at St. Elizabeths hospital, Adult Psychiatry Branch, NIMH, Washington DC, 1981-1983, and worked as a researcher at the Research Laboratories of the Finnish State Alcohol Company Alko 1984-1996. Thereafter, he acted as the professor of pharmacology at the University of Turku 1996-2002. He has spent sabbatical periods at the Center of Molecular Biology, University of Heidelberg, Germany 1991-1992, at the University of Mainz, Germany, at the Vanderbilt University Nashville TN and NIAAA Rockville MD USA 2000-2002. He chaired the Brain Research Society of Finland 2000-2002 and the Drug Research Foundation 2006-2008. He has been a member of the Finnish Academy of Science and Letters since 2007. He has published over 250 original articles and reviews in the fields of neuropsychopharmacology, neurobiology of addiction and brain diseases.

Abstract

Midbrain dopamine neurons are involved in motivational processes, including early phases of drug addiction. Even the first in vivo dose of various abused drugs induces glutamate receptor plasticity at the excitatory synapses of VTA dopamine neurons, while withdrawal from their chronic use is often associated with reduced activity of the dopamine neurons. More recent studies have suggested that there is heterogeneity in VTA dopamine neuron populations and that some neurons are actually activated by stress and aversive signals. The main fast inhibitory neurotransmitter system using GABA and $GABA_A$ receptors is functional in the VTA, and benzodiazepines that activate synaptic $GABA_A$ receptors produce glutamate neuroplasticity after one dose. Furthermore, gaboxadol (THIP), an agonist for extrasynaptic $GABA_A$ receptors, also induces similar persistent plasticity in dopamine neurons, but behaviourally it produces long-lasting aversion. Molecular and neuronal circuitry mechanisms of drug-induced neuroplasticity are being explored to understand the functions of different dopamine neuron populations.

20 December 2012, Thurs / 3.00pm / Centre for Life Sciences (CeLS) Seminar Room 1 / Convenor: A/P Lim Kah Leong