

5th World Congress on

Neurology and Therapeutics

March 14-16, 2016 London, UK



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HIV-1/AIDS-associated neuropathic pain

Neuropathic pain is a common neurological disorder in HIV-1/AIDS patients. Current therapies for HIV-associated neuropathic pain are largely ineffective. Multiple etiological factors may contribute to the pathogenesis of this pain syndrome, including HIV-1 infection and antiretroviral medicine. Analysis of the pain neural pathway in postmortem tissues of HIV-infected patients leads to identifying specific neuropathologies that may causally link to pain development in the patients. We are interested in understanding the pathogenic mechanism for the ultimate development of rationale-based therapeutic approaches. Our work has been focused on identifying the HIV-1 pathogenic factor and elucidating the molecular and cellular processes through which the factor causes neuropathic pain. We have undertaken an interdisciplinary approach in this research, including analyzing postmortem nervous tissues from HIV-1/AIDS patients, generating clinically relevant rodent models, and determining the molecular, cellular, behavioral and electrophysiological abnormalities in the model. Our results indicate that HIV-1 gp120 is a relevant viral factor for the HIV neuropathic pain. We also identify that Wnt signaling in the pain neural circuit plays a key role in mediating the activity of gp120 to cause major neuropathologies of HIV-associated pain such as neuro-inflammation, astroglial reaction and neuropathy. Our studies may provide novel insights into the molecular, cellular and circuitry mechanisms of HIV-associated neuropathic pain.

Biography

Shao-Jun Tang completed his PhD from the University of Toronto in Canada in 1998 and Post-doctoral studies from the HHMI/California Institute of Technology in USA in 2001. He is currently the William Willis Jr. MD PhD Distinguished Professor in Neuroscience and the Director of Neuroscience Graduate Program in the University of Texas Medical Branch.

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