

School of Pharmacy

Division of Pharmacology

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Academic Background:

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| Research Associate/Senior Postdoctoral | Scripps Research Institute, La Jolla, 1999-2003 |
| Ph.D. in Pharmacology | National Institutes of Health (NIH), 1997-1999 |
| M.S. in Immunology | Louisiana State University (LSU), 1997 |
| M.D. | Second Military Medical University, China, 1988 |
| | Second Military Medical University, China, 1983 |

Research Interests:

Cytokines are the signaling molecules in neuro-immune communication and are intimately involved in brain physiology and pathophysiology. Our goals are to elucidate what role these molecules play in neurological and neuropsychiatric disorders as well as normal neurodevelopment. Our current focus is on interferons (IFNs). Type I interferon IFN- α , for example, is a key first line anti-viral cytokine through Janus kinase (JAK)/signal transducer and activator of transcription (STAT) signaling pathway. As the most used cytokine clinically, type I IFN has been successfully used to treat patients with chronic HCV infection, a number of different malignancies and relapsing-remitting multiple sclerosis. In addition to its anti-viral, anti-tumor and immunoregulatory activity, however, IFN- α has also been implicated in the pathogenesis of several CNS disorders, such as NeuroAIDS (HIV dementia), CNS lupus, Aicardi-Goutières syndrome and Cree encephalitis. Furthermore, chronic systemic therapy of IFN- α in patients results in severe neuropsychiatric side effects including depression, anxiety and cognition impairments.

Our current study is to elucidate the mechanisms of neuropathogenic/neurotoxic actions of interferons (IFNs) employing integrated neurobehavioral, neuropathological, pharmacological, molecular and genetic approaches. There are two projects in progress in our laboratory: one project is to develop a mouse model for human depression resulting from chronic IFN- α therapy in which to investigate the neurobehavioral deficits due to chronic IFN- α treatment and elucidate the underlying mechanisms. The other project is to investigate interferons and IFN signaling in cerebellar development and to explore the possible direct link between infection/inflammation and medulloblastoma, the most common pediatric malignancy in the brain.

Techniques Used in Laboratory:

RNase protection assay (RPA); Western blot; immunohistochemistry; in situ hybridization; primary neuronal culture; global gene profiling (microarray analysis); application of genetically manipulated animals (both transgenic & knockout mice); enzyme-linked immunosorbent assay (ELISA); radioimmunoassay (RIA); depression- and anxiety-like behavior battery.

Representative Publications:

Wang, J. and Campbell, I.L. (2005). Innate STAT1-dependent genomic response of neurons to the anti-viral cytokine alpha interferon. **J. Virology** 79:8295-8302.

Wang, J., Lin, W., Popko, B. and Campbell, I.L. (2004). Induced production of interferon- γ in the developing brain causes cerebellar dysplasia with activation of the sonic hedgehog pathway. **Mol. Cell. Neurosci.** 27:489-496.

Dunn, A. J. & **Wang, J.** (2004). Cytokines and the brain. In: **Elsevier's Encyclopedia of Neuroscience** (Ed. Adelman, G. and Smith, B.H.), Elsevier Science Press, 3rd Edition.

Wang, J., Pham-Mitchell, N., Schindler, C. and Campbell, I.L. (2003). Dysregulated sonic hedgehog signaling and medulloblastoma consequent to IFN- α -stimulated STAT2-independent production of IFN- γ in the brain. **J. Clin. Invest.** 112:535-543

Wang, J., Schreiber, R.D. and Campbell, I.L. (2002). STAT1 deficiency unexpectedly and markedly exacerbates the pathophysiological actions of IFN- α in the CNS. **Proc. Natl. Acad. Sci. USA.** 99:16209-16214.

Wang, J., Valerie, C.A. and Campbell, I.L. (2002). Cytokines and chemokines as mediators of protection and injury in the central nervous system assessed in transgenic mice. **Current Topics Microbiol. Immunol.** 265:23-48

Griffiths, M.M., **Wang, J.**, Joe, B., Dracheva, S., Kawahito, Y., Shepard, J.S., Reese, V.R., McCall-Vining, S., Hashiramoto, A., Cannon, G.W., Remmers, E.F. and Wilder, R.L. (2000). Identification of four new quantitative trait loci regulating arthritis severity and one new quantitative trait locus regulating autoantibody production in rats with collagen-induced arthritis. **Arthritis Rheum.** 43:1278-1289.

Dracheva, S.V., Remmers, E.F., Chen, S., Chang, L., Gulko, P.S., Kawahito, Y., Longman, R.E., **Wang, J.**, Du, Y., Shepard, J., Ge, L., Joe, B., Kotake, S., Salstrom, J.L., Furuya, T., Hoffman, J., Cannon, G. W., Griffiths, M.M. and Wilder, R.L. (2000). An integrated genetic linkage map with 1,137 markers constructed from five F2 crosses of autoimmune disease-prone and -resistant inbred rat strains. **Genomics** 63:202-226.

Wang, J. and Dunn, A. J. (1999). The role of interleukin-6 in the activation of the hypothalamo-pituitary-adrenocortical axis induced by endotoxin and interleukin-1 β . **Brain Res.** 815:337-348.

Wang, J. and Dunn, A. J. (1998). Mouse interleukin-6 stimulates the HPA axis and increases brain tryptophan and serotonin metabolism. **Neurochem. Internl.** 33:143-154.

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