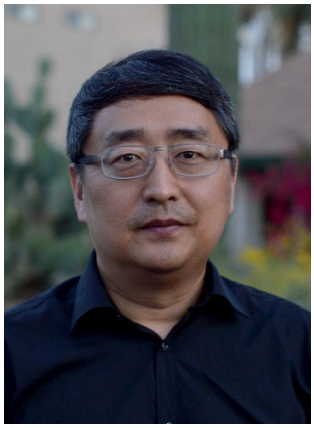
THE UNIVERSITY OF ARIZONA
COLLEGE OF MEDICINE TUCSON
Pharmacology

Presents

"MET receptor tyrosine kinase: from neurodevelopment to neurodegeneration"

BY



Shenfeng Qiu,
Professor, Basic Medical Sciences
University of Arizona, Phoenix
College of Medicine

Abstract: The long-term mission of my laboratory is to understand how genetic risk factors predispose an individual for neurodevelopmental disorders, including autism, by affecting the normal brain development trajectory and its physiological function. We strive to apply a 'precision medicine in psychiatry' approach to dissect how risk genes affect specific neuronal types and circuits across the protracted neurodevelopmental timeline. I am also keenly interested in neurocircuit basis of behavior, and molecular and synaptic basis of aging and neurodegeneration. One NIH-funded research project (K99-R00-R01 is to investigate the role of MET receptor tyrosine kinase, an established prominent autism risk factor, on forebrain development. Our findings suggest that MET signaling serves as a critical intrinsic neural mechanism that regulates dendritic spine formation and synaptogenesis. Current lab efforts focuses on important roles of MET in synapse pruning, maturation, refinement of circuit connectivity, and the emergence of social behavior, by utilizing several novel Met mutant mice lines created in our lab. In addition to its neurodevelopmental role of MET, MET signaling may play a key role in aging/degenerating brain. Recent findings indicate that MET protein, heavily expressed in the excitatory neurons during early brain development, is reduced in AD brain. The sole ligand for MET, hepatocyte growth factor (HGF), found to be primarily expressed in astrocytes, is elevated in the cerebrospinal fluid in AD patients, and is associated with small vessel pathology in AD and dementia. Activation of MET by HGF initiates a pleiotropic signaling that is neurotrophic and neuroprotective in multiple neurodegenerative mouse models ranging from multiple sclerosis, Parkinson's disease, to ALS. Built on these key literature and preliminary findings, we are currently testing the hypothesis that re-activating MET signaling promotes functional synaptogenesis and protects against synapse loss and degradation of circuit connectivity associated with aging, neurodegeneration, and other neurological conditions

Wednesday, September 11, 2024
11:00 am – Noon
AHSC - Room 8403
https://arizona.zoom.us/j/82268538546