CASE STUDY

Inflammation in Alzheimer's disease

Who: Melanie Das, PhD
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Melanie Das, PhD, is a Postdoctoral Scholar in Dr. Lennart Mucke's laboratory at the Gladstone Institutes. Her work aims to identify the mechanisms by which inflammation and abnormal neuronal activity contributes to synaptic loss in Alzheimer's disease. She received her PhD in Biomedical Sciences at Cedars-Sinai Medical Center in Los Angeles, California.

nCounter® Assay selection:

nCounter Mouse Neuroinflammation Panel

Project Summary:

Genome-wide association studies have identified numerous immune gene variants as risk factors for AD, implicating immune processes in AD pathogenesis. Yet, how exactly immune cells, particularly microglia, contribute to AD pathology remains unclear. Mounting evidence suggests a reciprocal regulation of neuronal activity and inflammatory processes. Normal neuronal activity during development drives microglia-mediated synaptic modulations. Neuronal network hyperactivity in epilepsy triggers microglial activation and inflammation. In turn, microglia and inflammatory mediators can regulate neuronal excitability and synaptic transmission, which may subsequently regulate network activity. Neuronal activity can regulate multiple inflammatory processes including the complement cascade, the NLRP3 inflammasome, and fractalkine and purinergic signaling pathways. NanoString's Neuroinflammation Panel will accelerate our study by allowing us to assess the contribution of network dysfunction in hAPP mice to all of these pathways. In addition, we hope to identify novel pathways activated in microglia and other glial and immune cells. The reproducibility of the NanoString assay will allow us to reliably repeat these experiments with other mouse models of AD and other manipulations of neural activity.

"The idea is to identify what inflammatory mediators are increased in our Alzheimer's disease model and whether drugs that decrease abnormal neuronal network activity can reduce these inflammatory processes. This panel will allow us to identify multiple inflammatory pathways that may be linked to neuronal activity."

Melanie Das, PhD

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JUL 2018 MK0887-1