September 10th, 2018 4-5pm - Speaker: Shelly Erickson, PhD, Research Assist. Professor, Division of Gerontology, UW. *Serum amyloid A proteins and their interactions with the blood-brain barrier in Alzheimer's disease.* Location: Rm 812, Bldg 1, VA Puget Sound.

Serum amyloid A (SAA) proteins accumulate in the brain parenchyma and CSF in humans with Alzheimer's disease. Current evidence shows that blood levels of SAA are elevated in Alzheimer's co-morbid conditions; circulating SAA can cross the intact blood brain barrier (BBB); SAA accumulates in the brain when overexpressed in the liver; hepatic overexpression of SAA alters neurobehavioral outcomes; and SAA can impair clearance of amyloid beta from the brain.

Dr. Erickson is interested in BBB dysfunction as a potential driver of Alzheimer's disease. Specifically, substances in the blood could interact with brain endothelial cells to modulate their function, via disruption, immune cell trafficking, transport, or communication with other CNS cells.

For her pilot project, Dr. Erickson will investigate questions related to SAA interactions with the BB in Alzheimer's disease, guided by the hypothesis: "Chronically increased SAA in blood leads to elevated SAA in the brain, which promotes neuroinflammatory response and increases the accumulation of amyloid and tau in the brain, and exacerbates cognitive decline in AD.
She aims to determine whether SAA overexpression exacerbates AD-associated neuropathology in a mouse model of AD. She will then determine whether SAA protein levels in the blood and brains of human subjects are associated with AD pathological burden and dementia, leveraging autopsy specimens from the ADRC/ACT studies.

In the future, Dr. Erickson plans to use isopleuripotent stem cells (iPSCs) to study the effects of SAA on HDL-mediated protection of the BB against inflammatory stimuli. This in vivo model of AD could provide more knowledge about the brain endothelial function. Potentially, iPSCs could be used as a screening tool for blood factors capable of modulating brain endothelial function. Collaborators are welcome to join in this effort.