

Nicole Crawford<sup>1</sup> • Peter Nelson, MD, PhD<sup>2</sup> • Adam Bachstetter, PhD<sup>3</sup>

<sup>1</sup>SCoBIRC, University of Kentucky • <sup>2</sup>Sanders-Brown Center on Aging, University of Kentucky • <sup>3</sup>SCoBIRC / Neuroscience, University of Kentucky

***Abstracts will be considered for both poster and platform presentations***

### ***Other neurodegenerative disorders***

Microglia are non-neuronal cells with important neuroprotective and potentially neurotoxic functions in the central nervous system. Little is known about how these cells change with age in the healthy human brain. In this experiment, human brains were studied to identify changes in the concentration and complexity of microglia that occur with age. We hypothesized that aging would lead to increased microglial activation but not necessarily changes in cell density. Moreover, we hypothesized greater microglia activation in the white matter than gray matter. Post-mortem tissue of the frontal cortex (area 9) was stained with IBA1 to label microglia. Digital microscopic images of the IBA1 stained tissue were generated using the Aperio ScanScope at 40x magnification. Ten regions of interest (ROI) (200um<sup>2</sup>) were selected randomly in both gray and white matter for each case. Microglia were counted both by hand and the positive pixel algorithm on the Aperio Software in the ROIs. More microglia were found in the white vs. gray matter; however, there was no difference between age groups in the number of microglia. There was a small, but statistically significant, increase in the IBA1 positive pixels in the white matter of samples from 70 year or older patients. Our results are in agreement with microglia changes seen as a function of normal aging in rodents. Furthermore, our results suggest that in the absence of neurological disease, aging is not sufficient to cause a profound increase in microgliosis. For future experimentation, we will increase our sample size and include analysis of the hippocampus.