



VirginiaTech®

School of Neuroscience

## Faculty Candidate

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**“The Normal Functions of Brain  
Pericytes and Blood-Brain Barrier and  
their Dysfunction in Alzheimer’s Disease”**

**February 28, 2017**  
**3:30pm – 4:30pm**  
**Bicomplexity Institute Room 145**

Pericytes are cells that wrap around capillaries, the smallest diameter blood vessels. They play an important role in supporting the blood-brain barrier (BBB), angiogenesis, phagocytosis of toxic metabolites, and have multipotent stem cell capabilities. These cells have also been shown to be contractile cells regulating cerebral blood flow (CBF); however this has been the matter of a nearly 150 year old debate. Recently, we found that the loss of pericytes in transgenic mice leads to neurovascular uncoupling and limits oxygen supply to brain. Furthermore, using optogenetics, I found that channelrhodopsin positive brain pericytes contract *in vivo* constricting the underlying capillary. It is important to understand the normal functions of brain pericytes, as these cells degenerate in normal aging and this loss is accelerated in many neurological diseases, including Alzheimer’s disease (AD), amyotrophic lateral sclerosis and stroke. Strikingly, disruption of the BBB and CBF also occurs in these diseases leading to blood-derived cells and toxins to enter the brain, and a reduction in delivery of oxygen and glucose, respectively. At the same time, this dysfunction impairs proper transport and clearance of molecules across the BBB, including amyloid-beta, a pathological hallmark of AD. It is likely that pericyte degeneration causes these disturbances, but little is known about how pericyte reduction alters overall brain function or what causes their loss. In the future, I would like to investigate causes of pericyte degeneration and BBB dysfunction in AD and I am also interested in post-translational modifications and expression level changes of key receptors at the BBB. Additionally, I am interested in utilizing iPSCs and CRISPR technology to repair AD related risk genes. Ultimately, identifying new therapeutic targets that prevent or repair BBB disruption may be the key to the treatment of neurodegenerative diseases, including AD.

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