Stephenson School of Biomedical Engineering
Seminar Series Presents

THE BLACK BOX OF CHRONIC VISCERAL PAIN: SHINING LIGHT ON NOVEL CIRCUITRY

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1:30 pm | Friday, Oct. 25, 2019 | Gallogly Hall, Room 127

BIO

Dr. Anthony Johnson is the clinical assistant professor of research in the OU Medicine Department of Neurology. He received his bachelor of science degree in biochemistry and microbiology, his master's degree in neuroscience and doctorate degree, all from the University of Oklahoma Health Sciences Center.

Dr. Johnson's research interests include stress remodels limbic circuits, pain, hypersensitivity, optogenetic and behavioral techniques in rat models, and brain circuitry. Honors include the American Neurogastroenterology and Motility Society Young Investigator Award, NIH Pain Consortium Mitchell Max Award for Research Excellence and College of Medicine Graduate Student Association Award for Basic Science Oral Presentation.

ABSTRACT:

Chronic pain affects as many as 20% of adults and 40% of veterans in the United States. In particular, chronic visceral pain, such as occurs in irritable bowel syndrome (IBS), significantly impacts the quality of life of patients due to a lack of effective therapies. Stress worsens symptoms in IBS patients and brain imaging studies have demonstrated abnormal limbic brain activity, suggesting a disordered brain-gut communication may contribute to the etiology of the disease. Optogenetic tools have been developed that allow for manipulation of brain circuits in rodent models to demonstrate the effect on behaviors. Our research has focused on using optogenetic tools in rat models of chronic stress-induced colonic pain to investigate the role of the amygdala in the regulation of the emotional exacerbation of visceral pain. We expressed either excitatory (channelrhodopsin) or inhibitory (halorhodopsin) opsins in the central nucleus of the amygdala and then activated the opsins at the bed nucleus of the stria terminals, a key integratory region for neuroendocrine stress signaling. In stress-naïve rats, 'turning-on' the central amygdala-bed nucleus pathway with channelrhodopsin induced colonic hypersensitivity. In stressed rats, 'turning-off' the central amygdala-bed nucleus pathway inhibited the stress-induced colonic hypersensitivity. Overall, we demonstrated that real-time optogenetic manipulation of the amygdala-bed nucleus pathway was sufficient to change colonic sensitivity in freely moving rats. These findings illustrate that stress-induced colonic hypersensitivity can be induced and maintained by central limbic neurocircuits. Thus, these studies provide a rationale to develop brain-directed therapies to decease chronic visceral pain.

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