RACHEL LEE, ALYSSA WOLFE, & MICHELLE BRIDI, Dept of Neuroscience, West Virginia University, Morgantown, WV, 26505. Exploring the mechanisms underlying sleep/wake-dependent control of excitatory and inhibitory synaptic transmission.

Sleep is highly conserved, suggesting that this behavioral state performs a crucial function. However, our understanding of this function remains incomplete. During sleep and wake, the ratio of synaptic excitation to inhibition (E/I) changes in opposite directions: decreasing during sleep and increasing during wake. This sleep-dependent E/I regulation is altered in mouse models of atypical conditions such as autism spectrum disorder (ASD). In humans, ASD also commonly presents with sleep disturbances. How sleep and E/I regulation are linked is unknown. Elucidating the underlying mechanism driving a change in E/I is crucial to revealing the underpinnings of how sleep plays a role in neuronal communication. We hypothesize that wake-active neuromodulation increases excitatory synaptic signaling and decreases inhibitory synaptic signaling during wake. Utilizing whole-cell patch clamp, we have investigated the effects of wake-active neuromodulators on the frequency and amplitude of miniature postsynaptic potentials following both sleep-rich and wake-rich periods in mice. This project will characterize how sleep and wake control the E/I ratio, leading to translatable research that uncovers how synaptic activity impacts public health.