My research interests center on the hypothesis that myelin pathology is a causal mechanism underlying neurodevelopmental disorders. During my DVM and PhD training at Auburn University, I studied AAV gene therapy and myelin pathology in cats with lysosomal storage disorders. In the Jordan lab, I study oligodendrocyte and myelin deficits in multiple high-confidence autism spectrum disorder models (Nlgn1, Shank3, and SynGAP1) and the effects of pro-myelinating drugs such as clemastine. I primarily use mouse and cell culture models to assess myelin amount, oligodendrocyte morphology, and behavior, and I have a special interest in microscopy and image analysis.

Lentiviral shRNA knockdown of Nlgn1 causes reduced MBP expression in oligodendrocyte-enriched culture. (A) Reduced immunostaining of synaptic protein Nlgn1 (green) and myelin basic protein (MBP, blue)) in primary oligodendrocytes infected with lentiviral shRNA designed to knock down Nlgn1. GFP expression in shRNA-infected cells in blue. Scale bar = 10µm.