ELAINE SHELTON, PHD is an Assistant Professor of Pediatrics and Pharmacology at Vanderbilt University Medical Center. She completed her PhD at Cincinnati Children's Hospital Medical Center, where her research focused on defining transcriptional regulators of heart valve development. She then moved to Vanderbilt University to undertake a postdoctoral fellowship in cardiovascular medicine. As a postdoc, she focused on understanding blood vessel development, repair, and remodeling, with the goal of identifying non-vascular cell types that could be used to form new vasculature or repair injured vessels.

In 2013, Dr. Shelton was recruited to the Division of Neonatology at Vanderbilt University Medical Center. Much of her current work focuses on fetal blood vessel development and cardiovascular transition at birth. Specifically, she is interested in understanding the molecular and genetic regulation of the ductus arteriosus, an essential fetal artery, in order to develop novel therapies to treat patent ductus arteriosus (PDA). The Shelton lab uses a combination of transcriptome analyses, high throughput screens, vessel myography assays, and in vivo mouse models to investigate the extent to which molecular/genetic/biomechanical factors regulate ductus development and identify factors that could serve as novel PDA therapeutics.

ABSTRACT: The ductus arteriosus (DA) is an essential fetal vessel connecting the pulmonary artery and aorta. In utero, the DA diverts blood flow away from the developing lungs and into the systemic circulation where gas exchange occurs within the placenta. After birth, the DA must permanently close to allow adequate perfusion of the newly inflated lungs. Failure of the postnatal DA to constrict results in PDA (patent ductus arteriosus), one of the most common congenital cardiovascular disorders. PDA disproportionately affects the most critically ill premature neonates, affecting 60-80% of infants weighing <1000g. The only drugs currently available to promote DA closure (indomethacin, ibuprofen, acetaminophen) non-specifically target the prostaglandin pathway, are associated with severe adverse side-effects, and are ineffective in ~30% of patients. Consequently, there is a significant need to identify novel DA-specific regulators of vascular tone in order to develop additional PDA therapeutic options.