Abstract

Congenital heart defects (CHD) are the most common birth defect. Multiple studies have uncovered a wide-array of non-synonymous mutations in the GATA4 gene of patients suffering from CHD. The GATA4 protein is a transcription factor that regulates gene expression during heart development. Our hypothesis is that non-synonymous mutations in GATA4 modulates its DNA-binding properties and alters its gene targets. My work in the Rodríguez-Martínez laboratory is to characterize the DNA binding properties of non-synonymous mutants of GATA4 and compare them to the 'wild-type' GATA4. As part of this project, I will use molecular biology tools to clone, express, and purify of GATA4 mutants. Once completed, our work will contribute to the understanding of the molecular consequences of GATA4 mutations in proper heart development.