**ACTIVE FUNDING:**

R01 HL033756 07/01/12-06/30/16
Role: Co-I
NIH NHLBI
Mucopolysaccharide Metabolism in Cardiac Anomalies
The major goals of this project are to determine if the heart defect (htd) gene is a prototype candidate for an embryonic segmentation gene; to determine if the fate of the luminal “face” of cushion mesenchyme is to expand into the definitive precursor valve leaflets; and to determine if a primary fate of cushions is to interact with the myocardium to complete septal development.

P20 GM103444-07 07/01-07/01/18
Role: Target Faculty
NIH/NIGMS
Sub-Project Title: Developmental And Biomechanical Mechanisms of Valve Tissue Formation
The goals of this project are to determine developmental mechanisms that contribute to mitral valve prolapse. Additionally, we aim to integrate biomechanical studies with development using novel bioreactors and cells derived from patients with mitral valve prolapse to gain a clear understanding for how valve developmental defects give rise to mechanically inferior adult valve tissues.

1R01 HL122906 09/01/14-08/31/15
Role: Co-I
NIH/NHLBI
Mechanisms of DMP Development and Atrioventricular Septation
The major goal of this project is to test the hypothesis that primary cilia are functioning as a signaling center in the pSHF/DMP and are critically important in the development of the DMP and in atrioventricular septation.

1R01 HL127692-01 04/01/15-03/31/20
Role: Institutional PI
NIH/NHLBI
Genetics and Mechanisms of Mitral Valve Prolapse
Mitral Valve Prolapse affects about 7 million Americans and increases the risk of endocarditis and congestive heart failure, but relatively little is known about the underlying mechanisms leading to this disease. The goal of our application is to study the genetics and mechanisms of this disease. Characterizing new pathways for mitral valve prolapse will provide new targets for drug discovery to prevent and treat this heart disease.

15GRNT25080052 07/01/15-06/30/17
Role: PI
AHA Grant in Aid
Etiology and Treatment for Mitral Valve Prolapse
Mitral valve prolapse is one of the most common disease in the human population and affects 1 in 40 people. Roughly 50% of people who have MVP require some aspect of clinical intervention ranging from medical management to valve replacement. To date, little is known regarding the etiology of the disease. Although MVP can be inherited through generations, disease genes have been elusive. We have recently identified DCHS1 as an MVP disease gene and have developed applicable models to study MVP in mice. This affords us the ability to ask questions that heretofore have not been possible and are critical if we are to improve medical options for patients with MVP.