Results of Foundation-backed research at Tufts to be presented at Heart Association colloquium

Important news for 2013 is that scientists at the upcoming American Heart Association Scientific Sessions in Dallas in November will review exciting research funded by the JTB Foundation at the Molecular Cardiology Research Institute at Tufts Medical Center.

This research focuses on the role genetics and genetic mutations play in heart defects.

Dr. Gorgon Huggins and his team study genetic modifiers for Hypertrophic Cardiomyopathy (HCM), a disease of the heart that can cause Sudden Cardiac Arrest. Current medical therapies -- beta-blocking drugs and calcium channel blockers -- decrease symptoms of heart failure from HCM but do not prevent it or alter the natural history of the disease, according to Huggins.

Foundation grants support the work of his team and enables it to hire Tufts medical student-trainees in the lab.

Huggins' research looks specifically at a gene called FHOD3 to determine its role in the disease. He has already found that people with HCM are more likely to carry a form of the FHOD3 gene with a different amino acid sequence than those who don't have the disease. He has speculated that this may have a deleterious effect on the function of the proteins that heart muscle cells use to generate contraction.

"In this work we further define the importance of this family of proteins in muscle development, which heightens the importance of our findings in human HCM," said Huggins. His team recognized the support from the Babbitt Foundation in its submission papers for the Dallas colloquium and will include it in the conference poster.

Huggins said this newest research, done on flies, hypothesizes that the gene being studied is required for normal muscle development in flies. "Flight muscles can be compared to mammalian cardiac muscle due to their involuntary contractions," noted Huggins.

Another avenue of research may show how a protein found in cells, transforming growth factor beta (TGF-b), might be the suspect in HCM. This protein controls cell proliferation and is a factor in cancer as well as heart disease.

Testing on mouse models has shown that stimulation of the TGF-b in cardiac fibroblast cells may prevent development of HCM. But this has to translate to human cells, Huggins emphasized.

"We have made good but somewhat slow progress on the TGF-b story," Huggins wrote in his report to the Foundation this summer. His team has been exposing HCM cardiac fibroblast cells to TGF-b, with "variable" response, he noted. Despite this, Huggins told the Foundation, the team is moving ahead with this line of investigation.

. One day, results of research funded by the John Taylor Babbitt Foundation could produce drugs that prevent HCM in genetically susceptible individuals as well as modify disease progression in patients with established HCM.