June 21, 2012

I would like to thank the John T. Babbitt Foundation for their support over the past year. The grant provided by the JTB has made possible tremendous accomplishments vital to our longterm Hypertrophic Cardiomyopathy (HCM) research. I would like to take this opportunity to review the accomplishments over the funding period. The grant from the JTB supported three key areas.

First, the grant was to support the salary of a recent graduate from college with aspirations of pursuing a career in biomedical sciences and/or medicine. Over the past year we have had the pleasure of working with Jenna Calvino who has been the principal person responsible for work on the Hypertrophic Cardiomyopathy Project. Jenna has done a remarkable job in terms of learning new techniques and significantly driving this effort to a successful completion. During the Summer of 2011 we supported Randy Kring and this summer we are also joined by Betty Shum, both rising second year students in the Tufts University School of Medicine. Betty was awarded a prestigious Williams Scholarship from the university to support her summer research experience studying HCM in the summer of 2012.

Second, the JTB grant provided support for the collection of blood and tissue samples from patients with HCM and for purification of DNA from existing samples that were frozen as well as new samples. With the JTB support we now have ~340 purified DNA samples. During this past year we also instituted a rigorous program for collection of muscle tissue taken from HCM patients at the time of surgery for heart failure. Recognizing that qualified academic partners could make important discoveries using these precious samples we have considered several research partnerships. Currently the samples are supporting research performed at the Mayo Clinic and the Beth Israel Deaconess Hospital in Boston.

Third, the JTB grant provided support for the analysis of a gene called FHOD3 (FHOD3 is an abbreviation for the gene named: forming homology 2 domain containing 3) in HCM. Our genetic studies indicated that people with HCM were more likely to carry a form of FHOD3 with a different amino acid sequence (amino acid 1151 encoded a Valine rather than an Isoleucine). We submitted a manuscript describing this discovery to several journals and we are now working to respond to requests from the manuscript reviewers for additional experiments. These additional studies include the analysis of the two FHOD3 protein forms and how they may have different effects on assembly and function of the protein mechanism that heart muscle cells use to generate contraction. The JTBF is formally acknowledged in the manuscript as providing funding for the experiments described in the manuscript.

In summary, I would like to sincerely thank the JTB for their support, which has been truly transformational to our HCM research efforts.

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