The Effects of Fetal Troponin T on the Cardiac Remodeling of Hypertrophic Transgenic Hearts

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Hypertrophic cardiomyopathy (HCM) is a genetic heart disease that affects 1 in 500 people in the United States, resulting in fibrotic hearts. This disease is very difficult to detect and nearly impossible to cure. The purpose of this research was to cross the Fetal TnT gene with RL50% gene in Mus musculus to determine if the Fetal TnT gene could alter the effects of the RL50% gene, which causes HCM. Genotypes of the offspring from RL50% and Fetal TnT Mus musculus were identified through polymerase chain reaction and agarose gel electrophoresis. Once offspring were 2 months old, body weights were taken and the heart tissue was harvested. Then, heart weights were recorded. Published research has shown an increase in heart to body weight ratios in subjects with HCM. Results from this investigation verified this work. Subsequently, heart slices were histologically prepared in order to ascertain the severity of cardiac fibrosis. The disease-free nontransgenic and Fetal TnT offspring exhibited no fibrosis. However, observations of the RL50% offspring displayed clear signs of fibrotic disease. The RL50%-Fetal TnT heart, or the double transgenic genotype, showed no fibrosis. These results support the hypothesis that the Fetal TnT gene remodeled the purebred hypertrophic hearts. This knowledge is compelling. Future research into the effects of the Fetal TnT gene as the age of experimental subjects increases is warranted. The potential to one day eliminate sudden cardiac mortality due to HCM could save countless lives.

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