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Low-dose oral *triiodothyronine* (T3) improves left ventricular function and myocyte survival following myocardial infarction in rats

Anthony Martin Gerdes, Viswanathan Rajagopalan, Youhua Zhang and **Yuefeng Chen**
New York Institute of Technology-College of Osteopathic Medicine, USA

Myocardial Infarction (MI) activates cardiac D3 deiodinase, contributing to low tissue T3 and Heart Failure (HF). Potential improvements in LV remodeling and function with a therapeutic T3 dose after MI are not clear.

We hypothesize that a safe, low-dose T3 treatment/monitoring regimen will lead to significant cardioprotection in rats following MI-induced HF.

MI was produced in adult rats by LAD ligation. T3 (4-5 μg/kg/day) in drinking water was started after MI and continued for 2 months (Mo). Vehicle (V) was used in MI controls (n=16-20/group).

Infarct size was similar in both MI groups. D3 mRNA expression increased in MI+V (2.7-fold) and was reversed in MI+T3 (0.49-fold). MI+T3 improved ejection fraction by 51% at 1 Mo and by 47% at 2 Mo post-MI as assessed by magnetic resonance imaging (MRI). Mean MRI wall thickness was increased at both latter time-points. Histologically, non-infarct area and wall thickness increased significantly (30% and 18% respectively). Non-infarct length was also increased. Remarkably, following MI, the incidence of atrial tachyarrhythmias that persisted following discontinuation of experimental atrial tachypacing was significantly diminished by 63% with T3. T3 did not affect heart rate. The selected dose led to feedback inhibition of Thyroid-Stimulating Hormone (TSH) but no significant change in serum T3.

Conclusion: Results demonstrate a safe and effective post-MI/HF T3 treatment strategy that dramatically improves LV function, atrial arrhythmogenesis, non-infarct tissue remodeling, and myocyte survival with no adverse effects. This study describes an effective, translatable, treatment/monitoring protocol for T3 treatment of MI.

Biography

Anthony Martin Gerdes has done PhD in Anatomy (1978), from University of Texas Medical Branch at Galveston. He was the Professor/Chair of Anatomy, University of South Dakota. Also the founding Scientist for Sanford Research-University of South Dakota. His Current position is Professor/Chair Biomedical Sciences, NYIT College of Osteopathic Medicine, Old Westbury, NY, 2011-present. Publications: ~120 peer reviewed journal articles. 2013 Distinguished Alumnus, Graduate School of Biomedical Sciences, UTMB at Galveston Anthony Martin Gerdes developed a precise method to determine cardiac myocyte shape. He then provided a comprehensive understanding of how cardiac myocytes remodel during growth, maturation, aging, cardiac hypertrophy, and heart failure (HF) from many etiologies. After demonstrating that low thyroid hormone function alone can cause heart failure, he showed remarkable beneficial changes in myocyte shape and vascular remodeling, reduced fibrosis, and improved LF function after thyroid hormone treatment of various models of HF (including ischemia, diabetes, hypertension).

agerdes@nyit.edu

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