

KIRSTEN BOGUNOVICH AND HOLLY RACINE, Dept. of Biomedical Sciences, West Liberty University, WV, 26074. Induced Thyrotoxicosis Elicits Protective Effects in Developing Hearts.

Maternal hyperthyroidism affects development in infants and can lead to conditions such as craniosynostosis, exophthalmos, and cardiac hypertrophy. Models for studying maternal hyperthyroidism, or thyrotoxicosis, are limited. Our lab has established an avian model to study the effects of induced thyrotoxicosis on embryonic development. Thyroid hormones (TH) cause inotropic and chronotropic changes in cardiac tissue, including increased cardiac output, increased blood volume, and hypertrophy. Therefore, we wanted to investigate the cardiac changes in our model. Fertilized chicken eggs are injected on embryonic days (E) 11 and 15 with either saline (control) or 25ng thyroxine (T<sub>4</sub>). Hearts were harvested on day E19, with halves of each sample used for histological staining with Masson's Trichrome and qRT-PCR analysis. The main objective was to observe systemic effects of our model by studying morphological and genetic changes in the heart following exposure by measuring expression of *THRα* (TR receptor), *ATP2A2* (calcium ATPases), and *MYH7* (Myosin heavy chain 7). We hypothesized that levels of all 3 markers would be upregulated, since THRα regulates transcription of these other cardiac markers due to fluctuating levels of TH. These are linked to ventricular hypertrophy when upregulated. However, our results contradicted our hypothesis. There was downregulation of all 3 genes, with a significant downregulation in *ATP2A2*. In combination with other data collected in our lab and related literature, we suspect that there is a cardiac protective effect occurring in the hearts following thyroxine exposure.

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