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Thyrotoxicosis is a condition of excess circulating thyroid hormones. Maternal thyrotoxicosis has been related to the development of craniosynostosis (CS) in approximately 3% of births. CS occurs when the cranial sutures of the skull fuse prematurely. However, the mechanism for thyroid hormone-induced CS in those cases is unclear. Currently, we are establishing an avian model of thyroxine-induced CS to study these mechanisms. Two groups of fertilized chicken eggs were injected with saline or 25 ng T₄ (thyroxine) on embryonic days 11 and 15 and collected on 19 prior to hatching. Data were collected from each group including: body mass and length, tibia and femur length, liver and heart mass, and circulating T₄ concentrations. Our study aimed to characterize all possible physiological changes associated with our treatment and determine the subset of positive cases. Following thyroxine exposure, 19-day old embryos were 4% shorter ($p < 0.05$) with a corresponding 8% decrease in tibia length ($p < 0.01$) and 9% decrease in femur length ($p < 0.01$) compared to the controls. Measured T₄ concentrations were 88% higher in serum samples following thyroxine-exposure compared to the controls ($p < 0.01$). Body, heart, and liver mass were not different between groups. We were then able to isolate positive cases from correlation plots based on clustering observed in anticipated regions. In conclusion these results support that there is a subset of samples within the treatment group that display the expected phenotypes. It is in this subgroup of data that our focus can be shifted to study the mechanism of thyroxine-induced CS.

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