

Haley M. Carter, Department of Biomedical Sciences, West Liberty University, West Liberty, WV, 26074, and Holly L. Racine, Department of Biomedical Sciences, West Liberty University, West Liberty, WV, 26074. The Effect of Induced Thyrotoxicosis on Avian Limb Development.

Thyroid hormone (TH) is essential for bone development. Altered TH levels such as hyperthyroidism, or thyrotoxicosis, results in the early maturation of the cartilaginous scaffold for long bone development, which can cause improper ossification. Maternal hyperthyroidism during embryonic development causes neonatal thyrotoxicosis and can therefore lead to stunted growth in infants. In our avian model of induced thyrotoxicosis, we injected saline (control) or 25ng thyroxine (T₄) in 0.1 mL dose into fertilized chicken eggs on embryonic (E) days E11 and E15. On E19, chicken embryo length was measured and limbs were collected. The left limbs were whole-mount stained with Alizarin Red (bone) and Alcian Blue (cartilage). Limbs were then imaged and tibias were measured using ImageJ. The tibias from the right limbs were further dissected, embedded in paraffin, cut (5µM), and stained with Safranin O (cartilage) and Fast Green (bone). Sections were examined to determine morphological changes in the cartilaginous long bone scaffold. Body length was 4.8% shorter following thyroxine exposure ($p < 0.01$). Tibias were 2.4% shorter, though not statistically significant ($p = 0.36$). Paraffin-embedded sections suggested increased chondrocyte hypertrophy, although further quantification is required. In conclusion, our data support thyroxine-induced changes in long bone morphology. Understanding how thyrotoxicosis can alter bone development *in utero* is important for developing potential targeted therapies for improving ossification in cases of neonatal thyrotoxicosis.

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