ANDREA DWIGGINS and HOLLY RACINE, Department of Biological Sciences, West Liberty University, West Liberty, WV 26074. Establishing a model for studying the effects of maternal hyperthyroidism *in utero*.

Thyroid hormones (THs), triiodothyronine (T₃) and thyroxine (T₄), play a critical role in growth and maintenance in all organ systems. Research suggests that maternal hyperthyroidism is a risk factor for craniosynostosis (CS in infants). CS is characterized by the premature fusion of the cranial sutures, which leads to debilitating side effects. The mechanism of TH-induced CS is obscure. The overall objective of this study is to enhance the knowledge about *in utero* thyroid-related mechanisms and how they relate to the development of CS. However, a model for study must first be established. It is hypothesized that a dose of T₄ between 50 pg and 0.5 ug will induce thyrotoxicosis in an avian model. Fertilized chicken eggs were injected beneath the chorioallantoic membrane with saline and 50pg, 0.5ng, 5.0ng, 50ng and 0.5ug T₄. Embryos (N=4-6 per group) were collected at E19. In addition to viability, body and heart mass were recorded. Hyperthyroidism is known to cause a decrease in body mass. Results showed a significant decrease in body mass with 50ng T₄ (p<0.001) while maintaining viability (92%). Hyperthyroidism is also known to cause cardiac hypertrophy. Results showed a significant increase in heart to body mass ratio with 50ng T₄ (p<0.001). In conclusion, the 50ng T₄ is the appropriate dose to maintain viability with accompanied phenotypic changes indicative of thyrotoxicosis. Determining an effective dosage regimen is important for establishing a model for studying the mechanisms of hyperthyroid-induced CS. Supported by the NASA West Virginia Space Grant Consortium (80NSSC20M0055).