Thyroid hormones (THs), triiodothyronine ($T_3$) and thyroxine ($T_4$), 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 $T_4$ 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 $T_4$. 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 $T_4$ ($p<0.001$) 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 $T_4$ ($p<0.001$). In conclusion, the 50ng $T_4$ 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).