1048-92-64 **Toby L Shearman*** (tshearman@vt.edu), 325 New Kent Road, Blacksburg, VA 24060, and **Pablo Diaz**, **Michael Gillespie**, **Justin Krueger**, **Jose Perez**, **Alex Radebaugh**, **Garret Vo** and **Christine Wheatley**. *A mathematical model of the immune system's role in obesity-related chronic inflammation.*

Obesity is quickly becoming a pandemic. The low-grade chronic inflammation associated with obesity leads to health risks such as cancer, heart disease, and type 2 diabetes mellitus. To better understand the progression of obesity-related chronic inflammation, mice were fed either a high fat or low fat diet over 140 days. At days 0, 35, 70, and 140, the percentages of macrophage subsets, CD4+ T cells, and regulatory-T cells infiltrating the intra-abdominal white adipose tissue (WAT) were examined. Monocyte chemoattractant protein-1 (MCP-1) mRNA expression in WAT was also quantified. Additionally, glucose-normalizing ability was examined by administering peritoneal glucose tolerance tests. A system of ordinary differential equations models this system. The model consists of 8 differential equations, has 25 parameters, and has 1 forcing function. Tools used to characterize the model include parameter estimation, sensitivity analysis, and stability analysis. Based on the data provided, the system describes the growth of adipocyte size and chronic inflammation over 105 days beginning at day 35, which is approximately when the adipose cells become hypertrophic. The model shows that without intervention, chronic inflammation escalates and the related health problems persist. (Received January 20, 2009)