Defining the Location of T-bet-expressing Myeloid Cells During Acute Intestinal *Toxoplasma gondii* Infection

**Melody Wickstrom**¹, Madison Schanz², Kimberly Larson¹,², Américo López-Yglesias³

¹Indiana University School of Medicine; ²Indiana State University; ³Indiana University School of Medicine, Department of Microbiology and Immunology

**Background/Objective:**
The protozoan parasite *Toxoplasma gondii* is the second leading cause of foodborne pathogen-related deaths in the United States. The transcription factor T-bet is indispensable for host immunity against *T. gondii*. The absence of T-bet results in rapid susceptibility during parasite infection. T-bet has been considered essential for T-cell-derived IFN-γ during *T. gondii* infection; yet, recent research has shown that T-bet is not required for lymphocyte-derived IFN-γ responses. Our preliminary research shows that T-bet-deficient mice succumb to parasite infection significantly quicker than mice lacking lymphocytes. This has led to our hypothesis that T-bet-dependent myeloid cells are critical for host resistance during acute intestinal *T. gondii* infection. The objective of this project was to define the location of the T-bet-expressing myeloid cells in the medial small intestines (MSI) of naïve and infected mice during acute mucosal parasite infection.

**Methods:**
We used immunofluorescence microscopy to determine the location of T-bet-expressing myeloid cells in the MSI of naïve and *T. gondii* infected mice. Mice were orally infected with 40 cysts of the ME49 strain of *T. gondii*. On days 0 and 5, one-inch MSI segments were harvested, fixed with 4% paraformaldehyde for at least one hour, and then frozen in OCT compound. Tissues were then cut into 8µm sections and placed onto slides for staining. Sections were stained for nuclei, CD11c, T-bet, and *T. gondii*.

**Results:**
Our results revealed T-bet-expressing CD11c+ cells in both the MSI and spleen on days 0 and 5 of *T. gondii* infection.

**Summary:**
These data indicate that T-bet-expressing myeloid cells are present in the MSI during *T. gondii* infection. Defining the position of these cells will allow us to determine T-bet’s role in mediating myeloid cell-dependent *T. gondii* clearance. Due to the limited treatment options for patients suffering from toxoplasmosis it is critical to define new mechanisms for eliminating *T. gondii*. 