RUTGERS HEALTH
School of Graduate Studies
Health Sciences Campus - Newark

Office of Student Affairs and Admissions
SGS at New Jersey Medical School
Rutgers, The State University of New Jersey
185 South Orange Avenue, MSB C-696
Newark, NJ 07103

http://njms.rutgers.edu/gsbs/
p. 973-972-4511
f. 973-972-7148

YOU ARE INVITED TO ATTEND THE DEFENSE OF THE DOCTORAL DISSERTATION

"THE FUNCTIONAL ROLE OF GRADED T-BET EXPRESSION IN CD11C+ B CELLS"

By

Rebecca L. Francis
Infection, Immunity and Inflammation Program
B.S. Biomedical Laboratory Science, 2016, Michigan State University, East Lansing, MI

Thesis Advisor: Jason Weinstein, PhD
Associate Professor
Department of Medicine

Wednesday, October 7th, 2026
Cancer Center, G1196
9:00 A.M.

Join Zoom presentation
https://rutgers.zoom.us/j/96869163470?pwd=VfsV5rg12TFj7uy4xgEQ5TuJbYLCFn.1

Meeting ID: 968 6916 3470
Password: 437092

ABSTRACT

Age-associated B cells (ABCs), defined by their expression of T-bet and CD11c, are key contributors to the type 1 humoral response in both acute and chronic disease. Their function in the immune response is suggested to be multi-fold. Most notably, ABCs are described as being precursors to antibody-secreting cells (ASCs); however, they also play roles as antigen-presenting cells (APCs) and localize to areas that allow for quick differentiation upon secondary exposure. These functions are inconsistent in the literature and represent functions performed by B cells in general. We aim to understand how ABC functions differ from other B cell subsets during acute viral progression and to uncover unique functional differences between ABCs of different tissues and origins. T-bet is an important transcription factor for driving type 1 responses in a variety of immune cells. In B cells, T-bet is known to promote IgG2c isotype switching and ASC differentiation, block germinal center development, and affect migratory abilities. However, additional functions specific to ABC's high and prolonged T-bet expression are still being explored. Therefore, we further investigated the specific influences of T-bet on CD11c+ B cells. By using a T-bet reporter mouse, we defined three populations of CD11c+ B cells based on reporter expression in the spleen: T-bet^{Neg}, T-bet^{Mid}, and T-bet^{Hi}. To assess what functions T-bet exerts, we performed flow cytometry and scRNA-seq to identify effector phenotypes and transcriptional programs of ABCs across tissues and T-bet levels. T-bet^{Hi} B cells are the presumed ABCs; however, antibody secretion functions and phenotypes are restricted to T-bet^{Mid} B cells, while T-bet^{Hi} and T-bet^{Neg} cells are primarily memory cells whose characteristics largely overlap, with general memory, ABC, and APC phenotypes. In addition, ABCs arise in peripheral tissues but only have T-bet^{Neg} and T-bet^{Pos} populations suggesting tissue-specific influences of T-bet. These phenotypes are consistent across infection and autoimmune settings. Our findings support a model in which CD11c+ B cells with low T-bet levels support established T-bet functions, particularly ASC differentiation. However, T-bet is not absolute in driving ABC functions as high and negative T-bet levels promote similar characteristics with a yet unknown contribution to the humoral response.