

May 20th, 12:30 PM

A Novel Population Of Natural Killer Cells Pplays A Critical Role in the Depletion of Splenic B2 B Cells During Experimental Africian Trypanosomiasis


Deborah Frenkel

University of Massachusetts Amherst, dfrenkel@vasci.umass.edu

Samuel J. Black

University of Massachusetts Amherst

Follow this and additional works at: http://escholarship.umassmed.edu/cts_retreat

 Part of the [Animal Sciences Commons](#), [Immunology of Infectious Disease Commons](#), [Parasitic Diseases Commons](#), and the [Parasitology Commons](#)



This work is licensed under a [Creative Commons Attribution-Noncommercial-Share Alike 3.0 License](#).

Deborah Frenkel and Samuel J. Black, "A Novel Population Of Natural Killer Cells Pplays A Critical Role in the Depletion of Splenic B2 B Cells During Experimental Africian Trypanosomiasis" (May 20, 2016). *UMass Center for Clinical and Translational Science Research Retreat*. Paper 20.

http://escholarship.umassmed.edu/cts_retreat/2016/posters/20

This material is brought to you by eScholarship@UMMS. It has been accepted for inclusion in UMass Center for Clinical and Translational Science Research Retreat by an authorized administrator of eScholarship@UMMS. For more information, please contact Lisa.Palmer@umassmed.edu.

A NOVEL POPULATION OF NATURAL KILLER CELLS PLAYS A CRITICAL ROLE IN THE DEPLETION OF SPLENIC B2 B CELLS DURING EXPERIMENTAL AFRICAN TRYPANOSOMIASIS

Deborah Frenkel PhD¹ Samuel J Black PhD¹

¹ University of Massachusetts, Department of Veterinary and Animal Sciences

Mice infected with *Trypanosoma brucei*, the causative agent of human sleeping sickness and a contributor to nagana in cattle, rapidly lose the capacity to mount VSG-specific antibody responses, and die with uncontrolled parasitemia. We have shown (Bockstal et al., 2011, PLOS Pathogens) that the loss of humoral immune competence in the infected mice results from depletion of developing and mature splenic B cells. We now report that *T. brucei*-induced splenic B cell depletion is dependent upon the presence of the pore forming molecule perforin which is present in the cytotoxic granules of cytotoxic T lymphocytes, natural killer T cells and natural killer cells, occurs in the absence of T cells (and natural killer T cells), i.e., in T cell receptor ($\alpha\beta\gamma\delta$)-/- mice, but does not occur in intact mice that are depleted of natural killer (NK) cells by treatment with monoclonal antibody specific for the NK1.1 differentiation antigen. In the intact mice, B cells are deleted after remission of the first *T. brucei* parasitemic wave. At this time natural killer cells are expressing the cytotoxic granule marker CD107a, indicating that they have degranulated, executing their effector function. Moreover, in vitro assays show that B cells from *T. brucei* infected mice are killed by natural killer cells from uninfected C57BL/6 mice but not efficiently killed by CD107a positive natural killer cells isolated from infected mice, which may be functionally exhausted.

Deborah Frenkel, Ph.D.

University of Massachusetts

Veterinary & Animal Sciences

460 Integrated Sciences Building

Amherst, MA 01003

413 522 6066

dfrenkel@vasci.umass.edu