Altered First Responders: Changes in Neutrophils in Chronic Leishmaniasis

Richard Davis
PhD Candidate

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283 EMRB
Richard Davis
Biographical Sketch

Richard, the middle of seven children, was raised in Draper, Utah. Growing up he had a deep interest in science and once, as an elementary school student, turned a dirt pit in his backyard into a 'science camp' featuring cool looking leaves and an ant hill. ‘Science camp’ was poorly attended by both friends and siblings, but this did not deter him from continuing to enthusiastically pursue science courses and projects through his grade school and undergraduate years.

Richard graduated from Brigham Young University in Provo, Utah with a bachelors degree in Clinical Laboratory Science. A class on Medical Parasitology got Richard intensely interested in human parasitic diseases. As an undergrad, he innovated and published a method that increased the sensitivity of blood smears for malaria parasites. Wanting to pursue a graduate degree studying parasites, he joined the lab of Dr. Mary Wilson studying the protozoan parasite Leishmania. His research included investigation of neutrophils' in vitro interactions with Leishmania parasites and traveling to Brazil to study neutrophils in patients with cutaneous leishmaniasis.

Richard has accepted a post doctoral fellowship in Clinical Microbiology at ARUP laboratories at the University of Utah that will begin July 2017, leading to board certification and a career directing a clinical microbiology laboratory.

Richard met his wife Celeste on their university ballroom dance company. They have three kids, Ellia, Ivy and Lennon, whose health and well-being is due mostly to Celeste's (often literally) tireless efforts. Besides clinical field work, discussing science with his lab mates and writing about himself in third person, Rich enjoys hiking, camping and running.

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*Leishmania* spp. parasites, most often transmitted by the bite of a sand fly vector, infect around one million new humans each year, with upwards of 12 million people living with leishmaniasis worldwide. The most severe form of the disease, visceral leishmaniasis, is the second most deadly parasitic disease after malaria.

My research has focused on the role and interaction of neutrophils (PMNs) and parasites. PMNs, generally thought of as short-lived "first responder" cells, are the most abundant leukocyte of the human immune system with billions of new neutrophils entering the circulation from the bone marrow daily. While PMNs are seen to be recruited early to sites of *Leishmania* infection in animal models of disease, PMNs during chronic infection, both at sites of infection and in circulation, have largely been unexamined.

I investigated how chronic leishmaniasis might influence neutrophil populations in human patients with cutaneous leishmaniasis (CL) and visceral leishmaniasis (VL). I performed studies at a rural leishmaniasis treatment center in Brazil, examining the phenotype of circulating PMNs. I identified a population of PMNs expressing surface MHC class II (called HLA-DR in humans), a molecule important for the activation of T cells and highly expressed on antigen-presenting cells. I determined that HLA-DR+ PMNs show increased markers of activation and found that the activation of PMNs by soluble stimuli or patient plasma can induce HLA-DR-expression on PMNs in vitro.

Besides investigating the characteristics of human HLA-DR+ PMNs, other areas of my research have included examining the recruitment and phenotype of PMNs in mice and canines (foxhounds) infected with *Leishmania* parasites. I also examined the uptake and activation of humans PMNs exposed to different *Leishmania* life-stages.

These studies suggest that *Leishmania* infection can have profound impacts on host PMNs and may alter their ability to influence adaptive immune responses.