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Serum mediated activation of macrophages reflects TcVac2 vaccine efficacy against Chagas disease

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Chagas disease is endemic in Latin America and an emerging infectious disease in the US. No effective treatments are available. TcG1, TcG2 and TcG4 antigens are highly conserved in clinically-relevant Trypanosomacruzi (Tc) isolates, and recognized by B and T cells in infected host. Delivery of these antigens as a DNA-prime/protein-boost vaccine (TcVac2) elicited lytic antibodies and type 1 CD8+T cells that expanded upon challenge infection, and provided >90% control of parasite burden and myocarditis in chagasic mice and dogs. Here in, we determined if peripheral blood can be utilized to capture the TcVac2-induced protection from Chagas disease. We evaluated the sera levels of TckDNA/Tc18SrDNA and murine mitochondrial DNA (mtDNA) as indicators of parasite persistence and tissue damage; and effect of sera on macrophage phenotype. Circulating TckDNA/Tc18SrDNA and mtDNA were decreased by >3-5-fold and 2-fold, respectively, in vaccinated/infected mice as compared to non-vaccinated/infected mice. Macrophages incubated with sera from vaccinated/infected mice exhibited M2 surface markers (CD16, CD32, CD200 and CD206), moderate proliferation, low oxidative/nitrosative burst, and regulatory/anti-inflammatory cytokine (IL-4+IL-10>TNF-α) response. In comparison, macrophages incubated with sera from non-vaccinated/infected mice exhibited M1 surface markers, vigorous proliferation, substantial oxidative/nitrosative burst, and proinflammatory cytokine (TNF-α>>IL-4+IL-10) response. Cardiac infiltration of macrophages and TNF-α and oxidant levels were significantly reduced in TcVac2-immunized chagasic mice.

Conclusion: Circulating TcDNA and mtDNA levels and macrophage phenotype mediated by sera constituents reflects *in vivo* levels of parasite persistence, tissue damage and inflammatory/anti-inflammatory state; and have potential utility in evaluating disease severity and efficacy of vaccines and drug therapies.

Biography

Nisha Jain Garg, Ph.D. is currently a Professor in the Departments of Microbiology & Immunology and Pathology in School of Medicine, and serves as Robert E. Shope, MD and John S. Dunn Distinguished Chair in Global Health, Director, Global Health Policy, Epidemics, International Organization and Associate Director, Center for Tropical Diseases at the University of Texas Medical Branch at Galveston. She also serves as a member of the NIH study sections and the American Society of Microbiology International Education Board; Associate editor of the American Journal of Pathology and the Journal of Neuroparasitology; and on the Editorial Boards of several journals. Recently she served as Senior Scientific Advisor at the US Agency for International Development USAID. She has developed a strong and successful research program in the field of tropical infectious cardiomyopathy.

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