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TcVac2^{R} (DNA/protein), but not TcVac1^{R} (DNA/DNA), is protective against *T. cruzi* infection and Chagas disease in dogs

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American trypanosomiasis (Chagas disease) is caused by the protozoan *Trypanosoma cruzi* of the family trypanosomatidae. Approximately, 40% of the infected patients develop a chronic debilitating illness of the cardiac system, characterized by clinically irreversible and progressive tissue destruction, and myocardial hypertrophy, eventually leading to heart failure and the patient's death. Dogs are an excellent model for studying the human disease. Further, the risk of *T. cruzi* infection in humans is increased by the presence of infected dogs in domestic environment, and strategies that can limit *T. cruzi* infection in the reservoir host would be effective in interrupting the parasite transmission to the vector, and consequently, to the human host. We will present the current status of vaccine development efforts and discuss our recent results related to testing the prophylactic and transmission-blocking efficacy of 2nd generation subunit vaccines, TcVac1^{R} (DNA/DNA) and TcVac2^{R} (DNA/protein) in dogs. Dogs (3-4 months old) vaccinated with TcVac1^{R} or TcVac2^{R} elicited strong IgM and IgG antibody response. Yet, the trypanolytic activity of antibodies elicited by TcVac2^{R} was significantly higher (up to 80%) compared to that observed in anti-sera from TcVac1-vaccinated dogs (46-50%). Upon challenge infection with *T. cruzi*, all dogs exhibited a rapid expansion of IgG response. Dogs vaccinated with TcVac2^{R} exhibited a significant decline in acute parasitemia, while TcVac1-vaccinated dogs failed to control acute parasitemia. Clinical exam (EKG analysis) of TcVac2^{R}-vaccinated dogs at 60 dpi detected mild alterations while control dogs and dogs given TcVac1^{R} were polysymptomatic and exhibited conduction problems, myocarditis, and or pericarditis. Anatomopathological analysis after necropsy (at 60 dpi) showed a decline in myocardial and colonic pathology in dogs vaccinated with TcVac2^{R}. Together, these results indicate that TcVac2^{R} would be effective in controlling *T. cruzi* infection and Chagas disease in dogs. These studies were supported in part by grants from American Heart Association (0855059F) and National Health Institute (AI072538) to NJG.

Biography

Dr. Nisha Jain Garg, Ph.D. is currently Professor in the Departments of Microbiology & Immunology and Pathology. Dr. Garg has developed a strong and successful research program in the field of tropical infectious cardiomyopathy. Her research efforts have led to >50 peer-reviewed journal articles on the concept of Chagas disease, and >15 extramurally funded projects. She serves as a member of the NIH study section 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 the Infection and Immunity Journal and the Journal of Geriatric Cardiology. Recently she served as Senior Scientific Advisor at the US Agency for International Development (USAID), engaged in implementation of Neglected Tropical Diseases Initiative of the US Government in Latin America.

Dr. Garg's lab efforts are targeted to win the human fight against Trypanosoma cruzi that causes Chagasic cardiomyopathy. Chagas disease is a major public health threat in Latin America and Mexico, and recognized as an emerging infectious disease in the U.S. Her ongoing translational research with multiple international collaborations focuses on identifying the potential vaccine candidates, and using these candidates to develop multi-component vaccine(s) that provide protection against different T. cruzi strains in multiple animal hosts and humans. Working with young students and scientists in the lab, Dr. Garg utilizes innovative approaches to understand the pathomechanisms of oxidative stress in progressive Chagas disease, and develop adjunct therapies that can prevent or arrest the chronic heart failure.