1. **Link between adipocyte physiology and chronic infectious diseases:** We have shown that adipose tissue acts as a reservoir for the pathogens like *T. cruzi* (parasite) and *M. tuberculosis* (bacterium) in both animals and humans during Chagas disease, and in animal models of TB disease, respectively. These pathogens target adipose tissue, invading adipocytes and using their fat stores for replication and as an energy source during acute infection. Indeed, my laboratory has used several different mouse models to demonstrate that adipocytes play a major role in determining parasitemia, cardiac parasite load, and cardiac pathology during acute *T. cruzi* infection. Also, we showed a significant association between adipocyte physiology and lung pathology during Mtbb infection.


2. **Role of diet and metabolic drugs in chronic infectious diseases:** We have provided abundant evidence that whole body lipid homeostasis is altered during acute *Trypanosoma cruzi* infection. Consistent with these observations, my laboratory also demonstrated that diet and metabolic drugs play important roles in the pathogenesis of Chagas disease both during acute and chronic stages. For instance, high fat diet and metformin increased infected mouse survival in the murine acute Chagas model compared with mice fed on control diet. We also demonstrated a link between diet, adipogenesis, lipolysis and survival during acute infection. These studies are particularly important as the epidemics of diabetes and obesity are emerging in the endemic regions of Chagas disease, where they have significant potential to alter the pathogenesis of Chagas disease.


e. Burke S, Nagajyothi F, Thi MM, Hanani M, Scherer PE, Tanowitz HB, Spray DC (2014) Adipocytes in both brown and white adipose tissue of adult mice are functionally connected via gap junctions:
3. **Role of host lipids in the pathogenesis of Chagasic cardiomyopathy:** Chagas disease is caused by the parasite *Trypanosoma cruzi*. Even after 100 years since its discovery, the mechanisms involved in the parasite invasion and the pathogenesis of Chagas disease are not completely understood. My laboratory demonstrated that *T. cruzi* has high affinity for host lipoproteins and utilizes the LDL receptor for invasion. These discoveries were a major turning point in understanding the pathogenesis of Chagas disease. The parasites bound to LDL bring in cholesterol into the cell during invasion, elevating intracellular cholesterol and causing cardiac lipidopathy. We demonstrated increased cholesterol accumulation in heart sections of cardiomyopathic Chagas patient, which is a novel mechanism for the pathogenesis of Chagas disease.


Complete List of Published Work in My Bibliography: (Jyothi F Nagajyothi and Fnu Nagajyothi)