

Simvastatin and Benznidazole-Mediated Prevention of Trypanosoma cruzi-Induced Endothelial Activation: Role of 15-epi-lipoxin A4 in the Action of Simvastatin

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© 2015 Campos-Estrada et al. Trypanosoma cruzi is the causal agent of Chagas Disease that is endemic in Latin American, afflicting more than ten million people approximately. This disease has two phases, acute and chronic. The acute phase is often asymptomatic, but with time it progresses to the chronic phase, affecting the heart and gastrointestinal tract and can be lethal. Chronic Chagas cardiomyopathy involves an inflammatory vasculopathy. Endothelial activation during Chagas disease entails the expression of cell adhesion molecules such as E-selectin, vascular cell adhesion molecule-1 (VCAM-1) and intercellular cell adhesion molecule-1 (ICAM-1) through a mechanism involving NF- κ B activation. Currently, specific trypanocidal therapy remains on benznidazole, although new triazole derivatives are promising. A novel strategy is proposed that aims at some pathophysiological processes to facilitate current antiparasitic therapy, decreasing treatment length or doses and slowing disease pro