

## Tropical Diseases 2020: Sulfotopes from *Trypanosoma cruzi* major or minor antigenic glycoproteins, are involved in parasite infection, and immunopathogenesis of experimental Chagas disease – Vilma G Duschak – Instituto Nacional de Parasitol

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**Statement of the Problem:** Chagas disease (ChD) constitutes a major endemic health problem in Latin America. The presence of sulfate-bearing-glycoproteins has been identified in *Trypanosoma cruzi*, they are targets of specific immune responses and subjects chronically infected with *T. cruzi* mount specific humoral immune responses to sulfated glycoproteins. Cruzipain (Cz), a major antigen. Containing a C-terminal domain (C-T), is responsible Crossbreeding transfers the kDNA mutations to the chicken progeny. The DNA-mutated chickens grow simple cardiomyopathy in adult life and die of heart failure.

The phenotyping of the lesions exposed that cytotoxic CD45, CD8+  $\gamma\delta$ , and CD8 $\alpha$ + T lymphocytes carry out the rejection of the chicken heart. This membrane flexibility enabled the prokaryote-derived eukaryote to develop key properties, including phagotrophy; an internal membrane system with peroxisomes, a cytoskeleton, and a nucleus; cell division; and sex. These results suggest that the inflammatory cardiomyopathy of Chagas' disease is a genetically driven autoimmune disease for the immunogenicity of the molecule in natural and experimental infection Synthetic anionic sugar conjugates containing Acute *Trypanosoma cruzi* infections can be asymptomatic, but chronically infected individuals can die of Chagas disease. The transfer of the parasite mitochondrial kinetoplast DNA (kDNA) minicircle to the genome of chagasic patients can explain the pathogenesis of the disease N-acetyl D glucosamine-6-sulfate (NAcGlc6-SO<sub>3</sub>) mimics the N-glycan-linked sulfated epitope (sulfotopo) displayed in the C-T. IgG2 antibody levels specific for sulfotopes are inversely correlated with Chagas disease severity.

Another sulfated glycoprotein a bifurcation between prokaryotes and eukaryotes was resolved circa 1 billion years ago. In that epoch, major suites of evolution occurred, including the origin of cellular structures and organelles with all the novelties included in the compendium of eukaryotic biology. With the evolution of phagotrophy with serine carboxypeptidase (SCP) activity was studied. Chagas' disease is the most lethal disease is considered incurable, and its high mortality rates translate to hundreds of thousands of deaths per year endemic infectious ailment in the Western Hemisphere, with a devastating effect upon populations in rural areas of Latin America. Chagas' heart disease typically kills people in the age range of 30 to 50 years.

**Methodology & Theoretical Orientation:** Native SCP copurifies with Cz from Concanavalin-A affinity columns. The Cz-SCP mixture was desulfated, ascribing the cross-reactivity between both molecules to the presence of sulfated groups. SCP-N-glycosidic chains after an editor-instigated inquisition regarding the authenticity of the kinetoplast DNA [kDNA] integration site, which was considered "open to alternative interpretations," Genotype modifications of the host's cells are associated with the pathogenesis of autoimmune Chagas' disease in the cross-kingdom, parasite-free chicken model system and the article can no longer be obtained from the Cell website as a legible transcript. Primary data on the integration of parasite DNA into vertebrate genomes have taken on a controversial nature were analysed by UV-MALDI-TOF-MS. Immunoblotting of lysates from the An accurate evaluation of the role played by the genotype alterations in the autoimmune rejection of self-tissues in Chagas' disease is achieved with the cross-kingdom chicken model system, which is refractory to *T. cruzi* infections After six years, the editor did not show experimental data to refute the original observations of kDNA integration. Now, unadulterated copies of the Cell article different parasite stages were confronted with SO<sub>3</sub>-specific antibodies; *in vivo* effects of sodium chlorate on Cz-sulfation and tissue damage in C-T-immunized-mice muscle-tissues were evaluated.

**Findings:** I) the presence of short-sulfated high-mannose-type oligosaccharidic chains was confirmed in SCP II) sulfotopes participate in trypanomastigotes infection of cardiac cells; iii) sulfotopes generate muscle tissue damage in BALB/c mice, in absence of infection. iv) sulfotopes from Cz and other sulfated glycoproteins participate in parasite infection and immune pathogenesis. v) Sulfotopes and their specific antibodies are responsible for the ultrastructural abnormalities observed in the outcome of the experimental ChD disease vi) a band with apparent molecular weight similar to SCP was highly recognized in trypanomastigotes: vi) SCP is a minor antigen recognized by most of chronic-Chagas- basic parasitologic, immunologic, molecular biology, genetic, clinical, and pathology aspects required to approach questions related to the pathogenesis of Chagas' disease. Life-long, cryptic *Trypanosoma cruzi* infections provide the grounds for the transfer of parasite mitochondrial minicircle sequences that accumulate and spread DNA insertions throughout the human host genome over time disease-patient's sera.

**Conclusion & Significance:** The shared sulfotopes between Cz and SCP, and the enhanced presence of sulfotopes in trypomastigotes, are involved in parasite-host relationship, in immunopathogenic and infection processes.