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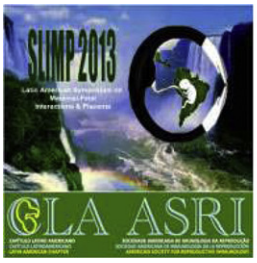

Abstracts for the V SLIMP – IV LASRI Meeting 2013

February 18-20th, 2013

V Latin American Symposium on Maternal Fetal Interaction & Placenta
IV Latin American Symposium on Reproductive Immunology

Raffain Palace Hotel Convention Center Foz do Iguaçu, Brazil

Support and Sponsorship

Abstract Outline V SLIMP – IV LASRI 2013

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G8

Implications of oxidative stress and placental structural alterations in *Trypanosoma cruzi* infection in vitro and ex vivo

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Human placenta participates in the control of Chagas congenital infection, which could be due by nitric oxide synthase (NOS) and peroxinitrites which are deleterious for *Trypanosoma cruzi* and placental tissue. We proposed to analyze the detachment of STB and its relation to oxidative stress and congenital transmission. Two experimental designs: *In vitro*: Placental villi explants co-cultured with and without *T. cruzi*. *Ex vivo:* placentas from chagasic mothers with (CT) and without (NC) congenital transmission. It was analyzed eNOS, Nitrotyrosine (NT) and detachment of STB. Detachment of STB was increased and induced by Tc and H_2O_2 *in vitro*, however it was not significantly higher in ex vivo placentas. These changes were associated to modifications in the expression of eNOS and NT both in vitro and ex vivo. The balance between deleterious effect on *T. cruzi*, and structural placental alterations produced by nitrosative stress, could participate in the infection of placenta by the parasite.
Grants: SECyT-UNC, SECyT-UNLaR, MINCyT-Córdoba.

Keywords: placenta, *Trypanosoma cruzi*, congenital Chagas, oxidative stress

G9

IL-10, TGF-β1 and IFN-γ modulate ICAM-1 expression and cytokine signaling pathways controlling *Toxoplasma gondii* infection in human BeWo cells

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Background: IL-10, TGF-β1 and IFN-γ are involved in the susceptibility of BeWo trophoblast cells to *Toxoplasma gondii* infection. Approved by ethics committee (Protocol #197/08), the present study investigated the effector mechanisms triggered by these cytokines in BeWo cells.
Methods: Cells were treated with these cytokines, infected with *T. gondii,* and analyzed for the susceptibility of infection, ICAM-1 expression, phosphorylation of STAT-1, STAT-3 and Smad2/3, and Th1/Th2/IL-17A cytokine production.
Results: IL-10 and TGF-β1 favored susceptibility to infection, but only TGF-β1 and IFN-γ increased ICAM-1 expression. These findings were associated with the phosphorylation of STAT-3 and STAT-1, but not with Smad2/3. IL-10 and TGF-β1 down modulated TNF-α and IL-6, while IFN-γ upregulated IL-6 and IL-17A release.
Conclusion: IL-10, TGF-β1 and IFN-γ regulate susceptibility to *T. gondii* in human trophoblast cells because differentially modulate ICAM-1 expression, cytokine production and distinct intracellular signaling pathways, contributing to the understanding of the vertical transmission of this parasite.
Financial support: FAPEMIG, CNPq and CAPES.

Keywords: trophoblast cells, *Toxoplasma gondii*, cytokines, ICAM-1 and signaling pathways