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Oral presentation

Co-infection with *Trypanosoma cruzi* (Chagas' disease agent) decreases HIV-1 transcription in human placenta

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Background

Several factors determine the risk of HIV mother-to-child transmission (MTCT), such as co-infections in placentae from HIV-1 positive mothers with other pathogens [1,2]. One of the most important endemic zoonosis in Latin America is Chagas' disease, caused by the protozoa *Trypanosoma cruzi*. MTCT of *T. cruzi* is today one of the main transmission routes in big cities [3]. The aim of the study was to determine whether *T. cruzi* modifies HIV infection at tissue or cellular level of the placenta.

Material and methods

Simple and double infections were carried out on a placental histoculture system (chorionic villi isolated from term placentae from HIV and Chagas negative mothers) and on choriocarcinoma BeWo cell line. We used trypomastigotes of *T. cruzi* (VD lethal strain, isolated from a child with MTCT), either purified from mice blood or from Vero cell cultures, 24h-supernatants of blood and cellular trypomastigotes, and the viral HIV-1DenvLuc+/VSV-G pseudotype. Viral replication was evaluated by luciferase activity quantification. Tissue viability was evaluated by hCG hormone secretion in histoculture supernatant. Quantification of soluble factors protein secretion and mRNA expression in histocultures were carried out by ELISAs and real time PCR respectively.

Results

Whole trypomastigotes, either from mice blood (mean ± SD; −92.13% ± 4.85) or from cell cultures (−97.33% ± 0.58), in co-infection with viral pseudotype decreased luciferase activity in placental histocultures. Similar results were obtained on BeWo cells. When supernatants from blood trypomastigotes were used on placental histocultures, luciferase activity presented a decrease of −85.50% ± 2.12, while supernatants from culture trypomastigotes presented lower effects (−65.50% ± 2.12). Tissue viability was not modified by viral and/or parasite infection. In co-infected histocultures, both protein secretion and mRNA expression of IL-10 were down regulated. Surprisingly, RANTES was increased.

Conclusions

Acute infection with *T. cruzi* and HIV-1 in this placentae *in vitro* system as well as in the trophoblast cell line decreased HIV-1 transcription. *T. cruzi* activity on HIV-1 replication seems to be caused not only by active infection but also by soluble factors shed by the parasite. A balance between proinflammatory and inhibitory cytokines/chimiokines might have a role in this phenomenon. Ongoing experiments are being conducted in order to elucidate the mechanisms involved in the impairment of HIV

replication by *T. cruzi* and their role on MTCT of both pathogens.

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