Effect of serum limitation in the extracellular hydrolysis of nucleotides and in the cytotoxicity of *Trichomonas vaginalis* against vaginal and prostate epithelial cells

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Trichomonas vaginalis is a flagellated protozoan that causes trichomoniasis, the most common non-viral sexually transmitted infection (STI) in the world. There is only two FDA-approved drugs for the treatment of trichomoniasis, metronidazole and tinidazole. Given the drug resistance and failures in the treatment, the search of new agents is essential. The purinergic signaling occurs when nucleotides and nucleosides, regulated by enzymes called ectonucleotidases, bind to specific receptors called purinoceptors. Ectonucleotidases are the enzymes involved in the hydrolysis of extracellular nucleotides and include NTPDase, ecto-5'-nucleotidase, and adenosine deaminase. These activities have been characterized in *T. vaginalis* by our group. The aim of this study was to investigate the effect of heat inactivated bovine serum (HIBS) limitation as adenosine source restriction in the cytotoxicity involved in the pathogenesis of trichomoniasis. The *T. vaginalis* isolates were cultivated in TYM medium at 37ºC supplemented with 10% HIBS and treated parasites were grown with 1.0% HIBS based on a kinetic growth assay. The cytotoxicity of *T. vaginalis* was measured by the release of lactate dehydrogenase (LDH) by vaginal epithelial cells (HMVII) and by prostate epithelial cells (DU145). The NTPDase activity was tested using ATP and ADP as substrates by malachite green colorimetric method. All isolates tested were cytotoxic against both lineages and the highest level of LDH release by cells occured at 5 h. All 1.0% HIBS-treated isolates showed lower numbers of trophozoites in relation to control up to 48 h. The NTPDase activity strongly increased in 1.0% HIBS-treated parasites (three-fold for ATP). Assays to determinate the effect of serum limitation in ecto-5'-nucleotidase and adenosine deaminase activities, as well as in the cytotoxicity exerted by *T. vaginalis* against host cells are in progress.