Iron Deprivation Decreases Adenosine Deaminase Activity in

**Trichomonas vaginalis**

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*Trichomonas vaginalis* is a flagellated protozoan that causes trichomoniasis, the most common non-viral sexually transmitted disease responsible for 170 million new cases annually worldwide. All living organisms require iron for many biological functions. *T. vaginalis* has multiple iron acquisition systems, and iron plays a key role in modulating the pathogenesis of trichomoniasis, such as increase of cytoadherence and resistance to complement. Extracellular nucleotides are released during stress, anoxia or injury, and the enzymes ectonucleoside triphosphate diphosphohydrolase (NTPDase) and ecto-5'-nucleotidase, break down ATP to adenosine, which is finally degraded to inosine by adenosine deaminase (ADA). The presence of these enzymes on *T. vaginalis* surface has been studied, and it suggests a role in the modulation of nucleotides/nucleosides levels during inflammation and immune response. The aim of this study was to investigate the effect of iron in *T. vaginalis* ADA activity. *T. vaginalis* ATCC and fresh clinical isolates were cultivated in trypticase-yeast extract-maltose (TYM) medium at 37°C. The treatments to test the effect of iron were performed using different sources: 50 µM 2,2-bipyridil (iron chelator), 200 µM ferrous sulfate, 25 µM hemoglobin, and 25 µM hemin. ADA activity in intact trophozoites was determined by colorimetric reaction based on ammonia production and specific activity was expressed as nmol NH₃/min/mg protein. Results showed a significant reduction in enzyme activity when the bipyridil chelator was used. Our findings show that iron deprivation decreases ADA activity in *T. vaginalis*, revealing the purinergic system involvement on *T. vaginalis* infection through adenosine degradation modulated by iron.

Key Words: adenosine deaminase, iron, purinergic system, *Trichomonas vaginalis*

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