**_Schistosoma mansoni_ infection associated with dyslipidemia in an experimental model: implications for visceral adipose tissue after praziquantel treatment**

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Schistosomiasis is a neglected disease that affects millions of people in endemic areas and it's heavily influenced by changes in the host's metabolic profile. To investigate the effects of praziquantel (PZQ) treatment on acute schistosomiasis infection in animals subjected to a high-fat diet (HFD), female Swiss webster mice were fed either an HFD or a standard diet (SD) for 34 weeks. The animals were subcutaneously infected with 100 *Schistosoma mansoni* cercariae (LE strain) in the 24$^{th}$ week, and received by gavage 60 mg/kg of PZQ in the 32$^{nd}$ week. Body mass (BM) and food intake (FI) were monitored weekly. At week 34 of the experiment mice were euthanized, blood samples were collected for biochemical analyses (lipids levels), and macrophages (MAC) were cultured for indirect evaluation of nitric oxide (NO) production. Visceral adipose tissue (VAT) was removed, fixed, histologically processed, and stained with Hematoxylin and Eosin for stereological and histopathological analyses. The results showed that HFD promoted a progressive and sustained increase in BM, high lipids levels compared to animals fed an SD, even with a reduction in FI, inducing dyslipidemia. However, the treatment appeared to attenuate the protective effect of the infection against excessive lipid accumulation in the host. When analyzed individually, both HFD and infection enhanced the inflammatory response in stimulated macrophages. However, the combination of these conditions revealed an inhibitory effect on NO production, suggesting a remodeling of the inflammatory response. Stereological and histopathological analyses indicated that HFD promoted adipocyte hypertrophy, while infection induced hyperplasia. Treatment, on the other hand, resulted in a mixed profile of these cellular changes. In conclusion, the interaction between HFD, acute schistosomiasis infection, and PZQ treatment impacts the course of the infection, modulating both metabolic and inflammatory parameters.

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