Targeted proteomics applied to serum from infected C57BL/6 mice with Schistosoma mansoni: emphasis on lipid metabolism

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Schistosomiasis is a tropical disease caused by parasites of the genus Schistosoma. Widely spread throughout the world, it currently affects 240 million individuals. S. mansoni is the only species described in Brazil, where 25 million people live in areas at risk of contracting the disease. The parasite has a complex life cycle, is capable of evading the immune system and causes relevant clinical manifestations in the vertebrate host. Previous work by our research group demonstrated that S. mansoni infection in the murine model (BALB/c lineage) promotes changes in the abundance of several serum constituents, including molecules related to inflammation, regulation and lipid transport. Continuing this line of investigation, the present study evaluated the histological aspect of adipose tissue and quantified, via targeted proteomics, 36 constituents in the serum of 56 mice of the C57BL6 lineage at 3, 5, 7 and 12 weeks post infection. Morphological analyzes of adipose tissue demonstrated that the infection promotes inflammation in this tissue, evidenced by an increase in the number of cell nuclei and a decrease in the area of adipocytes. These findings corroborate the proteomic data obtained, which showed an increase in the levels of proteins with inflammatory activity throughout the infection, such as ApoB-100, and a decrease in proteins related to the anti-inflammatory response, such as APOA-IV. Concomitant to this, an increase in CD5L protein was observed, which is related to lipolysis in adipocytes. The inflammatory profile observed in adipose tissue reflects systematically, with an increase in acute phase response proteins such as Alpha-1-antitrypsin, Alpha-1-acid glycoprotein 1, Serum amyloid and Kininogen II. Particularly, the abundance of the complement system showed that in schistosomiasis there is activation of the pathways, due to the increase in initial constituents (such as C3) in infected animals, but through some unknown mechanism there is interruption of the signaling cascade (evidenced by the decrease in components end of the pathway, such as C8) in infected animals. The infection also promotes changes in metal-binding proteins, such as Hemoglobin and Ceruloplasmin, and proteins related to the processing of associated components, such as Hemopexin and Serotransferrin. Taken together, the data obtained for the seroproteome in the S. mansoni infection model, in the C57BL6 lineage, reinforce previous findings regarding the establishment of tissue and systemic inflammation, changes in metabolism and lipid transport and hemostasis. Furthermore, this work contributes to the understanding of the complex host-parasite relationship and can direct new methodological approaches for diagnostic purposes, early detection of tissue changes and disease prognosis.

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