Proteomic analysis of Rattus norvegicus serum: insights into a non-permissive host response to a Schistosoma mansoni infection

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Schistosomiasis is a serious and neglected tropical disease, caused by parasites of the genus Schistosoma, which affects more than 350 million people globally, being considered the second with the greatest socioeconomic impact, behind only Malaria. In Brazil, S. mansoni is the only described species, and it is estimated that more than 25 million people live at risk of contracting the disease. The parasite\'s life cycle includes invertebrate and vertebrate hosts, with infection occurring in the aquatic environment, when the parasite actively penetrates the skin and begins to inhabit the circulatory system. Previous investigations from our research group have highlighted various proteomic alterations in the abundance of serum constituents from permissive hosts (BALB/c and C57BL/6 mice), related to signaling pathways, lipid transport, inflammation and immune response. Continuing the investigation on the host\'s serumproteome, the present work carried out a shotgun proteomic analysis by liquid nano chromatography coupled to mass spectrometry (shotgun), of control and infected Rattus norvegicus, a known S. mansoni resistant host at 3 weeks post infection. The compositional analysis of components uniquely abundant in infected individuals revealed constituents related to different functions, such as proteins associated with liver damage (Bile acid-CoA: amino acid N-acyltransferase, BACAT), Cytosolic 10-formyltetrahydrofolate dehydrogenase and Oncoprotein-induced transcript 3 proteins; response to oxidative stress such as Mitogen-activated protein kinase and Sulfotransferase 1A1 (ST1A1); response mediated by reactive oxygen species (Argininosuccinate lyase); Glutathione metabolism (Nitrilase 1, Deaminated glutathione amidase (dGSH amidase) and Glutathione S-transferase alpha-2); immune response (AT-rich interactive domain-containing protein 5B and CTP synthase (UTP-ammonia ligase), Myosin regulatory light chain 12B (Myosin RLC-B) and inflammation (Myosin light chain 12B and Myosin regulatory light polypeptide 9). These findings revealed that the infection promotes alterations in the host\'s proteome leading to a differential immune response, control of cellular damage, and hepatic metabolism. Additional analyses are underway to aid in the understanding of the complex molecular mechanisms supporting the self-cure response exhibited by the rat model of infection.

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