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The possible molecular mechanisms of *Schistosoma japonicum* soluble egg antigens in reverse fibrosis

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Hepatic stellate cells play a key role in the development of hepatic fibrosis. Activated hepatic stellate cells can be reversed to a quiescent-like state or apoptosis can be used to induce reverse fibrosis. Some studies have recently shown that *Schistosoma mansoni* eggs could suppress the activation of hepatic stellate cells and that soluble egg antigens from schistosome eggs could promote immunocyte apoptosis. Hence, our study attempts to assess the direct effects of *Schistosoma japonicum* soluble egg antigens on hepatic stellate cell apoptosis, and to explore the mechanism by which the apoptosis of activated hepatic stellate cells can be induced by soluble egg antigens, as well as the mechanism by which hepatic stellate cell activation is inhibited by soluble egg antigens. Here, it was shown that *S. japonicum*-infected mouse livers had increased apoptosis phenomena and a variability of peroxisome proliferator-activated receptor α expression. Soluble egg antigens induce morphological changes in the hepatic stellate cell LX-2 cell line, inhibit cell proliferation and induce cell-cycle arrest at the G1 phase. Soluble egg antigens also induce apoptosis in hepatic stellate cells through the TNF-related apoptosis-inducing ligand/death receptor and caspase-dependent pathways. Additionally, soluble egg antigens could inhibit the activation of hepatic stellate cells through peroxisome proliferator-activated receptor and the transforming growth factor signalling pathways. Therefore, our study provides new insights into the anti-fibrotic effects of *S. japonicum* soluble egg antigens on hepatic stellate cell apoptosis and the underlying mechanism by which the liver fibrosis could be attenuated by soluble egg antigens.

Biography

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