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CORRECTION

Correction to: Schistosomiasis—from immunopathology to vaccines

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The original version of this article inadvertently missed out to display the correct acknowledgement for Fig. 2. The corrected legend of Fig. 2 is given below.

The original article has been corrected.

The online version of the original article can be found at https://doi.org/10.1007/s00281-020-00789-x

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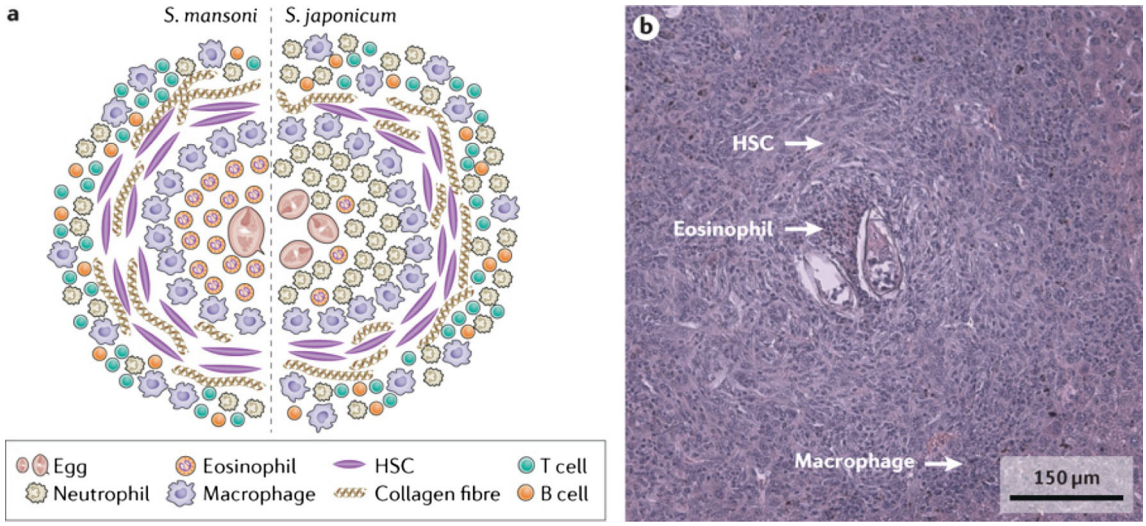

Fig. 2 Features of schistosome-induced granuloma formation. Adult schistosome worm pairs residing in mesenteric veins produce eggs some of which become entrapped in the host's liver (or other organs) tissue where they evoke a dominant CD4(+) T_H2 immune response mediated by IL-4 and IL-13. This leads to the development of granulomas and fibrosis with hepatic stellate cells, macrophages, lymphocytes, neutrophils, and eosinophils, all identified as major cellular contributors to these events. **a** Major cellular populations located within and adjacent to the hepatic granuloma induced in either *S. japonicum* or *S. mansoni* infection. Whereas a dense population of eosinophils are present at the core of a *S. mansoni*-induced hepatic granuloma, the core in a *S. japonicum* infection is comprised chiefly of neutrophils. Chemokine-binding proteins secreted by the eggs of *S. mansoni* eggs bind neutrophil chemoattractant C-X-C-motif chemokine ligand 8 (CXCL8), thereby blocking the infiltration of neutrophils to the granuloma. In contrast, these proteins do not bind to eosinophil chemoattractant CC-chemokine ligand 11 (CCL11) and, therefore, do not inhibit the recruitment of eosinophils. **b** A granuloma in the liver of a *S. mansoni*-infected mouse with hepatic stellate cells (HSCs). Part a adapted with permission from Chuah, C., Jones, M. K., Burke, M. L., McManus, D. P. & Gobert, G. N. Cellular and chemokine-mediated regulation in schistosome-induced hepatic pathology. Trends Parasitol. 30, 141–150 (2014), Elsevier. Part b courtesy of A. M. O. Kildemoes, University of Copenhagen, Denmark