Tissue-Specific Helminth Conditioning Protects Against African Trypanosome-Mediated Pathology

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Helminth endemic regions report lower morbidity or mortality to other infectious diseases. This has been suggested to be as a result of the lingering effect of innate and adaptive type 2 and regulatory responses elicited by helminth pathogens. Since gastrointestinal helminth infections and African trypanosomiasis are co-endemic in sub-Saharan Africa, it is plausible that host responses to intestinal worm infections may modulate responses to trypanosome infections. Here, we used a mouse model to determine the impact of prior acute high dose oral infection with the helminth pathogen *Trichuris muris* on the pathogenesis of an intradermal African trypanosome (*Trypanosoma brucei*) infection. Our data show that helminth co-infection reduced the parasitemia, clinical disease and inflammatory cytokine such as IFN-γ responses to subsequent *T. brucei* infection. Prior acute Th2 dominant helminth infection also provided protection against severe trypanosome induced pathologies in the skin (adipose tissue wasting and inflammation) and skin draining lymph nodes. However, splenic pathologies such as splenomegaly and B lymphocyte depletion were execrated in the co-infected mice. Together, our data suggest that the impact of gastrointestinal helminth infections on the host immune system modulate subsequent responses to co-infections with African trypanosomes, altering the disease pathogenesis.