Schistosoma mansoni-mediated suppression of allergic airway inflammation requires patency and Foxp3+ Treg cells.

The continual rise of asthma in industrialised countries stands in strong contrast to the situation in developing lands. According to the modified Hygiene Hypothesis, helminths play a major role in suppressing bystander immune responses to allergens, and both epidemiological and experimental studies suggest that the tropical parasitic trematode Schistosoma mansoni elicits such effects. The focus of this study was to investigate which developmental stages of schistosome infection confer suppression of allergic airway inflammation (AAI) using ovalbumin (OVA) as a model allergen. Moreover, we assessed the functional role and localization of infection-induced CD4(+)Foxp3(+) regulatory T cells (Treg) in mediating such suppressive effects. Therefore, AAI was elicited using OVA/adjuvant sensitizations with subsequent OVA aerosolic challenge and was induced during various stages of infection, as well as after successful anti-helminthic treatment with praziquantel. The role of Treg was determined by specifically depleting Treg in a genetically modified mouse model (DEREG) during schistosome infection. Alterations in AAI were determined by cell infiltration levels into the bronchial system, OVA-specific IgE and Th2 type responses, airway hyper-sensitivity and lung pathology.
Our results demonstrate that schistosome infection leads to a suppression of OVA-induced AAI when mice are challenged during the patent phase of infection: production of eggs by fecund female worms. Moreover, this ameliorating effect does not persist after anti-helminthic treatment, and depletion of Treg reverts suppression, resulting in aggravated AAI responses. This is most likely due to a delayed reconstitution of Treg in infected-depleted animals which have strong ongoing immune responses. In summary, we conclude that schistosome-mediated suppression of AAI requires the presence of viable eggs and infection-driven Treg cells. These data provide evidence that helminth derived products could be incorporated into treatment strategies that specifically target suppression of immune responses in AAI by inducing Treg cells.

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