IL-10 and TGF- β 1 Cytokines Stimulated by *Ascaris lumbricoides* Body Fluid Are Associated with Secretory IgA Levels Against Other Intestinal Pathogens Attenuating Diarrheal Symptoms

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To the Editor: It has been extensively reported that helminth-derived excretory/secretory products stimulate, nonspecifically, the production of TGF β and IL-10 regulatory cytokines by Foxp3⁺ T regulatory cells, generating a tolerant microenvironment in the gut, which allows them to persist for long periods of time preventing parasite-associated pathologies and modulating bystander inflammatory processes [1]. Thereby, the possible influence of *Ascaris lumbricoides* body fluid (ABF)-stimulated IL-10 and TGF- β cytokines on secretory IgA levels and diarrheal symptoms were studied in a group of Venezuelan slum preschool children. Clinical, parasitological, and bacteriological studies were carried out. sIgA levels in the sublingual/submandibular saliva fluids and cytokines in supernatants of ABF-stimulated whole blood cultures were measured by ELISA. Data were analyzed by nonparametric ANOVA. It was found that 69.2% of the children were helminth-parasitized with *A. lumbricoides* being the most prevalent parasite. By the time of examination, 32.7% had diarrheal symptoms mainly caused by *Giardia duodenalis* and *Escherichia coli* EPEC. Helminthic infection did not prevent from infections by other intestinal parasites. However, regardless of the infective pathogen, diarrheal symptoms were lower ($p < 0.0001$) among helminth-parasitized children. Values of ABF-stimulated TGF β 1 and IL-10 were higher ($p < 0.005$) among helminth parasitized children and correlated positively ($p < 0.001$) with total, anti-*G. duodenalis* and anti-*E. coli* EPEC sIgA values. Diarrhea symptoms were lower ($p < 0.001$) among children with elevated levels of sIgA. The results of this work

suggest that ABF-stimulated IL-10 and TGF- β may enhance nonspecifically the production of total and specific sIgA against other enteric pathogens. Through this mechanism helminths may favor the development of anti-inflammatory mechanisms driven by secretory IgA [2]. Besides, the stimulation of high levels of specific sIgA against *E. coli* EPEC and *G. duodenalis* would enhance prevention of mucosal adhesion of these pathogens [3, 4], thus limiting inflammation and ameliorating intestinal symptoms. Nevertheless, further studies are needed to confirm these preliminary results.

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Declarations

Conflict of Interest None.

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