

The Impact of antibiotic treatment on *Trichuris muris* Excretory/Secretory Products and their role in host-bacteria interactions

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Trichuriasis is a disease caused by soil-transmitted helminths of the genus *Trichuris*. They infect the cecum of mammals, invade epithelial mucosa, and cause bacterial translocation and dysbiosis. These parasites produce excretory/secretory products (ESPs), which play a role in modulating the host's immune response. This study aimed to extract ESPs from *T. muris* recovered from antibiotic-treated and untreated mice to determine whether antibiotic treatment alters the protein composition of ESPs. Additionally, we assessed the antimicrobial activity of these ESPs and their potential influence on bacterial interactions with T84 and Caco-2 cells *in vitro*. Mice were infected with *T. muris* and either received antibiotics or remained untreated. Parasites were maintained in RPMI medium at 37°C with CO₂ for 24 h. Some samples underwent MS/MS analysis for protein identification, followed by data extraction and quantification, including concentration measurements, KEGG pathway analysis, gene ontology, signal peptide identification, and network analysis. We also evaluated the ESPs' ability to inhibit bacterial growth and their interactions with *Escherichia coli* 042, *E. coli* DH5 α , and *Salmonella* c20 in T84 and Caco-2 cells for 6 h. We observed differences in the protein composition of ESPs between treated and untreated groups. The ESPs from antibiotic-treated mice exhibited a more complex protein profile, with a broader range of functional proteins. Some proteins identified in both groups displayed antimicrobial and immunomodulatory activities. However, antibiogram analyses revealed that ESPs did not inhibit bacterial growth, although *Staphylococcus aureus* exhibited altered aggregation behavior. Additionally, we detected changes in bacterial aggregation and invasive potential, particularly in *E. coli* 042 and *E. coli* DH5 α . Our findings suggest that the composition of ESPs varies depending on the host's microbiota, potentially influencing bacterial interactions within the host.

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