

Decoding the Microbial Influence: How Bacteria from Gut Microbiota Modulates *Giardia intestinalis* Morphology and Pathogenicity

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Giardiasis, an intestinal disease caused by the protozoan parasite *Giardia intestinalis*, affects over 300 million people globally each year. *G. intestinalis* infections are linked to structural and compositional changes in mucosal bacteria, which are part of the stable microbiota colonizing human mucosal surfaces. Evidence indicates that human microbiota can significantly alter the course of giardiasis in hosts. However, the cellular and molecular mechanisms during interactions of *G. intestinalis*, host cells, and microbiota remain unclear. This study aimed to evaluate the cellular effects on *G. intestinalis* cells following interactions with bacteria characteristic of both eubiotic and dysbiotic microbiota. Cell cultures were incubated in two ways: first, with isolated bacteria in Giardia medium to observe the generation time, and second, by co-incubating the parasites with bacteria. The cellular morphology and ultrastructure were examined using Scanning Electron Microscopy (SEM) and Transmission Electron Microscopy (TEM). Co-incubation of the parasites with bacteria revealed alterations in both growth and morphology. Exposure to *Enterococcus lactis* induced parasite lysis, damaging the plasma membrane without altering the structure of the ventral disc. In contrast, interaction with *Lactobacillus casei* resulted in wrinkling of the plasma membrane in the dorsal region and distortion at the edge of the ventral disc. However, co-incubation with the commensal bacterium *Cronobacter sakazakii* did not significantly affect the parasite's morphology, and the interaction with *Escherichia coli* appears to stabilize Giardia's growth. These preliminary results confirm that bacteria exert different morphological effects on *Giardia*. Overall, our findings suggest that eubiotic bacteria may negatively impact *G. intestinalis*, while commensal bacteria do not substantially harm the parasites.

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