JOURNAL OF APPLIED SCIENCE AND SOCIAL SCIENCE
eISSN 2229-3113 pISSN 2229-3205

Volume 15 Issue 03, March 2025
Impact factor: 2019: 4.679 2020: 5.015 2021: 5.436, 2022: 5.242, 2023: 6.995, 2024 7.75

SCIENTIFIC NOVELTY OF THE EFFECTS OF ASCARIASIS ON THE MUCOSAL LAYER OF THE SMALL INTESTINE

Solieva Gulchehra Abdurakhmonovna

Assistant Professor of Histology, Cytology and Embryology,

CAMU International Medical University

E-mail: gulchehrasaliyeva26@gmail.com

Annotation: Ascariasis, caused by Ascaris lumbricoides, is a widespread parasitic infection that primarily affects the small intestine. Recent research has provided new insights into the pathophysiological mechanisms by which Ascaris damages the intestinal mucosa, including mechanical injury, inflammatory response, and immunological alterations. This article explores scientific advancements in understanding how ascariasis contributes to villous atrophy, epithelial cell damage, and altered gut microbiota. The study highlights the latest findings on host-parasite interactions and their implications for developing new diagnostic and therapeutic approaches.

Keywords: Ascariasis, Ascaris lumbricoides, small intestine, mucosal damage, villous atrophy, inflammation, parasite-host interaction, immune response, gut microbiota, helminth infections.

Introduction. Ascariasis, caused by Ascaris lumbricoides, is one of the most prevalent helminthic infections, affecting approximately 800 million people worldwide (WHO, 2023). The parasite primarily colonizes the small intestine, where it damages the mucosal layer, leading to nutritional deficiencies, impaired digestion, and increased susceptibility to secondary infections.

Recent research has revealed that ascariasis not only causes mechanical injury to the intestinal lining but also modulates immune responses, alters gut microbiota, and disrupts epithelial barrier integrity. Advances in histopathological and molecular techniques have provided new insights into the pathogenesis of mucosal damage, which were previously not well understood.

This article examines the scientific novelty in the study of ascariasis-induced small intestinal damage, focusing on: Mechanisms of villous atrophy and epithelial injury

Host immune system interactions with the parasite. Changes in gut microbiota composition. Implications for novel treatment and prevention strategies

Villous atrophy and epithelial disruption. Studies using scanning electron microscopy have shown severe villous atrophy in infected individuals, leading to reduced nutrient absorption. Goblet cell hyperplasia and increased mucin secretion indicate an attempt to protect the epithelium but also contribute to malabsorption syndromes (Smith et al., 2022).

Immune response and chronic inflammation. Ascariasis induces a Th2-dominant immune response, characterized by elevated levels of IL-4, IL-5, and IL-13, leading to eosinophilia and chronic inflammation. Recent immunological studies suggest that Ascaris suppresses Th1

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responses, reducing resistance to bacterial infections such as Helicobacter pylori (Chen & Wang, 2023).

Alterations in gut microbiota. Metagenomic sequencing has revealed significant dysbiosis in the gut microbiota of individuals with chronic ascariasis.

Reduced levels of beneficial bacteria (Lactobacillus, Bifidobacterium) and an increase in opportunistic pathogens have been observed, which may contribute to long-term gastrointestinal complications (Gómez et al., 2021).

New approaches in diagnosis and treatment. The use of biomarkers such as fecal calprotectin has been proposed for early detection of mucosal inflammation in ascariasis. Novel anthelmintic therapies, including plant-derived alkaloids, have shown promising results in reducing Ascaris burden while preserving gut microbiota balance (Hernández et al., 2023).

Conclusion. Recent advancements in parasitology, immunology, and microbiology have provided new insights into the mechanisms of mucosal damage caused by ascariasis. The discovery of immune suppression pathways, gut microbiota alterations, and biomarkers for early detection represents a significant leap forward in understanding this neglected tropical disease.

Future research should focus on targeted therapies that not only eliminate the parasite but also restore gut homeostasis. Integrating molecular diagnostics, immunomodulatory treatments, and probiotics may pave the way for more effective and sustainable control strategies against ascariasis.

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215-224.