IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 22, Issue 8 Ser.2 (August. 2023), PP 01-03
www.iosrjournals.org

Immune Responses To Entamoeba Histolytica Infection: A Comprehensive Review

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Abstract:
Intestinal amebiasis, caused by the protozoan parasite Entamoeba histolytica, remains a significant global health burden. Despite the prevalence of this infection, many individuals exposed to the parasite remain asymptomatic, indicating the presence of host protective mechanisms.This review paper aims to explore and analyze the current scientific literature on the host's immune response and protective mechanisms against E. histolytica infection. The review covers multiple aspects, including mucin exocytosis, gut microbiota, antimicrobial peptides, immune response, vaccine development, epithelial tight junctions, Paneth cells, oxidative stress response, glycosylation, and the interplay between the gut microbiota and Muc2 mucin.The integration of these findings provides a holistic understanding of the host-parasite interaction and offers insights into potential strategies for preventing and managing amebiasis.

Key Words: *Intestinal amebiasis, Entamoeba histolytica. Host immunoresponce.*

Date of Submission: 25-07-2023 Date of Acceptance: 05-08-2023

I. Introduction

Intestinal amebiasis is a parasitic infection caused by *Entamoeba histolytica*, affecting millions of people globally. Although amebiasis can lead to severe gastrointestinal manifestations, a significant number of infected individuals remain asymptomatic carriers. The ability of some individuals to mount effective host protective mechanisms against E. histolytica highlights the complexity of the host-parasite interaction. Understanding these protective mechanisms is crucial for developing effective strategies to prevent and treat amebiasis.

Mucin Exocytosis and Goblet Cell Response

The intestinal mucin layer serves as a physical barrier against pathogens, including E. histolytica. Recent studies have shed light on the role of goblet cells in mucin exocytosis as a response to E. histolytica infection. Cornick et al. (2016) demonstrated that the cysteine protease 5 (EhCP5) of E. histolytica induces mucin exocytosis from colonic goblet cells via αvβ3 integrin activation (1). This process enhances the mucosal immune response, limiting the parasite's invasion and dissemination in the intestinal mucosa. Cornick et al. (2017) identify vesicle SNARE vesicle-associated membrane protein 8 (VAMP8) was specifically activated during E. histolytica infection, and ablation of VAMP8 caused devective mucin secretion. As a result, loss of VAMP8 elevated E. histolytica attachment to epithelial cells associated with enhanced cell death through apoptosis (2) . Leon-Coria, A. et al. (2020) in his study demonstrated that the absence of a protective mucus barrier alters microbiota to promote inflammation and intestinal permeability.(3)

Gut Microbiota and Immune Response

The gut microbiota plays a crucial role in modulating the host immune response.

Watanabe, K. et al. (2017) found that dysbiosis, which was shown by a lower Shannon diversity index of the gut microbiota, occurred symptomatic E. histolytica infection in children in native area, in mouse model, they show that dysbiosis induced by antibiotic pre-treatment elevated the severity of amebic as a result of decreased neutrophil activity and reduced IL-25 associated mucosal defense in the gut. In addition to, they demonstrated surface expression on neutrophils of CXCR2 was diminished in mice with dysbiosis, which resulted in decreased neutrophil recruitment to the gut. This study is of integral importance in amebiasis research for the discovery of a mechanism of microbiome-mediated resistance to amebiasis through neutrophil trafficking to the gut (4).

Ngobeni et al. (2017) investigated the correlation between Entamoeba species infection and the gut microbiome, identifying specific microbial communities associated with different parasite burdens and susceptibility to amebiasis (5). Gilchrist et al. (2016) explored the gut microbiome of children with diarrhea due

to E. histolytica infection, revealing potential associations between specific microbial communities and disease severity (6).

Burgess et al. (2020) discovered that the gut microbiome communicates with the bone marrow, influencing the host's susceptibility to amebiasis (7). They reported that specific gut microbial products can modulate bone marrow function, leading to altered immune responses against E. histolytica.

Antimicrobial Peptides and Immune Regulation

Antimicrobial peptides (AMPs) are necessary elements of innate host defense. Cobo et al. (2017) revealed the role of MUC2 mucin and butyrate in contributing to the synthesis of the AMP cathelicidin in response to E. histolytica-induced colitis (8). The study highlighted how mucin and short-chain fatty acids produced by the gut microbiota participate in the regulation of AMP expression and subsequent protection against the parasite.Noor et al. (2017) examine the function. of eosinophils and tumor necrosis factor-alpha (TNF-α) in interleukin-25 (IL-25)-mediated protection from amebic colitis (9). Their findings suggested that IL-25-induced TNF-α secretion contributes to mucosal immunity against E. histolytica.

Vaccine Development

The development of an effective vaccine is a promising strategy for controlling amebiasis. Abhyankar et al. (2017) studied the nano formulation of synergistic Toll-like receptor (TLR) ligands to enhance vaccination against E. histolytica (10). Their research demonstrated that the incorporation of specific TLR ligands in nanoparticles improved vaccine efficacy and elicited a robust immune response.In another study, Abhyankar et al. (2018) investigated the role of adjuvant composition and delivery route in shaping immune response quality and protective efficacy of a recombinant vaccine against E. histolytica (11). They found that different adjuvants and delivery routes elicited distinct immune responses, with potential implications for vaccine development strategies.

Epithelial Tight Junctions and Paneth Cell Functions

Epithelial tight junctions and Paneth cells are critical components of the mucosal barrier. Cuellar et al. (2017) explored how E. histolytica disrupts tight junction proteins, such as claudin-1 and claudin-2, at the intestinal epithelium, contributing to disease susceptibility (12). Understanding the mechanisms of tight junction disruption could provide new targets for therapeutic interventions.In a study by Cobo et al. (2018), the role of E. histolytica in altering ileal Paneth cell functions was investigated, both in intact and Muc2 mucin-deficient mice (13). The findings shed light on the interplay between the parasite and the host's innate immune system in the context of mucin deficiency.

Intestinal Inflammation and Bacterial Composition

Xiao et al. (2019) investigated the impact of lack in intestinal epithelial Reg4 on intestinal inflammation and alterations in the colonic bacterial composition during E. histolytica infection (14). The study revealed a potential role of Reg4 in regulating inflammation and modulating the gut microbiome during amebiasis.

Wojcik et al. (2018) conducted a genome-wide association study to reveal a genetic link between diarrhea-associated E. histolytica infection and inflammatory bowel disease (IBD) (15). Understanding the shared genetic factors between IBD and amebiasis could provide insights into common pathways involved in intestinal inflammation.

Oxidative Stress Response

Entamoeba histolytica faces oxidative stress in the host gut. Shaulov et al. (2018) demonstrated how Escherichia coli mediates resistance of E. histolytica to oxidative stress by triggering oxaloacetate production (16). This finding sheds light on the intricate interplay between commensal bacteria and the parasite's stress response.

Varet et al. (2018) investigated how enteric bacteria boost defenses against oxidative stress in E. histolytica (17). Their research suggested that gut microbial products can influence the parasite's ability to cope with oxidative stress, thus impacting the course of infection.

Glycosylation and Innate Host Defense

The gut microbiota affects Muc2 mucin O-glycosylation by regulating the differential expression of glycosyltransferases (18). Arike et al. (2017) revealed that specific microbial communities influence the glycosylation of Muc2 mucin, potentially affecting the protective function of the mucosal barrier (18).Leon-Coria et al. (2018) defined cooperative roles for colonic microbiota and Muc2 mucin in mediating innate host defense against E. histolytica (19). Their research highlighted the interdependence between the gut microbiota and the mucosal immune response against the parasite (19).

II. Conclusion

In conclusion, this comprehensive review paper has explored the host protective mechanisms to intestinal amebiasis, offering a thorough analysis of various aspects of the host-parasite interaction. The integration of findings from multiple studies provides a holistic understanding of the complex interplay between the host immune response, gut microbiota, antimicrobial peptides, and oxidative stress, which collectively contribute to the host's ability to fend off E. histolytica infection. Insights from this review can serve as a foundation for the development of novel therapeutic interventions and vaccines to combat this globally significant infectious disease. Continued research in this field is imperative to reduce the burden of amebiasis and improve public health outcomes worldwide.

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