LITERATURE REVIEW OPEN ACCESS

Molecular and Mucosal Immune Mechanisms in Infectious Gastritis and Gastroenteritis

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Abstract

Gastritis and gastroenteritis arise from pathogens whose virulence programmes interact with epithelial barriers and mucosal immunity through distinct mechanisms. This narrative review synthesizes selected evidence on Helicobacter pylori gastritis and gastroenteritis associated with norovirus and enterotoxigenic Escherichia coli (ETEC), while separately examining intestinal Candida albicans colonization and invasive disease in susceptible hosts. H. pylori uses urease, motility, adhesins, and a type IV secretion system to persist in the stomach; the effector CagA and the toxin VacA perturb epithelial integrity and inflammatory signalling. Norovirus attachment is influenced by histo-blood-group antigens and infection disrupts small-intestinal absorptive function, whereas viral variation limits durable protection. ETEC colonization factors enable mucosal adherence, and heat-labile and heat-stable enterotoxins raise cyclic AMP and cyclic GMP, respectively, producing CFTR-mediated secretory diarrhoea without substantial invasion. C. albicans is normally a gastrointestinal commensal; intestinal overgrowth or stool detection must not be equated with gastroenteritis. In profoundly susceptible hosts, yeast-to-hypha transition, candidalysin, adhesins, secreted aspartyl proteases, and biofilm formation may facilitate mucosal invasion, countered principally by epithelial sensing, Dectin-1/CARD9 signalling, neutrophils, and Th17 immunity. Because this is a narrative synthesis, conclusions depend on the scope and quality of the selected literature. Precise distinction among colonization, dysbiosis, mucosal invasion, and disseminated infection is essential for clinically meaningful interpretation.

Keywords: Helicobacter pylori; norovirus; enterotoxigenic Escherichia coli; Candida albicans; secretory diarrhoea; mucosal immunity

1. Introduction:

1.1. Background

Gastritis and gastroenteritis are inflammatory conditions of the gastrointestinal tract caused by various infectious agents, including bacteria, viruses, and fungi (1). One of the most common causes of gastritis is infection with *Helicobacter pylori* (*H. pylori*), whereas gastroenteritis can be triggered by infections with Norovirus, Enterotoxigenic *Escherichia coli* (ETEC), and *Candida albicans* (2). Each of these pathogens exhibits unique pathogenic mechanisms involving complex molecular and immune responses in the host. A thorough understanding of the molecular interactions between these pathogens and the host immune system could facilitate the development of more effective strategies for prevention, diagnosis, and treatment.

Infection with *Helicobacter pylori* is a leading cause of chronic gastritis and plays a crucial role in the development of peptic ulcers and gastric cancer (3). At the molecular level, *H. pylori* produces various virulence factors, such as CagA (cytotoxin-associated gene A) and VacA (vacuolating cytotoxin A), which induce epithelial cell damage and activate inflammatory responses. This infection triggers the production of pro-inflammatory cytokines, including IL-8, IL-1β, and TNF-α, through the activation of NF-κB signaling pathways and the NLRP3 inflammasome. The host immune system, involving neutrophils, macrophages, and T cells, attempts to eliminate the infection; however, in many cases, this immune response results in tissue damage and chronic inflammation (4).

Meanwhile, Norovirus-induced gastroenteritis is a leading cause of acute, self-limiting diarrhea. Norovirus has a single-stranded positive-sense RNA genome and binds to histo-blood group antigens (HBGA) on the surface of intestinal epithelial cells. The infection causes damage to intestinal microvilli, reduced fluid absorption, and excessive ion secretion (5). The immune response to Norovirus involves activation of cytotoxic T cells, type I interferon (IFN-α/β) production, and specific antibody responses to viral capsid proteins. However, the virus's ability to evade immune responses through antigenic mutations poses significant challenges for vaccine development (6,7).

Gastroenteritis caused by Enterotoxigenic Escherichia coli (ETEC) is a major cause of acute watery diarrhoea, particularly in settings with inadequate water and sanitation (8). ETEC heat-labile toxin activates adenylate cyclase and raises cyclic AMP, whereas heat-stable toxin activates guanylyl cyclase C and raises cyclic GMP. Both pathways enhance CFTR-mediated chloride secretion and reduce sodium absorption, producing secretory—not osmotic—diarrhoea (9). Mucosal secretory IgA can limit bacterial adherence and neutralize toxins, while innate receptors recognize bacterial surface structures (10).

Candida albicans commonly colonizes the gastrointestinal tract without causing disease (11). In hosts with severe immune impairment, epithelial injury, dysbiosis, prolonged broad-spectrum antimicrobial exposure, or intensive-care risk factors, fungal overgrowth may precede mucosal invasion or dissemination. Diagnostic terminology should therefore distinguish commensal carriage and dysbiosis from tissue-invasive intestinal candidiasis. Dectin-1/CARD9 pathways, neutrophils, and Th17-associated responses are central to mucosal antifungal defence (11,12).

Understanding the molecular and immunological aspects of various pathogens responsible for gastritis and gastroenteritis highlights the interplay between pathogen virulence factors and the host immune system in disease pathogenesis. The complexity of molecular pathways, as well as the roles of cytokines, inflammasomes, and immune cells in regulating inflammatory responses, presents opportunities for the development of more targeted therapies. Further research is needed to explore immunological and molecular interventions to reduce morbidity associated with these infections, particularly in vulnerable populations

1.2. Objectives

1. To compare the principal virulence and epithelial-injury mechanisms of H. pylori, norovirus, and ETEC.
2. To synthesize how innate and adaptive mucosal immunity shapes persistence, clearance, and tissue injury across these infections.
3. To distinguish gastrointestinal C. albicans colonization and dysbiosis from invasive intestinal candidiasis and identify the host conditions that modify risk.

1.3 Narrative Review Approach

This article uses a focused narrative-review framework to connect microbial virulence factors with epithelial and immune responses. Evidence was prioritized when it directly addressed human pathogenesis, clinically relevant experimental models, or established microbiological mechanisms. As no protocol was prospectively registered and no quantitative evidence synthesis was undertaken, the review should not be interpreted as a systematic estimate of prevalence or treatment effect.

[AUTHOR VERIFICATION REQUIRED: specify the databases searched, final search date, complete search terms, language limits, inclusion and exclusion criteria, screening process, and how disagreements were resolved. Remove any element that was not actually performed.]

2. Main Body:

2.1. Gastritis Caused by *Helicobacter pylori* Infection

Helicobacter pylori is a Gram-negative, microaerophilic bacterium that colonizes the gastric mucosa, often resulting in chronic gastritis. The pathophysiology of *H. pylori*-induced gastritis begins with its ability to survive and proliferate in the acidic gastric environment (3,4). Upon colonization, *H. pylori* releases virulence factors that disrupt the mucosal barrier and trigger inflammation (3).

The bacterium's motility, enabled by its polar flagella, facilitates penetration of the gastric mucus layer and adhesion to epithelial cells. Adhesins such as BabA and SabA mediate this attachment, enhancing bacterial persistence (13). Additionally, *H. pylori* secretes urease, which catalyzes the hydrolysis of urea into ammonia and carbon dioxide, neutralizing gastric acid in its microenvironment and creating a niche for its survival (3,13).

Chronic inflammation underpins the progression of gastritis. Bacterial toxins, such as cytotoxin-associated gene A (CagA) and vacuolating cytotoxin A (VacA), damage gastric epithelial cells and recruit inflammatory cells to the infection site (13). This activity induces the production of pro-inflammatory cytokines, including IL-1β, IL-6, and TNF-α, exacerbating tissue damage (4). Over time, sustained inflammation disrupts the gastric mucosal barrier, leading to epithelial apoptosis, mucosal atrophy, and, in some cases, intestinal metaplasia—a precursor to gastric cancer (14).

The molecular pathogenesis of *H. pylori*-induced gastritis relies on its ability to adapt to harsh gastric conditions, colonize the mucosa, and manipulate host cellular pathways through

diverse virulence factors and genetic mechanisms (15). Survival in the acidic gastric environment is achieved via urease activity, which neutralizes local gastric acid and contributes to tissue damage due to ammonia toxicity (16). The bacterial proton-gated urea channel (UreI) ensures urease activation only under acidic conditions, optimizing energy usage and survival (4,13).

H. pylori's helical shape and polar flagella enable navigation through viscous gastric mucus to reach the epithelial surface, where it adheres (17). Chemotactic sensors, such as TlpB, guide the bacterium toward less acidic, nutrient-rich niches (4).

Adhesion is critical for colonization. H. pylori expresses adhesins including BabA, SabA, HopQ, and AlpA/B. BabA binds Lewis-b antigens, whereas SabA recognizes sialylated glycans that increase during inflammation. CagA is not an adhesin; it is an effector protein encoded within the cag pathogenicity island and translocated into epithelial cells through a type IV secretion system. Once delivered, CagA alters SHP-2, junctional, polarity, and inflammatory signalling, including IL-8 production (18).

CagA also disrupts cell junctions and polarity through E-cadherin degradation and β-catenin pathway activation, fostering a pro-inflammatory, pro-carcinogenic environment. Additionally, CagA induces IL-8 secretion, recruiting neutrophils and amplifying local inflammation (4,18). VacA, a multifunctional toxin, disrupts epithelial integrity, induces apoptosis via mitochondrial cytochrome c release, and modulates immune responses, promoting bacterial survival (18).

H. pylori lipopolysaccharide (LPS) exhibits molecular mimicry, resembling host antigens to evade immune detection. Modified LPS dampens Toll-like receptor 4 (TLR4) activation, weakening innate immune responses (4). High genetic diversity in *H. pylori* supports its adaptability and pathogenicity. Horizontal gene transfer enables acquisition of virulence traits and antibiotic resistance, while phase variation allows evasion of host immune responses (4,16).

The host immune response to *H. pylori* infection involves both innate and adaptive immunity but often fails to clear the bacterium, resulting in chronic infection. Innate immunity recognizes *H. pylori* components through pattern recognition receptors (PRRs) such as TLRs, triggering downstream signaling pathways like NF-κB and inducing pro-inflammatory cytokine production (4).

Adaptive immunity plays a pivotal role in chronic infection. CD4+ T cells differentiate into Th1, Th17, and Treg subsets. Th1 cells produce IFN-γ, enhancing macrophage bactericidal activity, while Th17 cells release IL-17 to recruit neutrophils. Treg cells secrete IL-10 and TGF-β, suppressing immune responses and facilitating bacterial persistence. B cells produce antibodies against *H. pylori* antigens, but these antibodies are often ineffective in bacterial clearance and may exacerbate inflammation via immune complex formation (4).

Persistent *H. pylori* infection drives chronic inflammation, characterized by elevated IL-1β, TNF-α, and IL-6 levels. This inflammation damages gastric epithelium, contributing to gastritis, peptic ulcer disease, and gastric cancer. Host genetic polymorphisms, particularly in cytokine genes like IL-1β and TNF-α, influence disease susceptibility and severity (4,18).

2.2. Gastroenteritis Caused by *Norovirus* Infection

Norovirus is a non-enveloped, single-stranded RNA virus belonging to the *Caliciviridae* family (17). Its primary target is the small intestine, where it infects and damages epithelial cells, leading to malabsorption and secretory dysfunction. After ingestion, norovirus binds histo-blood group antigens (HBGA) on epithelial surfaces, facilitating viral entry (1,17).

Norovirus infection compromises the epithelial barrier, inducing vacuolization and apoptosis that disrupt villus integrity. This results in villus shortening, crypt hyperplasia, maldigestion, malabsorption, and diarrhea. Intestinal inflammation further increases permeability and fluid loss (5,17).

Clinically, norovirus infection manifests acutely with non-bloody watery diarrhea, nausea, vomiting, abdominal cramps, and low-grade fever. Vomiting, particularly in children, may result from virus-induced gastroparesis and autonomic dysfunction (20). Symptoms typically resolve within 1–3 days in immunocompetent individuals. Dehydration is the most common complication, especially in vulnerable populations such as children, the elderly, and immunocompromised patients (21,22).

Norovirus demonstrates efficient replication, relying on its positive-sense RNA genome comprising three open reading frames (ORFs). ORF1 encodes non-structural proteins essential for replication, including RNA-dependent RNA polymerase (RdRp). ORF2 encodes the major capsid

protein (VP1), responsible for antigenicity and HBGA binding, while ORF3 encodes the minor capsid protein (VP2), which stabilizes the viral capsid structure (6).

Host genetic susceptibility to norovirus infection correlates with HBGA expression. Non-secretor individuals, due to mutations in the FUT2 gene, exhibit partial resistance to certain norovirus strains. However, the virus's high mutation rate, driven by RdRp's error-prone nature, facilitates immune evasion and recurrent infections (5).

The immune response to norovirus involves innate and adaptive mechanisms. Innate immunity activates upon viral RNA recognition by PRRs like TLRs, leading to interferon production and the induction of antiviral gene expression. Pro-inflammatory cytokines, including IL-1β, IL-6, and TNF-α, promote viral clearance and inflammation (5).

Both humoral and cellular adaptive immunity play critical roles in controlling norovirus infections. Secretory IgA antibodies (sIgA), produced in the intestinal mucosa, represent the primary defense mechanism against viral entry and replication. These antibodies block the binding of viral capsid proteins to HBGA, thereby preventing epithelial cell invasion (7). Systemic IgG antibodies also develop post-infection, although their role in protection remains less well-defined. Despite the formation of neutralizing antibodies, immunity to norovirus is strain-specific and short-lived, lasting only a few months to years, which accounts for frequent reinfections (5,7).

The cellular immune response involves CD4+ and CD8+ T cells that recognize viral peptides presented by antigen-presenting cells (APCs). CD8+ T cells mediate cytotoxic activity, eliminating infected cells, while CD4+ T cells support B-cell activation and antibody production. In immunocompromised individuals, impaired T-cell responses can lead to prolonged infection and chronic viral shedding. Notably, noroviruses have evolved immune evasion strategies, including antigenic variability, suppression of type I IFN responses, and inhibition of apoptosis, enabling persistent infections in susceptible hosts (1,10).

2.3 Gastroenteritis Caused by Enterotoxigenic *Escherichia coli* (ETEC)

Enterotoxigenic *Escherichia coli* (ETEC) is a leading cause of traveler's diarrhea and pediatric gastroenteritis, particularly in developing countries. ETEC is transmitted via the fecal-oral route, typically through contaminated food or water (17,24). Following ingestion, the bacteria colonize the small intestine, where they adhere to the intestinal epithelium through colonization factors (CFs), which are hair-like fimbriae or pili structures. This attachment facilitates the secretion of enterotoxins, which are central to disease pathogenesis: heat-labile toxin (LT) and heat-stable toxin (ST) (9). These toxins disrupt ion transport in enterocytes, leading to secretory diarrhea without significant mucosal inflammation (24).

Clinically, ETEC-induced gastroenteritis presents as acute, watery, non-bloody diarrhea, often accompanied by nausea, abdominal cramps, and mild fever. Vomiting may occur but is less prominent compared to norovirus infections. Symptoms develop 12–72 hours after exposure and typically persist for 3–5 days (25). Severe fluid and electrolyte loss, especially in young children and malnourished individuals, can lead to dehydration, metabolic acidosis, and electrolyte imbalances, the most serious complications (8). Unlike invasive *E. coli* pathotypes, ETEC does not cause significant tissue damage, and stool samples generally lack leukocytes or blood (17).

ETEC pathogenesis is primarily mediated by two enterotoxins: LT and ST. LT, structurally and functionally similar to cholera toxin, consists of one A subunit and five B subunits. The B subunit binds to GM1 ganglioside receptors on enterocytes, facilitating the entry of the enzymatically active A subunit (17). Inside the cell, the A subunit activates adenylate cyclase via ADP-ribosylation of Gsα protein, leading to increased intracellular cyclic AMP (cAMP). Elevated cAMP stimulates the cystic fibrosis transmembrane conductance regulator (CFTR) chloride channels, resulting in chloride and water secretion into the intestinal lumen and subsequent diarrhea (9).

In contrast, ST is a small, heat-resistant peptide classified into STa and STb. STa binds and activates guanylyl cyclase C on the apical membrane of enterocytes, increasing intracellular cyclic GMP (cGMP). Elevated cGMP inhibits sodium absorption and enhances chloride secretion via CFTR, mimicking LT's effects but through a distinct signaling pathway (1,17). STb, less well-studied, also promotes chloride secretion and has been linked to prostaglandin E2 (PGE2) release. Notably, ETEC strains may produce one or both toxins, and diarrhea severity correlates with the type and amount of enterotoxins produced (26).

Colonization factors (CFs) are essential for ETEC adherence to the intestinal mucosa, facilitating toxin delivery. Over 20 CFs have been identified, with the most common being CFA/I,

CS1–CS6, and CS21. These CFs, encoded by plasmids, are expressed as fimbrial or non-fimbrial structures (9,24). The diversity of CFs and enterotoxins enables ETEC to adapt to various hosts and environmental conditions, contributing to its widespread prevalence. Furthermore, ETEC strains often harbor plasmids encoding antimicrobial resistance genes, complicating treatment and control efforts in endemic regions (27).

The host immune response to ETEC infection involves innate and adaptive mechanisms. Following colonization and toxin production, the innate immune system is activated via pattern recognition receptors (PRRs), such as toll-like receptors (TLRs), that recognize bacterial components like lipopolysaccharides (LPS) and flagellin. This activation triggers downstream signaling pathways, including NF-κB, and the production of pro-inflammatory cytokines like IL-8, IL-6, and TNF-α (10). However, ETEC is generally non-invasive, resulting in a limited inflammatory response compared to invasive *E. coli* pathotypes (24).

Adaptive immunity to ETEC involves antibody production targeting CFs and enterotoxins. Secretory IgA (sIgA) is the primary defense at mucosal surfaces, preventing bacterial adherence and neutralizing enterotoxins (10,17). Systemic IgG responses to LT and ST may also develop post-infection, contributing to immunity. However, ETEC immunity is strain-specific and short-lived, largely due to antigenic diversity in CFs and enterotoxins. This limited immunity explains the frequent reinfections observed in endemic areas, particularly among children (8,28).

2.4 Gastrointestinal Candida albicans: Colonization, Dysbiosis, and Invasive Disease

C. albicans is a frequent component of the gastrointestinal mycobiota, and its recovery from stool or mucosal surfaces generally represents colonization rather than infection (11). Bacterial dysbiosis, epithelial disruption, immunosuppression, neutropenia, intensive-care exposure, and broad-spectrum antimicrobial use can reduce colonization resistance and permit fungal expansion. These conditions increase risk, but fungal abundance alone does not establish that C. albicans is the cause of diarrhoea or mucosal inflammation (11,33,34).

Virulence depends on context-sensitive morphological and transcriptional programmes. Adhesins support epithelial attachment; yeast-to-hypha transition promotes penetration; secreted aspartyl proteases and phospholipases modify host substrates; and hypha-associated candidalysin can injure epithelial membranes and trigger danger signalling. Biofilms protect fungal communities from environmental stress and antimicrobial exposure. Experimental immunodeficient models demonstrate gastrointestinal colonization and dissemination, but direct extrapolation to immunocompetent human gastroenteritis is inappropriate (12,30,31).

Host defence integrates epithelial sensing with Dectin-1, Toll-like receptors, CARD9-dependent signalling, complement, neutrophil recruitment, and Th17-associated cytokines. Neutrophils are particularly important for controlling invasive hyphae, while IL-17 and IL-22 help maintain mucosal barrier function. Defects in these pathways can shift a commensal relationship toward mucosal invasion or bloodstream dissemination (11,12).

A clinically defensible diagnosis of invasive intestinal candidiasis requires compatible host risk, symptoms, and objective evidence of tissue invasion or dissemination; superficial cultures alone are insufficient. The distinction matters because unnecessary antifungal treatment may add toxicity and selection pressure, whereas delayed recognition of true invasive disease can be life-threatening. Future studies should define reproducible diagnostic criteria and determine when mycobiome changes are causal rather than secondary to inflammation.

3. Conclusion

H. pylori, norovirus, and ETEC cause gastrointestinal disease through distinct combinations of colonization, epithelial perturbation, toxin or effector activity, and host response. H. pylori persistence can drive chronic gastritis, ulceration, atrophy, and gastric carcinogenesis; norovirus typically produces acute vomiting and watery diarrhoea through small-intestinal dysfunction; and ETEC causes non-invasive secretory diarrhoea through cyclic-nucleotide-dependent ion transport. C. albicans requires different interpretation because gastrointestinal carriage is common: dysbiosis or overgrowth should be separated from objectively supported mucosal invasion or dissemination.

Across these conditions, mucosal protection depends on barrier integrity, pattern-recognition signalling, phagocytes, lymphocytes, and pathogen-specific antibodies. The same responses can also sustain tissue injury when infection persists or regulation fails. The most useful translational approach is therefore pathogen- and host-specific: eradicate H. pylori when indicated, prevent dehydration and transmission in acute viral or enterotoxigenic disease, and reserve invasive-candidiasis terminology and treatment for patients with compatible risk and diagnostic evidence. The

conclusions remain limited by the narrative search design and should be updated through a transparent, reproducible evidence-selection process.

4. Declarations

Author contributions: Devi Purnamasari Sasongko conceptualized the review, defined its scope, performed the literature synthesis, and prepared the original draft. Nieke Andina Wijaya contributed to the clinical and microbiological interpretation and manuscript revision. Fiona Paramitha contributed to the clinical interpretation and critical revision. All authors read and approved the final manuscript.

Funding: This work received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflicts of interest: The authors declare that they have no conflicts of interest relevant to the content of this article.

Acknowledgements: The authors thank the Faculty of Medicine, Universitas Negeri Surabaya, for academic and institutional support during the preparation of this manuscript.

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