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Article Review of Immunogenic Responses to Escherichia coli Infections

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I. Introduction

Escherichia coli (E. coli) is a well-known Gram-negative bacteria composed of single-celled organisms that can survive in a variety of conditions, both aerobic and anaerobic(Rahman, Rafid, and Chaity 2025). It belongs to the family Enterobacteriaceae and is a natural component of the gastrointestinal flora in humans and animals(Janda and Lopez 2021). It plays a crucial role in digesting, vitaminsynthesis, and maintaining microbial equilibrium in the gut. Although the majority of E. coli strains are innocuous, some pathogenic strains, commonly known as pathogenic E. coli, can cause a variety of disorders(Foster-Nyarko and Pallen 2022). The severity of these infections varies according on the strain, the location of infection, and the host's immunological response(Mukhopadhyay, Arranz-Solís, and Saeij 2020). These diseases include:

• UTIs are among the most prevalent infections in adults, particularly in women.

• Some strains, including EHEC and EPEC, can cause acute and persistent diarrhea.

• Immunocompromised persons are more susceptible to systemic infections, such as sepsis(Zhou et al. 2023).

Numerous virulence factors found in pathogenic E. coli strains allow them to adhere to host cells, secrete toxins, and evade the immune system. Important virulence variables consist of(Pakbin, Brück, and Rossen 2021):

• Lipopolysaccharides (LPS): a significant outer membrane component that, via pattern recognition receptors such as TLR4, elicits potent immunological responses(Zamyatina and Heine 2020).

• Bacterial adherence to gut and urinary tract epithelial cells is facilitated by fimbriae and pili(Flores and Rohn 2025).

• Toxins: they include α-hemolysin and Shiga toxin, which cause severe inflammation and tissue destruction(Acharya 2020).

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The severity and clinical outcome of E. coli infections varies greatly between individuals due to genetic differences in the host immune system. The immune system serves as the first line of defense, with innate immune cells such as macrophages and neutrophils identifying and removing pathogens, while adaptive immunity, mediated by T and B lymphocytes, provides long-term protection via antibody formation and immunologicalmemory(Saez et al. 2023).

Genetic factors play an important role in determining the effectiveness of these immune responses. For example, variations in cytokine genes like IL-6, TNF-α, and IL-10 or immunological receptors like TLRs can affect the host's capacity to manage infection, which can impact the severity of the illness and its clinical results(Mukherjee, Huda, and Sinha Babu 2019).

Host Immune Response to E. coli

The immune response to E. coli involves two main components:

1- Innate Immunity

• PRRs, like TLR4, recognize bacterial components.

• Activation of macrophages and neutrophils to phagocytize microorganisms.

• Pro-inflammatory cytokines (TNF-α, IL-6, IL-1β) promote immune cell recruitment(Gulati et al. 2019).

2- Adaptive Immunity

T cells are activated to provide cell-mediated immunity.

• Activation of B cells produces IgA and IgG antibodies, giving long-term protection.

• Creates immunological memory to prevent reinfection(Lam, Lee, and Farber 2024)

Pattern Recognition Receptors (PRRs) and Genetic Variability

Pattern Recognition Receptors (PRRs) are receptors on the surface or inside immune cells that recognize pathogen-associated molecular patterns (PAMPs), which are distinct molecular structures found on microorganisms. Escherichia coli's PAMPs include(Li and Wu 2021):

• LPS is a component of Gram-negative bacteria's outer membrane.

• Flagellin: a protein found in bacterial flagella.

• Peptidoglycan fragments are part of the bacterial cell wall.

The most relevant PRRs for E. coli infections include:

1- Toll-like receptors (TLRs):

• TLR4 identifies LPS and activates the NF-κB pathway, producing pro-inflammatory cytokines such TNF-α and IL-6(Ciesielska, Matyjek, and Kwiatkowska 2021).

• TLR5 identifies flagellin and helps boost immune responses, especially during intestinal infections(Feng et al. 2023).

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2- Nod-Like Receptors (NLRs):

• Detect bacterial components like muramyl dipeptide and activate the inflammasome to produce IL-1β and IL-18.3. C-type Lectin Receptor (CLR)(Qiu et al. 2025):

• Recognize carbohydrate structures on bacterial surfaces and regulate innate immune cell development(Cummings 2019).

Genetic Variability in PRRs

Genetic differences in PRRs can dramatically alter the host's immunological response to E. coli(You et al. 2023).

• TLR4 SNPs can diminish recognition of LPS, resulting in reduced cytokine production and increased susceptibility to severe infections(Silva et al. 2022).

• TLR5 polymorphisms can reduce flagellin detection, leading to a poorer immune response and increased risk of recurring infections(Mukherjee et al. 2019).

• NLR and CLR variations can change inflammasome activation and immune cell signaling, impacting the balance of protective immunity and excessive inflammation(Koumangoye 2022).

Cytokine Gene Polymorphisms

Non-conservative mutations in the coding area of genes can alter the function of the produced protein due to structural changes(Letchumanan and Say 2025). Exon sequences of cytokine and receptor genes are typically highly conserved. However, amino acid sequence variation has been observed for the IL-4 receptor, LTα (TNFβ), TGFβ, and GM-CSF receptor b in healthy individuals, as well as the IL-2 receptor g gene in individuals with severe combined immunodeficiency(Kubick et al. 2021)**.**Conservative mutations, which do not modify amino acid sequence, can still impact protein expression via altering mRNA splicing.mRNA stability and levels of gene transcription(Walker et al. 2023). Polymorphisms in the 5 and 3 regulatory sequences or introns of genes can significantly impact transcription by altering the structure of transcription factor binding sites in promoters, enhancers, and silencers in introns or remote regulatory sites(Degtyareva, Antontseva, and Merkulova 2021). Finally, they may modify the nuclear matrix's binding sites for architectural transcription factors, affecting promoter shape(van Steensel and Furlong 2019).

Polymorphisms in cytokine genes frequently occur within regulatory areas.

The reason for investigating cytokine gene polymorphisms in human disease can be generally summarized(Kozak et al. 2020).

The steps are as follows:

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- To enhance the understanding of the etiology and

pathology of human disease

- To discover potential indicators of susceptibility and severity,

and clinical outcomes.

- Identify potential markers for responders and nonresponses in therapeutic studies.
- Identify targets for therapeutic intervention.
- Developing new disease preventive measures and improving existing ones, such as vaccinations(Wang, Wu, and Sun 2022).

The Human Leukocyte Antigen Presentation

HLA antigens are classified into two types based on their structure and function, known as HLA Classes(Mahdi 2019).

The MHC consists of roughly 3.5 million base pairs and includes both Class I and Class II. The HLA Class I and HLA Class II genes cover roughly one-third of this length(Nehlsen 2022). Class III includes loci that regulate complement, hormones, intracellular peptide processing, and other developmental traits.The Class III area is not part of the HLA complex, but rather exists inside it(Carey, Poulton, and Poles 2019).

The components are linked to the actions of HLA antigens or have comparable regulatory mechanisms as HLA genes(Busch, Kollnberger, and Mellins 2019).

HLA molecules are divided into two major groups:

• HLA class I (HLA-A, HLA-B, and HLA-C) presents endogenous antigens to $CD8^+$ cytotoxic T lymphocytes(Bodade and Bodade 2020).

• HLA class II (HLA-DR, HLA-DQ, and HLA-DP) deliver foreign antigens, such as bacterial peptides, to $CD4^+$ helper T cells(Liu, Shao, and Fu 2021).

During an E. coli infection, antigen-presenting cells such as macrophages and dendritic cells process bacterial antigens and then present them via HLA class II molecules(Santa 2023). This mechanism activates $CD4^+$ T cells, which coordinate immunological responses by activating macrophages, encouraging B-cell development, and increasing antibody production(Hua and Hou 2020).

The large degree of variation in HLA genes results in significant variety in antigen-binding specificity between people(Kelly and Trowsdale 2019). Certain HLA alleles are

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better in presenting E. coli-derived peptides, resulting in strong T-cell activation and successful bacterial clearance(Chatzileontiadou et al. 2020). Other alleles, on the other hand, may convey antigens less efficiently, resulting in inadequate immune responses, greater vulnerability to infection, and more severe disease symptoms(Rich and Chaplin 2019).

HLA polymorphisms have been linked to differences in susceptibility to recurrent urinary tract infections, gastrointestinal infections, and E. coli-induced systemic consequences(Amegbletor 2022). Furthermore, alterations in HLA-mediated antigen presentation might alter the balance between protective immunity and excessive inflammation, influencing disease outcomes(Claiborne et al. 2019).

Understanding the role of HLA genetic diversity in E. coli infection is crucial for future immunogenetic research. It sheds light on host-specific immune responses, facilitates the identification of at-risk populations, and aids in the development of personalized therapy and immunization regimens based on individuals' genetic profiles(IBRAHIM n.d.).

Genetic susceptibility

Escherichia coli infection severity and disease outcome is shaped by genetic susceptibility As previously mentioned, despite exposure to similar strains of bacteria, individuals can experience exceptionally different presentations of illness, leading to asymptomatic colonization to life-threatening systemic effects e.g., sepsis(Denamur et al. 2021). These differences are largely attributed to the polymorphisms of host immune related genes that regulate pathogen recognition, inflammatory responses and immune regulation(Mukherjee et al. 2019).

Polymorphisms in the genes encoding pattern recognition receptors (PRRs)(such as TLR4 and TLR5) affect the ability to detect bacteria and trigger downstream immunological signaling. In carriers of certain PRR mutations, components of E. coli may not be effectively detected, leading to the poor inflammatory response with reduced or delayed activated of both innate and adaptive immune responses that allow more survival of the bacteria. Whereas hyper-responsive vs very responsive variations can overly activate pro-inflammatory pathways resulting in tissue damage and contributing to disease severity(Lin 2021).

Gene polymorphisms are repeatedly found to correlate with disease phenotypes. However, genetic variants of pro-inflammatory cytokines such as TNF-α and IL-6 can

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lead to a condition referred to as hyper-inflammation, which may result in organ dysfunction and systemic inflammation(Kany, Vollrath, and Relja 2019). Polymorphisms in genes encoding anti-inflammatory cytokines, especially IL-10, may not regulate the inflammatory response adequately leading to either exacerbated tissue damage or prolonged infection(Steen et al. 2020).

In addition to that, the diversity of HLA genes determines antigen presentation and the activation of adaptive immunity. Some HLA alleles are associated with more effective antigen presentation and bacterial clearance, others with more efficient T-cell activation and prolonged infection. Even for well-recognized uropathogenic E. coli virulence factors such as fimbriae and toxins, determination of which strains are more virulent or more resilient to immune clearance due to host genetic background have valuable implications, especially in well-studied conditions, for example among individuals who develop recurrent urinary tract infections(Medhasi and Chantratita 2022).

These and many other gene-gene interactions ultimately govern the host response to E. coli challenge, and therefore collectively determine the degree of disease. Elucidating genetic risk enables accurate risk stratification, infers particular high-risk groups for subsequent epidemiological screening, and allows tailoring of specific treatment and prevention strategies for the management of human E. coli-associated disorders(Elbaba 2025).

Clinical and Research Implications

There is great clinical and fundamental interest in studying the mechanisms of infection for this type of Escherichia coli. With the discovery of genetic variants that correlate with increased predisposition or severe disease, doctors will be able to identify patients at risk of severe or recurrent infections. Amongst immunosuppressed patients, neonates, and other elderlies, this pathway is advantageous (Deinhardt-Emmer et al. 2025).

The fine genetic diversity of the host clinical recommendations tips for personalised medicine Therapeutic targeting of treatment should be tailored according to optimal treatment success and successful immunomodulators combined with traditional antiinfections according to individual's immunogenetic status. This suggests that similar genetic differences in cytokines and PRRs may be useful in forecasting response to drug treatment and/or overall disease outcome(Jain et al. 2024).

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Immunogenetic research directs towards personalized vaccines and new immunotherapeutic approaches. Identification of key pathways in the host that protect against E. coli may guide therapies to boost protective immunity, but limit harmful inflammatory responses. Additionally, the integration of immunogenetics into future epidemiological and clinical research will start to provide more complete depictions of host–pathogen interactions(Kwok, Mentzer, and Knight 2021).

Merging clinical features with immunogenetics is an important step towards better prevention, diagnosis, and treatment of E. coli-associated infections which will improve patient outcomes and academic health systems strategies to advance public health objectives (Dhaure 2024).

Conclusion

Escherichia coli (E. coli) infections are a sequence of events with contributing factors of bacterial virulence and host immune response. Mounting evidence suggests that disease susceptibility is in part immunogenetic, providing insights into disease severity and outcomes in individual patients. Importantly, these genetic differences lead to changes in immune recognition, inflammation modulation, and adaptive immune activation that affect the nature and outcome of infection.

This work adds to the raw data reflects host-pathogen interaction and probably aids in understanding the variability in clinical presentations among infected individuals. Together, and through integration of immunogenetic insights, clinical practice and research can better accommodate risk prediction, treatment tailoring and, ultimately allow development of targeted vaccination and immunomodulatory strategies. Recent developments in immunogenetics will hopefully lead the field to some new potential in bettering prevention and control methods against this pathogen and ultimately alleviating some of the global disease burden related to E. Carroll and Hohmann outline future directions for E. coli research.

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