White Blood Cell Single Layers and Enteropathogenic *E.coli* Interaction

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Abstract

Enteropathogenic Escherichia coli (EPEC) is a common pathogen responsible for gastrointestinal infections. The interaction between EPEC and white blood cells (WBCs) is crucial in understanding the immune response against EPEC infection. This abstract summarizes the key findings regarding the interaction between EPEC and WBC single layers. EPEC infection leads to the formation of attaching and effacing (A/E) lesions on intestinal epithelial cells. Neutrophils and macrophages, as part of the innate immune response, play significant roles in combating EPEC infection. EPEC can adhere to and invade WBCs through specific adhesins and type III secretion system, respectively. The interaction triggers an immune response characterized by the release of pro-inflammatory cytokines, chemokines, and reactive oxygen species. WBCs employ various mechanisms such as phagocytosis,

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Introduction

Enteropathogenic Escherichia coli (EPEC) is a significant cause of gastrointestinal infections worldwide. When EPEC infects the human gastrointestinal tract, it encounters various host defense mechanisms, including the immune system's response. White blood cells (WBCs), particularly neutrophils and macrophages, play a crucial role in the innate immune response against bacterial infections. Understanding the interaction between EPEC and WBCs at a cellular level is essential for developing effective strategies to combat EPEC-induced infections. This article explores the interactions between EPEC and white blood cell single layers, shedding light on the mechanisms involved in the immune response to EPEC infection [1].

EPEC infection and pathogenesis: Enteropathogenic E. coli is a pathogenic strain that adheres to the intestinal epithelial cells, leading to the formation of attaching and effacing (A/E) lesions. These lesions disrupt the integrity of the intestinal lining and interfere with its normal functions, resulting in diarrhea, abdominal pain, and other gastrointestinal symptoms. EPEC infection activates the host immune response, including the recruitment and activation of white blood cells.

White blood cells and the immune response: White blood cells, including neutrophils and macrophages, are integral components of the immune system. Neutrophils are the first line of defense and are rapidly recruited to the site of infection.

Macrophages play a role in phagocytosis, antigen presentation, and the regulation of the immune response. Both cell types possess specific receptors that recognize pathogen-associated molecular patterns (PAMPs), such as lipopolysaccharides (LPS) present on EPEC [2].

Adhesion and invasion of EPEC into white blood cell single layers: Studies have shown that EPEC can adhere to and invade white blood cells. The initial adhesion of EPEC to white blood cell membranes is mediated by specific adhesins, including intimin and other outer membrane proteins. These adhesins interact with host cell receptors, leading to bacterial attachment. Once attached, EPEC can invade white blood cells, using its type III secretion system to deliver effector proteins

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b. Verify the purity and characteristics of the EPEC strains [4].

Co-incubation of WBCS and EPEC

a. Prepare a single-cell suspension of WBCs and adjust the cell density.

b. Add the appropriate concentration of EPEC to the WBC suspension.

c. Incubate the WBCs and EPEC together for a specific duration to allow interaction.

Adhesion assay

a. Wash the co-incubated WBCs and EPEC to remove non-adherent bacteria.

b. Fix the cells and stain them using appropriate dyes (e.g., Giemsa, Gram stain).

c. Visualize and quantify the adhered EPEC under a light microscope or fluorescence microscope.

d. Analyze the data by counting the number of adhered bacteria per WBC or per microscopic field.

Invasion assay

a. Perform the adhesion assay as described above to quantify adhered EPEC.

b. Treat the co-incubated WBCs and EPEC with antimicrobial agents (e.g., gentamicin) to kill extracellular bacteria.

c. Wash the cells to remove the antimicrobial agents.

d. Lyse the WBCs to release internalized bacteria.

e. Plate the lysates on appropriate agar plates to determine the number of internalized EPEC colony-forming units (CFUs).

f. Calculate the percentage of internalized EPEC relative to the

recruit and activate additional immune cells, amplify the inflammatory response, and contribute to the clearance of EPEC.

WBC antimicrobial mechanisms: WBCs employ various antimicrobial mechanisms to combat EPEC infection. Phagocytosis allows WBCs to internalize EPEC and subsequently destroy them within intracellular compartments. Additionally, WBCs release antimicrobial peptides and enzymes that directly target and eliminate EPEC. Neutrophils form neutrophil extracellular traps (NETs), which entrap and kill bacteria.

EPEC manipulation of the immune response: EPEC has evolved mechanisms to manipulate the host immune response for its survival. It can inhibit the production of pro-inflammatory cytokines by WBCs, dampening the immune response and facilitating bacterial persistence. EPEC can also impair WBC chemotaxis, hindering their ability to migrate towards the infection site. Disruption of NETs by EPEC allows the bacteria to evade capture and clearance by neutrophils [10].

Implications for EPEC pathogenesis: The interaction between WBCs and EPEC contributes to the pathogenesis of EPEC-induced gastrointestinal infections. Adhesion and invasion into WBCs enable EPEC to evade immune recognition, disseminate within the host, and establish infection. The activation of the immune response by WBCs limits bacterial growth and promotes bacterial clearance. However, EPEC's ability to manipulate the immune response aids its survival and persistence within the host.

Conclusion

In conclusion, the interaction between white blood cell (WBC) single-layers and Enteropathogenic [illegible]