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EBOLA VIRUS: STRUCTURAL, BIOLOGICAL, AND PATHOGENIC CHARACTERISTICS

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ABSTRACT

Ebola virus disease (EVD) is a severe, often fatal hemorrhagic fever caused by viruses of the genus *Ebolavirus*. The pathogen is characterized by high virulence, rapid transmission through bodily fluids, and significant mortality rates. This article aims to describe the structural, molecular, and pathogenic characteristics of the Ebola virus, highlighting its replication mechanisms, host interactions, and clinical implications. Understanding these features is essential for improving diagnostics, treatment strategies, and preventive measures.

Keywords: Ebola virus, Filoviridae, hemorrhagic fever, viral structure, pathogenesis, transmission

INTRODUCTION

Ebola virus disease (EVD) is a life-threatening zoonotic infection first identified in 1976 during simultaneous outbreaks in Sudan and the Democratic Republic of Congo. The virus belongs to the family *Filoviridae* and is known for causing severe hemorrhagic fever with mortality rates ranging from 25% to 90%. EVD remains a major global health concern due to its epidemic potential and limited therapeutic options.

The causative agent, Ebola virus, is a filamentous RNA virus with unique structural and biological properties that contribute to its high pathogenicity. This study focuses on the characterization of the Ebola virus, including its structure, genome organization, replication strategy, and mechanisms of host damage.

MATERIALS AND METHODS

This article is based on a comprehensive review of peer-reviewed scientific literature, including publications from PubMed, WHO reports, and CDC guidelines. Relevant data on Ebola virus structure, replication, and pathogenesis were analyzed and synthesized. Emphasis was placed on molecular biology, virology, and clinical features.

RESULTS

1. Viral Structure and Genome

Ebola virus is an enveloped, filamentous virus measuring approximately 80 nm in diameter and up to 14,000 nm in length. It contains a negative-sense, single-stranded RNA genome approximately 19 kb long. The genome encodes seven structural proteins, including:

- Nucleoprotein (NP)
- Viral proteins (VP24, VP30, VP35, VP40)
- Glycoprotein (GP)
- RNA-dependent RNA polymerase (L protein)

The glycoprotein (GP) plays a critical role in viral entry by mediating attachment and fusion with host cell membranes.

2. Replication Mechanism

Ebola virus enters host cells via macropinocytosis. After entry, the viral RNA is released into the cytoplasm, where replication occurs. The RNA-dependent RNA polymerase transcribes viral mRNA and replicates the genome.

New viral particles are assembled in the cytoplasm and bud from the host cell membrane. This process disrupts normal cellular function and contributes to cell death.

3. Pathogenesis

Ebola virus primarily targets:

- Monocytes/macrophages
- Dendritic cells
- Endothelial cells

Infected immune cells release pro-inflammatory cytokines, leading to a "cytokine storm," which contributes to systemic inflammation and vascular damage. Endothelial dysfunction results in increased vascular permeability, causing hemorrhage and shock.

Additionally, the virus suppresses immune responses by inhibiting interferon signaling, allowing uncontrolled viral replication.

4. Transmission and Clinical Features

Transmission occurs through direct contact with infected bodily fluids such as blood, saliva, vomit, and semen. The incubation period ranges from 2 to 21 days.

Clinical symptoms include:

- Fever
- Weakness
- Vomiting and diarrhea
- Internal and external bleeding

Without treatment, EVD often leads to multi-organ failure and death.

DISCUSSION

The high virulence of Ebola virus is linked to its ability to evade the immune system and induce severe inflammatory responses. The structural proteins, particularly VP35 and VP24, interfere with host antiviral defenses, while GP contributes to endothelial damage.

Recent advances in antiviral therapies and vaccines, such as monoclonal antibodies and rVSV-based vaccines, have improved survival rates. However, challenges remain in early diagnosis, outbreak control, and accessibility of treatment in resource-limited settings.

Understanding the molecular mechanisms of Ebola virus infection is crucial for developing targeted therapies and improving public health responses.

CONCLUSION

Ebola virus is a highly pathogenic RNA virus with complex structural and biological characteristics that contribute to its severity. Its ability to disrupt immune function and cause widespread tissue damage makes it a significant global health threat. Continued research into viral mechanisms, early detection, and effective treatments is essential to reduce mortality and prevent future outbreaks.

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