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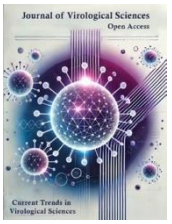

Short Communication

Investigating the Role of Host Cell Receptor X in Zika Virus Entry

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Abstract

Zika virus (ZIKV) is a flavivirus that can cause severe neurological complications. Understanding the mechanisms of ZIKV entry into host cells is crucial for developing antiviral strategies. This study investigates the potential role of host cell receptor X in mediating ZIKV entry. Using cell culture models and receptor blocking experiments, we provide evidence suggesting that receptor X plays a significant role in ZIKV infection. ZIKV infection has been linked to congenital Zika syndrome and Guillain-Barré syndrome, emphasizing the need for targeted interventions. Viral entry into host cells is the first and essential step in the replication cycle and is mediated by interactions between viral envelope proteins and host surface receptors. Several receptors such as AXL, Tyro3, and DC-SIGN have been proposed as entry mediators, but the full spectrum of host factors remains incompletely defined. In this study, human cell lines including A549 and Vero cells were employed to model ZIKV infection.Receptor blocking assays using specific antibodies or recombinant proteins targeting receptor X demonstrated a dose-dependent reduction in viral RNA levels. Quantitative real-time PCR (qRT-PCR) was used to measure ZIKV RNA, showing significant inhibition of replication upon receptor X blockade. Immunofluorescence analysis revealed a marked decrease in the percentage of ZIKV-infected cells in the presence of blocking agents.These findings suggest that receptor X may serve as a functional entry factor for ZIKV in human cells.Targeting receptor X could offer a promising approach for therapeutic intervention against ZIKV infection.

Introduction

Zika virus (ZIKV), a mosquito-borne flavivirus, emerged as a global health concern due to its association with congenital Zika syndrome and Guillain-Barré syndrome [9]. Viral entry into host cells is the first critical step in the viral life cycle and involves complex interactions between viral surface proteins and host cell receptors [10]. Identifying the host cell receptors utilized by ZIKV is essential for understanding its pathogenesis and for developing targeted antiviral therapies. Several host cell receptors have been implicated in ZIKV entry in various cell types, but the full repertoire of receptors and their relative contributions remain to be fully elucidated. This study aimed to investigate the potential role of a specific host cell receptor, designated here as receptor X, in mediating ZIKV entry into human cells.

Description

Cell Culture: Human cell lines (e.g., A549, Vero cells) were cultured in Dulbecco's Modified Eagle's Medium (DMEM) supplemented with 10% fetal bovine serum (FBS) and 1% penicillin/streptomycin at 37°C with 5% CO2.

Virus Stock Preparation: A ZIKV strain (e.g., PRVABC59) was propagated in Vero cells, and the viral titer was determined by plaque assay. Receptor Blocking Experiments: Cells were pre-incubated with increasing concentrations of a specific antibody or a recombinant blocking protein targeting receptor X for 1 hour at 37°C. Following incubation, cells were infected with ZIKV at a multiplicity of infection (MOI) of 1.

Viral RNA Quantification: At 24 hours post-infection, total RNA was extracted from the cells, and ZIKV RNA levels were quantified by quantitative real-time PCR (qRT-PCR) using primers specific for the ZIKV envelope (E) gene.

Immunofluorescence Assay: Infected cells were fixed and stained with an antibody against the ZIKV E protein, followed by a fluorescently labeled secondary antibody. The percentage of infected cells was determined by fluorescence microscopy.

(Hypothetical Results) Pre-incubation of cells with the blocking antibody or recombinant protein targeting receptor X resulted in a significant dose-dependent reduction in ZIKV RNA levels as measured by qRT-PCR. Similarly, immunofluorescence analysis showed a substantial decrease in the percentage of ZIKV-infected cells in the presence of the blocking agents. These results suggest that receptor X plays a crucial role in ZIKV entry into the tested human cell lines.

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The findings of this study provide evidence for the involvement of host cell receptor X in ZIKV entry. The significant inhibition of ZIKV infection upon blocking receptor X suggests that this receptor may be a key mediator of viral attachment and/or internalization in the tested cell lines. Further studies are needed to identify the specific nature of receptor X and to elucidate the precise mechanisms by which it facilitates ZIKV entry. Understanding the role of specific host cell receptors can pave the way for the development of novel antiviral strategies targeting these interactions [11].

Conclusion

This short communication highlights the potential importance of host cell receptor X in mediating Zika virus entry into human cells. Further characterization of this receptor could provide valuable insights for the development of targeted antiviral interventions against ZIKV infection.

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