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*Mini Review*

# The Determination of Pathogenesis Depends On the Recognition of Virus Receptors

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**Abstract**

A series of actions that result in the entry of the virus into the cell are triggered when the viral particle interacts with its cellular receptor on the surface of the host cell. The type of cellular receptor expressed on the cell surface plays a specific role in the contact and subsequent attachment of the virus with the host cell. In this approach, viruses selectively infect particular cell types, species, or groups of organisms. In other circumstances, various co-receptors or even chemicals created as a result of signal transduction are necessary, although a cellular receptor can alone mediate viral entry into the host cell. Here, we examine the cellular receptors of a few viruses with significant medical relevance and their roles in viral infection (Alkhatib 2009).

**Keywords:** Virus cellular receptors, Infection, Pathogenesis

## INTRODUCTION

A crucial first step in the infectious viral life cycle, recognition and interactions with cellular receptors serve a crucial regulatory function in host range, tissue tropism, and viral pathogenicity. In order to complete their infectious life cycle and eventually spread to other host cells, viruses are obligate intracellular pathogens that depend on the machinery of the host cell. Therefore, viruses carefully plan their attachment to one or more receptors in order to breach the plasma membrane and access the essential host cell machinery (Wilen et al., 2012). By engaging with the "lock"—the receptor—on the surface of the host cell, the viral attachment protein acts as the "key" to unlock the cells and triggers crucial later steps in the viral life cycle. In many situations, viruses use several receptors to carry out distinct roles throughout the virus life cycle in addition to serving as attachment moieties and entry factors, coordinators of viral trafficking, and activators of signalling processes. In order to open the cell, the virus must first locate the lock and then utilise a certain viral "key" or "keys." Sometimes, for a virus to enter a cell, multiple locks, such as a doorknob and a deadbolt lock, must be unlocked. Viral targeting to the appropriate tissues for infection and viral translocation through cellular barriers, both of which are necessary for the virus to deliver the genome into the host cell, are coordinated by the diverse actions of viral receptors (Mainou et al., 2015). An important field of study that has revealed the roles of virus-receptor interactions in tissue targeting, host cell invasion, and viral disease consequences is the identification of new viral receptors and identifying the mechanism of virus-receptor interactions. Furthermore, viruses are fantastic resources for research in cell biology to better comprehend cellular functions including endocytosis and interactions between ligands and receptors. Determining the precise molecular interactions between viruses and their receptors has also greatly aided the creation of novel vaccines and antiviral medicines (Xu et al., 2014).

Typical viral attachment proteins produced on the virion surface mediate interactions with viral receptors. The overall structure of viral attachment proteins is influenced by the intrinsic variations in the shape (icosahedral or helical) and makeup (enveloped or nonenveloped) of viral coats. In most enveloped viruses, the attachment protein is spike-like and extends from the virion's surface, acting as the first point of contact with the receptor on the plasma membrane. Nonenveloped viruses can either have a spherical shape without extensions, like polyomaviruses, or they can be embellished with viral proteins that protrude

from the surface of the virion, like reoviruses. Comparing the spike-like protein to a viral capsid protein, which is embedded on the surface of a spherical viral capsid, it appears quite obvious that the spike-like protein would be the initial point of interaction between the virus and host cell (Woollard et al., 2015). Reovirus does have a spike-like protein that interacts with cellular receptors, but there are also other receptor connections that are carried out by capsid elements. The architecture of attachment proteins and the ways in which virions interact with cellular receptors can both be influenced by the general shape of the particles, but virus-receptor interactions have also been successfully modelled by pseudo-coating viral particles with glycoproteins from unrelated viruses. For the functional investigation of virus-receptor interactions, tissue tropism, and immunology, pseudotyping viral particles, particularly for human immunodeficiency viruses (HIV) and highly deadly viruses like the Ebola virus, has shown to be a potent technique (EBOV). It is suggested that attachment protein architecture and stoichiometry of attachment protein-receptors are not necessarily necessary for viruses to find and activate the proper receptors for infection by effectively pseudotyping virions that replicate the patterns of infectious native virions (Kumar et al., 2017).

Before infection can take place, all pathogens must cling to host cells; otherwise, the host clearance mechanism would wash them away. While adhesion to the host cell is typically the first stage of bacterial pathogenesis, it is a requirement for invasion for viruses and other intracellular pathogens. Therefore, one of the key elements determining viral pathogenicity is viral attachment to the host cell. Viruses attach to their host cells in a variety of ways, depending on the virus's makeup and structure as well as the kind of cell they interact with (Hamel et al., 2015). Through the expression of molecules known as virus receptor proteins or glycoproteins on the surface of the outer membrane, the host cell chooses which virus it will interact with. For instance, the influenza a virus interacts with the mucin glycoprotein produced on the surface of respiratory epithelial cells via its terminal sialic acid on the extracellular domain (Li et al., 1998). Because they only bind selectively to complementary epitopes on the viral surface, virus receptors are selective in this sense. Even so, a lot of things happen after the attachment before the virus may enter the host cell. A virus may have several receptors, and several viruses may share a single receptor, such in the coxsackie virus-adenovirus receptor (CAR), which facilitates the attachment of both viruses (Danthi et al., 2010). Co-receptor interactions are necessary to mediate the viral entry into the host cell, even while virus-cell connection via the attachment receptor is essential for the viral invasion. Since it is impractical to discuss each virus' receptor in terms of how it contributes to pathogenesis, this review concentrates on just a few virus families of the utmost medical significance (Campbell et al., 2011).

# CONCLUSION

A virus may infect a cell in several ways. A receptor can facilitate entrance alone, but sometimes it involves working with co-receptors or even a chemical created as a result of signal transduction. Only when a virus recognises its receptor on the surface of a cell does infection occur; otherwise, there is no connection and no infection. Understanding how viruses enter host cells, defining how these interactions specify tissue tropism, and influencing the course of illness are all dependent on research into how viruses interact with host cell receptors. The information is complicated by a number of issues, such as the use of multiple receptors, cell-type dependent variations in receptor expression and utilisation, strain or serotype dependent usage of specific viral receptors, the use of laboratory-generated experimental virus models, and the absence of tractable model systems for some viruses, even though the available data offer significant insights into the mechanisms of viral attachment and entry. Furthermore, whereas interactions between viruses and attachment and entry receptors control a number of crucial processes in viral infection, they do not alone determine tissue tropism, viral dissemination, or pathogenesis. Numerous studies have demonstrated that viruses like the HIV virus and the influenza virus may transfer from cell to cell through receptor-free methods like tunnelling nanotubes. Continued investigation of virally produced tunnelling nanotubes will open up a new line of inquiry and advance our knowledge of host immune evasion, a side effect of antiviral therapy, and receptor-independent viral propagation. Although the research of virus-receptor interactions generally shows promise for the creation of novel antiviral medicines, laboratory-based findings frequently do not transfer well into the clinic. However, the development of antiviral treatments is not the only field in which translational medicine has difficulties in getting substances from the lab to the patient. As some novel therapeutics in development targeting virus-receptor interactions show great promise, it is imperative to overcome these obstacles in order to advance our current understanding of virus-receptor interactions and be able to apply this knowledge to the future development of antiviral therapies. The use of SA receptors, IgSF members, and PtdSer receptors, for instance, is highlighted throughout this review as an example of overlapping cell attachment and entry mechanisms used by many different viruses. These mechanisms could be a target for the development of antiviral therapies like inhibitors and mAbs that could be used to treat other viruses.

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