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Perspective

An Overview on the Structural Analysis of Filovirus

Mesele Admassie*

Department of Microbial, Cellular and Molecular Biology, Addis Ababa University, Addis Ababa, Ethiopia

ABOUT THE STUDY

The surface glycoprotein GP1,2 of filoviruses stimulates and promotes virus entrance into the host cell through a complex mechanism that is only partially understood. Numerous research groups have lately developed antiviral techniques aimed at inhibiting viral entrance based on current knowledge. This paper has outlined what is currently known about filovirus glycoproteins and compares their structures and functions. We explain the soluble GP derivatives sGP, ssGP, and peptide and discuss the structural aspects of the surface GP1,2 after briefly placing filoviruses in viral classification and nomenclature.

The purpose of this study is to emphasize the relationship between structural traits and their involvement in the various phases of the filovirus entry process, with a focus on Filoviridae peculiarities. Finally, we examine known and continuing antiviral techniques in order to link mechanisms of action to structure/function analyses and to develop effective anti-filovirus medicines. The Filoviridae family belongs to the Mononegavirales order of negative strand, non-segmented (NNS) RNA viruses. Highly deadly viruses found in these family groupings include those found in the Marburgvirus and Ebolavirus genera, which cause severe hemorrhagic fevers, as well as the genus Cuevavirus, which has only been discovered in the form of RNA sequenced from bats.

Viruses from a single species, Marburg marburgvirus, make up the Marburgvirus genus (Marburg virus-MARV). It was found in 1967 during linked epidemics in Frankfurt (Germany) and Belgrade (Yugoslavia) following the shipment of diseased monkeys from Uganda to Marburg (Germany). There are five virus species in the Ebolavirus genus. According to the new nomenclature, they are known as Zaire ebolavirus (Ebola virus-EBOV), which was the first ebolavirus species identified in 1976 in the Democratic Republic of the Congo (formerly northern Zaire) near the Ebola River, Sudan ebolavirus (Sudan virus-SUDV), Ta Forest ebolavirus (Ta Forest virus-TAFV), Bundibugyo ebolavirus (Bund ibugyo virus-BDBV) and Reston ebolavirus (Reston virus-RESTV) according to the new nomenclature.

While RESTV has yet to be identified as a human pathogen, the other species, such as MARV, are highly deadly, with death rates ranging from 25% to 90%. After the identification of sequences most likely belonging to a new filovirus, Lloviu cuevavirus (Lloviu virus-LLOV), possibly infecting bats in Asturias in 2002, the Cuevavirus genus was formed (Spain). Because it is a new member of the filovirus family, little is known about its biology and potential for human infection. Filoviruses cause an abrupt onset of symptoms such as fever, headache, myalgia, and gastrointestinal issues due to their high infectivity and capacity to weaken the immune system. Then, during the worst of the sickness, hemorrhagic signs can appear.

Shock, seizures, coagulopathy, and multi-organ failure develop later and, in many cases, are fatal. Unfortunately, there are no licenced antivirals or vaccinations available at this time, despite recent developments, although supportive treatments such as rehydration and temperature and pain control may aid patients in overcoming infection. Many efforts have recently been made to discover critical viral targets in order to disrupt the viral cycle and aid in the cure of illness. Filoviruses have a similar genetic structure. Their 19-kilobyte NNS RNA genome has seven primary genes that lead to the production of several viral proteins. All of these proteins are required for the establishment of an infection and the successful replication of the virus.

The single surface protein GP1,2, which requires attachment to components present on the surface of target Dendritic Cells (DCs), monocytes/macrophages, and endothelial cells of liver sinusoids and lymph node sinuses, initiates the earliest steps of cell infection. The virions are absorbed after attachment, and endosomal events cause fusion, allowing the viral particle content to be released into the cytoplasm. The nucleocapsid is made up of the genomic RNA in association with the nucleoprotein NP, two cofactors VP30 and VP35, and the big protein L, which together form a massive macromolecular complex that protects the RNA genome while also promoting genome replication and transcription. The RNA-dependent RNA polymerase (RdRp) activity of the L protein is required for both genome replication and transcription.

Furthermore, this protein has yet-to-be-identified enzymatic activity involved in RNA transcriptional modifications including

Correspondence to: Mesele Admassie, Department of Microbial, Cellular and Molecular Biology, Addis Ababa University, Addis Ababa, Ethiopia,, E-mail: mesele2006@gmail.com

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RNA capping and polyadenylation, which protect viral mRNA from destruction and detection by the host cell's innate immunity guardians. The nucleoprotein NP wraps around the NNS RNA and shields it from host nucleases. The transcription cofactor VP30 is involved, while the polymerase cofactor VP35 is involved. After viral genome replication and RNA transcription, nascent viral particles are formed at the cell surface membrane in a process mediated by the matrix protein VP40, and virus budding proceeds in a process that includes hijacking the host ESCRT machinery.