## Introduction:

Ebolaviruses are negative stranded RNA viruses that belong to the Filoviridae family and are endemic to regions of west and equatorial Africa. These public health pathogens are primarily transmitted by human-to-human contact with infected body fluids and corpses and causes severe and acute systemic disease with high mortality. Ebolaviruses have substantial epidemic potential, as shown by the 2013–16 west African outbreak.

This outbreak was unprecedented in scale, with more than 28 000 confirmed cases and 11 000 deaths.

Its economic impact on the west African region was crippling. This outbreak also showed that, in a context of resource-poor public infrastructure, a rapid transition from primarily affected rural villages to the urban areas of larger cities can occur. With considerable efforts from the affected countries and with international support, the outbreak was ultimately controlled. This outbreak was also unique in that it triggered the initiation and implementation of comprehensive research programmes into ebolavirus-related pathology, which has led to major scientific advances. This Seminar reviews available knowledge about the epidemiology, disease manifestation, pathophysiology, case management, and community control of these diseases.

**Virology**

Ebolaviruses belong to the genus *Ebolavirus* of the family Filoviridae in the order Mononegavirales, viruses whose genome consists of a single strand of RNA with negative polarity. The genus *Ebolavirus* contains five species with the taxonomic designations: *Bundibugyo ebolavirus* (Bundibugyo virus), *Reston ebolavirus* (Reston virus), *Sudan ebolavirus* (Sudan virus), *Taï Forest ebolavirus* (Taï Forest virus), and *Zaire ebolavirus* (Ebola virus). This taxonomy, revised in 2011, is emphasised because nearly identical terms have different meanings: *Ebolavirus* and *Zaire ebolavirus* refer to taxonomic classifications, whereas Ebola virus is a virus.

Only Bundibugyo, Sudan, and Ebola viruses have been associated with disease outbreaks in humans.

These outbreaks occurred mainly in South Sudan and Uganda for Bundibugyo and Sudan viruses, and in the Democratic Republic of Congo, Republic of Congo, and Gabon for Ebola virus. Henceforward, the disease caused by Bundibugyo virus is designated Bundibugyo virus disease, by Sudan virus as Sudan virus disease, and by Ebola virus as Ebola virus disease. The disease caused by any of these ebola viruses is called Ebola disease. Between 1976 and 2014, these three viruses had caused over 20 known outbreaks owing to human-to-human transmission, with a total of 2400 cases and 1600 deaths.

Overall case fatality has been 25% for Bundibugyo virus disease, 50% for Sudan virus disease, and 80% for Ebola virus disease.

The largest ever-recorded outbreak of Ebola virus disease occurred in west Africa from 2013–16 following a single Ebola virus introduction from the natural reservoir into the human population.

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Virus genomes from more than 5% of all recorded cases have been sequenced, which has allowed the spread of the disease to be reconstructed across country borders and the molecular clock of Ebola virus in the human host to be estimated at 1·2 × 10−3 substitutions per site per year.

This evolutionary rate overlaps with that of other RNA viruses. Few adaptive mutations, notably an alanine to valine exchange at GP position 82, have been selected in the outbreak strain because they enhance virus entry into human cells.

In addition, polymorphism in residue 544 has been identified as enhancing infection in other outbreaks.

However, no evidence has shown that the presence of these mutations or the accumulation of neutral substitutions measurably changed the clinical presentations, disease severity, or transmissibility of the virus