

FILOVIROSIS: CURRENT PERSPECTIVES ON THE DISEASE AND ONE HEALTH

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ABSTRACT

Filovirosis comprises severe zoonotic diseases caused by members of the family Filoviridae. The best-known human pathogens include viruses within the genera *Orthoebolavirus* and *Orthomareburgvirus*. These enveloped, negative-sense single-stranded RNA viruses can produce severe viral haemorrhagic fever, systemic inflammation, vascular dysfunction, coagulopathy and multiorgan injury. Fruit bats and other wildlife are central to the ecology of filoviruses, although reservoir relationships differ among virus species. Transmission to humans may occur through wildlife exposure, followed by person-to-person spread through contact with infectious body fluids. Diagnosis depends on appropriate clinical and epidemiological suspicion together with molecular and antigen-based testing performed under established biosafety and public-health systems. Management relies on intensive supportive care, infection prevention and control, and species-specific therapeutics or vaccines where evidence and regulatory authorization exist. The 2026 Bundibugyo virus disease outbreak in the Democratic Republic of the Congo, with international spread and a WHO-declared Public Health Emergency of International Concern, highlights the continuing global importance of filovirus preparedness. India has no documented indigenous Ebola outbreak in the current evidence base, but its extensive international connectivity makes importation preparedness, laboratory readiness, surveillance and One Health coordination essential. This review integrates virology, epidemiology, reservoir ecology, pathogenesis, diagnosis, treatment, vaccination, animal health and One Health considerations.

KEYWORDS: Bats; Bundibugyo virus; Ebola virus; Emerging infectious diseases; Filoviridae; filovirosis; Marburg virus; One Health; Veterinary public health; Viral haemorrhagic fever and zoonosis.

1. INTRODUCTION

Filoviruses are important emerging and re-emerging zoonotic pathogens capable of causing severe and sometimes fatal disease in humans and non-human primates. The family Filoviridae derives its name from the characteristic filamentous morphology of its virions. Filoviruses belong to the order *Mononegavirales* and possess linear, non-segmented, negative-sense RNA genomes. Filoviruses are enveloped, filamentous negative-sense single-stranded RNA viruses that include some of the most consequential zoonotic pathogens known to veterinary and human medicine. Human disease is principally associated with viruses in the genera *Orthoebolavirus* and *Orthomarburgvirus*. Clinical illness ranges from an acute febrile syndrome to severe systemic disease characterized by gastrointestinal involvement, vascular dysfunction, coagulopathy, shock and multiorgan failure. The combination of high pathogenic potential, zoonotic emergence and efficient transmission in healthcare and household settings makes filoviruses a major One Health concern.^[1,5,6]

Historically, filovirus outbreaks have been concentrated in sub-Saharan Africa, but globalization, population mobility, ecological disruption and expanding human–wildlife interfaces mean that preparedness is relevant well beyond endemic areas. The 2013–2016 West African Ebola epidemic demonstrated the capacity of an initially localized zoonotic event to produce sustained international public-health consequences. Imported infections have subsequently reinforced the need for points-of-entry surveillance and high-consequence infectious-disease preparedness.^[12]

The epidemiology is not static. In 2026, a large outbreak of Bundibugyo virus disease in the Democratic Republic of the Congo (DRC) became the most important ongoing filovirus event. WHO declared the event a Public Health Emergency of International Concern on 17 May 2026. By 20 September 2026, WHO reported 7,733 confirmed cases and 3,732 deaths in the DRC across 63 health zones, illustrating the continuing capacity of filoviruses to expand geographically and challenge health systems.^[31–33]

2. Taxonomy and Classification

Filoviridae belongs to the order Mononegavirales and comprises viruses with non-segmented, negative-sense RNA genomes. Modern taxonomy distinguishes several genera and species, including *Orthoebolavirus* and *Orthomarburgvirus* among the principal human-pathogenic groups. Taxonomic classification is important because biological properties, host range, geographic ecology and susceptibility to vaccines or therapeutics may differ between species.^[1,2]

Zaire ebolavirus is the species responsible for the largest number of recognized Ebola disease cases and has licensed vaccines and monoclonal antibody therapies. Sudan ebolavirus, Bundibugyo ebolavirus and other orthoebolaviruses have distinct epidemiological and medical-countermeasure evidence bases. Marburg marburgvirus causes Marburg virus disease and is biologically related to ebolaviruses but should not be treated as clinically or therapeutically interchangeable with them.^[1,5,13]

3. Morphology and Virion Structure

Filovirions are characteristically filamentous, although U-shaped, branched, circular and toroidal morphologies can occur. The virion consists of a host-derived lipid envelope containing viral glycoprotein (GP), a matrix layer dominated by VP40 and an internal nucleocapsid containing the viral genome and nucleoprotein-associated replication machinery.^[1,5]

The envelope glycoprotein mediates attachment and membrane fusion and is a major determinant of cell tropism and immune recognition (Figure 01). VP40 contributes strongly to virion assembly and budding. NP encapsidates genomic RNA, while VP35, VP30 and the L protein participate in transcription and replication. VP24 has important roles in virion biology and immune antagonism. These proteins collectively enable a compact genome to perform entry, replication, assembly and immune-evasion functions.^[5,6]

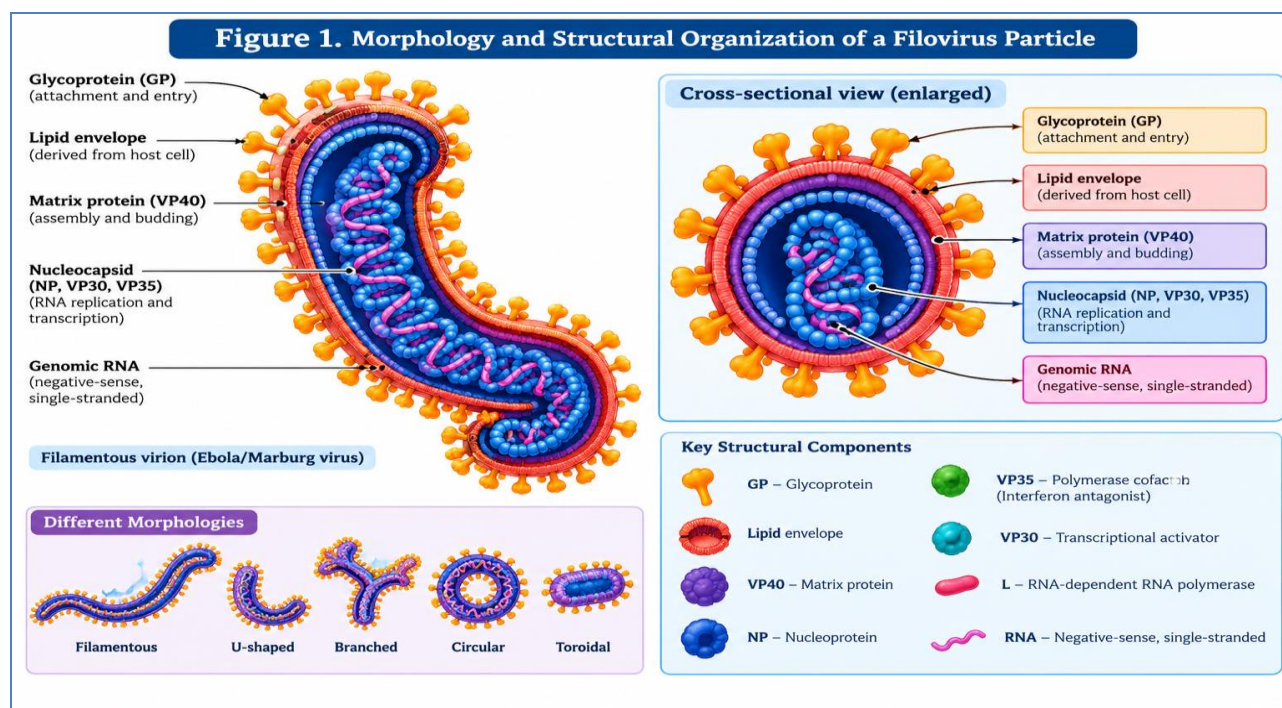


Figure 01: Generalized structure of a filovirus particle.

4. Genome Organization

Filoviruses possess a linear, non-segmented negative-sense RNA genome of approximately 19 kb, depending on species. In the conventional representation of the Ebola virus genome, the gene order is 3'–NP–VP35–VP40–GP–VP30–VP24–L–5' (Figure 02). The genome contains regulatory leader and trailer regions that participate in replication and transcription.^[1,5]

The negative-sense genome cannot directly function as messenger RNA and therefore must enter the host cell in association with the viral RNA-dependent RNA polymerase complex. Transcription produces viral mRNAs, while genome replication generates full-length antigenomic and genomic RNA templates. This organization permits coordinated production of structural, replication-associated and immune-modulatory proteins.

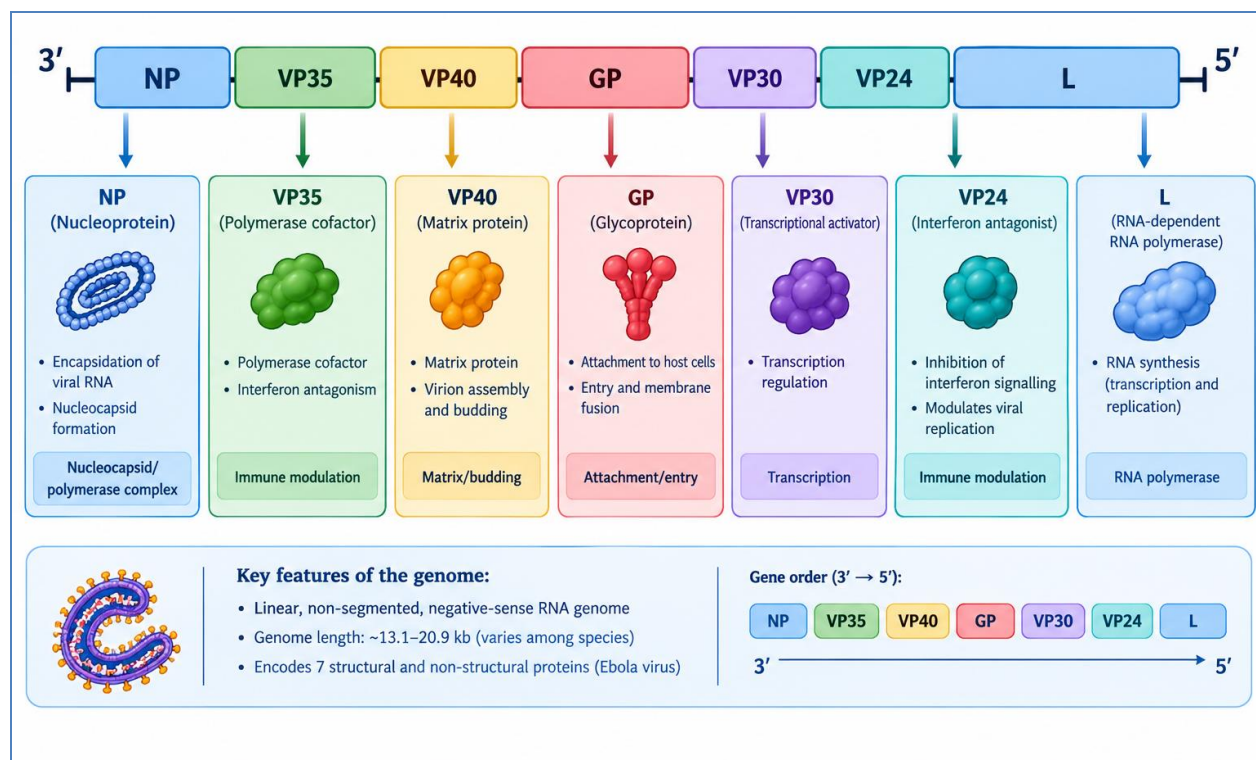


Figure 02: Simplified Filovirus (Ebola virus) genome.

5. Viral Proteins and Their Functions

NP is the principal nucleoprotein and encapsidates viral RNA. VP35 functions as a polymerase cofactor and interferon antagonist, while VP30 regulates transcription. VP40 is the major matrix protein and is central to assembly and budding. GP forms the surface spikes and mediates receptor engagement, endosomal processing and membrane fusion. VP24 contributes to assembly and interferon antagonism, whereas L is the RNA-dependent RNA polymerase (Table 01).^[5,6]

The multifunctional nature of these proteins explains why filoviruses can replicate efficiently while simultaneously interfering with innate antiviral responses. VP35 and VP24 are particularly important in suppression of interferon responses, allowing viral replication to proceed before an effective antiviral state is established.^[6,7]

Table 01: Major structural proteins of filoviruses.

Protein	Principal function	Relevance
NP	Encapsidates genomic RNA	Nucleocapsid formation and replication
VP35	Polymerase cofactor; interferon antagonist	RNA synthesis and immune evasion
VP40	Matrix protein	Assembly and budding
GP	Envelope glycoprotein	Attachment, entry and fusion
VP30	Transcriptional regulator	Viral transcription
VP24	Immune antagonist and structural protein	Assembly and interferon signalling interference
sGP	Soluble glycoprotein; immunological and pathogenic functions	
ssGP	Additional glycoprotein product generated through RNA editing	
L	RNA-dependent RNA polymerase	Transcription and genome replication

6. Viral Replication Cycle

Filovirus replication begins with attachment of GP to susceptible host-cell surfaces followed by uptake into endosomal compartments. Endosomal acidification and proteolytic processing of GP produce a form capable of interacting with intracellular receptors and promoting membrane fusion. The nucleocapsid is then released into the cytoplasm, where the viral polymerase complex initiates transcription.^[5,6]

Viral mRNAs are translated by host ribosomes, producing structural and non-structural proteins. When sufficient replication machinery is available, full-length antigenomic and genomic RNA are synthesized. Newly formed nucleocapsids associate with matrix and envelope components at the plasma membrane, where VP40-driven budding releases progeny virions. The cycle therefore links entry, cytoplasmic transcription, genome replication, assembly and budding.

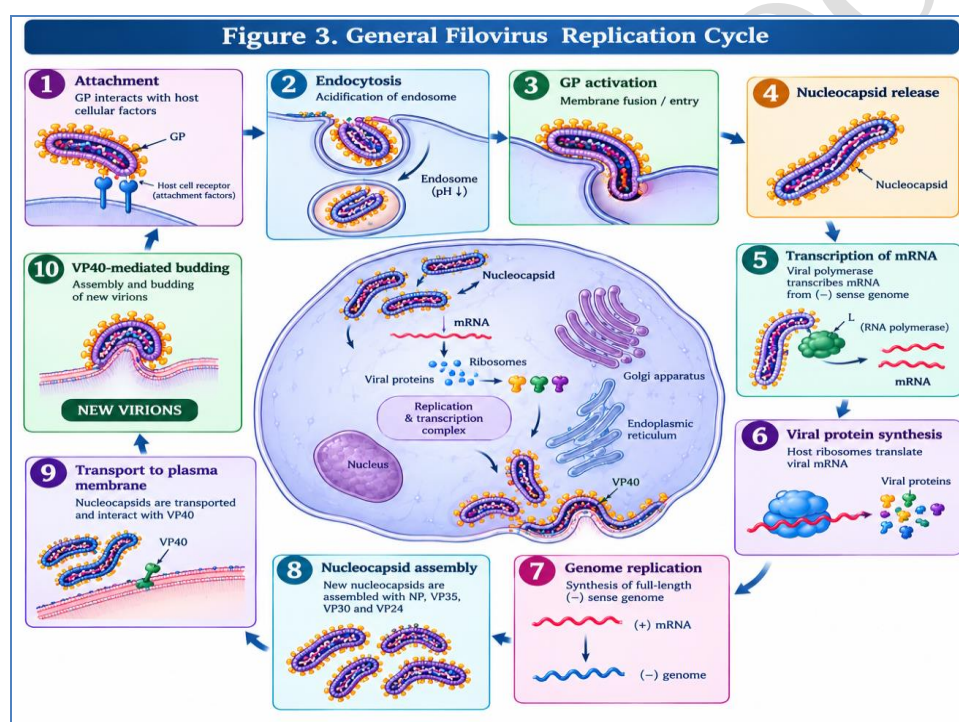


Figure 03: General replication cycle of filovirus.

7. Epidemiology and Geographical Distribution

Filovirus disease has historically been associated with tropical and subtropical regions of sub-Saharan Africa, where wildlife reservoirs, human–wildlife contact and healthcare vulnerabilities can intersect (Table 02 and Table 03). Outbreak frequency and geographic distribution vary among species. Ebola outbreaks have occurred in several Central and West African countries, whereas Marburg virus disease has been reported in countries including Uganda, Angola, Ghana, Equatorial Guinea, Tanzania and Rwanda in addition to earlier outbreaks.^[5,13]

Large outbreaks are often shaped by healthcare-associated transmission, funeral practices, mobility, delayed recognition and health-system constraints. Genomic surveillance has become increasingly useful for distinguishing independent spillover events from sustained transmission chains and for identifying geographic spread.^[12,15]

Table 02: Comparison of selected human-pathogenic filoviruses.

Virus/species group	Typical disease	Reservoir evidence	Medical countermeasures
Zaire ebolavirus	Ebola disease	Wildlife ecology; fruit bats strongly implicated	Licensed vaccine and monoclonal antibodies
Sudan ebolavirus	Ebola disease	Reservoir not definitively established	Candidate vaccines/therapeutics under development
Bundibugyo ebolavirus	Ebola disease	Reservoir not definitively established	No licensed species-specific vaccine or established specific treatment
Marburg marburgvirus	Marburg virus disease	Rousettus aegyptiacus strongly established	No broadly licensed specific countermeasure; candidates under development

Table 03: Comparative characteristics of Ebola and Marburg viruses.

Feature	Ebola viruses	Marburg virus
Family	Filoviridae	Filoviridae
Current principal genus	Orthoebolavirus	Orthomarburgvirus
Genome	Enveloped, non-segmented, negative-sense ssRNA	Enveloped, non-segmented, negative-sense ssRNA
Virion morphology	Pleomorphic, predominantly filamentous	Pleomorphic, predominantly filamentous
Important viruses	Ebola virus, Sudan virus, Bundibugyo virus, Taï Forest virus	Marburg virus
Reservoir evidence	Fruit bats suspected; definitive reservoir ecology unresolved	Strong evidence implicates Egyptian fruit bat (<i>Rousettus aegyptiacus</i>)
Zoonotic transmission	Contact with infected wildlife/body fluids	Contact with infected bats/animals/body fluids
Human-to-human transmission	Primarily direct contact with infectious body fluids	Primarily direct contact with infectious body fluids
Major clinical features	Fever, weakness, gastrointestinal disease, dehydration, vascular dysfunction, coagulopathy and potentially multiorgan failure	Similar acute febrile and gastrointestinal illness with severe systemic disease and possible haemorrhagic manifestations
Laboratory diagnosis	RT-PCR, antigen detection, serology and sequencing	RT-PCR, antigen detection, serology and sequencing
Specific licensed vaccine	Available for Ebola virus	No broadly licensed vaccine
Specific licensed therapeutics	Monoclonal antibody therapies available for Ebola virus disease	No broadly licensed specific therapy
Major prevention measures	Surveillance, isolation, IPC, vaccination where appropriate, contact tracing and safe burials	Surveillance, isolation, IPC, contact tracing and safe burials
Veterinary importance	Wildlife surveillance and investigation of zoonotic spillover	Particularly important because of the established bat reservoir

Source: ICTV [1]; WHO Ebola information [2]; WHO Marburg information [17]; WHO clinical-management guidance.^[10]

• Current Situation in India

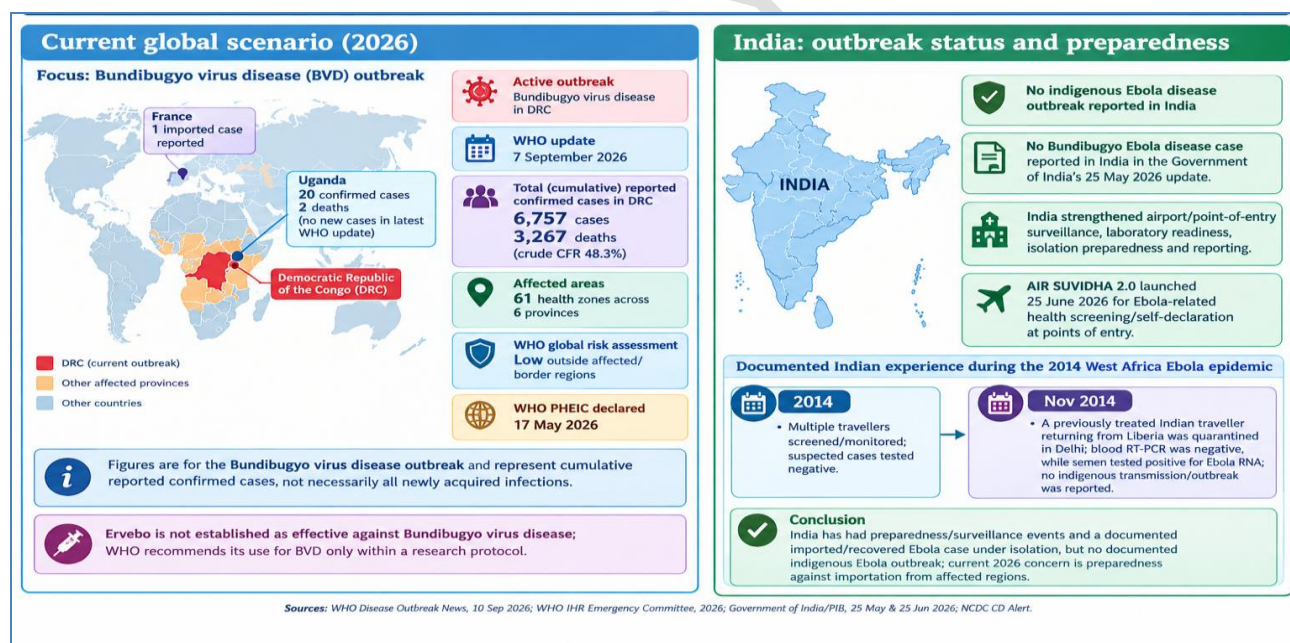
India is not an endemic country for Ebola or Marburg virus disease. NCDC's 2026 information states that no indigenous case of Ebola virus disease has been identified in India (Table 04) and describes the current risk as minimal while emphasizing institutionalized surveillance, screening and response preparedness.^[36]

Table 04: Current 2026 filovirus situation and India.

Parameter	Current evidence
Major ongoing outbreak	Bundibugyo virus disease, DRC
WHO emergency status	PHEIC declared 17 May 2026
DRC situation, 20 Sep 2026	7,733 confirmed cases; 3,732 deaths; 63 affected health zones
India	No documented indigenous Ebola outbreak in the cited NCDC evidence
Important Indian event	2014 recovered/imported Ebola case returning from Liberia
Primary preparedness priorities	Points of entry, surveillance, laboratory readiness, IPC, contact monitoring and One Health coordination

India has documented experience with imported/recovered Ebola infection (Figure 04). In November 2014, a 26-year-old Indian man returning from Liberia, who had previously been treated for Ebola, underwent screening at Delhi Airport. Blood, saliva and urine were negative, while Ebola viral RNA was detected in semen; he was isolated as a precautionary measure and subsequently managed through public-health procedures.^[36,38–40] No sustained indigenous transmission resulted from this event.

Preparedness in India should focus on points-of-entry surveillance, rapid clinical recognition, laboratory referral pathways, infection prevention and control, contact monitoring, risk communication and coordination through NCDC, IDSP, airport/port health authorities and designated healthcare facilities. The national surveillance architecture and Rapid Response Team mechanisms provide an important foundation for high-consequence infectious-disease preparedness.^[36,37]

**Figure 04: Current scenario of Filovirolosis in abroad and India.**

8. Reservoir Ecology and Zoonotic Spillover

Reservoir ecology is species-specific and remains incompletely resolved for some filoviruses. Egyptian fruit bats (*Rousettus aegyptiacus*) are strongly established in the ecology of Marburg virus, including virus isolation from naturally infected bats. Fruit bats have also been implicated in Ebola virus ecology, but the precise reservoir relationship for individual ebolavirus species remains less certain than the evidence for Marburg virus.^[3,4,14]

Spillover is best understood as a multistep process involving pathogen exposure, contact with a susceptible host, successful infection and onward transmission. Caves and mines, forest environments, hunting and handling of wildlife, and other high-contact interfaces can facilitate exposure. Land-use change, habitat disturbance and human mobility may alter the frequency and spatial distribution of such encounters.^[14,15]

9. Veterinary and One Health Significance

Although severe human disease dominates the public-health literature, filoviruses are fundamentally zoonotic infections and therefore require veterinary and wildlife-health participation. Non-human primates can develop severe disease and may become infected during natural or experimental exposure. Wildlife surveillance can contribute to understanding viral circulation, geographic risk and possible spillover conditions.^[16,26]

A One Health approach integrates human surveillance with veterinary investigation, wildlife ecology, environmental monitoring and laboratory science. Veterinary public-health institutions can contribute through wildlife health surveillance, biosafety expertise, sample referral systems, risk communication and outbreak preparedness. The 2026 Bundibugyo outbreak further demonstrates why cross-sector surveillance and international coordination are needed.^[31–35]

10. Modes of Transmission

Human-to-human transmission occurs primarily through direct contact with blood or other infectious body fluids of symptomatic or recently deceased patients (Figure 05). Exposure may occur through mucous membranes, non-intact skin or contaminated materials. Healthcare workers and family caregivers can be at increased risk when infection-prevention measures are inadequate.^[5,8,30]

Transmission associated with burial and funeral practices has been important in previous outbreaks. Filovirus RNA and infectious virus can persist in selected immune-privileged sites after recovery, and sexual transmission has been documented for Ebola virus. These findings justify survivor follow-up and risk-reduction counselling without implying that all recovered individuals remain infectious.^[18,25]

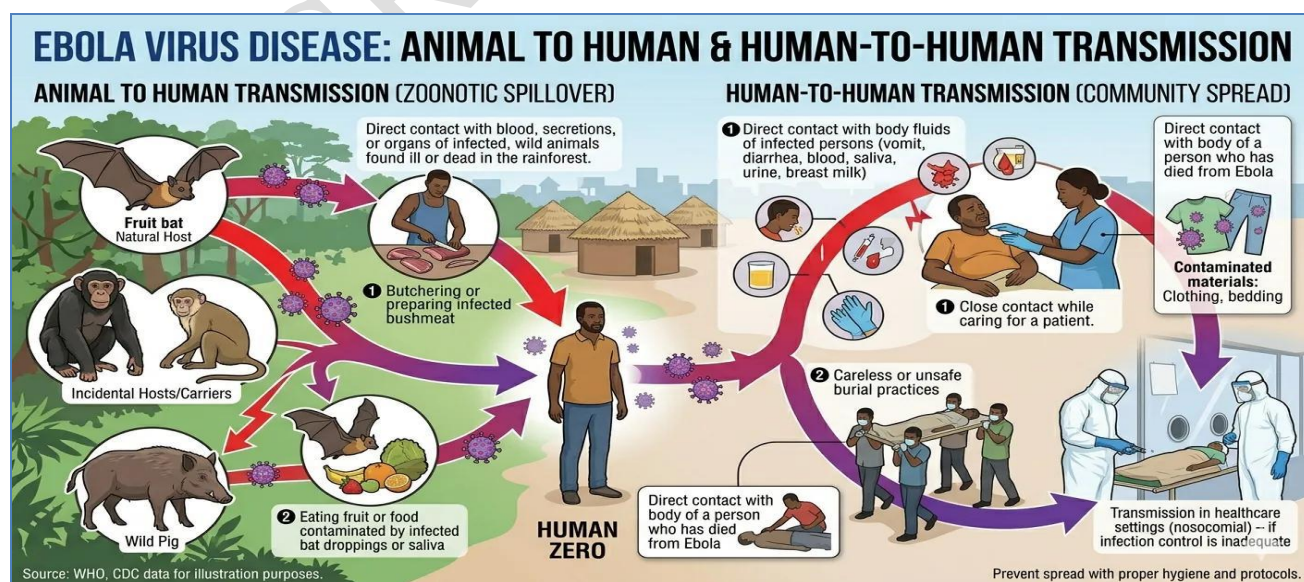


Figure 05: Transmission cycle of filoviruses.

11. Incubation Period and Clinical Course

The incubation period for Ebola disease and Marburg virus disease is generally several days to three weeks, depending on virus and exposure. The illness commonly begins with nonspecific fever, fatigue, headache and myalgia. Gastrointestinal symptoms such as vomiting and diarrhoea may become prominent as disease progresses, followed by dehydration, electrolyte disturbances and systemic inflammatory manifestations.^[5,8,13]

Severe disease may include bleeding manifestations, hypotension, shock, encephalopathy, hepatic and renal dysfunction, respiratory compromise and multiorgan failure. Hemorrhage is clinically important but is not present in every patient; severe disease can occur through profound systemic inflammation, vascular dysfunction and shock even without dramatic external bleeding.

12. Pathogenesis

Filovirus pathogenesis is multifactorial (Figure 06). After entry, viruses infect monocytes, macrophages and dendritic cells and disseminate to tissues including liver, spleen, lymphoid organs and other sites. Early infection of immune cells can alter cytokine signaling and antigen presentation, while viral replication in tissue compartments contributes to systemic disease.^[6,7]

A central feature is dysregulated host response. Excessive inflammatory mediator release, impaired adaptive immune responses, endothelial activation and vascular leakage can produce intravascular volume loss, coagulopathy and tissue hypoperfusion. The interaction between viral replication and host immunopathology therefore determines disease severity.^[7]

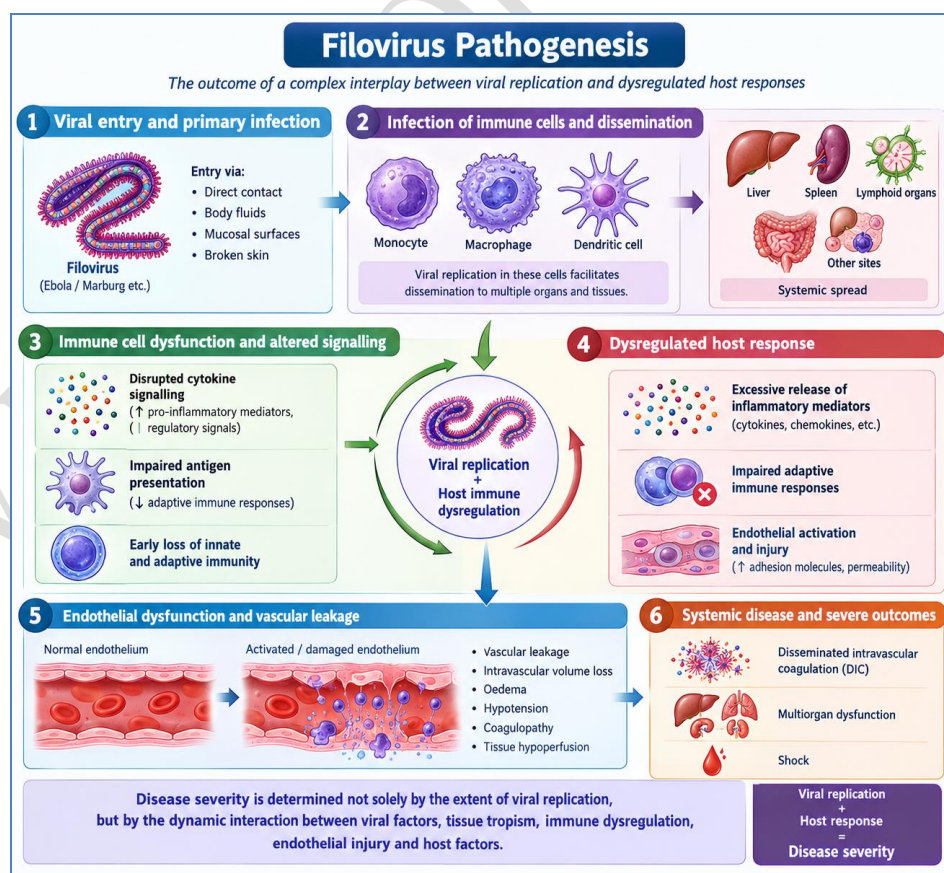


Figure 06: Pathogenesis of filovirus.

13. Immune Evasion

Filoviruses have evolved several mechanisms that interfere with innate antiviral immunity. VP35 can inhibit pathways involved in recognition of viral RNA and induction of type I interferon, while VP24 can interfere with downstream interferon signaling. These mechanisms reduce the ability of infected cells to establish an effective antiviral state.^[6]

Suppression of innate immunity is accompanied by dysregulated inflammatory responses. The resulting imbalance—insufficient early antiviral control together with excessive later inflammation—can contribute to widespread tissue injury. Understanding these mechanisms is important for development of antiviral agents, immunomodulators and next-generation vaccines.

14. Endothelial Dysfunction and Coagulopathy

Vascular endothelial injury and dysregulation contribute to increased vascular permeability and fluid loss. In severe disease, coagulation abnormalities may develop through interactions among endothelial activation, inflammatory mediators, platelet abnormalities, tissue injury and hepatic dysfunction.^[5,7]

Coagulopathy can progress to clinically significant bleeding and disseminated intravascular coagulation in severe cases. However, the extent and mechanism vary among patients and viruses. Supportive management therefore requires careful monitoring of hemodynamic status, electrolytes, renal function, coagulation and organ function.

15. Clinical Pathology

Laboratory abnormalities may include thrombocytopenia, leukocyte abnormalities, elevated transaminases, electrolyte disturbances, renal dysfunction and coagulation abnormalities. Findings evolve with disease stage and should be interpreted together with clinical and epidemiological information rather than used in isolation for diagnosis.^[5,19]

Because suspected filovirus disease is a high-consequence infectious-disease scenario, specimen collection, transport and testing must follow national and laboratory-specific biosafety and public-health procedures. Clinical laboratories should avoid ad hoc handling of potentially infectious specimens outside validated systems.^[20,30]

16. Diagnosis

Diagnosis requires integration of exposure history, compatible clinical illness and laboratory testing (Table 05). Molecular detection of viral RNA by reverse-transcription polymerase chain reaction (RT-PCR) is central to diagnosis during acute illness. Antigen detection can provide an additional diagnostic pathway, while serology is particularly useful for documenting immune responses in later disease or surveillance settings.^[19,20]

Laboratory confirmation should be performed through appropriately designated facilities and referral pathways. Virus isolation is a specialized research and public-health procedure rather than a routine diagnostic test. The diagnostic strategy must prioritize rapid confirmation while maintaining biosafety and minimizing exposure to laboratory personnel.

Table 05: Diagnostic approach to suspected filovirus disease.

Stage	Key action	Purpose
Clinical suspicion	Assess compatible illness plus exposure/travel history	Identify high-consequence infectious-disease risk
Immediate IPC	Isolate and apply appropriate precautions	Prevent healthcare-associated transmission
Laboratory confirmation	RT-PCR and/or antigen testing through designated pathways	Confirm or exclude infection
Serology	Use in appropriate later-stage or surveillance settings	Document immune response
Public-health response	Contact identification, monitoring and epidemiological investigation	Interrupt transmission

17. Differential Diagnosis

Early filovirus disease may resemble malaria, typhoid fever, severe bacterial sepsis, meningitis, dengue, yellow fever, Lassa fever, leptospirosis and other viral hemorrhagic fevers depending on geography and clinical presentation. Differential diagnosis should therefore be guided by exposure history, travel, epidemiological setting, local disease prevalence and clinical findings.

In endemic or outbreak settings, diagnostic algorithms should allow parallel assessment of more common treatable conditions while maintaining appropriate isolation and testing precautions for suspected filovirus disease. Rapid recognition is essential because delays can increase household and healthcare-associated transmission.

18. Treatment and Case Management

Supportive care remains fundamental to survival. Management may include careful fluid and electrolyte replacement, oxygen and hemodynamic support when indicated, nutritional support, treatment of secondary infections or complications, renal support where required, and correction of clinically significant coagulation abnormalities. Early supportive care can improve outcomes.^[8,9]

For Zaire ebolavirus disease, monoclonal antibody therapies such as Inmazeb (REGN-EB3) and Ebanga (ansuvimab/mAb114) have demonstrated benefit and are licensed in relevant jurisdictions.^[9,10] These treatments should not be generalized to all filoviruses. For Bundibugyo virus and Marburg virus disease, candidate vaccines and therapeutics remain species-specific and evidence-dependent.

19. Vaccination

The rVSV-ZEBOV vaccine (Ervebo) is licensed for prevention of Zaire ebolavirus disease and has demonstrated high effectiveness in ring-vaccination studies.^[11,21] Vaccination strategies are particularly important for outbreak response, where ring vaccination and targeted immunization can interrupt transmission chains.

Medical countermeasures should always be discussed by virus species. Evidence for Zaire ebolavirus cannot automatically be extrapolated to Bundibugyo virus, Sudan virus or Marburg virus. Candidate vaccines for other filoviruses require evaluation of safety, immunogenicity, efficacy and operational feasibility.^[22,23]

20. Current 2026 Evidence: Bundibugyo Virus Disease

The 2026 Bundibugyo virus disease outbreak is now a major documented filovirus event. WHO reported that the outbreak was detected in the Mongbwalu health zone in Ituri Province, DRC, and on 17 May 2026 the WHO Emergency Committee determined that the epidemic constituted a PHEIC.^[31]

WHO's 20 September 2026 situation report documented 7,733 confirmed cases and 3,732 deaths in the DRC, with transmission reported across 63 health zones. The affected provinces included Bas-Uélé, Haut-Uélé, Ituri, North Kivu, South Kivu and Tshopo. The geographic expansion toward the South Sudan border emphasized the importance of cross-border surveillance.^[32,33]

The current evidence also demonstrates the importance of species-specific language. Licensed Zaire ebolavirus vaccines and monoclonal antibodies should not be described as established treatments or preventive measures for Bundibugyo virus disease unless supported by species-specific evidence and current regulatory/public-health recommendations.^[31,34]

21. Infection Prevention and Control

Filovirus infection prevention requires early recognition, rapid isolation of suspected cases, appropriate personal protective equipment, safe injection and sharps practices, environmental cleaning, safe handling of linens and waste, and safe and dignified burial procedures. Healthcare workers require practical training, supervision and adequate supplies.^[8,30]

Community engagement is equally important. Misinformation, stigma and fear can delay presentation and undermine contact tracing. Risk communication should therefore be culturally appropriate, transparent and coordinated with trusted community leaders and local health authorities.

22. Persistence in Survivors

Ebola virus may persist in immune-privileged sites such as semen, ocular fluid and central nervous system compartments after clinical recovery. Persistence of viral RNA does not always mean that infectious virus is present, but it has important implications for survivor counselling and sexual transmission risk.^[18,25]

Survivor programmes should combine medical follow-up, psychosocial support, laboratory assessment were indicated and evidence-based risk-reduction advice. Such programmes also reduce stigma by emphasizing that survivors are not generally a continuing source of casual transmission.

23. Animal Models and Veterinary Research

Non-human primates, rodents and other experimental models have contributed substantially to understanding filovirus pathogenesis, vaccine efficacy and therapeutic candidates. Animal models can reproduce important features of human disease but differ in susceptibility, immune responses and clinical expression.^[26]

Veterinary research is also important for wildlife ecology, reservoir surveillance, biosafety and development of diagnostic platforms. Ethical use of animals, refinement of experimental protocols and careful translation of animal findings to human disease remain essential.

24. One Health Approach to Filovirus Prevention

A practical One Health strategy links human health, animal health and environmental health. Human components include surveillance, clinical recognition, laboratory diagnosis, treatment and vaccination where available. Animal-health components include wildlife surveillance, veterinary investigation and monitoring of susceptible species. Environmental components include habitat assessment, land-use monitoring and characterization of human–wildlife interfaces.^[15,16]

Integrated surveillance can identify risk before sustained human-to-human transmission becomes established. This is especially important in regions where wildlife exposure, healthcare access, insecurity and population mobility overlap.

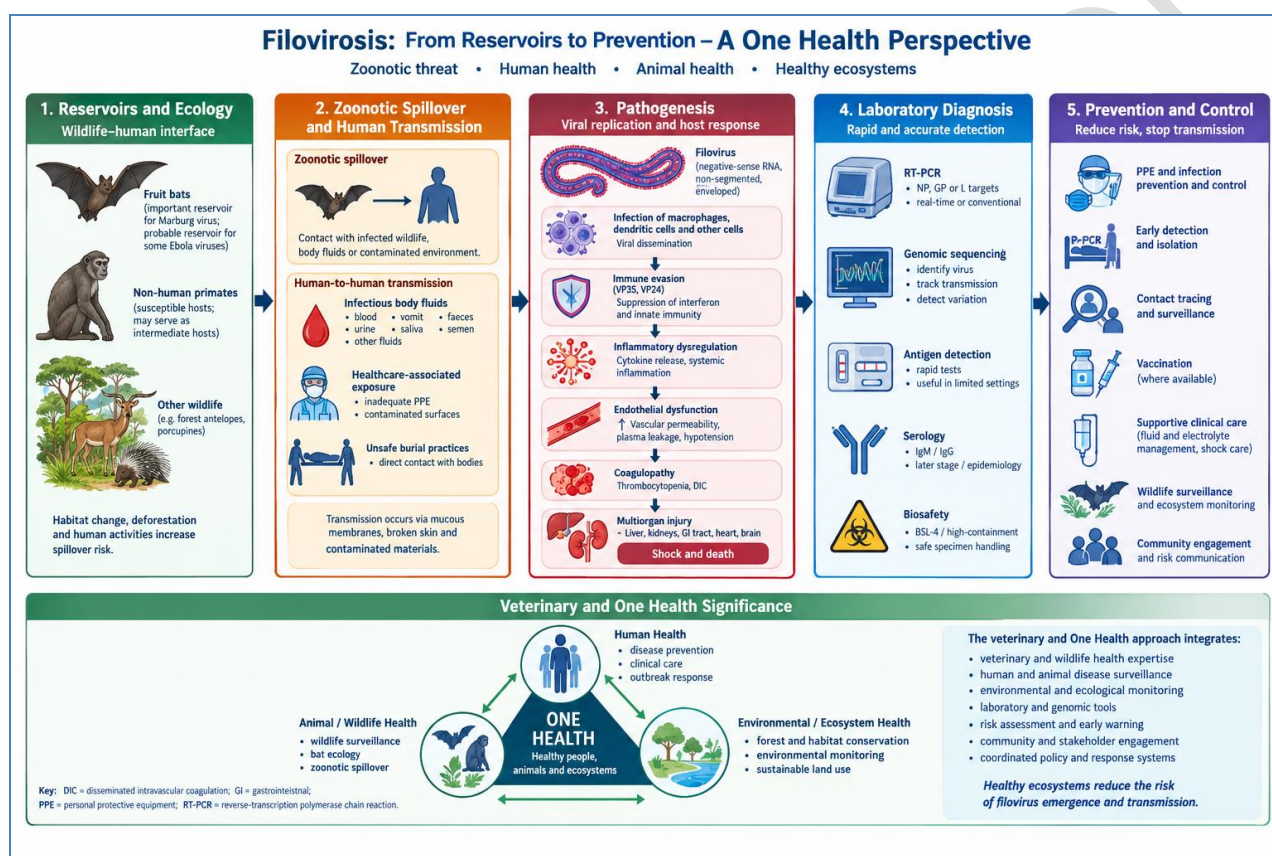


Figure 07: Schematic representation of the One Health pathway of filoviral disease.

25. Molecular Epidemiology and Genomic Surveillance

Genomic sequencing can identify transmission clusters, distinguish independent spillover events and monitor viral evolution. When combined with epidemiological data, genomic analysis can clarify transmission chains, identify geographic links and support outbreak-control decisions.

Portable and laboratory-based sequencing technologies are increasingly useful during outbreaks, but their value depends on sample quality, turnaround time, bioinformatics capacity and integration with field epidemiology. Genomic surveillance should therefore complement rather than replace conventional epidemiological investigation.

26. Challenges in Filovirus Control

Major challenges include incomplete understanding of reservoir ecology, delayed recognition, limited access to specialized diagnostics, healthcare infrastructure constraints, misinformation, community mistrust, insecurity, population displacement, ecological change and limited species-specific medical countermeasures.

The 2026 Bundibugyo outbreak illustrates how geographically dispersed transmission can complicate contact tracing and resource allocation. Sustained investment in laboratory capacity, infection prevention, field epidemiology, community engagement and cross-border coordination is therefore essential.^[31–35]

27. Future Research Priorities

Research priorities include development of broader-spectrum filovirus vaccines, antiviral therapeutics active against multiple species, rapid point-of-care diagnostics, improved animal and ecological models, wildlife genomic surveillance and integrated ecological forecasting. Long-term survivor studies are also needed to better characterize viral persistence and sequelae.

Research should increasingly adopt a One Health framework in which wildlife, livestock, human health and environmental data are analyzed together. Standardized data systems and rapid sharing of genomic and epidemiological information could improve outbreak detection and response.

28. CONCLUSION

Filovirosis remains a major One Health challenge because of the combination of zoonotic emergence, severe clinical disease, healthcare-associated transmission and complex ecological drivers. Advances in molecular diagnosis, genomics, vaccination and monoclonal antibody therapy have improved the response to selected filoviruses, particularly Zaire ebolavirus, but major gaps remain for other species.

The 2026 Bundibugyo virus disease outbreak demonstrates that filovirus threats remain dynamic and geographically consequential. For India, the evidence supports a preparedness-focused strategy based on surveillance, points-of-entry screening, laboratory readiness, infection prevention, trained healthcare workers, risk communication and coordinated One Health systems. Future progress will depend on species-specific medical countermeasures, improved understanding of wildlife ecology and integration of human, animal and environmental surveillance.

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