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Marburg Virus Disease: Virology, Pathogenesis, Diagnosis, Treatment, and Preventive Strategies

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Abstract

Marburg virus disease (MVD) is a severe and often fatal hemorrhagic fever caused by the Marburg virus, a member of the *Filoviridae* family. First identified in 1967 during simultaneous outbreaks in Germany and Serbia, the virus is a zoonotic pathogen whose natural reservoir is the African fruit bat, *Rousettus aegyptiacus*. Human infection typically occurs through contact with bat excreta in mines or caves, or via secondary transmission from infected individuals through direct contact with bodily fluids. The clinical course of MVD is characterized by an abrupt onset of non-specific symptoms including high fever, severe headache, and myalgia after a 5-10 day incubation period. This progresses to gastrointestinal symptoms and, in severe cases, hemorrhagic manifestations, jaundice, and multi-organ failure, with a high case fatality rate ranging from 23% to 90%. Diagnosis is challenging due to symptom overlap with other tropical diseases but is confirmed through RT-PCR, antigen-capture ELISA, or virus isolation in high-containment (BSL-4) laboratories. There are currently no approved vaccines or specific antiviral treatments for MVD. Management is primarily supportive, focusing on fluid balance, electrolyte correction, and treating complications. Prevention relies on public health measures, including avoiding contact with bats, implementing strict barrier nursing techniques in healthcare settings, and safe burial practices during outbreaks. Ongoing research into post-exposure prophylactics, such as experimental vaccines and antisense therapies, shows promise for future outbreak control.

Keywords: Marburg virus, hemorrhagic fever, Filoviridae, zoonotic infection, outbreak prevention

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1. Introduction

Marburg hemorrhagic fever (Marburg HF) is a rare but severe hemorrhagic fever which affects both humans and non-human primates. Marburg HF is caused by the Marburg virus, a genetically unique zoonotic (or, animal-borne) RNA virus of the filovirus family. The five species of Ebola virus are the only other known members of the filovirus family. (Smith et al., 2013)

Marburg virus was first recognized in 1967 when outbreaks of hemorrhagic fever occurred simultaneously in laboratories in Marburg and Frankfurt, Germany and in Belgrade, Yugoslavia (now Serbia). Thirty-one people became ill, initially, laboratory workers followed by several medical personnel and family members who had cared for them. Seven deaths were reported. The first people infected had been exposed to imported African green monkeys or their tissues while conducting research. One additional case was diagnosed retrospectively. (Brauburger et al., 2012).

The reservoir host of Marburg virus is the African fruit bat, *Rousettus aegyptiacus*. Fruit bats infected with Marburg virus do not to show obvious signs of illness. Primates (including humans) can become infected with Marburg virus, and may develop serious disease with high mortality. Further study is needed to determine if other species may also host the virus.

This *Rousettus* bat is a sighted, cave-dwelling bat widely distributed across Africa. Given the fruit bat's wide distribution, more areas are potentially at risk for outbreaks of Marburg HF than previously suspected. The virus is not known to be native to other continents, such as North America. (Towner et al., 2009).

Marburg HF typically appears in sporadic outbreaks throughout Africa; laboratory confirmed cases have been reported in Uganda, Zimbabwe, the Democratic Republic of the Congo, Kenya, Angola, and South Africa. Many of the outbreaks started with male mine workers working in bat-infested mines. The virus is then transmitted within their communities through cultural practices, under- protected family care settings, and under-protected health care staff. It is possible that sporadic, isolated cases occur as well, but go unrecognized. Cases of Marburg HF have occurred outside Africa, such as during the 1967 outbreak, but are infrequent. In 2008, a Dutch tourist developed Marburg HF after returning to the Netherlands from Uganda, and subsequently died. (Adjemian et al., 2007).

2. Literature Review

2.1 Classification of Marburg Virus Disease

Marburg virus disease (MVD) is the official name listed in the World Health Organization's International Statistical Classification of Diseases and Related Health Problems 10 (ICD-10) for the human disease caused by any of the two marburgviruses Marburg virus (MARV) and Ravn virus (RAVV). In the scientific literature, Marburg hemorrhagic fever (MHF) is often used as an unofficial alternative name for the same disease. Both disease names are derived from the German city of Marburg, where MARV was first discovered (Bogomolov, 2008).

2.2 Signs and Symptoms of Marburg Virus Disease

The most detailed study on the frequency, onset, and duration of MVD clinical signs and symptoms was performed during the 1998–2000 mixed MARV/RAVV disease outbreaks. A maculopapular rash, petechiae, purpura, ecchymoses, and hematomas (especially around needle injection sites) are typical hemorrhagic manifestations. However, contrary to popular belief, haemorrhage does not lead to hypovolemia and is not the cause of death (total blood loss is minimal except during labour). Instead, death occurs due to multiple organ dysfunction syndrome (MODS) due to fluid redistribution, hypotension, disseminated intravascular coagulation, and focal tissue necroses (Gear, 2009).

The clinical phases of Marburg Hemorrhagic Fever's presentation are described below. Note that phases overlap due to variability between cases.
Incubation: 2–21 days, averaging 5–9 days (Todorovitch et al., 2001).

Generalization Phase: Day 1 up to Day 5 from onset of clinical symptoms. MHF presents with a high fever (~40˚C) and a sudden, severe headache, with accompanying chills, fatigue, nausea, vomiting, diarrhoea, pharyngitis, maculopapular rash, abdominal pain, conjunctivitis, & malaise (Gear et al., 2005).

Early Organ Phase: Day 5 up to Day 13. Symptoms include prostration, dyspnea, edema, conjunctival injection, viral exanthema, and CNS symptoms, including encephalitis, confusion, delirium, apathy, and aggression. Hemorrhagic symptoms typically occur late and herald the end of the early organ phase, leading either to eventual recovery or worsening & death. Symptoms include bloody stools, ecchymoses, blood leakage from venipuncture sites, mucosal & visceral haemorrhaging, and possibly hematemesis (Gear *et al.*, 2008).

Late Organ Phase: Day 13 up to Day 21+. Symptoms bifurcate into two constellations for survivors & fatal cases. Survivors will enter a convalescence phase, experiencing myalgia, fibromyalgia, hepatitis, asthenia, ocular symptoms, & psychosis. Fatal cases continue to deteriorate, experiencing continued fever, obtundation, coma, convulsions, diffuse coagulopathy, metabolic disturbances, shock and death, with death typically occurring between Days 8 and 16 (Weidmann et al., 2007).

2.3 Causes of Marburg Virus Disease

MVD is caused by two viruses classified in the genus Marburgvirus, family *Filoviridae*, order *Mononegavirales:* Marburg virus (MARV) and Ravn virus (RAVV) (Feldmann et al., 2005).

Marburg viruses are endemic in arid woodlands of equatorial Africa. Most Marburg virus infections were repeatedly associated with people visiting natural caves or working in mines. In 2009, the successful isolation of infectious MARV and RAVV was reported from healthy Egyptian rousettes (*Rousettus aegyptiacus*) caught in caves. This isolation strongly suggests that Old World fruit bats are involved in the natural maintenance of marburgviruses and that visiting bat-infested caves is a risk factor for acquiring marburgvirus infections. Further studies are necessary to establish whether Egyptian rousettes are the actual hosts of MARV and RAVV or whether they get infected via contact with another animal and therefore serve only as intermediate hosts. Another risk factor is contact with nonhuman primates, although only one outbreak of MVD (in 1967) was due to contact with infected monkeys. Finally, a major risk factor for acquiring marburgvirus infection is occupational exposure, i.e. treating patients with MVD without proper personal protective equipment (Stille et al., 2009).

Contrary to Ebola virus disease (EVD), which has been associated with heavy rains after long periods of dry weather, triggering factors for spillover of marburgviruses into the human population have not yet been described (Grolla et al., 2005).

2.4 Genome of marburg virus disease

Like all monomegaviruses, marburgvirions contain non-infectious, linear nonsegmented, single-stranded RNA genomes of negative polarity that possesses inverse-complementary 3' and 5' termini, do not possess a 5' cap, are not polyadenylated, and are not covalently linked to a protein. Marburgvirus genomes are approximately 19 kb long and contain seven genes in the order 3'-UTR-NP-VP35-VP40-GP-VP30-VP24-L-5'-UTR. The genomes of the two different marburgviruses (MARV and RAVV) differ in sequence (Gibb et al., 2001).

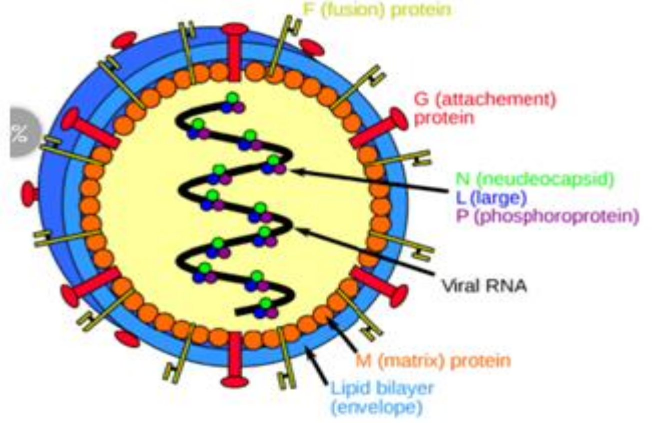

Figure 1: Genome of marburg virus disease
Source: (Barat et al., 2011).

Functions structural/accessory protein of Marburg virus

- **F (fusion) protein:-** It direct membrane fusion because paramyxovirus fusion is pH independent, virus entry occurs at host cell plasma membrane.
- **G (attachment) protein:-** It bind the receptor by attaching to the cell surface receptor and fusing viral and cell membrane.
- **N (Nucleocapsid):-** It binds to viral RNA and leads to formation of the helical nucleocapsid.
- **L (Large):-** It absorbs water and waste material before they are removed by defecation.
- **P (Phosphoprotein):-** It activates the hormone incline which indicates that there is a high concentration of glucose in the blood.
- **Viral RNA:** It translates protein into the host ribosomes.
- **M (Matrix) Protein:** - It plays an important structural role in viral assembly and also regulatory role in viral transcription.
- **Lipid bilayer (envelope):-** It is a universal component of all cell membranes. Its role is critical because its structural components provide the barrier that marks the boundaries of a cell.

2.5 Structure of marburg virus disease

Like all filoviruses, marburgvirions are filamentous particles that may appear in the shape of a shepherd's crook or in the shape of a "U" or a "6", and they may be coiled, toroid, or branched. Marburgvirions (Figs. 1 & 2) are generally 80 nm in width, but vary somewhat in length. In general, the median particle length of marburgviruses ranges from 795–828 nm (in contrast to ebolavirions, whose median particle length was measured to be 974–1,086 nm), but particles as long as 14,000 nm have been detected in tissue culture. Marburgvirions consist of seven structural proteins. At the center is the helical ribonucleocapsid, which consists of the genomic RNA wrapped around a polymer of nucleoproteins (NP). Associated with the ribonucleoprotein is the RNA-dependent RNA polymerase (L) with the polymerase cofactor (VP35) and a transcription activator (VP30). The ribonucleoprotein is embedded in a matrix, formed by the major (VP40) and minor (VP24) matrix proteins. These particles are surrounded by a lipid membrane derived from the host cell membrane. The membrane anchors a glycoprotein (GP1,2) that projects 7 to 10 nm spikes away from its surface. While nearly identical to ebolavirions in structure, marburgvirions are antigenically distinct (Grolla et al., 2005).

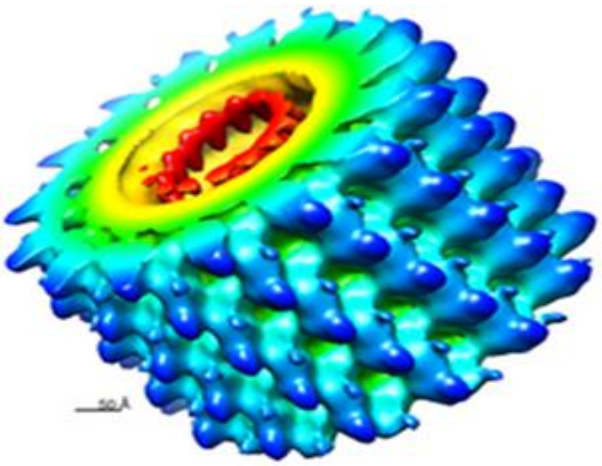

Figure 2: Marburg virus
Source: (Kiley et al., 2002)

2.6 Epidemiology of Marburg virus

The initial outbreak of Marburg virus disease (MVD) provided critical insights into its transmission patterns, host sources, and human-to-human spread, as summarized in Table 1 and illustrated in Figure 3.

2.6.1 1967 outbreak

MVD was first documented in 1967, when 31 people became ill in the German towns of Marburg and Frankfurt am Main, and in Belgrade, Yugoslavia. The outbreak involved 25 primary MARV infections and seven deaths, and six nonlethal secondary cases. The outbreak was traced to infected grivets (species Chlorocebus aethiops) imported from an undisclosed location in Uganda and used in developing poliomyelitis vaccines. The monkeys were received by Behringwerke, a Marburg company founded by the first winner of the Nobel Prize in Medicine, Emil von Behring. The company, which at the time was owned by Hoechst, was originally set up to develop sera against tetanus and diphtheria. Primary infections occurred in Behringwerke laboratory staff while working with grivet tissues or tissue cultures without adequate personal protective equipment. Secondary cases involved two physicians, a nurse, a post-mortem attendant, and the wife of a veterinarian. All secondary cases had direct contact, usually involving blood, with a primary case. Both physicians became infected through accidental skin pricks when drawing blood from patients. A popular science account of this outbreak can be found in Laurie Garrett's book The Coming Plague (Siegert et al., 2009).

2.6.2 1975 cases

In 1975, an Australian tourist became infected with MARV in Rhodesia (today Zimbabwe). He died in a hospital in Johannesburg, South Africa. His girlfriend and an attending nurse were subsequently infected with MVD, but survived (Kiley et al., 2002).

2.6.3 1980 cases

A case of MARV infection occurred in 1980 in Kenya. A French man, who worked as an electrical engineer in a sugar factory in Nzoia (close to Bungoma) at the base of Mount Elgon (which contains Kitum Cave), became infected by unknown means and died shortly after admission to Nairobi Hospital. The attending physician contracted MVD, but survived. A popular science account of these cases can be found in Richard Preston's book The Hot Zone (the French man is referred to under the pseudonym "Charles Monet", whereas the physician is identified under his real name, Shem Musoke) (Saijo, et al., 2005).

Table 1: Marburg virus disease outbreaks

Year	Country	Virus subtype	Cases	Death	Case fatality rate
1967	Germany	MARV	31	7	23%
1967	Yugoslavia	MARV	2	0	0%
1980	Kenya	MARV	2	1	50%
1987	Kenya	RAVV	1	1	100%
1988	Soviet union	MARV	1	1	100%
1990	Soviet Union	MARV	1	0	0%
1998-2000	Democratic Republic of Congo	MARV & RAVV	154	128	83%
2004-2005	Angola	MARV	252	227	90%
2007	Uganda	MARV & RAVV	4	1	25%
2008	Netherland of U.S	MARV	2	1	50%
2012	Uganda	MARV	18	9	50%
2014	Uganda	MARV	1	1	100%

Source: (Peterson et al., 2004).

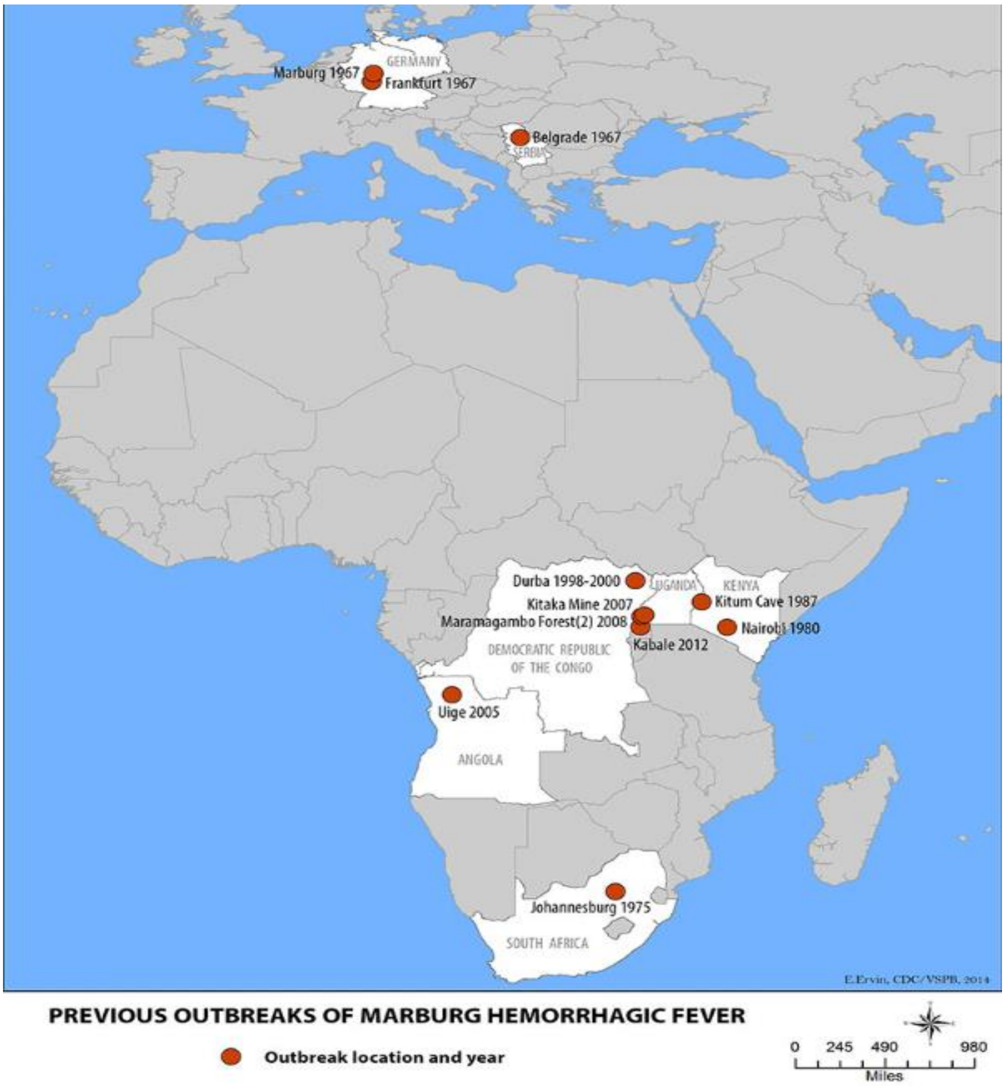

Figure 3: Previous outbreaks of Marburg virus
Source: (Johnson et al., 2006).

2.6.4 1987 case

In 1987, a single lethal case of RAVV infection occurred in a 15-year-old Danish boy, who spent his vacation in Kisumu, Kenya. He had visited Kitum Cave on Mount Elgon prior to travelling to Mombasa, where he developed clinical signs of infection. The boy died after transfer to Nairobi Hospital. A popular science account of this case can be found in Richard Preston's book The Hot Zone (the boy is referred to under the pseudonym "Peter Cardinal" (Geisbert and Jahrling, 2005).

2.6.5 1988 laboratory infection

In 1988, researcher Nikolai Ustinov infected himself lethally with MARV after accidentally pricking himself with a syringe used for inoculation of guinea pigs. The accident occurred at the Scientific-Production Association "Vektor" (today the State Research Center of Virology and Biotechnology "Vektor") in Koltsovo, USSR (today Russia). Very little information is publicly available about this MVD case because Ustinov's experiments were classified. A popular science account of this case can be found in Ken Alibek's book Biohazard (Gear et al., 2008).

2.6.6 1990 laboratory infection

Another laboratory accident occurred at the Scientific-Production Association "Vektor" (today the State Research Center of Virology and Biotechnology "Vektor") in Koltsovo, USSR, when a scientist contracted MARV by unknown means (Hofmann and Kunz, 2008).

2.6.7 1998–2000 outbreak

A major MVD outbreak occurred among illegal gold miners around Goroumbwa mine in Durba and Watsa, Democratic Republic of Congo from 1998 to 2000, when co-circulating MARV and RAVV caused 154 cases of MVD and 128 deaths. The outbreak ended with the flooding of the mine (Ksiazek, 2001).

2.6.8 2004–2005 outbreak

In early 2005, the World Health Organization (WHO) began investigating an outbreak of viral hemorrhagic fever in Angola, which was centered in the northeastern Uíge Province but also affected many other provinces. The Angolan government had to ask for international assistance, pointing out that there were only approximately 1,200 doctors in the entire country, with some provinces having as few as two. Healthcare workers also complained about a shortage of personal protective equipment such as gloves, gowns, and masks. Médecins Sans Frontières (MSF) reported that when their team arrived at the provincial hospital at the center of the outbreak, they found it operating without water and electricity. Contact tracing was complicated by the fact that the country's roads and other infrastructure have been devastated after nearly three decades of civil war and the countryside remained littered with land mines. Americo Boa Vida Hospital in the Angolan capital Luanda set up a special isolation ward to treat infected people from the countryside. Unfortunately, because MVD often results in death, some people came to view hospitals and medical workers with suspicion and treated helpers with hostility. For instance, a specially-equipped isolation ward at the provincial hospital in Uíge was reported to be empty during much of the epidemic, even though the facility was at the center of the outbreak. WHO was forced to implement what it described as a "harm reduction strategy", which entailed distributing disinfectants to affected families who refused hospital care. Of the 252 people who contracted MVD during outbreak, 227 died (Havemann and Schmidt, 2001; Stille and Böhle, 2001).

2.6.9 2007 cases

In 2007, four miners became infected with marburgviruses in Kamwenge District, Uganda. The first case, a 29-year-old man, became symptomatic on July 4, 2007, was admitted to a hospital on July 7, and died on July 13. Contact tracing revealed that the man had had prolonged close contact with two colleagues (a 22-year-old man and a 23-year-old man), who experienced clinical signs of infection before his disease onset. Both men had been admitted to hospitals in June and survived their infections, which were proven to be due to MARV. A fourth, 25-year-old man, developed MVD clinical signs in September and was shown to be infected with RAVV. He also survived the infection (Roddy et al., 2010).

2.6.10 2008 cases

On July 10, 2008, the Netherlands National Institute for Public Health and the Environment reported that a 41-year-old Dutch woman, who had visited Python Cave in Maramagambo Forest during her holiday in Uganda suffered of MVD due to MARV infection, and had been admitted to a hospital in the Netherlands. The woman died under treatment in the Leiden University Medical Centre in Leiden on July 11. The Ugandan Ministry of Health closed the cave after this case. On January 9 of that year an infectious diseases physician notified the Colorado Department of Public Health and the Environment that a 44-year-old American woman who had returned from Uganda had been hospitalized with a fever of unknown origin. At the time, serologic testing was negative for viral hemorrhagic fever. She was discharged on January 19, 2008. After the death of the Dutch patient and the discovery that the American woman had visited Python Cave, further testing confirmed the patient demonstrated MARV antibodies and RNA (Martini et al., 2009).

Viral Replication

The viral replication cycle occurs in the cytoplasm of infected cells and follows the general strategy of non-segmented negative-strand RNA viruses but with distinct features that contribute to its pathogenic potential.

Attachment and Entry

Replication begins with the attachment of viral glycoprotein (GP) to host cell receptors. C-type lectins, TIM-1, and the endosomal receptor Niemann–Pick C1 (NPC1) have been identified as essential for entry (Huang et al., 2022; Iheukwumere *et al*., 2025a). The virus enters host cells via macropinocytosis, a process dependent on actin remodeling, after which the viral particle is trafficked to endosomes. In the acidic environment of late endosomes, host cysteine proteases (cathepsins B and L) cleave GP, exposing binding domains that interact with NPC1. This interaction facilitates fusion between the viral and endosomal membranes, releasing the viral ribonucleoprotein complex (vRNP) into the cytoplasm (Kleine-Holthaus et al., 2023; Iheukwumere *et al.*, 2025b).

Transcription and Translation

The vRNP complex contains the negative-sense genomic RNA encapsidated by nucleoprotein (NP), along with polymerase cofactor VP35, transcription factor VP30, and the RNA-dependent RNA polymerase (L protein). The polymerase begins transcription at the 3′ leader region, producing capped and polyadenylated monocistronic mRNAs via a sequential "start-stop" mechanism (Cross et al., 2020; Iheukwumere *et al*., 2025c). These viral mRNAs are then translated by host ribosomes to generate structural proteins (NP, VP35, VP40, GP, VP30, VP24, and L) and regulatory proteins.

Genome Replication

Once sufficient NP is synthesized, the polymerase switches from transcription to replication. The full-length positive-sense antigenome is synthesized, which then serves as a

2.7 Signs and Symptoms of Marburg virus

After an incubation period of 5-10 days, symptom onset is sudden and marked by fever, chills, headache, and myalgia. Around the fifth day after the onset of symptoms, a maculopapular rash, most prominent on the trunk (chest, back, stomach), may occur. Nausea, vomiting, chest pain, a sore throat, abdominal pain, and diarrhea may then appear. Symptoms become increasingly severe and can include jaundice, inflammation of the pancreas, severe weight loss, delirium, shock, liver failure, massive hemorrhaging, and multi-organ dysfunction.

Because many of the signs and symptoms of Marburg hemorrhagic fever are similar to those of other infectious diseases such as malaria or typhoid fever, clinical diagnosis of the disease can be difficult, especially if only a single case is involved. The case-fatality rate for Marburg hemorrhagic fever is between 23-90% (Egbring et al., 2001).

Pathogenesis

The disease is characterized by high case fatality rates, systemic immune dysregulation, and multiorgan failure. The pathogenesis of MARV is multifactorial, involving viral replication dynamics, immune evasion mechanisms, and extensive tissue damage.

Initial Infection and Cellular Tropism

MARV primarily enters the body through mucosal surfaces, broken skin, or via contact with infected bodily fluids. Following entry, the virus targets monocytes, macrophages, and dendritic cells as its primary sites of replication (Olejnik et al., 2020). These antigen-presenting cells serve as reservoirs for viral amplification and dissemination throughout the body. Hepatocytes, endothelial cells, and fibroblasts are also infected, contributing to systemic pathology (Huang et al., 2022; Iheukwumere *et al*., 2024a).

Viral Immune Evasion

Effective immune evasion is a hallmark of MARV pathogenesis. The viral protein VP35 acts as a strong

template for producing new genomic RNAs. These nascent genomes are immediately encapsidated by NP, forming progeny vRNPs. The presence of VP35 as a cofactor enhances replication efficiency, while VP30 regulates the balance between transcription and replication (Olejnik et al., 2020; Iheukwumere *et al*., 2025d).

Assembly and Budding

Viral assembly takes place at the plasma membrane, mediated primarily by matrix protein VP40, which orchestrates the recruitment of vRNPs and other structural proteins to budding sites. The GP is incorporated into the host plasma membrane through the secretory pathway, where it forms spikes on the viral envelope. VP24 and VP35 also contribute to nucleocapsid condensation and immune evasion. Budding is completed through a process involving the host's endosomal sorting complexes required for transport (ESCRT) machinery, leading to the release of filamentous virions (Westover et al., 2022; Iheukwumere *et al*., 2025e; Iheukwumere *et al*., 2025f).

interferon (IFN) antagonist by inhibiting RIG-I-like receptor (RLR) signaling and preventing the production of type I IFNs (Valmas and Basler, 2020). VP24 further impairs antiviral responses by blocking nuclear import of phosphorylated STAT1, thereby suppressing IFN-stimulated gene expression (Kleine-Holthaus et al., 2023; Iheukwumere *et al*., 2024b). These strategies allow the virus to replicate unchecked during the early stages of infection, contributing to rapid viral spread.

Cytokine Storm and Inflammation

The unchecked replication of MARV in macrophages and dendritic cells triggers aberrant pro-inflammatory cytokine and chemokine release, leading to a "cytokine storm." Elevated levels of tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and other inflammatory mediators promote vascular leakage and endothelial dysfunction (Westover et al., 2022; Iheukwumere *et al*., 2024c). This dysregulated immune activation plays a central role in the development of hemorrhagic manifestations and shock.

Endothelial Damage and Coagulopathy

MARV pathogenesis also involves direct and indirect damage to endothelial cells. Viral replication in endothelial tissues, combined with inflammatory mediator release, results in disruption of vascular integrity. The coagulation system is severely impaired, leading to disseminated intravascular coagulation (DIC), thrombocytopenia, and widespread hemorrhage (Cross et al., 2020; Iheukwumere *et al*., 2024d). This contributes to the characteristic bleeding and organ ischemia seen in advanced disease stages.

Multiorgan Failure

Widespread viral replication, coupled with immune dysregulation, leads to significant damage in multiple organs, particularly the liver, spleen, and kidneys. Hepatic necrosis impairs clotting factor synthesis, further exacerbating coagulopathy, while splenic and lymph node destruction compromises adaptive immune responses (Westover et al., 2022; Iheukwumere *et al*., 2024e). Renal tubular necrosis contributes to fluid imbalance and metabolic failure, culminating in multiorgan dysfunction and, in many cases, death.

2.8 Risk of Exposure

People who have close contact with African fruit bats, humans patients, or non-human primates infected with Marburg virus are at risk. Historically, the people at highest risk include family members and hospital staff who care for patients infected with Marburg virus and have not used proper barrier nursing techniques. Particular occupations, such as veterinarians and laboratory or quarantine facility workers who handle non-human primates from Africa, may also be at increased risk of exposure to Marburg virus (Nikiforov et al., 2004).

Exposure risk can be higher for travelers visiting endemic regions in Africa, including Uganda and other parts of central Africa, and have contact with fruit bats, or enter caves or mines inhabited by fruit bats (Martini, 2001).

3. Marburg Virus

3.1 Replication of Marburg virus

The marburgvirus life cycle begins with virion attachment to specific cell-surface receptors, followed by fusion of the virion envelope with cellular membranes and the concomitant release of the virus nucleocapsid into the cytosol. The virus RdRp partially uncoats the nucleocapsid and transcribes the genes into positive-stranded mRNAs, which are then translated into structural and nonstructural proteins. Marburgvirus L binds to a single promoter located at the 3' end of the genome. Transcription either terminates after a gene or continues to the next gene downstream. This means that genes close to the 3' end of the genome are transcribed in the greatest abundance, whereas those toward the 5' end are least likely to be transcribed. The gene order is therefore a simple but effective form of transcriptional regulation. The most abundant protein produced is the nucleoprotein, whose concentration in the cell determines when L switches from gene transcription to genome replication. Replication results in full-length, positive-stranded antigenomes that are in turn transcribed into negative-stranded virus progeny genome copies. Newly synthesized structural proteins and genomes self-assemble and accumulate near the inside of the cell membrane. Virions bud off from the cell, gaining their envelopes from the cellular membrane they bud from. The mature progeny particles then infect other cells to repeat the cycle (Gear, 2009). Figure 4 shows marbug infection in human. Astrain of marbug virus is shown in Fig. 5

3.2 Diagnosis of marburg virus disease

The diagnostic process relies on a combination of clinical evaluation, laboratory-based molecular techniques, antigen and antibody detection methods, and supportive post-mortem analyses.

Clinical Evaluation

Early symptoms of MVD, including fever, headache, malaise, myalgia, and gastrointestinal disturbances, are nonspecific and overlap with other endemic diseases (Westover et al., 2022). Hemorrhagic signs, such as bleeding gums, petechiae, and gastrointestinal bleeding, may develop during later stages. While clinical suspicion is crucial in outbreak-prone areas, confirmatory laboratory testing remains indispensable (Iheukwumere *et al*., 2024f).

Figure 4: Marburg virus replication (Martini, 2001).

Molecular Diagnostics

Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) is the gold standard for detecting MARV during the acute phase of infection. Real-time quantitative RT-PCR (qRT-PCR) allows for both detection and viral load monitoring, which is essential for disease management and prognosis (Kleine-Holthaus et al., 2023; Iheukwumere *et al*., 2025g). Molecular assays can detect viral RNA in blood, saliva, or other body fluids within days of symptom onset.

Antigen Detection

Enzyme-linked immunosorbent assays (ELISAs) designed for MARV antigen capture provide rapid diagnostic capability during acute infection. Antigen detection is particularly useful in resource-limited settings, as it offers a quicker turnaround compared to molecular methods, though it may be less sensitive (Huang et al., 2022; Iheukwumere *et al*., 2025h).

Serological Methods

Serology plays a role in both acute and retrospective diagnosis. **IgM-capture ELISA** is employed to confirm recent infection, as IgM antibodies appear within days after symptom onset. **IgG detection** confirms past infection or seroconversion during convalescence, supporting epidemiological surveillance and outbreak investigations (Valmas and Basler, 2020; Iheukwumere *et al*., 2025i).

Virus Isolation and Culture

Although rarely used in routine diagnostics due to biosafety concerns, virus isolation in cell culture remains the definitive confirmatory method. This procedure can only be performed in biosafety level-4 (BSL-4) laboratories, limiting its widespread application (Olejnik et al., 2020; Iheukwumere *et al*., 2025j).

Immunohistochemistry and Post-Mortem Diagnosis
In fatal cases, immunohistochemistry (IHC) can detect viral antigens in tissue samples, providing valuable insights into viral tropism and confirming cause of death. This method complements molecular testing and aids in understanding disease pathology (Cross et al., 2020).

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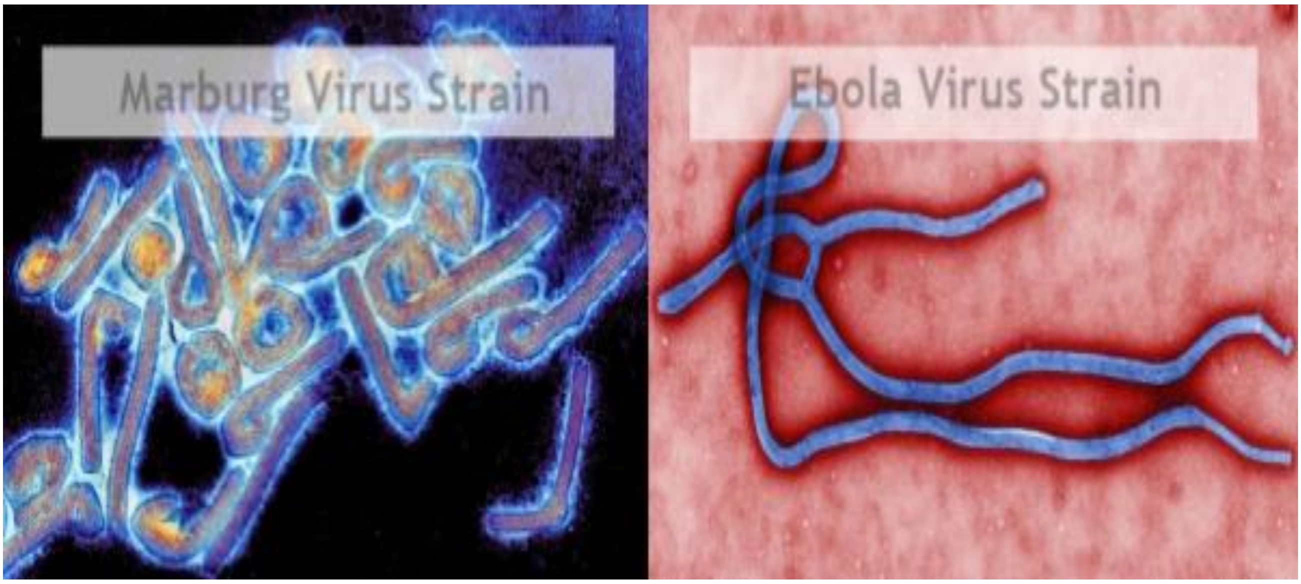

Figure 5: Marburg Virus strain (Peterson et al., 2006).

3.3 Prevention of marburg virus disease

There are currently no Food and Drug Administration-approved vaccines for the prevention of MVD. Many candidate vaccines have been developed and tested in various animal models. Of those, the most promising ones are DNA vaccines or based on Venezuelan equine encephalitis virus replicons, vesicular stomatitis Indiana virus (VSIV) or filovirus-like particles (VLPs) as all of these candidates could protect nonhuman primates from marburgvirus-induced disease. DNA vaccines have entered clinical trials. Marburgviruses are highly infectious, but not very contagious. Importantly, and contrary to popular belief, marburgviruses do not get transmitted by aerosol during natural MVD outbreaks. Due to the absence of an approved vaccine, prevention of MVD therefore relies predominantly on behavior modification, proper personal protective equipment, and sterilization/disinfection (Roddy et al., 2010).

Preventive measures against Marburg virus infection are not well defined, as transmission from wildlife to humans remains an area of ongoing research. However, avoiding fruit bats, and sick non-human primates in central Africa, is one way to protect against infection. Measures for prevention of secondary, or person-to-person, transmission are similar to those used for other hemorrhagic fevers. If a patient is either suspected or confirmed to have Marburg hemorrhagic fever, barrier nursing techniques should be used to prevent direct physical contact with the patient. These precautions include wearing of protective gowns, gloves, and masks; placing the infected individual in strict isolation; and sterilization or proper disposal of needles, equipment, and patient excretions.

In conjunction with the World Health Organization, CDC has developed practical, hospital-based guidelines, titled: Infection Control for Viral Haemorrhagic Fevers In the African Health Care Setting. The manual can help health-care facilities recognize cases and prevent further hospital-based disease transmission using locally available materials and few financial resources. Marburg hemorrhagic fever is a very rare human disease. However, when it occurs, it has the potential to spread to other people, especially health care staff and family members who care for the patient. Therefore, increasing awareness in communities and among health-care providers of the clinical symptoms of patients with Marburg hemorrhagic fever is critical. Better awareness can lead to earlier and stronger precautions against the spread of Marburg virus in both family members and health-care providers. Improving the use of diagnostic tools is another priority. With modern means of transportation that give access even to remote areas, it is possible to obtain rapid testing of samples in disease control centers equipped with Biosafety Level 4 laboratories to confirm or rule out Marburg virus infection (Pringle, 2005).

3.4 Endemic zones

The natural maintenance hosts of marburg viruses remain to be identified unequivocally. However, the isolation of both MARV and RAVV from bats and the association of several MVD outbreaks with bat-infested mines or caves strongly suggests that bats are involved in marburg virus transmission to humans. Avoidance of contact with bats and abstaining from visits to caves is highly recommended, but may not be possible for those working in mines or people dependent on bats as a food source (Bogomolov, 2008).

3.5 During outbreaks

Since marburgviruses are not spreading via aerosol, the most straightforward prevention method during MVD outbreaks is to avoid direct (skin-to-skin) contact with patients, their excretions and body fluids, or possibly contaminated materials and utensils. Patients ought to be isolated but still have the right to be visited by family members. Medical staff should be trained and apply strict barrier nursing techniques (disposable face mask, gloves, goggles, and a gown at all times). Traditional burial rituals, especially those requiring embalming of bodies, ought to be discouraged or modified, ideally with the help of local traditional healers (Pinzon et al., 2005).

3.5.1 In the laboratory

Marburgviruses are World Health Organization Risk Group 4 Pathogens, requiring Biosafety Level 4-equivalent containment. Laboratory researchers have to be properly trained in BSL-4 practices and wear proper personal protective equipment.

3.6 Treatment of marburg virus disease

There is currently no effective marburgvirus-specific therapy for MVD. Treatment is primarily supportive in nature and includes minimizing invasive procedures, balancing fluids and electrolytes to counter dehydration, administration of anticoagulants early in infection to prevent or control disseminated intravascular coagulation, administration of procoagulants late in infection to control hemorrhaging, maintaining oxygen levels, pain management, and administration of antibiotics or antimycotics to treat secondary infections. Experimentally, recombinant vesicular stomatitis Indiana virus (VSIV) expressing the glycoprotein of MARV has been used successfully in nonhuman primate models as post-exposure prophylaxis. Novel, very promising, experimental therapeutic regimens rely on antisense technology: phosphorodiamidate morpholino oligomers (PMOs) targeting the MARV genome could prevent disease in nonhuman primates. Leading medications from Sarepta and Tekmira both have been successfully used in European humans as well as primates (Nikiforov et al., 2004).

3.7 Prognosis

Prognosis is generally poor. If a patient survives, recovery may be prompt and complete, or protracted with sequelae, such as orchitis, hepatitis, uveitis, parotitis, desquamation or alopecia. Importantly, MARV is known to be able to persist in some survivors and to either reactivate and cause a secondary bout of MVD or to be transmitted via sperm, causing secondary cases of infection and disease (Peterson et al., 2004).

Of the 252 people who contracted Marburg during the 2004–2005 outbreak of a particularly virulent serotype in Angola, 227 died, for a case fatality rate of 90%.

Although all age groups are susceptible to infection, children are rarely infected. In the 1998–2000 Congo epidemic, only 8% of the cases were children less than 5 years old (Saijo, et al., 2005).

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