



The Waves of Ebola Virus: A Historical and Epidemiological Perspective with the Role of the Armed Conflicts

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Abstract

Ebola hemorrhagic fever (EHF) is an endemic disease in many African countries, first recorded in Sudan (now South Sudan) and the Democratic Republic of the Congo (DRC) in 1976. During these initial outbreaks, the case fatality rate (CFR) reached 53% in South Sudan and 88% in the DRC, underscoring the extreme virulence of Ebola virus disease (EVD) from its earliest appearances. Over the decades following these events, including the 2018–2020 outbreak—the second largest in the history of the disease—EVD resulted in 3,470 cases and 2,287 deaths. Armed conflicts have been a major factor in facilitating transmission, as population displacement from conflict zones or migration for economic reasons can trigger new waves of infection across borders. In this context, the ongoing conflict in Sudan and the large number of refugees crossing into Egypt pose a potential challenge for the Egyptian health sector, despite the absence of officially reported cases to date. Objective: This review synthesizes the historical evolution and epidemiology of EVD in conflict-affected regions and discusses implications for surveillance, early diagnosis, and outbreak control among displaced populations.

Keywords: Hemorrhagic fever; Trans-boundary diseases; Diseases and armed conflicts.

1. Introduction

Recent decades have witnessed the negative effects of pathogens on a wide scale that extended to include the entire world, as happened in the Covid-19 pandemic [1]. At the regional level, many African countries suffered from dangerous diseases during the twentieth century and extending to the twenty-first century, which had negative effects on the public health of the population of the affected areas. The list of

diseases included Human Immunodeficiency Virus (HIV), Rift Valley Fever (RVF), and Lassa fever [2–5]. Ebola virus disease (EVD) is a severe and often fatal illness in humans, first identified in 1976 during simultaneous outbreaks in South Sudan and the Democratic Republic of the Congo (DRC). These initial epidemics revealed the extraordinary virulence of the virus, with case fatality rates (CFR) of 53% and 88%, respectively, highlighting the urgent need for research into its transmission dynamics, clinical presentation, and effective control measures. Since then, EVD has re-emerged repeatedly across Central and West Africa, causing both localized and large-scale epidemics that tested the resilience of fragile health systems and the capacity for international response [6–8]. Beyond its clinical and epidemiological dimensions, Ebola has been shaped by complex socio-political contexts, particularly in conflict-affected regions, where displacement, migration, and health infrastructure breakdown have amplified transmission risks [9]. In this review, we synthesize the historical evolution and epidemiology of EVD, with emphasis on how political instability and population displacement influence outbreak dynamics, while also highlighting gaps in surveillance, diagnosis, and control strategies in resource-limited settings.

2. Taxonomic Position of Ebola Virus

The Genus Ebolavirus (EBO) comprises negative-stranded RNA viruses and is classified under the family Filoviridae and order Mononegavirales based on the latest taxonomic conditions. EBO is endemic to Africa, with its origins primarily traced to the central and western regions of the continent [10–11]. Initially, following a brief period of isolation of the Marburg virus (MBG) and the subsequent discovery of the EBO, taxonomic suggestions emerged due to their shared morphological features. Based on physicochemical and morphological data, both MBG and EBO were proposed to be classified under the Family Rhabdoviridae, as per the widely accepted

propositions among taxonomists at that time [12-13]. Thorough scrutiny of the morphological traits of MBO and EBO viruses led to the identification of distinguishing features that set them apart from their taxonomically related counterparts, such as rhinoviruses. Notably, the central axial channel in MBO and EBO was found to be considerably smaller in diameter compared to rhinoviruses. Furthermore, other morphological attributes, including body length, and chemical properties like protein structure, provided additional support for the taxonomic differentiation between Ebola and Marburg viruses on one side, and rhinoviruses on the other [14]. During the early stages of classification, certain researchers speculated that mature viruses had a doughnut-like shape, leading them to name the family containing these viruses as 'torovirus.' However, this term was short-lived since electron microscope examination after isolating EBO revealed that both MBG and EBO viruses exhibit elongated tubular shapes. Consequently, the term 'tuburnaviruses' was adopted to better align with the morphological characteristics of this virus family [15-16]. No formal proposal for the classification of MBG and EBO viruses was submitted to the International Committee on Taxonomy of Viruses (ICTV) until 1979. During a virus's congress in that year, discussions centered around the characteristics of these viruses, leading to the naming of this viral group as Filoviridae. The name was derived from the Latin word "filo" meaning "filament" chosen as an alternative to the term 'tuburnaviruses' based on early electron microscope examinations [14]. Figure (1) shows the degrees of relatedness between the two families, Filoviridae and Rhabdoviridae, which, as previously mentioned, led to the inclusion of MBG and EBO viruses in the Rhabdoviridae family. Five strains of Ebola viruses have been identified in Africa under genus *Ebolavirus*, classified based on the geographical location of the outbreaks, which are: (1) Zaire Ebola Virus (EBOV). (2) Sudan Ebola Virus (SUDV). (3) Bundibugyo Ebola Virus (BDBV). (4) Tai Forest ebolavirus (TAFV) and (5) Reston Ebola virus (RESTV); The first four strains have posed significant threats to both human and nonhuman primates, leading to viral hemorrhagic fevers with fatality rates of up to 90% [17-18].

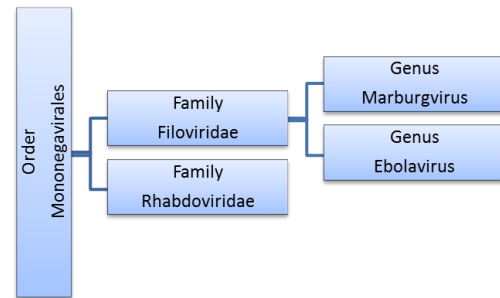


Figure 1. The taxonomic position of the Ebola virus and the relatedness of Filoviridae and Rhabdoviridae

3. Chronological review of Ebola Strains outbreaks in Africa

3.1 Early Indications and Pre-1976 Evidence

For nearly five decades since the first documented appearance of the Ebola virus in 1976 [19], both local and international health institutions have systematically monitored subsequent outbreaks (fig 2). These efforts have included recording the number of infections and associated clinical symptoms, assessing fatalities and case fatality rates, and providing medical care and treatment to affected populations while implementing precautionary measures, which remain a cornerstone of preventive medicine. Since the initial identification of Ebola hemorrhagic fever (EHF) in 1976 [19], viruses belonging to the genus *Ebolavirus* have spread across multiple countries, and outbreaks are no longer restricted to the African continent. This section provides an overview of the historical trajectory of Ebola outbreaks and their epidemiological manifestations within Africa.

3.2 The First Documented Outbreaks (1976–1979

Languon and Quaya [29] reported that although the first recognized outbreak of Ebola occurred in 1976, fact-finding investigations into Ebola as a viral pathogen have suggested the possible emergence of hemorrhagic fever in the DRC as early as 1972. This was based on the detection of viral antibodies in a physician who was likely infected during the autopsy of a medical student who had suffered from EHF. Such evidence indicates that the epidemiology of the Ebola virus may predate the first officially documented outbreak. However, retrospective reviews of medical records aimed at confirming earlier, undocumented events remain unreliable due to the risk of misdiagnosis, since clinical manifestations of EVD can resemble those of other infectious diseases. Moreover, limitations in the accuracy and reliability of antibody detection methods at that time further complicate the interpretation of such findings. The first officially reported outbreak of EHF occurred in southern Sudan between June and November 1976.

The earliest infections were reported in Nzara, primarily among workers in a cotton factory. Subsequent transfer of patients to the hospital in Maridi contributed to a worsening of the situation, as cases in Maridi alone accounted for nearly 75% of the total infections. By the end of the outbreak, a total of 284 cases (Table 1) had been recorded, including 151 deaths [19]. Coinciding with the outbreak of hemorrhagic fever in southern Sudan, another epidemic emerged in the northern part of the DRC between September 1 and October 24, 1976, in the Yambuku region. A total of 318 cases were reported, the majority of which occurred within a 70 km radius centered on Yambuku. The CFR was exceptionally high, reaching 88% of all confirmed infections [20]. These two outbreaks were the main starting point for the spread of the virus on a wider geographical scale that included other countries within a regional area that is confined in Central and West Africa, careful examination of the virus isolates from these two outbreaks showed that the isolates are of two different strains of the same species, these differences, from a genetic perspective, may be the reason for the virulence of the EBOV strain, which is evident from the higher number of infected people and the case fatality rate compared to the South Sudanese SUDV strain. The hemorrhagic fever virus was named Ebola, after the Ebola River located in the north of the DRC, in the region that witnessed the second outbreak, according to chronology [21]. On June 1977 an individual case appeared, consequently that does not fall under the concept of outbreak, the case was a nine-year-old girl from the village of Bonduni in the Democratic Republic of the Congo; She was suffering from a fever and her temperature rose to 39.5 degrees Celsius, accompanied by abdominal pain and bloody vomiting she died 28 hours after Tandala Hospital entrance [22-23]. Between July 31 and October 26, 1979, an outbreak caused by the SUDV occurred in the same rural area that had been affected during the earliest recorded epidemic in Nzara the outbreak was limited, as infections appeared in five families. The outbreak began with the entrance of a 45-year-old man to Nzara hospital, on the second of August 1979, when he had symptoms three days before entering the hospital but he soon died on the 5th of August, failure to implement strong precautionary measures the symptoms of hemorrhagic fever appeared on three of the relatives of those who had contact with him, and who later died as a result of their infection, the matter extended to four other families, as well as the death of two nurses from the medical staff of Nazara Hospital, the total outcome of this outbreak for the infected was 34 cases, and the death rate reached 65%, the limited number of infected people may be due to remedial failure in implementing the

precautionary measures that the local medical staff made; In September, about one month before the end of the outbreak, the entire area was isolated, and a supported medical team From the WHO provides health care [22, 24].

3.3 The Period of Latency (1980–1993)

After announcing the end of the second outbreak of the disease in Sudan in October 1979, no new cases of hemorrhagic fever were detected in Africa, and this continued until 1994, as if these 15 years seem more like a latency phase or the end of an activity cycle. No explanations were provided about the reasons behind the disappearance for this long period or what are the factors for the re-emergence of outbreaks [22, 25 – 28].

3.4 The Re-emergence and New Strains (1994–2001)

On November 24, 1994, a 34-year-old female scientist showed symptoms of a disease like those of dengue fever. She suffered from fever, rash, and diarrhea. She was evacuated to Switzerland, where she recovered. Diagnostic tests revealed that she was exposed to a new strain of Ebola virus TAFV, which was transmitted to her during the autopsy of a recently dead chimpanzee in the Taï Forest in Côte d'Ivoire [26, 30-32]. The TAFV outbreak in Côte d'Ivoire coincided with another outbreak in Gabon. During the outbreak in Gabon, which extended from November 1994 to February 1995, the symptoms that patients developed were thought to be due to yellow fever virus YF alone, based on clinical symptoms, and routine biochemical testing initially supported this belief, the results of a blood serum test using polymerase chain reaction (PCR) for 49 people tested positive because there was already a YF epidemic at that time. But some symptoms were not consistent with yellow fever, and screening of serological tests led to the detection of antibodies to Ebola virus in the isolates. Partial sequencing testing of the GP and L genes also confirmed that the isolated virus was indeed of the Zaire strain. This outbreak consisted of two waves. The first wave occurred in November and December 1994, and infection occurred in three gold extraction camps: Mikoka, Andok, and Minkibi, which are located in the vicinity of the rainforest. The second wave of the outbreak began in early 1995, and the last recorded case was on February 9, while the Gabonese Health Authority stated that the outbreak officially ended on February 17. The total number of cases of this outbreak, resulting from its two waves was 52 cases of infection, of which 31 patients died [26, 29, 32-33]. The re-emergence of EHF disease again in 1994, after a period of interruption in Ebola outbreaks within Africa since that date and over a period of twenty years until 2014, outbreaks continued to appear in some countries of Central and West Africa [31]. The outbreaks in the period between 1994 and the

declaration of the end of the Uganda outbreak of (2012-2013) were characterized by the limited number of people infected. According to the data in (Table 1), the largest number recorded in that period was in the Uganda outbreak of (2000-2001), where the number of infected people was 425 and deaths were 224 (53%) [27].

3.5 The West African Epidemic (2013–2016)

On December 2013, a two-year-old boy died in southeastern Guinea from hemorrhagic fever as a result of infection with the Ebola virus of the Zaire strain. This was followed by the appearance of symptoms of the disease on members of the child's family and health care workers who were in contact with the case. The disease was not diagnosed correctly, or its cause was determined, and the disease was not announced as a new outbreak until March 23, 2014, approximately three months after the death of this child and the infection was transmitted to a number of his family members as well as those in charge of the health care system [37-38]. This outbreak represents a special case in the history of the disease, as it is distinguished from its predecessors by its wide geographical scope. It did not exceed the borders of the state in the regional scope of Central and West Africa, but it reached the point where it crossed the African continent, where the outbreak affected many African countries, namely Guinea, Liberia, Sierra Leone, and the Democratic Republic of the Congo, Mali, Nigeria, Senegal, and European countries such as Italy, Spain, and UK, in addition to the USA [34]. This was accompanied by a very large number of infections and deaths, with the total number of patients at the end of that outbreak reaching 28,616 cases, while deaths exceeded 11,000, the largest part of which went to Guinea, Liberia, and Sierra Leone, while the rest of the countries did not have infections exceed the threshold of twenty cases, as is the case in Nigeria [31, 39].

3.6 The Recent and Limited Outbreaks (2017–2023)

The largest outbreak of Ebola hemorrhagic fever in the history of the disease was declared over on January 20, 2016 [40, 41]. After the end of the largest outbreak, a group of outbreaks occurred on the continent in the period between 2017 and the beginning of 2023, where the beginning of the last outbreak was announced on September 20, 2022 as a result of the emergence of the Ebola virus of the Sudanese strain, until the Ugandan Ministry of Health announced the end of that outbreak on January 11, 2023. The majority of outbreaks that followed the largest outbreak in the history of 2013-2016 were limited outbreaks in terms of the number of cases and deaths or in terms of the area covered

by the outbreak, with the exception of the 2018-2020 outbreak, which is the second largest outbreak in the history of the disease, which resulted in 3,470 cases of viral infection and 2,287 infections case of death [35-36].

Table 1: Ebola Outbreaks Chronology (1976-2014)

Outbreak	Species	Cases	Fatalities	CFR (%)	Ref
South Sudan (1976)	SUDV	284	151	53	[18]
DRC (1976)	EBOV	318	280	88	[47]
DRC (1977)	EBOV	1	1	100	[21]
South Sudan (1979)	SUDV	34	22	65	[23]
Cote d'Ivoire (1994)	TAFV	1	0	0	[30]
Gabon (1994-1995)	EBOV	52	31	60	[28]
DRC (1995)	EBOV	315	250	79	[27]
Gabon (1996)	EBOV	37	21	57	[28]
Gabon (1996-1997)	EBOV	60	45	75	[34]
Uganda (2000-2001)	SUDV	425	224	53	[26]
Gabon and ROC (2001-2002)	EBOV	124	97	78	[28]
ROC (2002-2003)	EBOV	143	128	89	[28]
ROC (2003)	EBOV	35	29	83	[30]
South Sudan (2004)	SUDV	17	7	41	[27]
ROC (2005)	EBOV	12	10	83	[33]
DRC (2007)	EBOV	264	187	71	[33]
Uganda (2007-2008)	BDBV	131	42	32	[33]
DRC (2008-2009)	EBOV	32	15	47	[33]
Uganda (2011)	SUDV	1	1	100	[34]
DRC (2012)	BDBV	38	13	34	[33]
Uganda (2012)	SUDV	11	4	36	[33]
Uganda (2012-2013)	SUDV	6	3	50	[28]
Guinea (2013-2016)	EBOV	28,646	11,323	40	[35]

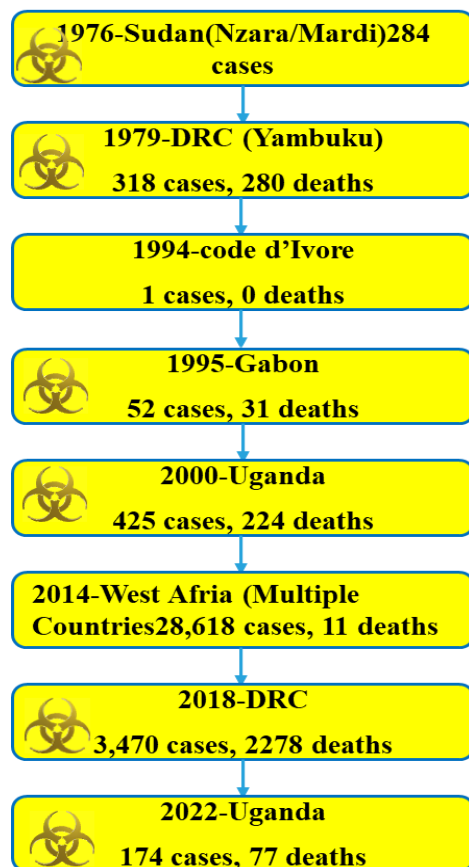


Figure 2. Chronological Timeline of Ebola Virus outbreaks

4. Ebola virus reservoirs, infection and transmission

4.1 The Recent and Limited Outbreaks (2017–2023)

Ebola is a zoonosis, there is a prevailing belief based on the hypothesis that fruit bats such as *Hypsignathus monstrosus*, *Epomops franqueti*, and *Myonycteris torquata* of the Pteropodidae family represent the main hosts of EBO; In contrast humans and non-human primates act as secondary hosts for the virus, causing severe disease symptoms that can lead to the death of the secondary host, and this is what some studies relied on to explain the decline in the numbers of non-human primates such as chimpanzees and African gorillas in the recent period [42-48] (fig3)

4.2 Early Studies to Identify the Natural Host

After an outbreak in 1995 in Kikwit and its surroundings in the Democratic Republic of Congo, 3066 samples of vertebrates were collected from habitats in the focus of the outbreak and its surroundings in order to identify the main reservoir of the virus from the Zaire strain, and the tests showed negative results. The failure to detect antibodies may be due to intermittent contact

between humans and the virus. Or the restrictions that can be imposed on the size of samples from rare species that are likely to be a reservoir for the virus [45].

4.3 Field Evidence Supporting Fruit Bats as the primary host

A subsequent field studies that extended from 2001 to 2005 through a number of expeditions that coincided with outbreaks in Gabon and the Democratic Republic of the Congo that resulted in the death of groups of primates, 1030 samples were collected of different animal species to be examined (Table 2) with the aim of identifying the reservoir of the virus among these species, the results showed conclusive evidence of the presence of species of fruit bats carrying the virus, with no symptoms of infection appearing [42, 49]. Laboratory tests of three species of bats, *E. franqueti*, *H. monstrosus*, and *M. torquata*, confirmed the presence of immunoglobulin G (IgG) of the Ebola virus in the blood serum of the tested bats [50].

Table 2: Collected animal specimens to identify the EBO reservoir [29].

Organism	Number of Samples
Bats	679
Birds	222
Small terrestrial vertebrates	129
Σ	1030

4.4 Ecological and Evolutionary Implications of Bat Reservoirs

Bats are also distinguished by their ability to fly, as they can cover large areas through this feature, and this may allow them to get rid of their droppings, which may be a container for the virus, which increases the possibility of the disease being transmitted to areas far from places where bats congregate, such as caves or African tropical forests, which increases the risk of transmission of the disease potential for large-scale outbreaks [26]. It is also noted that it is the same ecosystem in which great ape primates live, so exposure to bat droppings or to the remains of fruit and traces of bat saliva on them is the most likely means of transmitting the infection to great apes or even to humans. From the perspective of the evolution of Ebola viruses, there was a genetic differentiation between the different Ebola strains and the ability of this variation to cause infection among humans, as strains such as the Zaire strain and the Sudan strain are capable of causing disease in humans, while the Reston strain only infects great apes [51]. While recognizing the hypothesis that bats are the main reservoirs of viruses, given their ability to fly and cross natural barriers, such as the rivers spread throughout tropical Africa, this can be

explained by genetic variation between breeds based on the concept of development within individuals of a single species due to natural barriers [52]. Although bats represent the most likely reservoir for the Ebola virus, and although there is some preliminary evidence to suggest this, this topic has not yet been adequately addressed [41]. Therefore, we can conclude that it is possible that humans or even higher non-human primates interact directly or indirectly with bats, potentially representing the primary transmission source of the virus from bats to monkeys or even humans, especially since most of the infections occurred within the African tropical rainforests (fig 3).

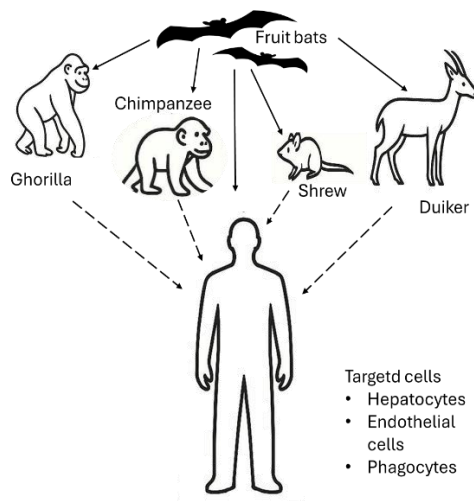


Figure 3. Transmission pathway from bats to primates

4.5 Transmission Pathways from Reservoirs to Humans

Assuming it is accepted that bats are the main source of transmission of the virus and causing infection, the secondary routes of infection are no less important, especially since they are confirmed by evidence, in contrast to the hypothesis that bats are the original source of infection, and the transmission of the Ebola virus through direct contact with the infected person is the most common way of transmitting the virus. As well as the consumption of infected bushmeat [53-54], sexual relations are also a means of horizontal transmission of the virus from one person to another, especially with the possibility of separating the virus from semen for a period that can reach 61 days. Possible sexual transmission occurred in March 2015 in a 44-year-old woman who, prior to her death, reported having had unprotected sex with an Ebola survivor 7 days before symptoms appeared [55] (fig 3).

4.6 Mechanisms of Infection and Viral Replication

The virus enters the body through any breaks in the skin or through mucous surfaces and targets many cells within the host's body [56]. The main targets of the virus are stem cells, liver cells, and endothelial cells [57]. During the incubation period, which may reach 21 days, the virus works to localize itself within the host's body and copies itself through a complex mechanism in order to ensure its survival [41]. At the cellular level, viruses multiply within the cell membranes of infected cells by budding. Laboratory studies on the human liver of some infected people who died as a result of the infection have indicated that this has also been done on monkeys and guinea pigs. Even experiments on tissue cultures have shown the existence of a pattern of virus reproduction by budding and the formation of branches parallel or perpendicular to the cellular membranes, which contain the viral protein [58].

4.7 Clinical Manifestations and Diagnostic Challenges

Usually, symptoms of infection appear after a period ranging between 6 to 12 days from the infection. The symptoms are mostly a high temperature with pain in the joints and muscles. At a relatively advanced stage, there is intestinal pain accompanied by nausea, vomiting, and diarrhea, as well as shortness of breath and cough [41]. One of the most important problems lies in the symptoms that appear on the person infected with Ebola hemorrhagic fever, which is the similarity of these symptoms with symptoms of other diseases, especially since the bleeding that characterizes Ebola disease does not appear in a maximum of 30% of patients, which means that 70% or more of those infected are at risk of being diagnosed. Their condition constitutes a mistake, and therefore the diagnosis must be made in the event of overlapping symptoms with other diseases, not by monitoring the apparent clinical symptoms only, despite their importance, because diseases such as Lassa fever, malaria, and typhoid have the same clinical symptoms resulting from infection with the Ebola virus, and this may be the case. It leads to a worsening of the condition or even to death [59-60].

5. The Impact of Armed Political Unrest on the Ebola Outbreak

5.1 Political Instability and Weak Health Infrastructure

The Ebola virus, responsible for hemorrhagic fever, has been endemic in Africa for decades, with the first recorded case occurring in the 1970s. While many African countries are experiencing successive waves of Ebola outbreaks, they are also experiencing political unrest and military conflicts that have led to

the collapse of government sectors, including the healthcare system. This has led to inadequate care for those infected, and the extreme difficulty of implementing measures under extreme political circumstances.

5.2 Correlation Between Conflict and Outbreak Hotspots

In West African countries that have been hotspots for the disease since the first recorded outbreak, waves of the disease in these countries coincided with the unrest sweeping these countries, which may indicate a causal relationship between armed political conflict and the emergence of hotspots of the disease [61-62].

5.3 Case Study: The 2018–2020 Outbreak in Eastern DRC

The 2018–2020 Ebola outbreak in eastern DRC was preceded by an incident on December 27, 2018, when protesters attacked an Ebola treatment center. The disruption contributed to the occurrence of 21 suspected and confirmed cases of the disease [62–63]. With repeated attacks on Ebola treatment centers, patients fled due to armed operations in North Kivu and Ituri provinces, and health care workers (174 workers) were attacked by warring groups [62]. On February 24, 2019, treatment centers in Katwa and Butembo were destroyed and staff evacuated, with 4 patients in the operational area fleeing into the forests.

5.4 Implications of the West African Civil Wars for Ebola Outbreak Management

During June–August of the same year, the Ugandan Ministry of Health and the World Health Organization (WHO) announced in a joint statement the rapid spread of the disease in areas where displaced people were gathering in border areas fleeing the war [62]. With the civil wars in Liberia and Sierra Leone, the health systems collapsed tragically, making control measures to limit the spread of the virus extremely difficult, especially with the almost complete absence of governmental and international financial support through international organizations concerned with health in conflict areas [64-65].

6. Public Health Awareness and Preventive Strategies in Egypt

6.1 Impact of the Sudan Conflict and Population Displacement

The armed conflict in Sudan on April 15, 2023, impacted more than 3.4 million civilians displacement to neighboring countries, including the Arab Republic of Egypt. According to official reports from the Egyptian government, Sudanese refugees in Egypt constituted (35.3%) of the total

number of people displaced by the conflict [66]. The escalating conflict between the two parties to the conflict in Sudan has led to the collapse of basic services, including health care institutions within the country. WHO reports indicate that 70-80% of health facilities are not operating at full capacity; the crisis has further worsened with the spread of cholera and malaria due to the destruction of infrastructure, especially sanitation, the occurrence of floods, and the decline in vector control.

6.2 Emerging Health Risks and Implications for Egypt's Public Health System

The emergence of the polio virus type (cVDPV2) has been observed, but concerns have increased with the detection of strains linked to cVDPV2 in Egypt, in which displacement has played a role, which warns of the possibility of other cross-border diseases [67-68].

Although no Ebola cases have been reported during the ongoing conflict in Sudan, the large-scale displacement of populations raises serious concerns about the cross-border spread of infectious diseases. Displaced people often move under conditions that favor the transmission of pathogens, including poor sanitation, limited access to medical care, and high population density in temporary shelters. This situation highlights the potential introduction of diseases previously unseen in Egypt, including Ebola virus disease (EVD). Therefore, it is essential to establish comprehensive preventive measures to mitigate the risk of disease importation and safeguard public health adopted through.

6.3 Proposed Preventive and Control Measures in Egypt

1. Establishing an Early Warning and Surveillance System

The creation of an integrated early warning system is critical to rapidly detect and respond to emerging health threats. Border hospitals should be linked to the Egyptian Centers for Disease Control and Prevention (CDC) and medical research institutions to facilitate timely data sharing, case tracking, and laboratory confirmation. Enhanced surveillance at border regions would enable the rapid identification of suspected Ebola cases before they spread further.

2. Strengthening Border Health Security

Strict health measures should be implemented at southern border crossings. This includes establishing new medical units, conducting health screening, and collecting detailed questionnaires from displaced persons to identify potential exposure risks. Polymerase chain reaction (PCR) testing should be administered for suspected cases, with immediate

isolation protocols based on established response frameworks and previous outbreak data.

3. Monitoring Regional Ebola Hotspots

Egypt should adopt a proactive regional monitoring approach by closely following developments in areas affected by Ebola outbreaks. Early awareness of emerging clusters will allow Egyptian health authorities to coordinate with international organizations, prepare medical facilities, and ensure the availability of diagnostic and treatment resources.

7. Conclusion

Ebola virus is a hemorrhagic fever. Fruit bats (Family: Pteropodidae) are believed to be the primary host of the virus, which is transmitted to humans as secondary hosts through human interactions with primary hosts, leading to death in the event of infection. Political unrest and armed conflicts encourage the spread of the disease due to the destruction of infrastructure and the disruption of preventive measures. This causal relationship has been clearly demonstrated in the conflicts in Sierra Leone and Liberia, and today with the deterioration of the situation in Sudan, which has a long history of Ebola, the resulting strong possibility of the disease returning, especially with the uncontrolled flow of displaced people under the influence of fighting towards the southern Egyptian border. Concerns are growing, requiring the Egyptian health sector, in coordination with international organizations, to take proactive measures, even if no confirmed cases have been recorded to date.

8. References

1. Bamgboye EL, Omiye JA, Afolaranmi OJ, Davids MR, Tannor EK, Wade S, Niang A, Were A, Naicker S. COVID-19 pandemic: is Africa different?. *Journal of the National Medical Association*. 2021 Jun 1;113(3):324-35.
2. <https://doi.org/10.1016/j.jnma.2020.10.001>
3. Chippaux JP, Chippaux A. Yellow fever in Africa and the Americas: a historical and epidemiological perspective. *Journal of Venomous Animals and Toxins including Tropical Diseases*. 2018 Sep 21;24:20. <https://doi.org/10.1186/s40409-018-0162-y>
4. Howlett WP. Neurological disorders in HIV in Africa: a review. *African health sciences*. 2019 Aug 20;19(2):1953-77. <https://doi.org/10.4314/ahs.v19i2.19>
5. Mylne AQ, Pigott DM, Longbottom J, Shearer F, Duda KA, Messina JP, Weiss DJ, Moyes CL, Golding N, Hay SI. Mapping the zoonotic niche of Lassa fever in Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene*. 2015 Aug 1;109(8):483-92. <https://doi.org/10.1093/trstmh/trv047>
6. Bird BH, Ksiazek TG, Nichol ST, MacLachlan NJ. Rift Valley fever virus. *Journal of the American Veterinary Medical Association*. 2009 Apr 1;234(7):883-93. <https://doi.org/10.2460/javma.234.7.883>
7. Kamorudeen RT, Adedokun KA, Olaninmoye AO. Ebola outbreak in West Africa, 2014–2016: Epidemic timeline, differential diagnoses, determining factors, and lessons for future response. *Journal of infection and public health*. 2020 Jul 1;13(7):956-62. <https://doi.org/10.1016/j.jiph.2020.03.014>
8. O'Leary A, Jalloh MF, Neria Y. Fear and culture: contextualising mental health impact of the 2014–2016 Ebola epidemic in West Africa. *BMJ global health*. 2018 Jun 1;3(3):e000924. <https://doi.org/10.1136/bmjgh-2018-000924>
9. Roshania R, Mallow M, Dunbar N, Mansary D, Shetty P, Lyon T, Pham K, Abad M, Shedd E, Tran AM, Cundy S. Successful implementation of a multicountry clinical surveillance and data collection system for Ebola virus disease in West Africa: findings and lessons learned. *Global Health: Science and Practice*. 2016 Sep 28;4(3):394-409. <https://doi.org/10.9745/GHSP-D-16-00186>
10. Chapwanya M, Lubuma J, Terefe Y, Tsanou B. Analysis of war and conflict effect on the transmission Dynamics of the tenth Ebola outbreak in the Democratic Republic of Congo. *Bulletin of Mathematical Biology*. 2022 Dec;84(12):136. <https://doi.org/10.1007/s11538-022-01094-4>
11. Siegert R. Marburg-, Lassa-und Ebola-Virus als Erreger hämorrhagischer Fieber. *DMW-Deutsche Medizinische Wochenschrift*. 1978 Jul;103(29):1176-81. <https://www.thieme-connect.com/products/ejournals/abstract/10.1055/s-0028-1129225>
12. Malvy D, McElroy AK, de Clerck H, Günther S, van Griensven J. Ebola virus disease. *The Lancet*. 2019 Mar 2;393(10174):936-48. [https://doi.org/10.1016/S0140-6736\(18\)33132-5](https://doi.org/10.1016/S0140-6736(18)33132-5)
13. Webb PA, Johnson KM, Wulff H, Lange JV. Some observations on the properties of Ebola virus. *Ebola virus haemorrhagic fever*. 1978:91-4.
14. Peters D, Müller G, Slenczka W. Morphology, development, and classification of the Marburg virus. In *Marburg virus disease 1971* (pp. 68-83). Berlin, Heidelberg: Springer Berlin Heidelberg. https://doi.org/10.1007/978-3-662-01593-3_10
15. Kiley MP, Bowen ET, Eddy GA, Isaacson M, Johnson KM, McCormick JB, Murphy FA, Pattyn SR, Peters D, Prozesky OW, Regnery RL. Filoviridae: a taxonomic home for Marburg and

- Ebola viruses. Intervirology. 1982 Jan 1;18(1-2):24-32. <https://doi.org/10.1159/000149300>
16. Almeida JD, Waterson AP, Simpson DI. Morphology and morphogenesis of the Marburg agent. In Marburg virus disease 1971 (pp. 84-97). Berlin, Heidelberg: Springer Berlin Heidelberg. https://doi.org/10.1007/978-3-662-01593-3_11
17. Simpson DI, Zuckerman AJ. Marburg and Ebola: viruses in search of a relation. Nature. 1977 Mar 17;266(5599):217-8. <https://doi.org/10.1038/266217a0>
18. de La Vega MA, Stein D, Kobinger GP. Ebolavirus evolution: past and present. PLoS pathogens. 2015 Nov 12;11(11):e1005221. <https://doi.org/10.1371/journal.ppat.1005221>
19. The Centers for Disease Control and Prevention., 2016. 2014-2016 Ebola Outbreak in West Africa, <https://www.cdc.gov/vhf/ebola/outbreaks/2014-west-africa/index.html>
20. Report of a WHO/International Study Team, 1978. Ebola haemorrhagic fever in Sudan, 1976. Bulletin of the World Health Organization, 56(2), p.247. <https://iris.who.int/handle/10665/261727>
21. International Commission, 1978. Ebola haemorrhagic fever in Zaire, 1976. Bull. World Health Organ., 56, pp.271-293. <https://iris.who.int/handle/10665/261733>
22. Peters, C.J. and Peters, J.W., 1999. An introduction to Ebola: the virus and the disease. The Journal of Infectious Diseases, 179(Supplement 1), pp.ix-xvi. <https://doi.org/10.1086/514322>
23. Tucker, C.J., Wilson, J.M., Mahoney, R., Anyamba, A., Linthicum, K. and Myers, M.F., 2002. Climatic and ecological context of the 1994-1996 Ebola outbreaks. Photogrammetric engineering and remote sensing, 68(2), pp.147-152.
24. Heymann, D.L., Weisfeld, J.S., Webb, P.A., Johnson, K.M., Cairns, T. and Berquist, H., 1980. Ebola hemorrhagic fever: Tandala, Zaire, 1977-1978. Journal of Infectious Diseases, 142(3), pp.372-376. <https://doi.org/10.1093/infdis/142.3.372>
25. Baron, R.C., McCormick, J.B. and Zubeir, O.A., 1983. Ebola virus disease in southern Sudan: hospital dissemination and intrafamilial spread. Bulletin of the World Health Organization, 61(6), p.997. <https://pmc.ncbi.nlm.nih.gov/articles/PMC2536233/>
26. Arthur, R.R., 2002. Ebola in Africa-discoveries in the past decade. Eurosurveillance, 7(3), pp.33-36. <https://doi.org/10.2807/esm.07.03.00342-en>
27. Pigott, D.M., Golding, N., Mylne, A., Huang, Z., Henry, A.J., Weiss, D.J., Brady, O.J., Kraemer, M.U., Smith, D.L., Moyes, C.L. and Bhatt, S., 2014. Mapping the zoonotic niche of Ebola virus disease in Africa. Elife, 3, p.e04395. <https://doi.org/10.7554/eLife.04395>
28. Legrand, J., Grais, R.F., Boelle, P.Y., Valleron, A.J. and Flahault, A., 2007. Understanding the dynamics of Ebola epidemics. Epidemiology & Infection, 135(4), pp.610-621. <https://doi.org/10.1017/S0950268806007217>
29. Changula, K., Kajihara, M., Mweene, A.S. and Takada, A., 2014. Ebola and Marburg virus diseases in Africa: increased risk of outbreaks in previously unaffected areas?. Microbiology and immunology, 58(9), pp.483-491. <https://doi.org/10.1111/1348-0421.12181>
30. Languon, S. and Quaye, O., 2019. Filovirus disease outbreaks: a chronological overview. Virology: research and treatment, 10, p.1178122X19849927. <https://doi.org/10.1177/1178122X19849927>
31. Le Guenno, B., Formenty, P., Wyers, M., Gounon, P., Walker, F. and Boesch, C., 1995. Isolation and partial characterisation of a new strain of Ebola virus. The lancet, 345(8960), pp.1271-1274. [https://doi.org/10.1016/S0140-6736\(95\)90925-7](https://doi.org/10.1016/S0140-6736(95)90925-7)
32. Krejcova, L., Michalek, P., Chudobova, D., Heger, Z., Hynek, D., Adam, V. and Kizek, R., 2014. West Africa Ebola outbreak 2014, J. Metallomics Nanotechnol, 2015, pp.6-12.
33. Khan, A.S., Tshioko, F.K., Heymann, D.L., Le Guenno, B., Nabeth, P., Kerstiëns, B., Fleerackers, Y., Kilmarx, P.H., Rodier, G.R., Nkuku, O. and Rollin, P.E., 1999. The reemergence of Ebola hemorrhagic fever, Democratic Republic of the Congo, 1995. The Journal of infectious diseases, 179(Supplement_1), pp.S76-S86. <https://doi.org/10.1086/514306>
34. Georges, A.J., Leroy, E.M., Renaut, A.A., Benissan, C.T., Nabias, R.J., Ngoc, M.T., Obiang, P.I., Lepage, J.P.M., Bertherat, E.J., Bénoni, D.D. and Wickings, E.J., 1999. Ebola hemorrhagic fever outbreaks in Gabon, 1994-1997: epidemiologic and health control issues. The Journal of infectious diseases, 179(Supplement_1), pp.S65-S75. <https://doi.org/10.1086/514290>
35. CDC. 2023 Outbreaks chronology: History of Ebola virus disease outbreaks. <https://www.cdc.gov/vhf/ebola/outbreaks/history/chronology.html> (accessed 31 August 2023).
36. Hussein, H.A., 2023. Brief review on ebola virus disease and one health approach. Heliyon. <https://doi.org/10.1016/j.heliyon.2023.e19036>

37. Coltart, C.E., Lindsey, B., Ghinai, I., Johnson, A.M. and Heymann, D.L., 2017. The Ebola outbreak, 2013–2016: old lessons for new epidemics. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 372(1721), p.20160297. <https://doi.org/10.1098/rstb.2016.0297>
38. Hayden, E.C., 2014. The ebola questions. *Nature*, 514(7524), p.554. <https://doi.org/10.1038/514554a>
39. Baize, S., Pannetier, D., Oestereich, L., Rieger, T., Koivogui, L., Magassouba, N.F., Soropogui, B., Sow, M.S., Keita, S., De Clerck, H. and Tiffany, A., 2014. Emergence of Zaire Ebola virus disease in Guinea. *New England Journal of Medicine*, 371(15), pp.1418-1425. <https://www.nejm.org/doi/full/10.1056/NEJMOA1404505>
40. Koroma, V., 2014. Ebola's lost ward (vol 513, pg 474, 2014). *Nature*, 516(7531), pp.298-298.
41. WHO. 2016. Ebola situation report: January 20, 2016. WHO, Geneva, Switz. <http://apps.who.int/ebola/current-situation/>ebola-situation-report-20-january-2016>
42. Boisen, M.L., Hartnett, J.N., Goba, A., Vandi, M.A., Grant, D.S., Schieffelin, J.S., Garry, R.F. and Branco, L.M., 2016. Epidemiology and management of the 2013–16 West African Ebola outbreak. *Annual review of virology*, 3, pp.147-171. <https://doi.org/10.1146/annurev-virology-110615-040056>
43. Leroy, E.M., Kumulungui, B., Pourrut, X., Rouquet, P., Hassanin, A., Yaba, P., Délicat, A., Paweska, J.T., Gonzalez, J.P. and Swanepoel, R., 2005. Fruit bats as reservoirs of Ebola virus. *Nature*, 438(7068), pp.575-576. <https://doi.org/10.1038/438575a>
44. Leendertz, S.A.J., Gogarten, J.F., Düx, A., Calvignac-Spencer, S. and Leendertz, F.H., 2016. Assessing the evidence supporting fruit bats as the primary reservoirs for Ebola viruses. *EcoHealth*, 13, pp.18-25. <https://doi.org/10.1007/s10393-015-1053-0>
45. Olson, S.H., Reed, P., Cameron, K.N., Ssebidde, B.J., Johnson, C.K., Morse, S.S., Karesh, W.B., Mazet, J.A. and Joly, D.O., 2012. Dead or alive: animal sampling during Ebola hemorrhagic fever outbreaks in humans. *Emerging health threats journal*, 5(1), p.9134. <https://doi.org/10.3402/ehth.v5i0.9134>
46. Leirs, H., Mills, J.N., Krebs, J.W., Childs, J.E., Akaibe, D., Woollen, N., Ludwig, G., Peters, C.J. and Ksiazek, T.G., 1999. Search for the Ebola virus reservoir in Kikwit, Democratic Republic of the Congo: reflections on a vertebrate collection. *The Journal of infectious diseases*, 179(Supplement 1), pp.S155-S163. <https://doi.org/10.1086/514299>
47. Baron, R.C., McCormick, J.B. and Zubeir, O.A., 1983. Ebola virus disease in southern Sudan: hospital dissemination and intrafamilial spread. *Bulletin of the World Health Organization*, 61(6), p.997. <https://pmc.ncbi.nlm.nih.gov/articles/PMC2536233/>
48. Vogel, G., 2003. Can great apes be saved from Ebola? *Science*, 300(5626), pp.1645-1646. <https://doi.org/10.1126/science.300.5626.1645>
49. Laupland, K.B. and Valiquette, L., 2014. Ebola virus disease. *Canadian Journal of Infectious Diseases and Medical Microbiology*, 25, pp.128-129. <https://doi.org/10.1155/2014/527378>
50. Walsh, P.D., Abernethy, K.A., Bermejo, M., Beyers, R., De Wachter, P., Akou, M.E., Huijbregts, B., Mambounga, D.I., Toham, A.K., Kilbourn, A.M. and Lahm, S.A., 2003. Catastrophic ape decline in western equatorial Africa. *Nature*, 422(6932), pp.611-614. <https://doi.org/10.1038/nature01566>
51. Geisbert, T.W., Hensley, L.E., Larsen, T., Young, H.A., Reed, D.S., Geisbert, J.B., Scott, D.P., Kagan, E., Jahrling, P.B. and Davis, K.J., 2003. Pathogenesis of Ebola hemorrhagic fever in cynomolgus macaques: evidence that dendritic cells are early and sustained targets of infection. *The American journal of pathology*, 163(6), pp.2347-2370. [https://doi.org/10.1016/S0002-9440\(10\)63591-2](https://doi.org/10.1016/S0002-9440(10)63591-2)
52. Jones, M.E., Schuh, A.J., Amman, B.R., Sealy, T.K., Zaki, S.R., Nichol, S.T. and Towner, J.S., 2015. Experimental inoculation of Egyptian rousette bats (*Rousettus aegyptiacus*) with viruses of the Ebolavirus and Marburgvirus genera. *Viruses*, 7(7), pp.3420-3442. <https://doi.org/10.3390/v7072779>
53. Calvignac-Spencer, S., Schulze, J.M., Zickmann, F. and Renard, B.Y., 2014. Clock rooting further demonstrates that Guinea 2014 EBOV is a member of the Zaïre lineage. *PLoS currents*, 6. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4073806/>
54. Feldmann, F. and Feldmann, H., 2013. Ebola: facing a new transboundary animal disease?. In *Vaccines and Diagnostics for Transboundary Animal Diseases* (Vol. 135, pp. 201-209). Karger Publishers. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4813513/>
55. Wong, S., Lau, S., Woo, P. and Yuen, K.Y., 2007. Bats as a continuing source of emerging infections in humans. *Reviews in medical virology*, 17(2), pp.67-91. <https://doi.org/10.1002/rmv.520>
56. Christie, A., Davies-Wayne, G.J., Cordier-

- Lasalle, T., Blackley, D.J., Laney, A.S., Williams, D.E., Shinde, S.A., Badio, M., Lo, T., Mate, S.E. and Ladner, J.T., 2015. Possible sexual transmission of Ebola virus—Liberia, 2015. *Morbidity and Mortality Weekly Report*, 64(17), p.479. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4584553/>
57. CDC. 2009. Arboviruses and related zoonotic viruses. In *Biosafety in Microbiological and Biomedical Laboratories*, ed. LCW Chosewood, E Deborah, pp. 233–67. Atlanta: CDC
 58. Ito, H., Watanabe, S., Takada, A. and Kawaoka, Y., 2001. Ebola virus glycoprotein: proteolytic processing, acylation, cell tropism, and detection of neutralizing antibodies. *Journal of virology*, 75(3), pp.1576-1580. <https://doi.org/10.1128/jvi.75.3.1576-1580.2001>
 59. Ellis, D.S., Stamford, S., Tovey, D.G., Lloyd, G., Bowen, E.T.W., Platt, G.S., Way, H. and Simpson, D.I.H., 1979. Ebola and marburg viruses: II. Their development within vero cells and the extra-cellular formation of branched and torus forms. *Journal of Medical Virology*, 4(3), pp.213-225. <https://doi.org/10.1002/jmv.1890040307>
 60. Johnson, K.M., McCormick, J.B., Webb, P.A., Smith, E.S., Elliott, L.H. and King, I.J., 1987. Clinical virology of Lassa fever in hospitalized patients. *Journal of Infectious Diseases*, 155(3), pp.456-464. <https://doi.org/10.1093/infdis/155.3.456>
 61. Schieffelin, J.S., Shaffer, J.G., Goba, A., Gbakie, M., Gire, S.K., Colubri, A., Sealfon, R.S., Kanneh, L., Moigboi, A., Momoh, M. and Fullah, M., 2014. Clinical illness and outcomes in patients with Ebola in Sierra Leone. *New England journal of medicine*, 371(22), pp.2092-2100. <https://doi.org/10.1056/nejmoal1411680>
 62. Kraemer MU, Pigott DM, Hill SC, Vanderslott S, Reiner RC, Stasse S, Brownstein JS, Gutierrez B, Dennig F, Hay SI, Wint GW. Dynamics of conflict during the Ebola outbreak in the Democratic Republic of the Congo 2018–2019. *BMC medicine*. 2020 Dec;18:1-0. <https://doi.org/10.1186/s12916-020-01574-1>
 63. Chapwanya M, Lubuma J, Terefe Y, Tsanou B. Analysis of war and conflict effect on the transmission Dynamics of the tenth Ebola outbreak in the Democratic Republic of Congo. *Bulletin of Mathematical Biology*. 2022 Dec;84(12):136. <https://doi.org/10.1007/s11538-022-01094-4>
 64. Wells CR, Pandey A, Ndeffo Mbah ML, Gaüzère BA, Malvy D, Singer BH, Galvani AP. The exacerbation of Ebola outbreaks by conflict in the Democratic Republic of the Congo. *Proceedings of the National Academy of Sciences*. 2019 Nov 26;116(48):24366-72. <https://doi.org/10.1073/pnas.1913980116>
 65. Kruk ME, Rockers PC, Williams EH, Varpilah ST, Macauley R, Saydee G, Galea S. Availability of essential health services in post-conflict Liberia. *Bulletin of the World Health Organization*. 2010 Jul;88(7):527-34. <https://doi.org/10.2471/blt.09.071068>
 66. Omondi T, Sheriff ID. Sierra Leone's long recovery from the scars of war. *Bull World Health Organ*. 2010;88:725-6. <https://doi.org/10.2471/blt.10.031010>
 67. UNICEF., 17 March 2025. Situation of the Sudanese Displaced Population in Egypt 2023/2024. <https://knowledge.unicef.org/resource/situation-sudanese-displaced-population-egypt-20232024/revisions/36331/view>
 68. Venkatesan P. Burden of infectious diseases in Sudan. *The Lancet Microbe*. 2025 Jan 1;6(1). <http://doi.org/10.1016/j.lanmic.2024.101035>
 69. SUDAN FI. 2024. Unprecedented hunger, mental health tragedy, and gender-based violence: THE CRISIS FOR CHILDREN AND FAMILIES IN SUDAN. <https://reliefweb.int/report/sudan/unprecedented-hunger-mental-health-tragedy-and-gender-based-violence-crisis-children-and-families-sudan>

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