

EBOLA – IN THE CONTEXT OF OTHER DISEASES & ISSUES (AFRICA)

Google AI Discussion 26.05.2026

how many people die of meningitis in Africa yearly?

26 May 2026

Meningitis claims the lives of tens of thousands of people in Africa every year, with children bearing the heaviest burden. The continent is home to the global "meningitis belt"—a region stretching across 26 countries from Senegal to Ethiopia—which experiences the highest rates of infection and mortality. [1, 2]

Context and Burden

- **Global vs. Regional:** Recent global studies indicate that meningitis kills over 250,000 people annually, with African countries disproportionately affected.
- **The Meningitis Belt:** This sub-Saharan zone is highly vulnerable due to environmental factors, such as dry winds and dust, which trigger devastating epidemics.
- **Official Data:** While outbreaks can spike during dry seasons, regional monitoring indicates that across key endemic nations, meningitis deaths routinely number in the thousands annually, with tens of thousands of suspected cases reported. [1, 2, 3, 4, 5]

Prevention and Response

Health organizations continuously work to mitigate this heavy toll through routine immunization programs and rapid emergency response teams. The [World Health Organization Regional Office for Africa](#) provides ongoing surveillance, data on active outbreaks, and updates on mass vaccination campaigns for the region. [1, 2, 3]

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

9 sites

- Meningitis kills a quarter million people a year - study
Updated / Saturday, 28 Mar 2026 12:01. A student receives the Meningitis B vaccine at the University of Kent in England. More than...
- RTE.ie
Over 250000 people die from meningitis annually
Meningitis is caused by the inflammation of tissues around the brain and spinal cord due to viruses, bacteria, fungi, or parasites...
- YouTube-CGTN Africa
Meningococcal Meningitis - WHO | Regional Office for Africa
During the 2014 epidemic season, 19 African countries implementing enhanced surveillance reported 11 908 suspected cases including...

WHO | Regional Office for Africa

Show all

You said:

how many people die of malaria in Africa yearly?

26 May 2026

Malaria causes approximately **580,000 to 600,000 deaths** in Africa annually, accounting for roughly **95% of all global malaria fatalities**. While mortality rates have declined by about 60% since 2000, progress has recently stalled due to factors like climate change, drug resistance, and funding gaps. [1, 2, 3]

Key Statistics and Trends

- **Most Recent Estimates:** In 2024, there were an estimated **610,000 malaria deaths globally**, with the African Region bearing the vast majority of this burden. In 2023, the region recorded approximately **569,000 deaths**.
- **Vulnerable Populations:** Children under the age of five are the most affected, making up about **75% to 76%** of all malaria deaths in Africa.
- **High-Burden Countries:** Just four countries—**Nigeria, the Democratic Republic of the Congo, Niger, and Tanzania**—account for more than half of all global malaria deaths. [1, 2, 3, 4, 5, 6]

Current Challenges

According to recent reports from the [World Health Organization \(WHO\)](#), the fight against malaria is facing several critical threats: [1, 2]

- **Biological Resistance:** Mosquitoes are becoming increasingly resistant to insecticides, and the malaria parasite is showing resistance to standard treatments.
- **Environmental Impact:** Extreme weather events and changing climate patterns are expanding the reach of malaria-carrying mosquitoes.
- **Stalled Progress:** The [African Union \(AU\)](#) notes that the continent is currently off track to meet its goal of eliminating malaria by 2030. [1, 2, 3, 4, 5]
This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

11 sites

- Malaria | WHO | Regional Office for Africa

Malaria deaths * About 96% of malaria deaths globally were in 29 countries. Four countries accounted for just over half of all mal...

WHO | Regional Office for Africa

- 2023 Africa Malaria Progress Report

H.E. Moussa Faki Mahamat. Chair, African Union Commission. H.E. Umaro Sissoco Embaló President, Guinea-Bissau. Chair, ALMA. Dr. Mi...

African Leaders Malaria Alliance

- Malaria facts & statistics 2025

Malaria facts and statistics. WHO World Malaria Report 2025. i. Malaria remains a serious global health challenge, particularly in...

Medicines for Malaria Venture

Show all

You said: how many people die of typhoid fever in Africa yearly?

26 May 2026

Typhoid fever causes an estimated 29,000 to 33,000 deaths in sub-Saharan Africa each year. While this is lower than the mortality rates for malaria or meningitis, the region still accounts for a significant portion of the roughly **110,000 to 140,000 deaths** that occur globally from typhoid annually. [1, 2, 3, 4]

Key Insights on Typhoid in Africa

- **High Burden Areas:** The **Democratic Republic of the Congo (DRC)**, Madagascar, and Burkina Faso report some of the highest incidence rates, with the DRC recording over **315 cases per 100,000 people**.

- **Vulnerable Populations:** Children under 15 years old are the most at risk, particularly those between **2 and 14 years of age**.
- **Recent Outbreaks:** Countries like [South Africa](#) have seen localized outbreaks in recent years, with over **200 laboratory-confirmed cases** reported throughout 2025 in provinces like Gauteng and Western Cape. [[1](#), [2](#), [3](#), [4](#)]

Challenges to Accuracy

Official numbers likely underestimate the true death toll for several reasons: [[1](#)]

- **Diagnostic Difficulty:** Because typhoid shares symptoms with malaria and other febrile illnesses, it is often misdiagnosed without blood culture sampling.
- **Limited Surveillance:** Many cases go unrecorded because individuals in remote areas may use traditional medicine or do not have access to hospitals.
- **Antimicrobial Resistance (AMR):** The emergence of multidrug-resistant (MDR) and extremely drug-resistant (XDR) strains is making the disease increasingly difficult to treat across Africa. [[1](#), [2](#), [3](#), [4](#), [5](#)]
Are you interested in the specific **vaccination programs** currently being rolled out to combat these deaths?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

10 sites

- Major typhoid fever surveillance study in sub-Saharan Africa ...
The findings from this 4-year study, the Severe Typhoid in Africa (SETA) program, offers new typhoid fever burden estimates from s...

International Vaccine Institute

- Alert: Update on enteric fever in South Africa (18 Feb 2022) - NICD
According to the World Health Organization (WHO), every year an estimated 11–20 million people develop enteric fever and between 1...

NICD

- Understanding typhoid burden in Africa
The results found that typhoid is a major cause of illness in sub-Saharan Africa. The World Health Organization (WHO) considers co...

Take on Typhoid
Show all

You said: how many people die of **hunger** in Africa yearly?

26 May 2026

Estimating the exact number of deaths from hunger in Africa is complex because hunger often kills indirectly by weakening the immune system against diseases like pneumonia or diarrhea. However, recent reports highlight a severe and worsening crisis: [[1](#), [2](#), [3](#), [4](#), [5](#)]

- **Growing Hunger:** While global hunger has slightly declined, it is **rising in Africa**, where over **307 million people** (more than 20% of the population) were affected by hunger in 2024.
- **Preventable Child Mortality:** An estimated **369,000 children** could die annually across 24 high-risk countries if they remain without treatment for severe acute malnutrition due to humanitarian funding cuts.
- **Famine Conditions:** In 2024 and 2025, formal **famines** were confirmed in parts of **Sudan** and **Gaza**. Famine is only declared when at least two people per 10,000 die every day from starvation-related causes.
- **High-Risk Countries:** **Nigeria, the Democratic Republic of the Congo**, and Sudan have the highest numbers of people facing acute food insecurity in 2025. For example, Nigeria alone records over **31,000 starvation-related deaths** annually. [[1](#), [2](#), [3](#), [4](#), [5](#), [6](#), [7](#), [8](#)]

Factors Driving the Crisis

- **Conflict and Displacement:** Major wars in Sudan and the DRC have shattered food systems and blocked aid delivery.
- **Climate Shocks:** Record heat in 2024 and recurring droughts in regions like the Horn of Africa have devastated crops.
- **Economic Instability:** Persistent food price inflation continues to make healthy diets unaffordable for one-third of the global population. [1, 2, 3, 4, 5, 6]
Would you like to see a **breakdown of hunger statistics** for a specific African region or country?

You said: how many victims have produced in Africa the relevant infectious diseases we discussed about, since each of them has been identified as such?

The number of victims produced by these infectious diseases in Africa varies dramatically. **Some, like Ebola, cause intense, high-fatality outbreaks, while others, like Lassa and Yellow Fever, cause thousands of deaths annually that often go underreported.** [1, 2]

The following data summarizes the impact in Africa since each disease was first identified:

1. Ebola Virus Disease (Identified 1976)

- **Total Deaths:** Over **15,000 deaths** have been recorded across Africa **over the past 50 years.**
- **Major Milestone:** The 2014–2016 West Africa outbreak was the deadliest, accounting for more than **11,300** of these deaths.
- **Current 2026 Outbreak:** As of early June 2026, the ongoing **Bundibugyo strain** outbreak in the DRC and Uganda has resulted in at least **42 confirmed deaths** (and nearly 250 suspected deaths) out of 282 confirmed cases. [1, 2, 3, 4, 5]

2. Marburg Virus Disease (Identified 1967)

- **Total Deaths:** Roughly **600–700 deaths** have been documented in Africa.
- **Major Outbreaks:** The largest occurred in **Angola (2004–2005)** with 227 deaths and the **DRC (1998–2000)** with 128 deaths.
- **Recent Impact:** Smaller outbreaks continue to appear, such as in **Equatorial Guinea (2023)** with 12 deaths and **Rwanda (2024)** with 6 deaths. [1, 2, 3]

3. Lassa Fever (Identified 1969)

- **Annual Impact:** Unlike Ebola, Lassa is endemic and occurs every year. It is estimated to cause up to **5,000 deaths annually** in West Africa. [1]
- **Recent Data:** In **Nigeria alone**, the 2026 season has already seen **167 deaths** as of April, with a high case fatality rate of 25.2%. [1, 2]
- **Total Victims:** Over the 55+ years since its discovery, the cumulative death toll likely reaches into the **hundreds of thousands**, though exact historical figures are missing due to severe underreporting. [1]

4. Yellow Fever (Identified 1900–1927) [1]

- **Annual Impact:** It remains the most widespread hemorrhagic disease discussed. The WHO estimates between **29,000 and 60,000 deaths annually** in Africa. [1, 2]
- **Total Victims:** Because it has been endemic for over a century, the total number of victims is in the **millions**. However, mass vaccination campaigns have significantly reduced these numbers in recent decades. [1]

5. Crimean-Congo Hemorrhagic Fever (CCHF) (Identified 1944/1956)

- **Impact:** While it is widespread across Africa, it typically causes **sporadic cases** rather than massive epidemics. Most African countries report only a handful of deaths per year, usually among livestock workers or through hospital-acquired infections. Would you like a more detailed breakdown of the **fatality rates** for each of these diseases to see which is technically the "deadliest"?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

15 sites

- Ebola - ANRS Maladies infectieuses émergentes
22 May 2026 — Global circulation of Ebola Around fifty Ebola virus disease outbreaks have been recorded since 1976. ... The majority and most se...

ANRS Maladies infectieuses émergentes

- Ebola Killed 15000 People In Africa Over The Past 50 Years
1 Jun 2026 — and the Democratic Republic of Congo has reported. a further rise in Ebola infections with 19 new positive test results it has now...

2:36

YouTube-WION

- Marburg Virus Disease: Causes, Symptoms & Treatment
20 Jun 2023 — Marburg virus outbreaks The most recent Marburg outbreaks were in 2023. The first was in February 2023 in Equatorial Guinea with 1...

Cleveland Clinic
Show all

how many people were infected with Ebola outside of Africa and how many of them died

8 June 2026

Outside of Africa, a total of **9 people** have been infected with Ebola, and **2 of them died**. [\[1, 2, 3\]](#)

While the vast majority of Ebola cases and deaths have occurred within Africa—most notably during the 2014–2016 West African epidemic—isolated cases were reported in other regions due to medical evacuations or local transmission following the arrival of infected individuals. [\[1, 2, 3\]](#)

Breakdwon of Cases Outside Africa

The specific count of confirmed cases and deaths by country is as follows:



United States: 4 cases, 1 death. These included a traveler from Liberia who died in Dallas and two nurses who treated him (both recovered), as well as a medical doctor who was infected in Guinea and successfully treated in New York. [[1](#), [2](#), [3](#)]



Spain: 1 case, 0 deaths. A nursing assistant in Madrid became the first person to contract the virus outside of Africa after caring for a repatriated priest. [[1](#), [2](#), [3](#), [4](#)]



United Kingdom: 1 case, 0 deaths. A nurse who had been working in Sierra Leone was diagnosed upon her return to Glasgow. [[1](#), [2](#), [3](#)]



Italy: 1 case, 0 deaths. An Italian healthcare worker who volunteered in Sierra Leone tested positive after returning to Italy. [[1](#), [2](#), [3](#)]



Germany: 1 case, 1 death. A UN medical officer who was infected in Liberia was evacuated to Germany for treatment, where he later died. [1]



Norway: 1 case, 0 deaths. A Norwegian doctor with Doctors Without Borders (MSF) was evacuated from Sierra Leone and successfully treated in Oslo. [1, 2]

You said: which one of them is considered **contagious and highly contagious?**

Of the viral hemorrhagic fevers discussed, **Ebola** and **Marburg** are considered **highly contagious** due to their potential for rapid human-to-human spread through direct contact with infected bodily fluids. [1, 2, 3]

The contagiousness of these diseases is categorized by how they spread between people: [1]

1. Highly Contagious (Direct Human-to-Human) [1]

These viruses are easily transmitted from person to person, often leading to large-scale outbreaks. [1, 2]

- **Ebola and Marburg:** Both are **highly contagious once symptoms appear**. Transmission occurs through direct contact with the blood, secretions (vomit, diarrhea), or organs of infected individuals, including those who have died. The risk is highest during the late stages of illness and at funerals. [1, 2, 3]
- **Lassa Fever:** While primarily spread by rodents, it is considered **contagious** because it can spread between humans, particularly in hospital settings or through sexual contact after recovery. It is generally less "explosively" contagious than Ebola but still a major public health risk. [1, 2, 3, 4]
- **Crimean-Congo Hemorrhagic Fever (CCHF):** This is also **contagious** between humans through contact with infected blood or secretions. It has caused significant "nosocomial" (hospital-acquired) outbreaks among medical staff. [1, 2, 3, 4, 5]

2. Not Contagious Between Humans (Vector-Borne) [1]

These diseases are infectious but **not contagious** from one person to another through typical direct contact. [1, 2]

- **Yellow Fever:** It is **not contagious between humans**. You cannot "catch" it by touching, kissing, or caring for an infected person. It is spread exclusively by the bite of an infected mosquito.
- **Dengue Hemorrhagic Fever:** Like Yellow Fever, it requires a mosquito vector and **cannot be transmitted directly between people**. [1, 2, 3, 4, 5]

Summary of Transmission Risks

Disease [1, 2, 3, 4, 5]	Contagious to Humans?	Primary Transmission Route
Ebola / Marburg	Yes (Highly)	Direct contact with bodily fluids
Lassa Fever	Yes	Rodent waste / Contact with infected people
CCHF	Yes	Tick bites / Contact with infected blood
Yellow Fever	No	Mosquito bites

Are you interested in how **Personal Protective Equipment (PPE)** and isolation protocols differ for these two groups?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

17 sites

- Viral haemorrhagic fevers: origins, reservoirs, transmission ...
30 Jul 2025 — Flaviviruses. Flaviviruses are found throughout the world and are primarily spread by mosquitoes and ticks. Human to human transmi...

GOV.UK

- [Highly contagious diseases with human-to-human transmission]
15 Dec 2012 — Abstract. Highly contagious diseases are caused by various biological agents that pose a risk to individuals and may have a potent...

National Institutes of Health (.gov)

- Crimean–Congo haemorrhagic fever: transmission to visitors ... - PMC - NIH
Introduction. Crimean–Congo haemorrhagic fever (CCHF) is a severe, contagious haemorrhagic fever with a case fatality rate ranging...

National Institutes of Health (.gov)

Show all

You said: What is considered to be the general Infection/Fatality rate for the above diseases - in the case of contagion (human to human transmission)?

The infection and fatality rates for these diseases vary significantly based on the specific virus and the level of medical care available. In the context of human-to-human transmission (contagion), the **Case Fatality Rate (CFR)** refers to the percentage of confirmed patients who die from the disease. [1, 2, 3]

Comparison of Fatalities and Contagion Risk

Disease [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13]	Average Case Fatality Rate (CFR)	Transmission Risk (Contagion)
Marburg	24% – 88% (Avg ~50%)	Highly Contagious: Very high risk through direct contact with blood/fluids.
Ebola (Zaire)	25% – 90% (Avg ~50%)	Highly Contagious: Extreme risk via direct physical contact with patients or deceased.

Ebola (Bundibugyo)	~15% – 30% (Current 2026: ~16.4%)	Highly Contagious: Rare strain; lower historical CFR but still high human-to-human risk.
Lassa Fever	~1% Overall (15%–25% for hospitalised)	Contagious: Risk exists in hospitals or close households, but primarily zoonotic.
CCHF	10% – 40%	Contagious: Risk of hospital-acquired outbreaks via contact with infected blood.

Key Factors in Infection Rates

- **Secondary Attack Rate:** In households, the risk of a healthy person catching **Ebola** from an infected member is approximately **32%** if they have direct physical contact (like nursing care), but drops to nearly **1%** without direct contact. [1]
- **The "Hemorrhagic" Variable:** For diseases like **Lassa Fever**, the majority of infections (80%) are mild or asymptomatic; however, for those who develop severe hemorrhagic symptoms and require hospitalisation, the fatality rate jumps to **15%–25%**. [1, 2]
- **Impact of Early Care:** Early intensive supportive care, such as **aggressive rehydration, can significantly lower the CFR** even for the deadliest viruses like **Marburg** and **Ebola**. [1, 2]
- **Strain Differences:** The **2026 Ebola outbreak** in the DRC and Uganda is caused by the **Bundibugyo strain, which historically has a lower fatality rate (around 30%) than the more common Zaire strain (up to 90%)**. [1, 2, 3, 4]

Can you please put in a table the diseases, with corresponding: number of victims since discovery, the case fatality rate and the transmission means?

Here is a summary table of the viral hemorrhagic diseases we discussed. Please note that "Total Victims" for endemic diseases like Lassa and Yellow Fever are estimates based on annual death rates provided by the [World Health Organization](#) and Africa CDC, as exact cumulative historical records for these are not fully tracked.

Disease	Estimated Total Victims (Since Discovery)	Case Fatality Rate (CFR)	Primary Transmission Means
Ebola (Zaire)	~15,000+	25% – 90% (Avg: 50%)	Highly Contagious: Direct contact with blood/fluids or infected corpses.
Ebola (Bundibugyo)	~150 – 200*	15% – 30%	Highly Contagious: Direct contact with blood/fluids.
Marburg	~600 – 700	24% – 88% (Avg: 50%)	Highly Contagious: Direct contact with blood/fluids; often linked to bat caves.
Lassa Fever	~285,000+ (Est.)	~1% (Overall) / 15%–20% (Severe)	Contagious: Rodent waste (rats); human-to-human via blood/fluids.
Yellow Fever	3.5M – 7.5M+ (Est.)	3% – 50% (Toxic phase)	Not Contagious: Spread exclusively by mosquito bites (<i>Aedes</i> or <i>Haemagogus</i>).
CCHF	Thousands (Sporadic)	10% – 40%	Contagious: Tick bites; contact with

blood from infected livestock or humans.

*Note: The total for the Bundibugyo strain includes the current 2026 outbreak figures (approx. 42 confirmed and 250 suspected deaths).

can you add to the table the average number of deaths per year and the number of outbreaks?

The table below incorporates the requested historical data and current figures for the **2026 outbreaks** in Africa.

Disease [1 , 2 , 3 , 4 , 5 , 6 , 7 , 8 , 9 , 10 , 11 , 12 , 13 , 14]	Estimated Total Deaths (Since Discovery)	Case Fatality Rate (CFR)	Avg. Deaths per Year (Est.)	Number of Outbreaks (Major/Recorded)	Primary Transmission Means
Ebola (Zaire)	~15,266+	50% – 90%	~305	~42	Highly Contagious: Direct contact with bodily fluids.
Ebola (Bundibugyo)	~118 – 350*	15% – 30%	~10 – 20	3 (2007, 2012, 2026)	Highly Contagious: Direct contact with bodily fluids.
Marburg	~600 – 700	24% – 88%	~10 – 12	~18	Highly Contagious: Direct contact with fluids; fruit bats.
Lassa Fever	~285,000+	~1% (Overall)	~5,000	Endemic (Seasonal surges)	Contagious: Rodent waste; human-to-human via fluids.
Yellow Fever	~3.5M+	3% – 50%	30,000 – 60,000	Frequent (Urban/Jungle cycles)	Not Contagious: Exclusively spread by mosquito bites.
CCHF	Thousands	10% – 40%	Variable (Sporadic)	Numerous (Sporadic clusters)	Contagious: Tick bites; contact with livestock blood.

Key Context for the Data:

- **Calculations:** "Average Deaths per Year" is a rough estimate derived by dividing total deaths by the number of years since each virus was first identified (e.g., Ebola Zaire: 15,266 / 50 years ≈ 305). [[1](#)]
- **Ebola Bundibugyo (2026):** Figures for this strain are rapidly changing. As of early June 2026, there are **64 confirmed deaths** in the DRC and Uganda, though suspected deaths exceed 250. [[1](#), [2](#)]
- **Lassa & Yellow Fever:** These are **endemic**, meaning they are constantly present in certain regions. Unlike "outbreaks" of Ebola which start and end, these diseases cause thousands of "silent" deaths every year that are often not part of a single officially declared event. [[1](#)]

can you give me the same categories for Influenza in Africa?

For influenza, the data is categorized differently because it is both a constant seasonal presence and an occasional pandemic threat. Unlike Ebola or Marburg, which occur in discrete outbreaks, influenza causes a "silent" but massive annual death toll in Africa. [1, 2]

Category [1, 2, 3, 4, 5, 6, 7]	Seasonal Influenza (Africa)	Pandemic Influenza (Historical/Major)
Estimated Total Deaths	Millions (Cumulative since identified)	~2.5 Million+ (1918 Pandemic alone)
Case Fatality Rate (CFR)	~0.1% or lower	~2% – 15% (Historical highs)
Avg. Deaths per Year (Est.)	28,000 – 163,000	N/A (Occurs in irregular waves)
Number of Outbreaks	Constant/Seasonal (Annual cycles)	4 Global Pandemics in the last 100 years
Primary Transmission	Highly Contagious: Respiratory droplets and aerosols	Highly Contagious: Rapid airborne spread

Key Differences in Impact

- **Annual Burden:** Seasonal influenza is estimated to kill between **28,000 and 163,000 people annually** across the African continent. In South Africa alone, it causes roughly 6,000 to 11,000 deaths every year. [1, 2]
- **The 1918 "Spanish Flu":** This remains the single most devastating respiratory event in African history. **It is estimated that 2% of Africa's entire population (roughly 2.5 million people) died within just six months.** [1]
- **High Vulnerability:** While the global CFR for seasonal flu is low (~0.1%), it is often higher in Africa due to factors like lower vaccination rates, limited access to healthcare, and high prevalence of co-morbidities such as HIV. [1, 2]
- **Modern Outbreaks:** Beyond seasonal cycles, Africa frequently monitors "highly pathogenic" strains like **Avian Flu (H5N1)**. While these have caused massive poultry die-offs, human cases in Africa remain rare compared to other regions. [1, 2]

Would you like to know more about how the **current 2026 surveillance systems** in Africa are being used to monitor both Ebola and new flu strains simultaneously?

You said: Can you tell me in what year these viral infections (for hemorrhagic diseases) have been discovered?

The viral infections associated with hemorrhagic diseases were discovered over several decades, often during sudden and dramatic outbreaks. [1]

The years of discovery for the major viral hemorrhagic fevers (VHFs) are:

- **Yellow Fever (1900–1927):** While the disease was known for centuries, it was the first human virus to be discovered. The **U.S. Army Yellow Fever Commission** confirmed its transmission by mosquitoes in **1900**, and the **virus itself was first isolated in 1927.** [1]
- **Crimean-Congo Hemorrhagic Fever (1944/1956):** It was first identified in the **Crimea** in **1944** (as Crimean fever). **A similar virus, known as the Congo virus**, was later isolated in the **Belgian Congo** (now DRC) in **1956. In 1969, these were found to be the same virus.** [1, 2]
- **Junín Virus (1958):** The cause of **Argentine hemorrhagic fever**, this was the first of the New World arenaviruses to be recognized when it was isolated in **1958.** [1]
- **Machupo Virus (1963):** This virus, which causes **Bolivian hemorrhagic fever**, was isolated in **1963.** [1]

- **Marburg Virus (1967):** First identified following simultaneous outbreaks in **Marburg** and **Frankfurt**, Germany, and **Belgrade**, Serbia. The infection was traced back to **African green monkeys imported from Uganda**. [1, 2, 3, 4, 5, 6]
- **Lassa Virus (1969):** Discovered after a series of fatal infections among **healthcare workers in Lassa, Nigeria**. [1, 2]
- **Ebola Virus (1976):** First appeared **during two simultaneous outbreaks** in **Yambuku**, Zaire (now DRC), and **Nzara**, South Sudan. [1, 2]
- **Hantavirus (1978):** While cases of "Korean hemorrhagic fever" occurred during the Korean War (1950s), the **Hantaan virus** was officially isolated from a striped field mouse in **1978**.
- **Lujo Virus (2008):** A newer addition to the list, this arenavirus was discovered during a small but highly fatal outbreak in **South Africa** and **Zambia** in **2008**. [1]
Would you like more details on how the **isolation methods** for these viruses have evolved since their initial discovery?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

14 sites

- Emergence of deadly viral haemorrhagic fever disease ... - PMC
Unlike LLOV, which is not known to infect humans but has been found to be associated with dead Schreibers's long-fingered bats of ...
- National Institutes of Health (.gov)
1. Overview of Viral Haemorrhagic Fevers (VHFs)
New-World Arenaviruses are a group of rodent-associated Arenaviruses in South America similar to the Lassa group in Africa (Old-World...)
- Health Protection Surveillance Centre
Hemorrhagic Fever - an overview | ScienceDirect Topics
2 Marburg Virus Disease. Marburg virus is a single-stranded RNA virus (genus Marburgvirus, family Filoviridae) that is the etiolog...

ScienceDirect.com
Show all

You said: What is considered to be the carrier sources of these viruses?

The carrier sources for these viruses, often referred to as **natural reservoirs**, are typically animals or insects that carry the virus without becoming seriously ill themselves. Humans are generally "accidental" hosts who become infected through contact with these carriers or their waste. [1, 2, 3]

The primary carrier sources are categorized as follows:

1. Bats (Filoviruses)

Bats are the most widely recognized reservoir for the deadliest hemorrhagic fevers. [1]

- **Ebola Virus: Fruit bats** from the *Pteropodidae* family are considered the most likely natural reservoir.
- **Marburg Virus: The Egyptian fruit bat** (*Rousettus aegyptiacus*) has been definitively identified as the natural host, often found in caves and mines. [1, 2, 3]

2. Rodents (Arenaviruses & Hantaviruses)

Rodents are the primary source for viruses that spread through contact with their urine, droppings, or saliva. [1, 2]

- **Lassa Fever:** Specifically carried by the **multimammate rat** (*Mastomys natalensis*), which often lives in and around human homes in West Africa. [1, 2]
- **Hantaviruses:** Carried by **various species of mice and voles**, depending on the region (e.g., the striped field mouse in Asia/Europe). [1, 2]

- **South American Hemorrhagic Fevers:** Viruses like **Junín (Argentina) and Machupo (Bolivia)** are each carried by **specific local rodent species**. [1, 2]

3. **Insects and Ticks (Arthropod Vectors)**

Many hemorrhagic fevers rely on an insect or tick to "bridge" the gap between the animal reservoir and humans. [1, 2]

Mosquitoes: These are the primary vectors for **Yellow Fever, Dengue, and Rift Valley Fever**. **They often pick up the virus from infected monkeys or livestock** before biting a human.

Ticks: The **Hyalomma tick** is both a reservoir and the primary vector for **Crimean-Congo Hemorrhagic Fever**. [1, 2, 3, 4]

4. **Livestock and Primates (Incidental Hosts)**

While not the original "reservoir," these animals can become infected and serve as a significant source of transmission to humans. [1, 2]

- **Livestock: Cattle, sheep, and goats** are major sources of **Rift Valley Fever and Crimean-Congo Hemorrhagic Fever**, often infecting humans during slaughter or veterinarian work. [1, 2]
- **Non-human Primates: Monkeys and apes** are susceptible to **Ebola and Yellow Fever**. **Handling or eating "bushmeat"** from these animals is a common way for an outbreak to start in humans. [1, 2, 3, 4]

what tests are used in the actual outbreak to definitely identify ebola?

To definitely identify Ebola in the current **2026 Bundibugyo outbreak**, medical teams use specialized molecular tests because standard symptoms are too similar to malaria or typhoid. [1, 2]

The primary tools currently being used include:

- **Gold Standard: RT-PCR (Reverse Transcription-Polymerase Chain Reaction):** This is the definitive test used to confirm cases. It detects the virus's genetic material in a blood or plasma sample. Because this outbreak is caused by the rare **Bundibugyo strain**, labs must use specific assays like the [RealStar Filovirus Screen](#) or [Roche's newly developed PCR test](#), as common tests for the Zaire strain often fail to detect it. [1, 2, 3, 4, 5]
 - **GeneXpert Systems:** Widely used in African labs, these machines provide rapid results. However, **many existing GeneXpert cartridges in the DRC were designed only for the Zaire strain**. [Cepheid](#) is currently deploying updated cartridges that can specifically identify the Bundibugyo species. [1, 2]
 - **Multi-Pathogen Panels:** In some regions, clinicians use broad panels like the [Biofire Warrior Panel](#), which can test for several different hemorrhagic fevers (including Ebola, Marburg, and Lassa) simultaneously. [1]
 - **Post-Mortem Testing:** For individuals who have already died, healthcare providers use **oral swabs** to test for the virus rather than drawing blood, which reduces the risk of infection for the response team. [1]
- Important Timing: The virus typically only reaches detectable levels in the blood 3 to 10 days after symptoms begin. If a patient's first test is negative but symptoms persist, they must be tested again at least 48 hours later to be definitively cleared**

Would you like to know more about the **challenges of transporting these samples** from remote mining areas like Mongbwalu to the main labs?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

13 sites

- Diagnostics of Ebola virus - PMC - NIH

Reverse transcription–polymerase chain reaction (RT-PCR) Thanks to its specificity and sensitivity, RT-PCR is considered the Gold ...

National Institutes of Health (.gov)

- Ebola Disease Outbreak in the Democratic Republic of the Congo and Uganda | HAN

19 May 2026 — For suspected cases, request testing for BVD and other VHF from CDC (Atlanta, Georgia) or the Laboratory Response Network (LRN). ...

Centers for Disease Control and Prevention | CDC (.gov)

- Laboratory guidance and resources for Ebola disease ... - ECDC

Sampling and diagnostics * The recommended sample type for testing BDBV is whole blood or plasma for patients, and oral swab for d...

European Centre for Disease Prevention and Control

Show all

what symptoms are specific to ebola and no other infectious disease?

There are no symptoms that are exclusive to Ebola and found in no other disease. Ebola belongs to the family of Viral Hemorrhagic Fevers (VHFs), and many of its symptoms overlap with other tropical diseases like Marburg virus disease, Lassa fever, Malaria, and Typhoid fever. [1, 2, 3, 4]

Because early signs are nonspecific, clinicians often cannot distinguish Ebola from other infections without specialized laboratory testing. However, the disease typically follows a unique and rapid progression through specific phases: [1, 2, 3, 4, 5]

- **Dry Phase (Early):** Sudden onset of **fever (typically over 38°C), severe fatigue, muscle pain, and sore throat.** [1, 2]
- **Wet Phase (Mid-stage):** As the illness progresses (usually after 4–5 days), patients develop **severe watery diarrhea, persistent vomiting, and intense abdominal pain.** [1, 2]
- **Hemorrhagic Phase (Late):** Only in the most severe cases does "unexplained **bleeding**" occur, such as **bleeding from the gums, nose, or gastrointestinal tract.** [1, 3, 4, 5]
- **Rare Indicators:** Some historical outbreaks have noted **hiccups** as a discriminating sign in fatal cases, though this is not a universal diagnostic marker. [1]

Because of this overlap, medical experts at the [CDC](#) and WHO emphasize that isolation should be based on **exposure history** (contact with infected people or animals) rather than specific symptoms alone. [1]

Do you have any **specific symptoms** in mind that you've heard associated with Ebola, or would you like to see how **diagnostic tests** differ for these diseases?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

12 sites

- Ebola disease - World Health Organization (WHO)

24 Apr 2025 — Symptoms. The incubation period or interval from infection to onset of symptoms varies from 2 to 21 days. The symptoms of Ebola di...

World Health Organization (WHO)

- Signs and Symptoms of Ebola Disease - CDC

23 Apr 2024 — Key points * Ebola disease symptoms appear 2 to 21 days after someone is exposed to the virus. * "Dry" symptoms, like fever and we...

Centers for Disease Control and Prevention | CDC (.gov)

- Ebola Disease - WHO | Regional Office for Africa
Symptoms. Ebola diseases symptoms can be sudden. They include fever, fatigue, muscle, pain, headache and sore throat. This is foll...

WHO | Regional Office for Africa
Show all

You said: is the hemorrhagic phase common to any other infectious disease?

Yes, the hemorrhagic phase is characteristic of a specific group of illnesses known as Viral Hemorrhagic Fevers (VHFs). While Ebola is the most well-known, many other infectious diseases caused by different virus families follow a similar progression from initial fever to severe bleeding. [1, 2, 3]

Common infectious diseases that include a hemorrhagic phase include: [1]

- **Marburg Virus Disease:** A close relative of Ebola (Filoviridae family) that causes **nearly identical severe bleeding symptoms**, usually appearing toward the end of the first week of illness. [1, 2]
- **Lassa Fever:** Endemic to West Africa, it can cause **hemorrhaging from the gums, nose, or eyes in severe cases.** [1, 2, 3]
- **Dengue Hemorrhagic Fever (DHF):** A severe form of the mosquito-borne Dengue virus that involves **internal bleeding and a "critical phase" where plasma leaks from blood vessels.** [1]
- **Crimean-Congo Hemorrhagic Fever (CCHF):** A tick-borne disease where the **hemorrhagic phase** typically begins on the third to fifth day of illness. [1]
- **Yellow Fever:** In its "toxic stage," this virus can cause **internal bleeding and jaundice** (yellowing of the skin), which gives the disease its name. [1]
- **Hantavirus:** Certain strains, particularly those in Europe and Asia, cause **Hemorrhagic Fever with Renal Syndrome (HFRS)**, which involves **bleeding alongside kidney failure.** [1, 2]

Key Differences in Other Infections

While bleeding is a dramatic symptom, it is not always the primary cause of death in these diseases. **For many VHFs, mortality is more often caused by multi-organ failure or septic shock resulting from damaged blood vessels and extreme fluid loss.** Additionally, some non-viral infections like severe **Malaria or Leptospirosis (a bacterial infection) can also occasionally cause hemorrhagic symptoms that mimic these viruses.** [1, 2, 3, 4, 5]

Would you like to know how doctors distinguish between these diseases since their **early-stage symptoms** are so similar?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

14 sites

- Viral hemorrhagic fevers - Symptoms and causes - Mayo Clinic
12 Nov 2024 — Overview. Viral hemorrhagic (hem-uh-RAJ-ik) fevers are infectious diseases that can be life-threatening. They can damage the walls...
Mayo Clinic
- Hemorrhagic fever viruses: Pathogenesis, therapeutics, and ...
Introduction. Hemorrhagic fever viruses (HFVs) are highly infectious RNA viruses that can lead to viral hemorrhagic fever (VHF) in...
National Institutes of Health (.gov)
- About Viral Hemorrhagic Fevers - CDC
15 Apr 2024 — At a glance * Viral hemorrhagic fevers (VHFs) are a group of diseases that are caused by several distinct families of viruses. * T...

Centers for Disease Control and Prevention | CDC (.gov)

Show all

You said: yes please, but also please tell me what causes Lassa Fever also?

Because the early symptoms of viral hemorrhagic fevers (VHFs) are so similar, doctors cannot rely on symptoms alone for a diagnosis. Instead, they use a combination of **epidemiological history** and **specialized laboratory testing**. [1, 2, 3]

How Doctors Distinguish Between VHFs

Since **Ebola, Marburg, and Lassa fever all start with "flu-like" symptoms (fever, fatigue, and muscle aches)**, doctors use the following factors to differentiate them: [1, 2]

1. **Exposure History:** This is the most critical tool. Doctors ask about:
 - o **Travel:** Did the patient visit West Africa (common for Lassa) or Central/East Africa (more common for Ebola/Marburg)?
 - o **Animal Contact:** Was the patient near bats (Ebola/Marburg) or rodents/rodent droppings (Lassa)? [1, 2]
2. **Gold-Standard Lab Tests:** Definitive diagnosis requires testing in high-security laboratories:
 - o **PCR (Polymerase Chain Reaction):** This is the preferred method as it detects the virus's genetic material in the blood within days of symptoms appearing.
 - o **ELISA (Antibody testing):** This looks for specific antibodies (IgM and IgG) that the immune system produces in response to a specific virus. [1, 2, 3, 4, 5]
3. **Unique Complications:** Sometimes, specific "late-stage" symptoms provide clues:
 - o **Deafness:** About 25%–30% of Lassa fever survivors experience sudden hearing loss, which is far less common in Ebola or Marburg cases.
 - o **Mortality Rates:** Ebola and Marburg tend to have much higher fatality rates (up to 90%) compared to Lassa fever (about 1% overall, though higher in hospitalized cases). [2, 3, 4, 5]

What Causes Lassa Fever?

Lassa fever is caused by the **Lassa virus**, which is an *Arenavirus*. Unlike Ebola, which is primarily spread between humans, Lassa fever is a **zoonotic disease**, meaning it is primarily spread from animals to humans. [1, 2, 3, 4, 5]

- **The Reservoir:** The primary carrier is the **multimammate rat** (*Mastomys natalensis*). These rats live in and around human homes in West Africa. [1, 3]
- **Transmission to Humans:**
 - o **Direct Contact:** Touching or being scratched by an infected rat.
 - o **Contaminated Food:** Eating food that has been tainted by rat urine or droppings.
 - o **Aerosolization:** Breathing in tiny particles of rat waste (e.g., while sweeping a floor where rats have been). [1, 2, 3]
- **Human-to-Human Spread:** While less common than rat-to-human transmission, it can happen through direct contact with an infected person's blood, vomit, or other bodily fluids, particularly in healthcare settings without proper protection. [1]

Would you like to know about the **antiviral treatments** used for Lassa fever that are not effective for Ebola?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

17 sites

- Lassa fever - World Health Organization (WHO)
5 Dec 2024 — Diagnosis. It can be difficult to clinically distinguish Lassa fever from other infectious diseases such as malaria, typhoid fever...

World Health Organization (WHO)

- Lassa fever : symptoms, treatment, prevention - Institut Pasteur

How is Lassa fever diagnosed? It is difficult to distinguish Lassa fever from other viral hemorrhagic fevers such as Ebola and yel...

Institut Pasteur

- Lassa Fever: What healthcare providers need to know ...

27 Feb 2025 — then move on to infection control measures to consider when caring for an LF patient treatment. and finally a few words on postexp...

18m

YouTube-Centers for Disease Control and Prevention (CDC)

Show all

have there been any incidents between people and their healthcare providers during ebola outbreaks

24 May 2026

Yes, severe and deadly incidents have occurred between communities and healthcare providers during Ebola outbreaks. These have primarily taken place in conflict-ridden regions of the Democratic Republic of the Congo (DRC), often driven by community mistrust, misinformation, and disputes over safe burial protocols. [1, 2, 3]

The most prominent incidents include:

- **Fatalities and Violence:** During the 10th Ebola response in the DRC, data compiled by [Insecurity Insight](#) detailed over 480 threats and attacks. This included at least 25 health workers killed, 27 abducted, and dozens of medical facilities targeted or destroyed by arson.
- **Targeted Assassinations:** Notable direct attacks against healthcare workers resulted in multiple casualties. For example, a [World Health Organization](#) epidemiologist was shot and killed during a 2019 raid on a hospital in Butembo, and multiple vaccinators and support staff were killed in Biakato Mines.
- **Hospital Arson and Riots:** Violent clashes have erupted when families rejected official claims that a relative died from Ebola. Because the disease is highly contagious, authorities mandate safe, controlled burials—which often conflicts with local traditions. In several instances, crowds have thrown stones, attacked ambulances, and set fire to medical tents or Ebola isolation centers. [1, 2, 3, 4, 5]
The violence has been heavily documented as a major logistical and medical challenge, severely restricting the ability of medical staff to track the disease and administer vaccines safely. [1, 2]

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

7 sites

- Attacks on Health Care During the 10th Ebola Response in ...

While the majority of attacks on health care have occurred in the context of the Ebola response and in Ebola-affected areas, many ...

Insecurity Insight

- Attack On Ebola Hospital In Eastern Congo Echoes Past Violence Against Health Workers

The fire raised dark memories for veterans of DRC Ebola outbreak response in North Kivu and Ituri a few years ago, the second-larg...

Health Policy Watch

- Public health intervention amidst conflict: Violence, politics ...
Highlights. • Violence aggravated Ebola transmission, but responded to politization of medical teams. Frames of public health resi...

ScienceDirect.com
Show all

You said: what were the main reasons for these incidents?

24 May 2026

The incidents between community members and healthcare providers during Ebola outbreaks have been driven by a complex mix of political, cultural, and socio-economic factors. [1]

Main reasons for these incidents include:

- **Political Resentment and Election Exclusion:** In the Democratic Republic of the Congo (DRC), violence surged after three regions were excluded from the 2018 presidential elections due to the Ebola outbreak. This led many to believe the disease was a political fabrication used to disenfranchise local populations.
- **Deep Mistrust and Misinformation:** Widespread rumors suggested that Ebola was "fake" or a business venture created for profit by international aid agencies and the government. This institutional mistrust led to the avoidance of formal care and resistance to vaccination campaigns.
- **Cultural Clashes over Burial Protocols:** Highly infectious bodies must be buried according to strict medical protocols to prevent transmission. However, these "Safe and Dignified Burials" often conflict with traditional practices like washing and touching the deceased. Violent clashes frequently occurred when families felt their religious or cultural requirements were being disrespected by response teams.
- **Abuse of Power and Exploitation:** The sudden influx of cash and international responders created opportunities for corruption, including sexual violence and exploitation during recruitment or work processes. Some local health workers were targeted due to disputes over hiring or supplier subcontracting.
- **Weaponization of the Outbreak:** In conflict zones, armed groups sometimes used attacks on health workers as a way to gain attention or resources. The use of armed escorts by health teams often backfired, as it associated medical staff with government security forces that communities already feared. [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13]

Would you like to know more about how **Safe and Dignified Burial (SDB)** protocols were eventually adapted to improve community relations?

All responses may include mistakes. [Learn more](#)

14 sites

- Effect of a safe and dignified burial intervention on Ebola virus ...
Ebola virus is transmitted through direct and indirect contact with blood or bodily fluids, and the bodies of those who die from t...

The Lancet

- Attacks on Health Care During the 10th Ebola Response in ...
The influx of Ebola responders and associated cash flows allowed for an abuse of power by intermediaries and managers and exposed ...

ReliefWeb

- How the fight against Ebola became part of the conflict in eastern DRC
Within this context, the payments to Congolese security forces had two critical consequences. First, they undermined the most impo...

ReliefWeb
Show all

You said: have there been any incidents between people and their healthcare providers during marburg outbreaks

24 May 2026

While incidents between the public and healthcare providers during Marburg outbreaks have occurred, they are generally less frequent and less violent than those documented during large-scale Ebola epidemics in conflict zones. Historically, friction has stemmed from fear, mistrust, and cultural clashes over medical protocols. [1]

Key incidents and documented tensions include:

- **Hospital Avoidance (Angola, 2004–2005):** During the largest Marburg outbreak on record, widespread fear led many people to view hospitals as the source of the disease rather than a place for cure. Healthcare providers worked under extremely difficult conditions, often facing hostility from families who believed that admitting a relative to a treatment center was a "death sentence."
- **Safe and Dignified Burial (SDB) Disputes:** Similar to Ebola, Marburg requires strict burial protocols because bodies remain highly infectious. In recent outbreaks, such as those in [Ethiopia \(2025\)](#) and [Tanzania \(2023\)](#), unsupervised or traditional burials were identified as major transmission drivers. Tension often arises when health teams intervene to prevent traditional mourning rituals, though recent responses have increasingly used Community Health Workers (CHWs) to mediate these interactions and prevent violence.
- **Occupational Risk and Stigma:** Healthcare workers are often the primary victims of Marburg outbreaks due to [nosocomial \(hospital-acquired\) transmission](#). In the [2024 Rwanda outbreak](#), the majority of cases were healthcare workers in intensive care units. This high infection rate can sometimes lead to [stigma from the community](#), as people may fear that health staff are carriers of the virus.
- **Rumors and Misinformation:** Public health agencies like the WHO and [Africa CDC](#) emphasize "Risk Communication and Community Engagement" (RCCE) specifically to counter **rumors that healthcare providers are intentionally spreading the disease or profiting from the response**. [1, 2, 3, 4]

Recent Trends:

In recent outbreaks (Rwanda 2024, Ethiopia 2025/2026, and Equatorial Guinea 2023), government and international responses have focused on [high-level engagement](#) with community leaders to prevent the kind of active violence seen in previous Ebola responses. [1, 2, 3, 4]

Would you like to know more about the **specific experimental vaccines** being used to protect healthcare workers in these outbreaks?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)



7 sites

- Marburg virus disease - World Health Organization (WHO)
Once introduced in the human population, Marburg virus can spread through human-to-human transmission via direct contact (through ...



World Health Organization (WHO)

- CDC warns doctors to watch for Marburg virus | What to know

The CDC has issued a health advisory urging physicians to be highly vigilant for cases of the Marburg virus following recent overs...

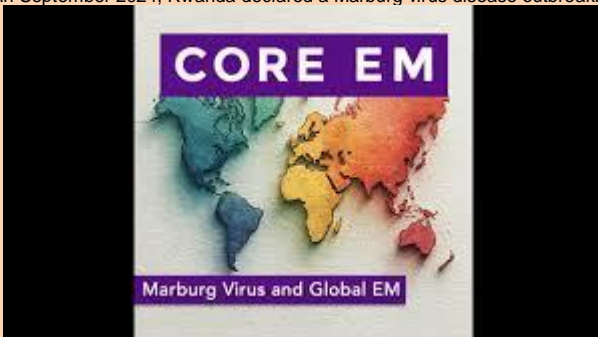


1:30

YouTube-11Alive

- Episode 215: Marburg Virus and Global EM

In September 2024, Rwanda declared a Marburg virus disease outbreak. Marburg virus is in the same family as Ebola and is a viral h...



4m

YouTube-Core EM
Show all

You said: were there such incidents with the new ebola outbreak in Africa (2026)?

Yes, several violent incidents have been reported during the current **2026 Ebola outbreak** in the Democratic Republic of the Congo (DRC). These attacks have primarily targeted medical facilities and response teams in the eastern provinces, driven by **a lack of trust and clashes over burial protocols**. [\[1, 2, 3\]](#)

Specific reported incidents include:

- **Storming of Health Centers:** In May 2026, residents reportedly stormed the **Rwampara health centre**. The group demanded the bodies of deceased relatives for traditional burials, despite warnings from medical staff that the bodies were highly contagious. [\[1, 2\]](#)
- **Arson of Medical Tents:** An isolation tent at a hospital in **Mongbwalu**, provided by **Doctors Without Borders (MSF)**, was intentionally set on fire following the death of a patient suspected of having Ebola. [\[1\]](#)
- **Attacks on Clinics:** Reports indicate that **attacks on medical facilities have intensified, occasionally forcing patients and staff to flee for safety**. [\[1\]](#)

Factors Fueling the Tension

This outbreak is particularly volatile because it is caused by the **rare Bundibugyo strain**, for which there is currently **no licensed vaccine** or specific treatment. The lack of medical tools, combined with the region's ongoing **armed conflict**, has led to: [\[1, 2, 3, 4\]](#)

- **Deep Mistrust:** Some community members view the restrictive health measures—such as the **prohibition of gatherings over 50 people—as politically motivated or unnecessary**. [\[1, 2\]](#)
 - **Security Obstacles:** The **WHO** has noted that insecurity in the northeast has hindered emergency operations, making it difficult for health workers to maintain consistent **contact tracing**. [\[1, 2, 3\]](#)
 - **Stigma and Fear:** High infection rates among medical staff—with at least **16 health workers** reportedly infected as of early June—have increased fears that hospitals are hubs for the virus. [\[1, 2\]](#)
- Would you like to know more about the **Bundibugyo strain** and why existing Ebola vaccines are not effective against it?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

11 sites

- Epidemic of Ebola Disease caused by Bundibugyo virus in the ...
17 May 2026 — Unusual clusters of community deaths with symptoms compatible with Bundibugyo virus disease (BVD) have been reported across severa...
- World Health Organization (WHO)
Why are people attacking Ebola clinics? - NPR
29 May 2026 — Focus on death and burials Although the DRC has roughly 450 tribes and lots of different death customs, Rukengeza says, it is, gen...
- NPR
DR Congo Ebola outbreak: Nurses discharged after full recovery
1 Jun 2026 — As of Sunday, there were 210 confirmed cases of infection reported in the country, with 17 confirmed deaths. Nearly 350 suspected ...

UN News
[Show all](#)

You said: How many of the medical staff died among the infected ones?

As of early June 2026, there have been at least **seven reported deaths among medical staff** during the current Ebola outbreak in the Democratic Republic of the Congo (DRC) and Uganda. [\[1, 2\]](#)

According to reports from health authorities and international monitoring agencies:

- **DRC:** At least **four healthcare workers** have died in the eastern provinces of Ituri and North Kivu. One of the early victims was a [healthcare professional](#) believed to be the index case (the first identified patient). [1, 2, 3]
- **Aid Workers:** On May 26, the British Medical Journal reported the deaths of **three Red Cross workers** in the DRC. [1]
- **Infections:** In total, at least **21 healthcare workers** have contracted the virus across both countries—16 in the DRC and five in Uganda. [1, 2]
- **Recoveries:** In a positive development, [UN News](#) reported on June 1 that **four nurses and one laboratory worker in the DRC** were discharged after fully recovering from the virus. [1]
The high infection rate among medical staff has been attributed to a [critical shortage](#) of Personal Protective Equipment (PPE) and initial delays in identifying the rare [Bundibugyo strain](#), which has no licensed vaccine. [1, 2, 3]

Would you like more information on the **international response efforts** to supply PPE to hospitals in the affected health zones?

what are the major incidents between people and health care providers in the history of ebola outbreaks?

Historically, major incidents between the public and healthcare providers during Ebola outbreaks have been driven by community mistrust, political conflict, and clashing burial traditions. These incidents have ranged from social ostracization to fatal targeted attacks. [1, 2]

1. The 10th Ebola Outbreak (DRC, 2018–2020)

This outbreak **saw the most documented violence against health workers in history due to its location in an active conflict zone.** [1, 2]

- **Over 480 Attacks:** Data from [Insecurity Insight](#) recorded at least 483 attacks on healthcare, resulting in the deaths of 25 health workers and the abduction of 27 others. [1, 2]
- **Arson of Treatment Units:** In February 2019, Ebola treatment centers in **Butembo** and **Katwa** were partially destroyed by arson, **forcing organizations like Doctors Without Borders (MSF) to suspend operations in those areas.** [1]
- **Assassination of WHO Staff:** Dr. Richard Mouzoko, a World Health Organization epidemiologist, was shot and killed during an attack on a hospital in Butembo in April 2019. [1]

2. Current 2026 Outbreak (DRC & Uganda)

Escalating violence has significantly hampered the ongoing response to the rare **Bundibugyo strain.** [1, 2]

- **Storming Health Centers:** Residents recently stormed the **Rwampara health centre to forcibly take bodies of relatives for traditional burials.** [1, 2]
- **Burial Team Attacks:** In June 2026, health workers attempting to perform a safe burial in eastern DRC were attacked by angry residents, who forced the teams to [abandon the coffin.](#) []
- **Facility Destruction:** Multiple medical tents, including those in **Mongbwalu**, have been set on fire because of **rumors that health facilities were causing more deaths than they were preventing.** [1, 2, 3]

3. West African Epidemic (2014–2016)

While widespread, incidents here were often characterized by fear and the breakdown of healthcare systems. [1, 2, 3]

- **The Womey Massacre (Guinea, 2014):** Eight members of an Ebola education team, including health officials and journalists, were killed by villagers in the town of **Womey who feared the team was spreading the virus.** [1, 2]
- **Social Ostracization:** Frontline health workers faced intense discrimination and [mental health challenges](#) from their own communities, who often feared they were carriers of the disease. [1, 2, 3]

4. Early Outbreaks (1976–1990s)

In the earliest outbreaks, incidents were less about violence and more about a lack of medical understanding. [1]

- **Hospital-Acquired Transmission:** In the 1976 Zaire outbreak, the use of contaminated needles in clinics led to high infection rates among medical staff, fueling the perception that hospitals were "death traps". [1, 2]



what poisons can cause internal bleeding in humans

Internal bleeding in humans can be caused by various poisons, most commonly those that interfere with the body's ability to clot blood or those that cause direct physical damage to tissues and organs.

Anticoagulant Substances

Certain substances function by interfering with the biological processes that allow blood to clot. By inhibiting the action of Vitamin K, these agents prevent the synthesis of essential clotting factors, which can result in spontaneous and widespread internal hemorrhaging.

National Institutes of Health (.gov) +4

- **Clinical Anticoagulants:** When used outside of strict medical supervision or in cases of accidental overdose, medications designed to thin the blood can cause life-threatening bleeding in the gastrointestinal tract or the brain.
- **Long-Acting Rodenticides:** Many modern pest control products contain highly potent anticoagulants. These are designed to remain active in a biological system for an extended period, leading to severe internal bleeding that may not manifest symptoms until several days after exposure.

National Institutes of Health (.gov) +4

Heavy Metals and Corrosive Irritants

- **Metallic Toxins:** Exposure to certain heavy metals can cause acute damage to the lining of the digestive system. This often results in severe gastrointestinal hemorrhage, accompanied by symptoms such as intense abdominal pain and vomiting.

- **Corrosive Agents:** Strong acids and alkaline compounds, frequently found in industrial and household cleaning products, cause immediate chemical burns. These burns can lead to the perforation of internal organs and significant internal bleeding.

Poison Control +4

Biological and Environmental Toxins

- **Plant-Derived Toxins:** Certain naturally occurring toxins found in specific seeds or beans can cause profound cellular damage. Depending on the method of exposure, this can lead to hemorrhaging in the lungs, intestines, or other major organs.
- **Toxic Fungi:** Ingestion of certain poisonous mushrooms can lead to acute organ failure. As the liver or kidneys cease to function, the body loses its ability to regulate blood consistency, leading to secondary bleeding complications.

Mayo Clinic +3

Industrial Solvents and Pesticides

- **Chemical Solvents:** Various industrial chemicals can cause rapid degradation of the liver. Because the liver is responsible for producing the proteins that clot blood, this damage can lead to systemic bleeding issues.
- **Pesticide Exposure:** Certain classes of pesticides primarily target the nervous system but have also been associated with complications involving the intestinal lining.

Springer Nature Link +3

If there is a concern regarding a potential exposure, it is critical to contact emergency services or a poison control center immediately for professional medical guidance.

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)
32 sites

- Upper gastrointestinal hemorrhage caused by superwarfarin ...

DISCUSSION. Superwarfarins, a class of rodenticides with brodifacoum and bromadiolone as their representative, are long acting ant...

National Institutes of Health (.gov)

- Acute liver failure - Symptoms and causes - Mayo Clinic

Herbal supplements. Herbal drugs and supplements, including kava, ephedra, skullcap and pennyroyal, have been linked to acute live...

Mayo Clinic

- Rodenticide poisoning leading to cerebral hemorrhage: A case report

Abstract * Rationale: Anticoagulant rodenticides (ARs) are a substantial fraction of murine types. AR poisoning causes bleeding fr...

National Institutes of Health (.gov)

Show all

what would happen in the body if a **higher dose of anticoagulant** would be ingested (how would this impact the macrophages, the blood cells, the endothelial cells, the liver)?

An overdose of an anticoagulant disrupts the body's delicate balance between bleeding and clotting, affecting several cell types and organs simultaneously.

Impact on Cells and Tissues

- **Macrophages:** These immune cells can actually be affected in a few ways. Some anticoagulants, like **heparin**, have been shown to have an **anti-inflammatory effect** on macrophages, potentially reducing their production of inflammatory signals. However, in the event of an overdose that causes internal bleeding, macrophages will be rapidly recruited to the sites of injury to begin the process of clearing dead cells and debris.

- **Blood Cells:**
 - **Platelets:** While anticoagulants don't typically destroy platelets directly, **certain types (like heparin)** can trigger a rare but dangerous immune response called **Heparin-Induced Thrombocytopenia (HIT)**, where the body's own immune system attacks and depletes its platelet supply.
 - **Red Blood Cells:** A high dose of some laboratory **anticoagulants (like EDTA)** can cause red blood cells to **swell or even burst (lysis)**. Clinically, an overdose leads to a massive loss of these cells through internal hemorrhaging into organs, joints, or the gastrointestinal tract.
- **Endothelial Cells:** These cells line your blood vessels and usually work with anticoagulants to keep blood flowing. While the drugs themselves don't typically "rupture" the vessels, an overdose can **impair the endothelium's ability to switch to a 'pro-clotting' state** when a small injury occurs. This means even minor, everyday wear-and-tear on your vessels can lead to significant, uncontrolled leaks.

Cleveland Clinic +7

Impact on the Liver

The liver is the primary "factory" for the proteins that help your blood clot. An anticoagulant overdose impacts it in two major ways:

1. **Production Shutdown:** **Anticoagulants like warfarin** work by blocking the liver's ability to use Vitamin K. In an overdose, the liver can no longer produce four essential clotting factors (II, VII, IX, and X), effectively "starving" the blood of its ability to thicken.
2. **Direct Damage:** While rare, extremely high doses of certain oral anticoagulants can cause **drug-induced liver injury (DILI)** or acute liver failure. This is often an idiosyncratic (unpredictable) reaction where the drug or its metabolites become toxic to the liver cells (hepatocytes).

National Institutes of Health (.gov) +4

Would you like to know more about the **specific antidotes** used by doctors to reverse these effects during an emergency?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)
13 sites

- **Anticoagulants (Blood Thinners) - Cleveland Clinic**

Type I: This type of HIT causes a slight decrease in platelets, a type of blood cell that is instrumental in blood's ability to cl...

Cleveland Clinic

- **Warfarin - LiverTox® - NCBI Bookshelf - NIH**

Liver injury due to warfarin therapy is rare, but clinically apparent acute liver injury attributable to it has been reported. Liv...

National Institutes of Health (.gov)

- **Liver injury from direct oral anticoagulants - PMC - NIH**

ARTICLE HIGHLIGHTS * Research background. Drug-induced liver injury (DILI) can be caused by any prescribed drug and is a significa...

National Institutes of Health (.gov)

Show all

are the lymphocytes affected also?

Yes, **lymphocytes are affected**, though the impact depends heavily on the specific type of anticoagulant and the dose. Unlike red blood cells, which may simply be lost through bleeding, lymphocytes experience complex changes in their movement, survival, and function.

ScienceDirect.com +2

1. Changes in Circulation (The "Homing" Effect)

- **Heparin-Induced Lymphocytosis:** High doses of **heparin** can actually cause the number of circulating lymphocytes in the blood to **increase**. This isn't because more are being made, but because heparin blocks their ability to "home" or exit the blood into the lymph nodes. They essentially get "stuck" in the highway of the bloodstream.
- **Warfarin-Induced Decrease:** Conversely, high doses of **warfarin** have been observed to **decrease total lymphocyte counts** in some studies, alongside other white blood cells.

ScienceDirect.com +4

2. Suppression of Immune Function

- **Inhibited Activation:** High levels of anticoagulants can act as **immunomodulators**. Warfarin, for example, has been shown to inhibit the activation and proliferation of **T-lymphocytes** by interfering with Interleukin-2 (IL-2) production.
- **Cellular Damage:** Extremely high doses of warfarin have been reported to cause **mitochondrial damage** in lymphocytes, leading to impaired cell viability and even lysis (rupture) of the cells.

National Institutes of Health (.gov) +3

3. Indirect Impact via Inflammation

- **Anti-inflammatory Effects:** Anticoagulants like heparin can reduce the ability of lymphocytes to adhere to and move through the **endothelial walls** (the lining of your blood vessels) to reach sites of infection or injury. While this can be beneficial in treating inflammatory diseases, an overdose could potentially weaken the body's immediate localized immune response.

ScienceDirect.com +2

Summary of Cellular Impact during Overdose

Cell Type	Primary Impact of Anticoagulant Overdose
Lymphocytes	Blocked migration (heparin) or reduced activation and counts (warfarin).
Macrophages	Recruitment to bleeding sites and reduced inflammatory signaling [Previous turn].
Red Blood Cells	Massive loss through internal bleeding; potential lysis (bursting) at extreme doses [Previous turn].
Endothelial Cells	Loss of "sealant" function, making vessels prone to leaking from minor stress [Previous turn].

Would you like to explore how these cellular changes contribute to **specific symptoms**, such as bruising or organ dysfunction?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)
11 sites

- The effect of unfractionated heparin on circulating lymphocyte ...

Introduction. Heparin is used for inhibition of blood clotting but also has effects on inflammation. Heparin has been reported to ...

ScienceDirect.com

- Immunomodulating activity of heparin - PubMed

Abstract. Aside from its well-known anticoagulant action, heparin has many other biologic effects. Recent data emphasize the immun...

National Institutes of Health (.gov)

- Heparin-induced Leukocytosis Requires 6-O-Sulfation and Is ... - PMC

As shown in Fig. 3A, the numbers of CFSE+ lymphocytes homing to the peripheral and mesenteric lymph nodes were reduced by ~90% in ...

how these cellular changes contribute to specific symptoms, such as bruising or organ dysfunction?

An anticoagulant overdose leads to specific symptoms like bruising and organ dysfunction because the cellular changes discussed previously essentially "break" the body's natural defense systems.

1. Bruising: A Failure of "Plugging and Sealing"

Bruising is the most common symptom of an overdose. At the cellular level, this happens because:

LITFL • Life in the Fast Lane +1

- **Capillary Leaking:** Small blood vessels (capillaries) are constantly experiencing tiny breaks from everyday movements. Normally, platelets and clotting factors seal these immediately.

British Heart Foundation +1

- **Clotting Failure:** In an overdose, the clotting factors are depleted (liver shutdown) or blocked (direct anticoagulants). Those tiny breaks don't seal, allowing blood cells to leak into the surrounding tissue.

British Heart Foundation +3

- **The Discolouration:** The leaked red blood cells eventually die and are broken down by **macrophages**. This breakdown process turns the hemoglobin into biliverdin and bilirubin, creating the changing colors of a bruise.

British Heart Foundation

2. Organ Dysfunction: "Slow Smothering" or Acute Damage

Organ failure occurs when the internal bleeding and cellular dysfunction become widespread:

- **Hypoxia (Oxygen Starvation):** As blood leaks out of vessels, there are fewer **red blood cells** available to carry oxygen to vital organs like the brain, kidneys, and liver. Over time, this leads to organ starvation and failure.

AccessMedicine

- **Compression Damage:** In some cases, blood can pool inside an organ or in the spaces around it (like the brain or joint spaces). This pressure physically compresses the organ, cutting off its own blood supply.

National Institutes of Health (.gov) +1

- **Endothelial Breakdown:** The **endothelial cells** (lining the vessels) lose their ability to act as a barrier. This makes blood vessels "porous," leading to fluid leaking into tissues, which can cause swelling and further impair organ function.

ScienceDirect.com +2

- **Rare Tissue Death (Skin Necrosis):** Specifically with **warfarin**, an overdose can cause a paradoxical effect where small blood clots form *before* the blood thins. These clots block blood flow to the skin and fat, causing painful skin death (necrosis).

National Institutes of Health (.gov) +2

3. Immune Compromise

The suppression of **lymphocytes** and **macrophages** during an overdose means that even if a person survives the initial bleeding, their body is less able to fight off infections or heal the damage caused by the hemorrhage.

Frontiers

Would you like to know about the **emergency treatments** used to reverse these effects, such as Vitamin K or Prothrombin Complex Concentrates (PCC)?

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)
13 sites

- Can blood-thinners make you bruise more easily?

24 Apr 2026 — Can blood-thinners make you bruise more easily? Medicines like warfarin, apixaban, aspirin and clopidogrel stop blood from clottin...

British Heart Foundation

- Side effects: Anticoagulant medicines - NHS

Contents. ... A possible side effect of anticoagulants is excessive bleeding (haemorrhage), because these medicines increase the t...

nhs.uk

- Warfarin-induced skin necrosis: a narrative review of clinical ... - PMC

12 Sept 2025 — Figure 2. ... The effect of warfarin on protein C and protein S. The initiation of warfarin therapy leads to a rapid decline in pr...

National Institutes of Health (.gov)

Show all

are the eyes affected also?

Yes, the eyes are highly susceptible to the effects of an anticoagulant overdose. Because the eyes contain some of the body's most delicate and densely packed micro-vessels, the loss of clotting ability can lead to several visible and dangerous conditions.

National Institutes of Health (.gov) +2

Visible Changes (External)

- **Subconjunctival Hemorrhage:** This is the most common ocular symptom. It appears as a bright red patch or a completely bloodshot "white" of the eye. While often painless, an overdose can cause these bleeds to be much more extensive or recurrent.

Mayo Clinic +5

- **Jaundice (Yellowing):** If the overdose leads to liver failure or significant breakdown of leaked blood cells, the whites of the eyes (sclera) may turn yellow.

nhs.uk

- **Bloody Tears (Haemolacria):** In rare and extreme cases of systemic anticoagulant poisoning (often seen with rodenticide ingestion), blood can actually leak into the tear ducts, causing "tears of blood".

Emergency Home Vet +1

Internal Damage (Vision-Threatening)

- **Intraocular Hemorrhage:** Bleeding can occur deep inside the eye, including the **vitreous** (the gel-like center), the **retina** (the light-sensing back), or the **choroid**. This can cause a sudden loss of vision or the appearance of "floaters" and dark spots.

American Academy of Ophthalmology +2

- **HypHEMA:** Blood may pool in the front chamber of the eye (between the cornea and iris). This can block vision and significantly increase pressure inside the eye.

National Institutes of Health (.gov) +1

- **Retrobulbar Hemorrhage:** Bleeding can occur *behind* the eyeball within the eye socket (orbit). This is a **medical emergency** because the pooling blood can compress the optic nerve, potentially leading to permanent blindness within hours if not treated.

Summary of Eye Impact

Symptom	Description	Severity
Red Spot (Subconjunctival)	Broken tiny vessels on the surface.	High visibility, usually low risk.
Vitreous/Retinal Bleed	Bleeding inside the eye globe.	High risk ; can cause vision loss.
HypHEMA	Blood pooling in the front of the eye.	High risk ; affects vision and pressure.
Retrobulbar Hemorrhage	Pressure-building bleed behind the eye.	Emergency ; risk of permanent blindness.

Would you like to know how medical professionals **diagnose** these internal eye bleeds during a suspected overdose?

This is for informational purposes only

is aspirin an anticoagulant

25 June 2026

No, aspirin is not an anticoagulant. While both are types of "blood thinners," they work in fundamentally different ways: [\[1, 2, 3, 4\]](#)

- **Antiplatelet (Aspirin):** Aspirin stops blood cell fragments called platelets from clumping together to form a clot. It is typically used to prevent heart attacks and strokes. [\[1, 2\]](#)
- **Anticoagulant:** These medications (like warfarin, apixaban, or heparin) slow down the clotting process by targeting specific proteins in the blood (the "clotting cascade"). They are often used to treat or prevent deep vein thrombosis (DVT) or strokes in patients with atrial fibrillation. [\[1, 2, 3, 4\]](#) For a full breakdown of how these medications differ and what they are used for, you can check out the [Cleveland Clinic Health Essentials](#) guide on blood thinners.

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

6 sites

- Is aspirin an anticoagulant? - Walgreens The Thread
No, aspirin is not an anticoagulant. Anticoagulants are medicines that delay the clotting of blood. They are used to help reduce t...

Walgreens The Thread

- **Is Aspirin a Blood Thinner? What You Should Know - GoodRx**
GoodRx is NOT insurance. So how does aspirin thin blood? Aspirin is an antiplatelet medication. This means that aspirin blocks blo...

GoodRx

- **Is Aspirin a Blood Thinner? - Cleveland Clinic Health Essentials**
Is Aspirin a Blood Thinner? Yes, aspirin is a commonly prescribed blood thinner that can lower cardiovascular disease risk — but i...

Cleveland Clinic Health Essentials

Show all

You said: can chloroquine cause bleeding?

25 June 2026

Yes, chloroquine can cause unusual bleeding and bruising, though it is a rare side effect. Unlike aspirin, chloroquine does not directly thin the blood, but it can occasionally suppress the bone marrow where blood cells are produced. [1, 2, 3, 4]

How Chloroquine Causes Bleeding

According to the [Mayo Clinic drug guide](#), chloroquine can cause serious blood disorders. [1, 2]

- **Thrombocytopenia:** Chloroquine can cause a drastic drop in blood platelets (the cells responsible for clotting). When platelets are too low, minor injuries can cause severe bleeding. [1, 2, 3, 4, 5]
- **Bone Marrow Suppression:** In rare cases, the drug causes bone marrow failure or pancytopenia (a severe drop in red blood cells, white blood cells, and platelets combined). [1]

Signs of Bleeding to Watch For

If you or someone you know is taking chloroquine, seek medical attention right away if you notice any of these warning signs: [1]

- **Skin changes:** Frequent, unexplained bruising or tiny red spots on the skin (petechiae).
- **Bodily fluids:** Blood in the urine, dark/tarry stools, or regular nosebleeds.
- **Oral bleeding:** Bleeding gums when brushing teeth or eating. [1, 2, 3, 4]

Drug Interactions

The risk of bleeding increases significantly if chloroquine is taken alongside actual blood thinners, aspirin, or Nonsteroidal Anti-inflammatory Drugs (NSAIDs) like ibuprofen. [1, 2, 3]

Are you **currently taking chloroquine**, or looking into its side effects for a **specific medical condition**? Knowing if you are pairing it with **other medications** can help me provide more tailored information.

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

13 sites

- Chloroquine (oral route) - Side effects & dosage - Mayo Clinic
Incidence not known * Anxiety. * attempts at killing oneself. * back, leg, or stomach pains. * black, tarry stools. * bleeding gum...

Mayo Clinic

- Chloroquine (Aralen) - Uses, Side Effects, and More - WebMD
Stop taking chloroquine and get help right away if you have any of the following symptoms of blood disorders. * Fever. * Shortness...

WebMD

- Chloroquine Side Effects: Common, Severe, Long Term
Hematologic * Rare (0.01% to 0.1%): Bone marrow failure/depression (including aplastic anemia, agranulocytosis, pancytopenia, thro...

Drugs.com

[https://viroliegy.com/2022/09/26/the-ebola-virus-part-1/The Ebola "Virus" Part 1](https://viroliegy.com/2022/09/26/the-ebola-virus-part-1/The Ebola)



A comment was recently left on one of my posts that caught my attention:

If you s are all so confident viruses don't exist, head over to an African country and expose yourself to ebola. Let me know how that works out for you. I'll be waiting. Then again you're all too to prove me wrong.

For some reason, there are people out there who seem to believe that in order for one to prove "viruses" do not exist (a proving non-existence logical fallacy), one must either inject themselves with "infected" blood or expose themselves to "infected" individuals. These people feel that dissecting the studies which have been presented as evidence for the existence of these fictional pathogenic entities is just not convincing enough. In this particular instance, I was told to go to Africa and expose myself to an Ebola patient. This person proudly declared "Prove to me I'm wrong by infecting yourself." Of course if I were to become sick after exposing myself to an Ebola patient, that would in no way prove a "virus" was the cause as environmental toxins, stress and fatigue from travel, changes in nutritional and sleeping habits, etc. could all be potential factors leading to dis-ease. If I were not to get sick, these people will fall back on rescue devices such as naturally occurring antibodies or asymptomatic infection as the likely explanation for any lack of dis-ease. It would ultimately be a fruitless exercise.

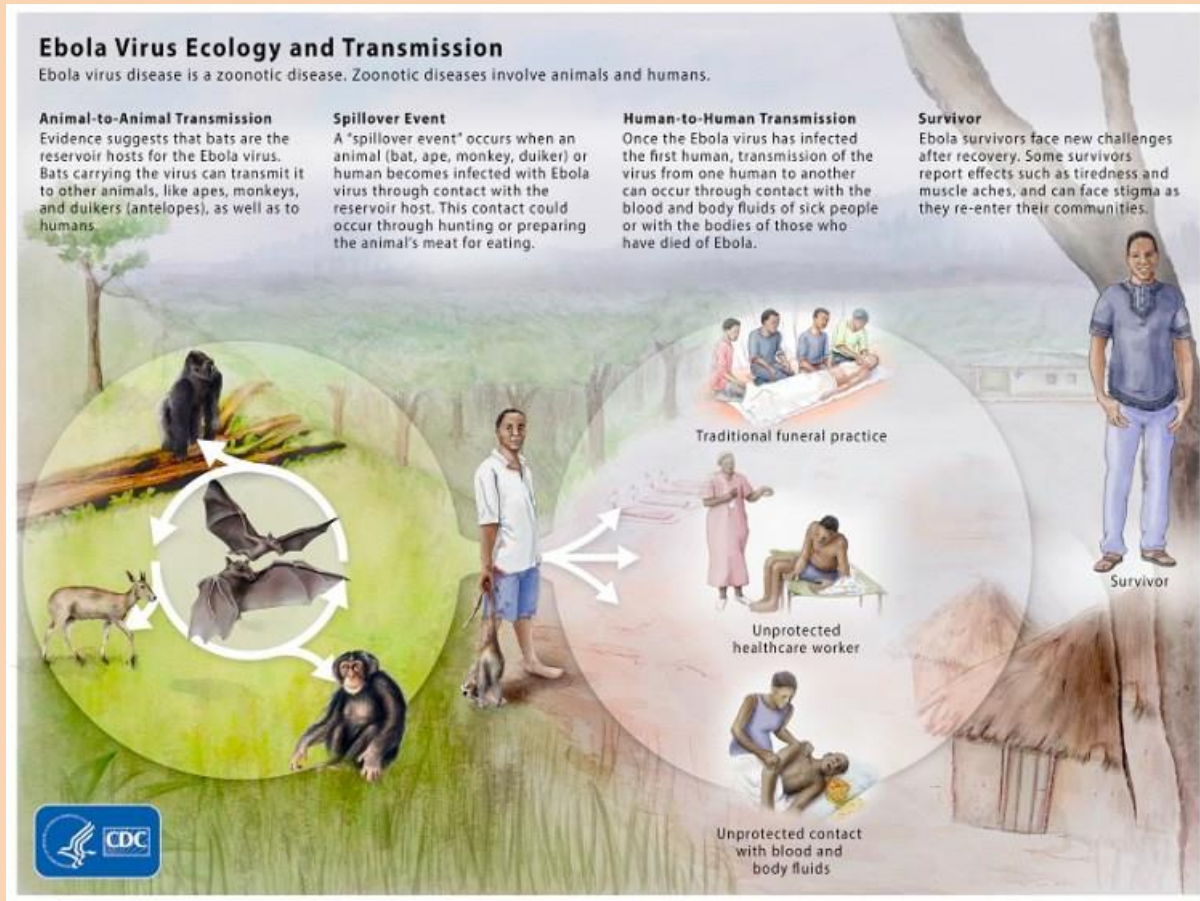
Unfortunately for those who make illogical demands like the example above, the burden of proof is on the one who makes the positive claim. If one states that a "virus," exists, the onus is on that person to prove the existence of that particular "virus." The best way to do so would be by presenting the foundational studies relating to said "virus." It is interesting to me that these people never look to the scientific literature to try and establish that these "viruses," such as Ebola, were proven to exist in the first place as claimed. However, I understand their frustration in trying to present the original studies as proof as the scientific evidence simply does not exist anywhere within any of these papers.

Regardless, Ebola is one of what I typically see as the three main “viruses” people love to challenge those who critique virology with, the other two being HIV and rabies which I’ve covered previously. According to their demands, in order to disprove virology, we must overcome the hurdle of the “Big Three” by physically exposing ourselves to sickened patients. Unfortunately, while I would love a safari, dropping everything and flying to Africa in order to expose myself to imaginary “viruses” isn’t really a realistic option for me at this moment in time. However, why incur travel expenses when I can just pick apart the pseudoscientific evidence used as proof for the existence of said “virus” (that these people are unwilling to look at and submit themselves) instead?

This breakdown of the Ebola fraud will be presented in two parts. Part one is focused on the “isolation” of the Ebola “virus” by three individual groups of researchers in 1976. I have provided the three papers submitted, one from each group, to see if there is any scientific evidence for an Ebola “virus” contained within them. Did the researchers adhere to the scientific method? Was there any attempt to properly purify and isolate the particles assumed to be the Ebola “virus” directly from the fluids of a sick host? Were these purified particles used to expose a susceptible host in a natural way? Were the electron microscope images only of the assumed “viral” particles and nothing else? Did the researchers carry out the proper control experiments? We shall find out.

The second part explores the inconsistencies in the WHO’s 23-page report released in 1978 summarizing the research conducted in 1976. There are many different holes within the narrative that require further examination. It will also look at other potential causes for the symptoms experienced by the patients that were bizarrely overlooked by the WHO.

What is the Ebola “Virus?”



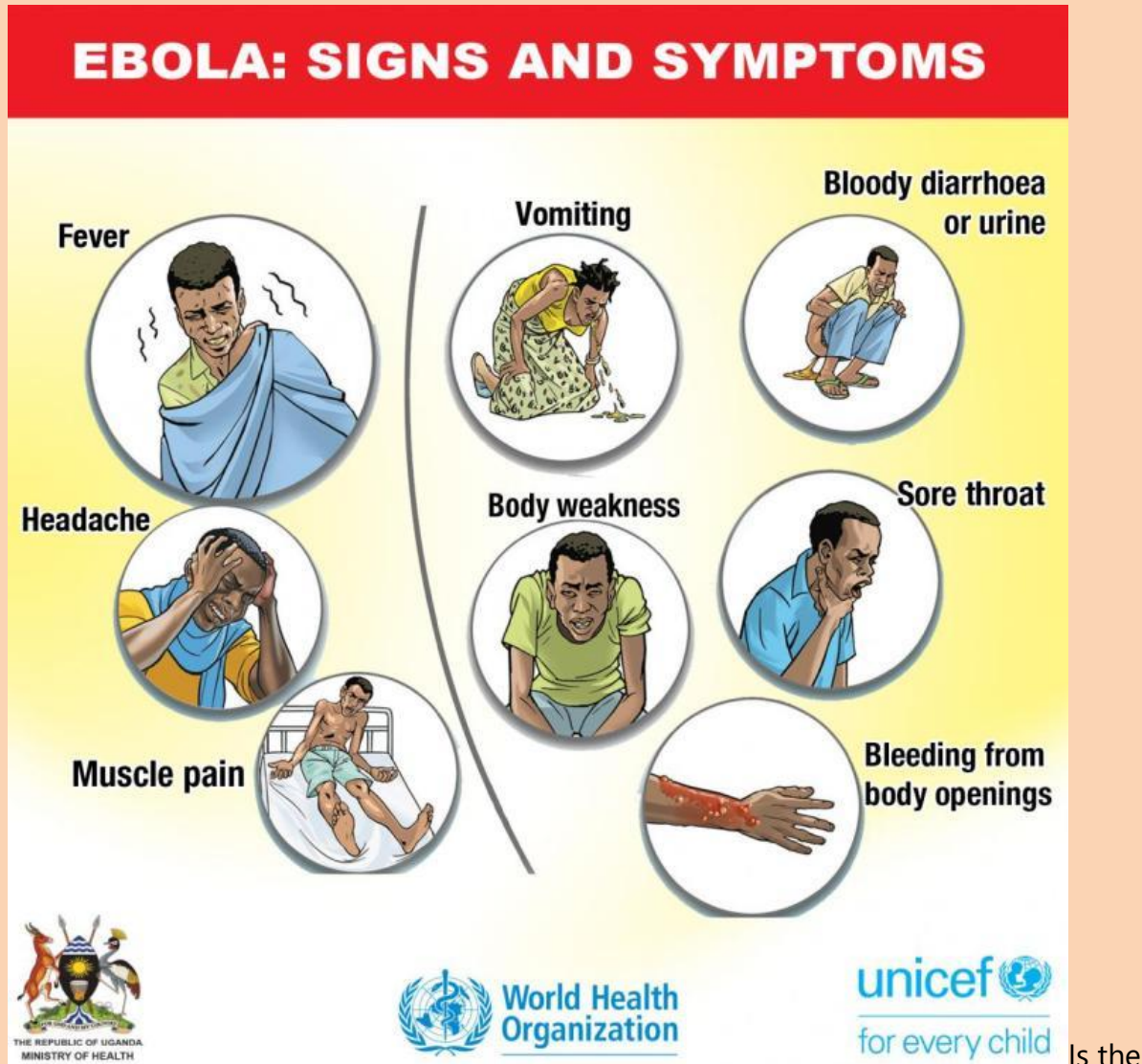
According to the CDC, the Ebola "virus" was discovered in Zaire in 1976 (after an outbreak of haemorrhagic fever). The "virus" occasionally likes to make appearances from time to time as outbreaks in Africa. It is said to first spread to humans through contact with an infected animal. After that, the "virus" transfers human-to-human by way of contact with an infected person's bodily fluids:

"Ebola virus was first discovered in 1976 near the Ebola River in what is now the Democratic Republic of Congo. Since then, the virus has been infecting people from time to time, leading to outbreaks in several African countries. Scientists do not know where Ebola virus comes from. Based on similar viruses, they believe EVD is animal-borne, with bats or nonhuman primates being the most likely source. Infected animals carrying the virus can transmit it to other animals, like apes, monkeys, duikers and humans.

The virus first spreads to people through direct contact with the blood, body fluids and tissues of animals. Ebola virus then spreads to other people through direct contact with body fluids of a person who is sick with or has died from EVD. This can occur when a person touches these infected body fluids or objects that are contaminated with them. **The virus then gets into the body through broken skin or**

mucous membranes in the eyes, nose, or mouth. People can get the virus through sexual contact with someone who is sick with or has recovered from EVD. The virus can persist in certain body fluids, like semen, after recovery from the illness.”

<https://www.cdc.gov/vhf/ebola/about.html>



Is the image of the guy dropping a deuce on the floor really necessary WHO?

According to the WHO, we find that this “rare but severe, often fatal illness” can only be spread by those who are symptomatic. The Ebola “virus” is associated with a list of non-specific symptoms, and is often confused in diagnosis with many other diseases and even pregnancy(!):

Symptoms

The incubation period, that is, the time interval from infection with the virus to onset of symptoms, is from 2 to 21 days. **A person infected with Ebola cannot spread the disease until they develop symptoms.**

Symptoms of EVD can be sudden and include:

- Fever
- Fatigue
- Muscle pain
- Headache
- Sore throat

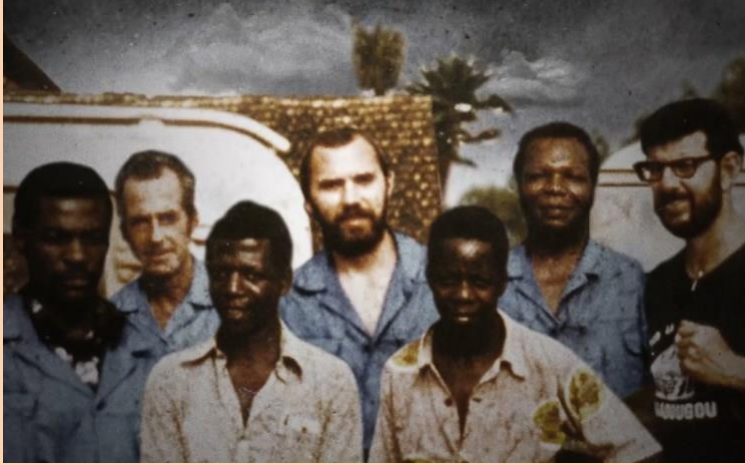
This is followed by:

- Vomiting
- Diarrhoea
- Rash
- Symptoms of impaired kidney and liver function
- In some cases, both internal and external bleeding (for example, oozing from the gums, or blood in the stools).
- Laboratory findings include low white blood cell and platelet counts and elevated liver enzymes.

Diagnosis

It can be difficult to clinically distinguish EVD from other infectious diseases such as malaria, typhoid fever and meningitis. Many symptoms of pregnancy and Ebola disease are also quite similar. Because of risks to the pregnancy, pregnant women should ideally be tested rapidly if Ebola is suspected.

https://www.who.int/news-room/fact-sheets/detail/ebola-virus-disease?gclid=Cj0KCQjwj7CZBhDHARIsAPPWv3cNnRDMQ6A8_meGwLE7XzuMOX1WvF97TctCB1A3nu1AfVv0MxnTwi4aAimkEALw_wcB



Dr. Piot and the "virus" hunters!

Irregardless of the non-specific symptoms and the similarities of the disease to pregnancy, researchers in 1976, seeing the same signs and symptoms of haemorrhagic fever associated with many conditions, felt for some reason that they had a new "virus" on their hands. To determine that there was a new "viral" outbreak occurring in Zaire, samples from a sick nurse were sent to Dr. Peter Piot in Belgium, a man who had just graduated medical school in 1976 and was training to be a clinical microbiologist. He is credited as one of the researchers who ultimately discovered the new "virus," even though the "virus" he "found" looked exactly like the Marburg "virus," a different "virus" discovered in 1967 which presents with a similar set of symptoms:

The Scientist Who Discovered Ebola

"While working in a lab at the Institute of Tropical Medicine in Antwerp, Belgium, Piot received a cheap plastic thermos containing two vials of blood and some melted ice. Also inside was a handwritten note from a Belgian doctor based in Zaire (presently the Democratic Republic of Congo). **The note explained that the blood had been taken from a Belgian nun working in Zaire. She and two hundred others in a remote region of Zaire had become seriously sick with a mysterious illness.** The thermos had been flown on a commercial flight from Zaire's capital city in one of the passenger's carry-on bags! Upon opening the thermos, **Piot and his colleagues were greeted with a slushy mix of melted ice and blood. Of the two vials only one had remained intact while the other had shattered en route.**

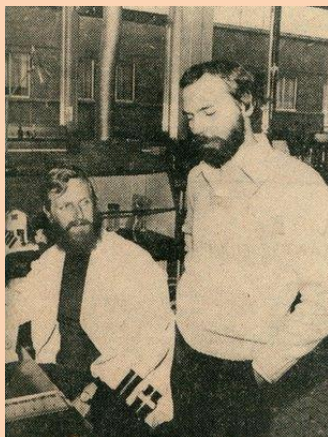
Piot and his team suspected the unknown illness to be yellow fever. The Institute of Tropical Medicine was qualified to handle yellow fever. Little did they know that the as yet to be named Ebola virus was lurking inside the thermos. In those days biosafety protocols were not as strict as they are today. **Wearing only thin latex gloves, the scientists removed a sample of blood from the undamaged vial and carried out**

standard tests on it. The blood sample was screened for known microbes, yellow fever, and several hemorrhagic-fever viruses such as Lassa, Marburg, and dengue. None of potential microbes or viruses were found in the blood. Piot also injected mice with samples of the nun's blood. After a weeks time all of the mice were dead.

When the scientists examined the blood under a microscope they were surprised by what they saw. **"We saw a gigantic worm like structure- gigantic by viral standards," explains Piot. The only other known virus that was of similar size and shape was Marburg virus.** Marburg had first appeared in 1967 when 31 laboratory workers became sick with hemorrhagic fever after coming into contact with infected monkeys. In 1976 only three facilities outside of the Soviet Union were qualified to deal with fatal viruses safely: Porton Down near London, Fort Detrick in Maryland, and what is now the CDC in Atlanta. **The World Health Organization ordered the Belgian scientists to send their blood samples to the CDC lab, the world's reference center for hemorrhagic viruses at the time. After analysing the virus, the CDC confirmed that the sample contained a brand new hemorrhagic virus.** Dr. Piot says that he experienced a feeling of "incredible excitement" with the discovery of Ebola."

"In retrospect, Dr. Piot says that he was "lucky not to get infected, not only in the laboratory but later on when I was drawing blood from patients and touching them." Following his work with Ebola, Dr. Piot conducted research on the AIDS epidemic in Africa and later became the founding executive director of UNAIDS, the Joint United Nations Programme on HIV/AIDS. Dr. Piot is currently the Director of the London School of Hygiene and Tropical Medicine."

https://www.nature.com/scitable/blog/viruses101/the_scientist_who_discovered_ebola/



Dr. Piot caught performing the art of sleeping while standing.

Interestingly, while Dr. Piot was widely given credit for discovering the Ebola "virus," there was some controversy surrounding this claim as there were many others said to be

involved in the discovery process. Even though Dr. Piot and Co. claimed “isolation” of the identical-to-Marburg-in-every-way “virus,” they did not know whether or not it was in fact a new “virus.” After insistence by the WHO, researchers at the CDC were sent samples to investigate whether or not Piot’s team had found something new:

History credits this man with discovering Ebola on his own. History is wrong

“But Pattyn and his colleagues didn’t know what they were looking at. **They saw a lasso-shaped virus that resembled Marburg virus — the cause of a similar type of hemorrhagic fever that was discovered nine years earlier — but didn’t have the ability to determine for sure whether what they observed was something new.**

The Antwerp lab was not equipped to work on deadly viruses like Marburg, so the World Health Organization instructed Pattyn to send the samples to the British military laboratories at Porton Down. **Scientists there started studying it, but also sent a sample to the CDC.**

The Atlanta team was able to show that the Yambuku outbreak was caused by a previously unknown virus, not Marburg. Webb ran the critical tests.

“Certainly those who should get credit for discovery actually knew they discovered something new,” recalled Dr. Joel Breman, a CDC epidemiologist in 1976 who led the field investigation of the outbreak at Yambuku. “Knowing what this is, different from anything else — that is the discovery of a new organism.”

The Antwerp, Porton Down, and CDC teams co-published papers describing their roles in the Ebola discovery in the March 12, 1977, issue of the Lancet. There were 15 authors in all.”

“In the interview with STAT, Piot acknowledged credit for the **actual discovery belongs to Johnson and the CDC team.**

He noted, though, that he and others in Pattyn’s lab felt they had the right to describe themselves as co-discoverers, **because of the work they did to isolate the virus from the original blood sample.**”

History credits this man with discovering Ebola on his own. History is wrong



Who deserves credit for the ebola "virus" discovery? No one. ☐♂

Three separate teams made up of 15 researchers were said to be involved in the Ebola "virus" discovery process. There was Dr. Piot's group in Antwerp, the British military group at Porton Down, and the CDC group in Atlanta. It was decided that Piot's group was able to claim that they "isolated" the Marburg-like "virus" while the CDC was given credit as the final say in the determination of the "isolate" as a new "virus" rather than the same ol' Marburg seen in 1967. In spite of the drama involved in properly crediting the discovery of Ebola to the correct individuals, when one examines the three studies submitted to the *Lancet* in 1977 by all of the researchers involved, one realizes that the question really shouldn't be about who deserves credit for discovering a new "virus" at all. **It becomes quite clear upon reading the studies that not a single one of these researchers deserve this recognition as nowhere in any of the papers are particles assumed to be the Ebola "virus" ever properly purified and isolated directly from the fluids of a sick host and then proven pathogenic in a natural way.**

For instance, in Dr. Piot's study, what we see is that the blood he received from the ill nurse was never shown to contain any "virus." At no point in time was any purification process (i.e. centrifugation, filtration, precipitation, etc.) ever described anywhere in the paper, thus there is no evidence of assumed "viral" particles being found directly in the fluids of the nurse. All that is described is **the usual cell culturing process utilizing Vero cells from African green monkeys, medium 199, and 7.5% calf serum. This is the exact opposite of purification and isolation as many foreign materials and contaminants are not separated but instead added together into a petri dish.** The resulting **cytopathogenic effect (CPE)** observed in the cell cultures was initially determined to be non-specific until the researchers switched the medium at day 5 to one more susceptible to producing CPE and incubated the culture for another week, thus creating the effect that they wanted to see. **The pathogenicity studies involved injecting the blood into the brains and stomachs of newborn mice and claiming any deaths were the result of the "virus" rather than the unnatural method of injecting the blood into the animals.** Indirect antibody results were non-specific as well and actually triggered for yellow fever, which is what Dr. Piot stated was his initial suspicion. The particles seen under Electron microscopy were not distinct new entities

and were actually identical to those associated with the Marburg "virus." No controls were performed using materials from healthy hosts nor those diagnosed with similar symptoms of disease. Upon conclusion, Dr. Piot and Co. stated it was possible that all they had done was "isolate" the Marburg "virus," yet he speculated it may have been a serologically distinct relative or perhaps a new "virus" belonging to the same group of "viruses" as rabies. In other words, they used the same indirect methods and gathered non-specific results and somehow this alerted the WHO that a new "virus" was the cause:

Isolation of Marburg-like Virus From A Case of Haemorrhagic Fever

A 42-year-old woman (patient M.E.) fell ill on Sept. 23, 1976, in Yambuku, Equateur Province, Zaire. She was transported by air on Sept. 25 to Kinshasa, where a haemorrhagic syndrome gradually developed. Clotted blood taken on the 5th day of illness was sent on ice to the Institute of Tropical Medicine, Antwerp. The sample arrived in the evening of Sept. 29 and was kept in the refrigerator.

The next morning serum was inoculated into 6 young adult mice by intracerebral and intraperitoneal routes, into 2 litters of newborn mice intracerebrally, and into 10 tubes of Vero-cell cultures (grown in medium 199 containing 7.5% calf serum).

The serum was tested by complement fixation for Lassa-virus antibodies (the result was negative) and **by neutralisation on Vero cells for antibodies against yellow-fever virus (antibodies were present at 1/30 dilution).**

RESULTS OF INOCULATIONS

Mice

One animal was found dead on the 4th day and a second on the 5th day. Brains were taken from these animals and the survivors on the 5th day.

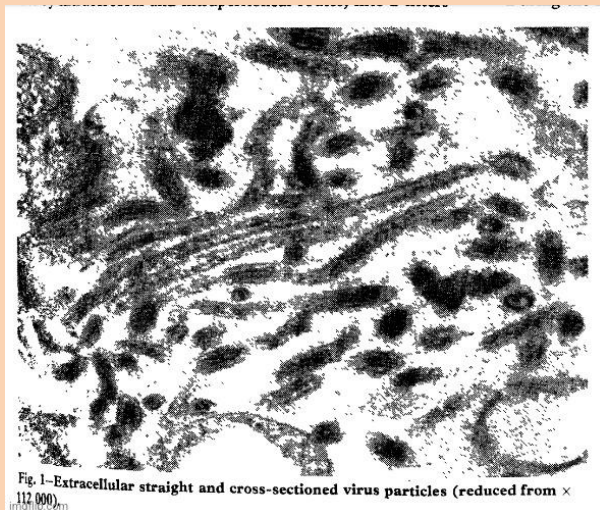
Newborn Mice

On the fifth day of observation one animal was found dead and partially eaten in each litter. In one litter several mice had disappeared on days 6 and 7, leaving only one animal. In the second litter, however, in which the animals had been very healthy during the whole observation period, only three young mice were left: one dead, one paralysed, and one very sick. The brains of these animals were removed and sent to the Microbiological Research Establishment, Porton, for further study.

Vero Cells

During the first 4 days of observation some cells in the bottom of most tubes became detached from the glass surface. Though this was first interpreted as a partial cytopathic effect, it did not increase during the following days and it was then judged to be non-specific. On day 5 the tissue-culture medium was changed to the succinate/succinic-acid buffered medium (as described by Plaisner et al.1) without serum. In our experience this medium permits the observation of Vero cells for several weeks, while many arboviruses produce a cytopathic effect in these conditions. On day 11 a very striking cytopathic effect was observed in these cultures, with most cells still attached to the glass. The cytopathic effect was almost complete on day 12.

ELECTRON-MICROSCOPY FINDINGS



The supernatant fluid of three tubes was decanted and they were filled with 3% glutaraldehyde for 30 min. The cells were then scraped off in a small amount of glutaraldehyde, rinsed with cacodylate-buffered sucrose (7.5%), postfixed in 1% phosphate-buffered osmium tetroxide, and prepared by the albumin coagulation method. Blocking staining was performed with 0.5% uranyl acetate, followed by dehydration and embedding in Spurr's low-viscosity medium. **Electron-microscopic examination of ultrathin sections of this material revealed extracellular straight and cross-sectioned virus particles morphologically similar to Marburg virus (fig. 1).** Intracellular nucleocapsids were also seen, some of them apparently originating in vesicles (figs. 2 and 3).

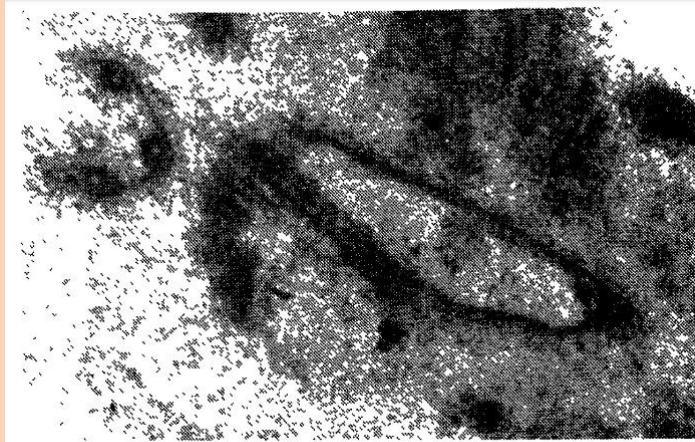


Fig. 2—Virus particles originating at intracellular membranes (reduced from $\times 112\ 000$).

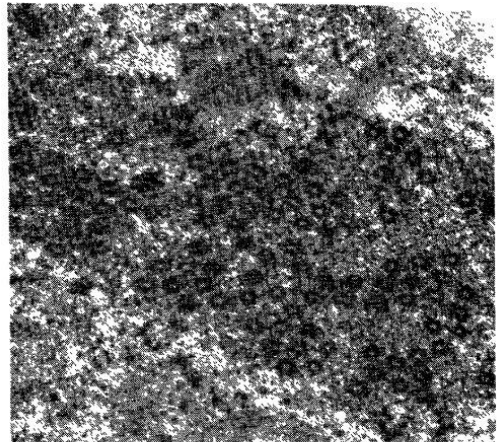


Fig. 3—Intracellular cross-sectioned virus particles (reduced from $\times 112\ 000$).

At the same time sections of the liver of the patient from whom this virus had been isolated and who had died on Oct. 1 became available. **Although the ultra-structure of this tissue was very poorly preserved, similar virus particles were observed.**

CONCLUSION

It was concluded that the agent responsible for the epidemic of haemorrhagic fever in Central Africa **was either Marburg virus or a virus serologically different from it but belonging to the same virus group, either rhabdovirus or torovirus.**

doi: 10.1016/s0140-6736(77)92002-5.



Is that Anthony Fauci?

As the WHO wanted separate investigations of the findings by labs which were better equipped to handle the more dangerous "virus" assumed to have been identified in Antwerp, the British military in Porton Down was called upon to confirm the results. In the Porton Down paper, once again we see **an absence of any mention of putifying and isolating the particles assumed to be a "virus."** We get the same cell culture experiments using Vero cells as done by Piot's team, even going so far as using material sent from Antwerp. Porton Down acquired from Antwerp the blood of acute-phase patients, the cell culture materials, and the brains from inoculated mice for their own

mad science experiments. None of the ingredients used for the culturing process were detailed and even though there is a mention of controls, they remain undefined. **During their cell culture experiments, the researchers noted slight cytopathogenic effects which they attributed to the toxic materials used for inoculation.** Eventually, three of the cultures changed to a more acidic color and caused illness when injected into the stomachs of young guinea pigs, thus signaling to the researchers that a "virus" was present. **The other pathogenicity studies performed included injecting the brains of mice killed in Antwerp into the brains and stomachs of newborn mice in Porton Down and claiming success when the mice eventually died.**

While the researchers assumed that they had a new "virus," the structures seen in EM from the livers in guinea pigs looked identical to that seen in guinea pigs and monkeys inoculated during the Marburg "virus" experiments in 1967 and 1975. The cell culture supernatant contained elongated sinuous structures which resembled the structures seen in baby-hamster kidney cells after infection with Marburg "virus." In fact, the researchers admitted that the disease and lesions produced in guinea pigs by the new agents resembled those in guinea pigs inoculated with early passage levels of Marburg "virus." For all intents and purposes, the researchers should have, if anything, concluded that they had "isolated" the Marburg "virus." While that conclusion would have been just as fraudulent, at least it would have lined up with what the indirect pseudoscientific evidence pointed towards:

Viral Haemorrhagic Fever in Southern Sudan and Northern Zaire

BETWEEN July and September, 1976, sporadic cases of fever with haemorrhagic manifestations were reported in the areas of Nzara, Maridi, and Lirangu in the southern Sudan. The first cases are believed to have been in agricultural settlements. An outbreak of a similar disease was also reported from the zone of Bumba in northern Zaire. As the epidemic increased in intensity, **the disturbingly high percentage of cases reported among hospital personnel suggested direct person-to-person spread of infection.** **The illness began with an acute fever, malaise, sore throat, muscular pains, vomiting, and diarrhoea. Those severely affected had epistaxis, subconjunctival haemorrhages, haemoptysis, hsematemesis, and melaena.** Some patients also had a body rash, tremors, and convulsions.

SOURCES OF SPECIMENS

Specimens from the northern Zaire outbreak were referred to the Microbiological Research Establishment, Porton, by Prof. S. R. Pattyn of the Institute of Tropical

Medicine, Antwerp. They were an acute-phase serum (no. 718), cell-culture materials and brains from suckling mice which had already been inoculated with the serum. We later received a specimen of liver from the same patient and also 5 acute-phase blood specimens from Zaire via Professor Pattyn. Specimens from the southern Sudan were mainly collected at Maridi Hospital and sent to us directly by Dr Babiker el Tahir, Dr D. H. Smith, Dr K. Jones, and Dr M. Cornet, who were there to investigate. They consisted of 3 throat swabs, 3 urine specimens, 6 acute-phase blood specimens, and convalescent serum specimens. These specimens were sent on dry ice or in liquid nitrogen. **Three laboratories engaged in preliminary studies on the aetiological agent reported the isolation of a virus which was morphologically similar to Marburg virus.**

RESULTS OF ATTEMPTS AT VIRUS ISOLATION

Virus isolation from the original human material was attempted in: (1) culture preparations of Vero cells; (2) suckling mice inoculated intraperitoneally (i.p.) and intracerebrally (i.c.); and (3) young guineapigs (200-250g) inoculated i.p.

Isolation in Guineapigs

So far 5 isolations of the aetiological agent have been obtained in guineapigs: 4 from specimens from northern Zaire and 1 from a specimen from southern Sudan. Guineapigs inoculated with these specimens became febrile 105°F (40-5°C) after an incubation period of 4-7 days. **The febrile illness lasted 4-5 days during which the guineapigs failed to thrive and looked ill. 1 of the 12 guineapigs inoculated with original material died on the 12th day after inoculation. The other 11 guineapigs slowly recovered** and were subsequently shown to have antibodies detectable by fluorescent antibody tests at titres ranging from 1/64 to 1/128. When whole heparinised blood from febrile guineapigs was inoculated i.p. into other guineapigs it produced a similar febrile illness.

Histopathological Findings

Liver.-There were numerous foci of necrosis which had no consistent lobular distribution and consisted of groups of liver cells undergoing hyaline degeneration and necrosis. In some of the degenerating cells small pleomorphic eosinophilic bodies were present in the cytoplasm which were periodic-acid/Schiff positive and stained bright red with the Machiavello technique but did not stain metachromatically with Giemsa. Kupffer cells were enlarged, some sinusoids contained lymphocytes, and the periportal areas were heavily infiltrated by lymphoreticular cells.

Spleen and lymph-nodes.-There was widespread depletion of the lymphoid tissue of the follicles, which contained small zones of necrosis. Large numbers of macrophages were

accumulated in the sinuses.

Lungs.—Changes in the lungs were slight: localised thickening and infiltration of interalveolar septa by lymphoreticular cells.

Other organs.—No lesions were detected in the brain, kidneys, or adrenal glands.

Electron Microscopy of Liver

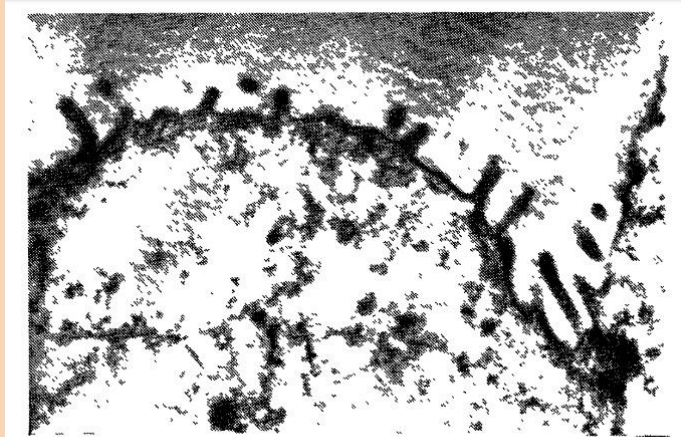


Fig. 1—Infected guineapig liver, bile canaliculi (reduced from $\times 40\,000$): virus particles budding from cell membrane.

Small pieces of liver from a guineapig killed 5 days after inoculation were fixed in 1% osmium tetroxide. Ultrathin sections were stained with uranyl acetate and lead citrate and examined under the electron microscope. **Fig. 1 shows structures strikingly similar to those seen in the livers of guineapigs and monkeys infected experimentally with Marburg virus.**

Isolation in Mice

The brains from suckling mice that became sick after being inoculated with acute-phase serum by Professor Pattyn in Antwerp were reinoculated into four litters of suckling mice. The mice began dying on the 5th day and were all dead by the 9th day. This mouse passage material has not yet been further investigated. We propose to inoculate this material into guineapigs to see whether the characteristic infection develops before we attempt further studies in mice.

Cell-culture Studies

Isolation from the original serum and blood specimens was also attempted in preparations of cultured Vero cells. **A partial cytopathic effect was seen under low-power microscopical examination. This effect did not progress to complete destruction of the cell sheet and could be attributed to a toxic effect of the specimens inoculated. There was, however, a distinct colour change in the medium of three of these cultures. By the 6th or 7th day after inoculation they became**

noticeably more acid than the control cultures. When young guineapigs were inoculated with these three cell cultures a febrile illness developed after 4-6 days.

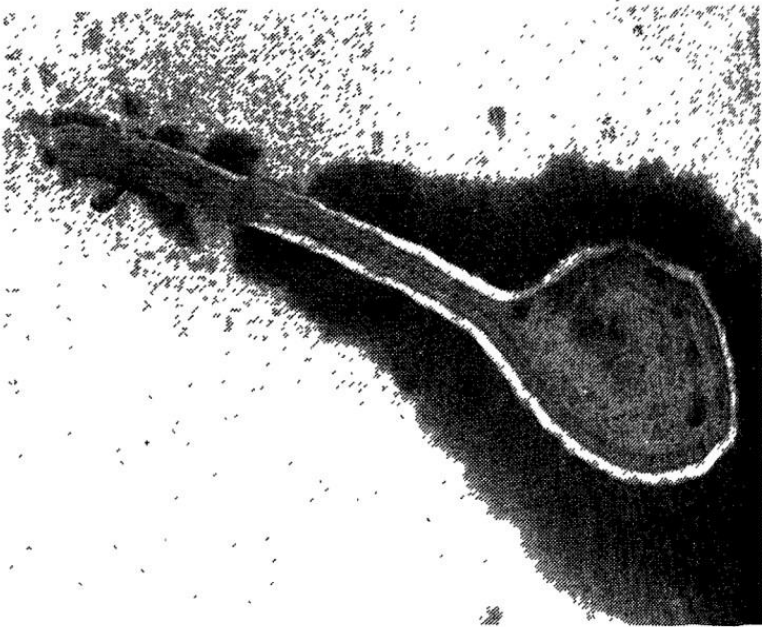


Fig. 2—Infected tissue-culture fluid from Vero cells (reduced from $\times 110\ 000$): internal appearance of virus particle with bleb-like structure.



Fig. 3—Infected tissue-culture fluid from Vero cells (reduced from $\times 139\ 000$): branching viral particle.

Electron microscopical examination of the cell-culture fluid revealed elongated sinuous structures (figs 2 and 3) **which resembled the structures seen in baby-hamster kidney cells after infection with Marburg virus.'**

CONCLUSIONS

Electron microscopy of infected guineapig livers and cell-culture material revealed structures with a striking resemblance to Marburg virus. The disease and lesions produced in guineapigs by the new agents resemble those in guineapigs inoculated with early passage levels of Marburg virus. Lesions in a liver sample taken at necropsy from one of the Zaire patients were very similar to those produced in the liver of experimentally infected guineapigs. **As yet, we have no positive evidence to suggest that the viruses isolated from northern Zaire and southern Sudan are related serologically to the Marburg virus strain isolated in 1967.** 18 convalescent sera collected in the Sudan did have fluorescent antibody titres ranging from 1/4 to 1/128 against one of the Zaire virus isolates. **Although this evidence is slight it suggests that both outbreaks have been caused by viruses which are related if not identical.** Studies are in progress to determine the relationship between the new isolates from Zaire and the Sudan with the Marburg strain isolated in 1967.

doi: 10.1016/s0140-6736(77)92001-3.



Searching hard for that "virus."

The insistence by the WHO for a re-examination of the evidence eventually led to the CDC team in Atlanta also becoming involved in order to have the final say as to whether the "isolates" in Antwerp consisted of a new "virus" or not. In the CDC paper, it is stated that Porton Down sent an aliquot of blood to the CDC. As in the previous studies, this specimen was also inoculated onto Vero cells. Once again, no purification procedures were detailed nor were the exact make-up of the cell culturing materials utilized ever provided. After observing a "distinct" CPE, the unpurified supernatant fluid was used for EM imaging. **The researchers stated that they observed large numbers of filamentous particles, which were approximately 100 nm in diameter and varied in**

length from 300 nm to more than 1500 nm. This is quite a large difference in the size range of the particles which would show that they are not homologous and could potentially have been many different "viruses" and/or microbes. The researchers also noted that, in all details, these particles were indistinguishable from those seen in the previous Marburg "virus" outbreaks. Even liver tissue examination showed the same structures seen in human and guinea pig livers from the 1967 and 1975 Marburg outbreaks. The only evidence for a proposed difference between what was eventually declared Ebola and the Marburg "virus" were results from indirect immunofluorescence antibody tests which, as the name implies, is a form of INDIRECT evidence using non-specific chemical reactions. In fact, the researchers admit that there was a weak reaction between the Marburg and Zaire samples. However, based on the weak serological evidence (which contradicted previous Marburg findings) and **in spite of the numerous admissions to identifying the same particles as associated with the Marburg "virus," the CDC had the final say that the Ebola "virus" was in fact a new "virus."** They declared so despite the fact that it is clear that not a single one of the 3 groups of researchers actually purified and isolated an Ebola "virus" to begin with:

Isolation and Characterization of a New Virus Causing Acute Haemorrhagic Fever in Zaire

AN outbreak of haemorrhagic fever with an exceptionally high mortality-rate occurred in southern Sudan and northern Zaire with peak case-rates in September, 1976. A W.H.O. International Commission operated in Sudan and Zaire from October onward. Blood and tissue specimens from persons with haemorrhagic disease were sent to laboratories in Belgium and England, and findings from these laboratories. appear in the accompanying reports. **While these specimens were being studied, Mr E. T. W. Bowen (Microbiological Research Establishment, Porton Down) sent an aliquot of an acute blood specimen from a patient in Zaire (no. 718, patient M.E.) to the Center for Disease Control, Atlanta, for additional study.**

This specimen, and all subsequent acute specimens, were inoculated into Vero (African green monkey) cells. Three days later a distinct cytopathic change (focal rounding and refractility) was evident, and an aliquot of supernatant fluid was removed for negative contrast electron microscopy.

ELECTRON MICROSCOPY OF CELL CULTURES

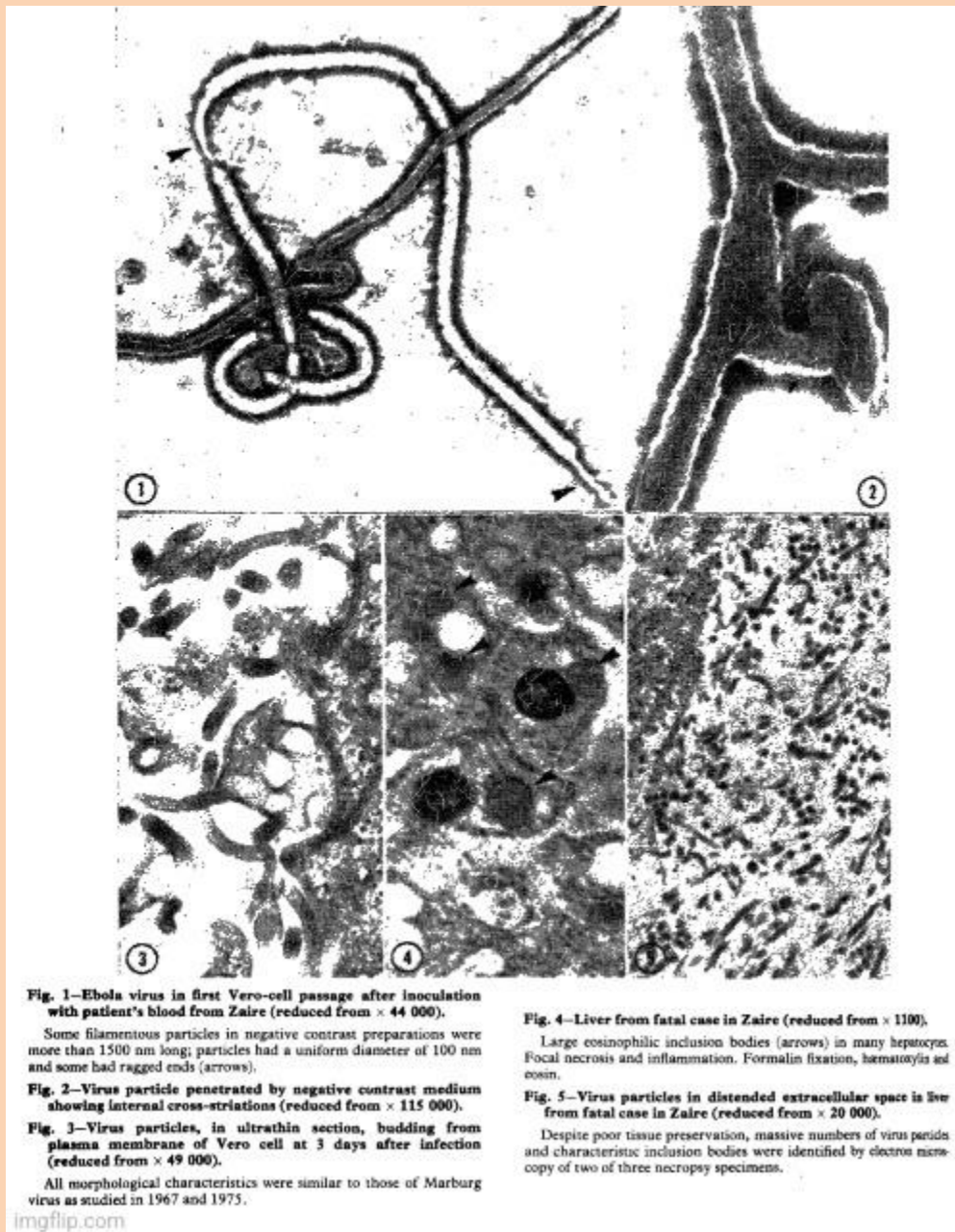


Fig. 1—Ebola virus in first Vero-cell passage after inoculation with patient's blood from Zaire (reduced from $\times 44\ 000$).

Some filamentous particles in negative contrast preparations were more than 1500 nm long; particles had a uniform diameter of 100 nm and some had ragged ends (arrows).

Fig. 2—Virus particle penetrated by negative contrast medium showing internal cross-striations (reduced from $\times 115\ 000$).

Fig. 3—Virus particles, in ultrathin section, budding from plasma membrane of Vero cell at 3 days after infection (reduced from $\times 49\ 000$).

All morphological characteristics were similar to those of Marburg virus as studied in 1967 and 1975.

Fig. 4—Liver from fatal case in Zaire (reduced from $\times 1100$).

Large eosinophilic inclusion bodies (arrows) in many hepatocytes. Focal necrosis and inflammation. Formalin fixation, haematoxylin and eosin.

Fig. 5—Virus particles in distended extracellular space in liver from fatal case in Zaire (reduced from $\times 20\ 000$).

Despite poor tissue preservation, massive numbers of virus particles and characteristic inclusion bodies were identified by electron microscopy of two of three necropsy specimens.

Carbon-coated grids were sequentially floated on droplets of the cell-culture fluid and then on 2% sodium silicotungstate pH 7. **Large numbers of filamentous virus particles were seen** (fig. 1). They were approximately 100 nm in diameter and varied in length from 300 nm to more than 1500 nm. Many had terminal blebs. Particles had regular surface projections approximately 10 nm long, and when stained they were seen to have internal cross-striations indicative of a helical core structure (fig. 2). **In all details, these particles were indistinguishable from Marburg virus particles studied in 1967** (isolates from Germany) and 1975 (isolate from South Africa). Two characteristics were more prominent in the 1976 Zaire isolate: there was more branching

of the filamentous particles (fig. 1); and more evidence of envelope continuation beyond the ends of the more rigid internal structure (fig. 1, arrow).

Vero cells infected with the same isolate from Zaire were examined also by thin-section electron microscopy. Filamentous virus particles were found budding from the plasma membrane of cells (fig. 3), and many of the cells contained inclusion bodies. These intracytoplasmic inclusions were complex and distinct, and consisted of a finely fibrillar, or granular ground substance which condensed into tubular structures. The latter have been considered to be the internal helical structure of mature virus particles. These tubules were sectioned randomly, some in cross-section, some linearly. **The virus particles in these sections were identical to those observed in the 1967 and 1975 isolates.**

POSTMORTEM LIVER SPECIMENS

Evidence of infection was seen by light microscopy in three postmortem human liver specimens from Zaire (received in formalin). Infection of two of these was confirmed by electron microscopy. Focal eosinophilic hepatocellular necrosis with modest inflammatory infiltration was prominent. Large eosinophilic inclusions were present in many intact hepatocytes, especially near sites of severe necrosis (fig. 4). These rather smooth and refractile inclusions were so characteristic that they have diagnostic significance. Plastic-embedded formalin-fixed necropsy specimens were examined with the electron microscope. **Although preservation of liver tissue was poor, large numbers of filamentous virus particles and inclusion bodies (masses of tubules) were found (fig. 5) which were indistinguishable from those in Marburg virus-infected human and guineapig livers studied in 1967 and 1975.**

ANTIGENIC COMPARISON WITH MARBURG

TABLE I—COMPARISON OF RECIPROCAL I.F.A. TITRES OF MARBURG ('67, '75) AND MARBURG-LIKE ('76) VIRUS DISEASE SERA

Year of illness	Country	Human sera	Time after onset	Antigen	
				Marburg '67	no. 718 '76
1967	Germany	U	5 mo.	128	<10
		K	5 mo.	64	<8
1975	South Africa	DO	1 mo.	64	<4
		MC	4 mo.	64	<4
1976	Sudan	no. 8	± 12 days	<2	16
		no. 9	± 12 days	<2	<2
	Zaire	no. 5	1 mo.	4	160

imgflip.com

An antigenic difference between this isolate and Marburg '67 was demonstrated by indirect immunofluorescence (I.F.A.). An infected Vero-cell suspension was placed in drops on slides, air dried, and then acetone-fixed for 10 min at room temperature. Slides were stored at -70°C until tested. Marburg '67 antigen slides, prepared in like manner, were used for comparison. Reciprocal titres obtained with convalescent human sera drawn during the 1967, 1975, and 1976 outbreaks are listed in table I. **With the exception of a weak reaction to Marburg antigen at a 1/4 dilution of the Zaire convalescent serum, the new isolate was distinct from Marburg virus.** The homologous Marburg titres of 128 and 64 obtained with '67 and '75 antigens and antisera were comparable to those reported by Wulff and Conrad.

TABLE II—COMPARISON OF I.F.A. TITRES OF GUINEAPIGS IMMUNISED (SINGLE INJECTION) AGAINST MARBURG ('67) AND MARBURG-LIKE ('76) VIRUSES

Immunising agent	Guineapig sera	Days after inoculation	I.F.A. titres with antigen of:	
			Marburg '67	no. 718 '76
Marburg '67	G.P. 16	12	>640	5
Marburg-like '76	G.P. 1	10	2	256

A single-injection immune serum to the new agent was prepared in guineapigs, and reciprocal I.F.A. tests were performed with available similar reagents for Marburg virus. Reciprocal titres (table 2) further confirmed the distinctness of the two viruses. Although one of two early convalescent sera from Sudan gave a positive reaction with the Zaire antigen (table I) further work is needed to determine whether the haemorrhagic-disease agents from the two countries are identical.

EBOLA VIRUS

With the concurrence of Prof. S. R. Pattyn, Institute of Tropical Medicine, Antwerp, and Mr E. T. W. Bowen, Microbiological Research Establishment, Porton Down, the name Ebola virus is proposed for this new agent. Ebola is a small river in Zaire which flows westward, north of Yambuku, the village of origin of the patient from whom the first isolate was obtained. In deference to the countries involved and to the lack of specific knowledge of the original natural source of the virus, it is also suggested that no names of countries or specific towns be used.



Looking hard or hardly looking?

In Summary:

- Ebola “virus” was **first discovered in 1976** near the Ebola River in what is now the Democratic Republic of Congo
- Since then, the “virus” has been said to infect people **from time to time**, leading to outbreaks in several African countries
- The “virus” **first spreads to people through direct contact** with the blood, body fluids and tissues **of animals**
- Ebola “virus” then spreads to other people **through direct contact with body fluids** of a person who is sick with or has died from EVD
- However, a person infected with Ebola **cannot spread the disease until they develop symptoms**
- **It can be difficult to clinically distinguish EVD from other infectious diseases such as malaria, typhoid fever and meningitis**
- **Many symptoms of pregnancy and Ebola disease are also quite similar**
- **Regular symptoms include:**
 - **Fever**
 - **Fatigue**
 - **Muscle pain**
 - **Headache**
 - **Sore throat**
 - **Vomiting**
 - **Diarrhoea**
 - **Rash**
 - **Symptoms of impaired kidney and liver function** In some cases, both internal and external bleeding (for example, oozing from the gums, or blood in the stools)
 - **Laboratory findings include low white blood cell and platelet counts and elevated liver enzymes**

- In 1976, Dr. Peter Piot received a thermos containing blood vials with a note which explained that the **blood had been taken from a Belgian nun working in Zaire**
- She and two hundred others in a remote region of Zaire had become **seriously sick with a mysterious illness**
- Piot and his colleagues were greeted with a **slushy mix of melted ice and blood** as, of the two vials, only one had remained intact **while the other had shattered en route**
- Dr. Peter Piot and his team suspected the unknown illness **to be yellow fever**
- Wearing **only thin latex gloves**, the scientists removed a sample of blood from the undamaged vial and carried out standard tests on it
- The blood sample was screened for known microbes, yellow fever, and several hemorrhagic-fever “viruses” such as Lassa, **Marburg**, and dengue
- **None** of the potential microbes or “viruses” were found in the blood
- Under Electron Microscope, Dr. Piot stated: **“We saw a gigantic worm like structure-gigantic by viral standards.”**
- The only other known “virus” that was **of similar size and shape was Marburg “virus”**
- *In other words, Piot and Co. found the exact same particles claimed to be Marburg but stated that they did not find Marburg in the blood samples...*
- In retrospect, Dr. Piot said that he was **lucky not to get infected**, not only in the laboratory but later on when he was drawing blood from patients and touching them
- Piot and Co. saw a lasso-shaped “virus” **that resembled Marburg “virus”** — the **cause of a similar type of hemorrhagic fever** that was discovered nine years earlier — but didn’t have the ability to determine for sure **whether what they observed was something new**
- Scientists in Porton Down started studying the sample, **but also sent a sample to the CDC after insistence by the WHO**
- The CDC’s Atlanta team was said to show that the Yambuku outbreak **was caused by a previously unknown “virus,” not Marburg**
- The Antwerp, Porton Down, and CDC teams co-published papers describing their roles in the Ebola discovery **in the March 12, 1977, issue of the Lancet**
- In the interview with STAT, Piot acknowledged **credit for the actual discovery belongs to Johnson and the CDC team**

**ISOLATION OF MARBURG-LIKE VIRUS FROM
A CASE OF HÆMORRHAGIC FEVER IN ZAIRE**

S. PATTYN
G. VAN DER GROEN
G. COURTEILLE

W. JACOB
P. PIOT

*University of Antwerp and Institute of Tropical Medicine,
Antwerp, Belgium, and Clinique Ngaliema, Kinshasa, Zaire*

- Serum from the sick nurse was inoculated into 6 young adult mice by **intracerebral and intraperitoneal routes**, into 2 litters of newborn mice **intracerebrally**, and into 10 tubes of **Vero-cell cultures (grown in medium 199 containing 7.5% calf serum)**
- The serum was tested by complement fixation for "Lassa-virus" antibodies (the result was negative) and by neutralisation on Vero cells for antibodies against yellow-fever "virus" (**antibodies were present at 1/30 dilution**)
- During the first 4 days of observation some cells in the bottom of most tubes became detached from the glass surface and **although this was first interpreted as a partial cytopathic effect**, it did not increase during the following days and it was then **judged to be non-specific**
- On day 5 **the tissue-culture medium was changed** to the succinate/succinic-acid buffered medium (as described by Plaisner et al.1) without serum
- On day 11 a very striking cytopathic effect was observed in these cultures, with most cells still attached to the glass **and the cytopathic effect was almost complete on day 12**
- *In other words, **Piot and Co. were not getting the CPE that they wanted to see so the medium was changed to one known to produce CPE with "arboviruses" and by day 12 they got the effect that they wanted to see***
- Electron-microscopic examination of ultrathin sections of this material revealed extracellular straight and cross-sectioned "**virus**" **particles morphologically similar to Marburg "virus"**
- Although the ultra-structure of liver tissue was very poorly preserved, **similar "virus" particles were observed**
- It was concluded that the agent responsible for the epidemic of haemorrhagic fever in Central Africa **was either Marburg "virus" or a "virus" serologically different from it but belonging to the same "virus" group, either "rhabdovirus" or "torovirus"**

**VIRAL HÆMORRHAGIC FEVER IN SOUTHERN
SUDAN AND NORTHERN ZAIRE**

Preliminary Studies on the Aetiological Agent

E. T. W. BOWEN

G. LLOYD

W. J. HARRIS

G. S. PLATT

A. BASKERVILLE

E. E. VELLA

*Microbiological Research Establishment, Porton, Salisbury,
Wiltshire, England*

- Specimens from the northern Zaire outbreak were referred to the Microbiological Research Establishment, Porton, by Prof. S. R. Pattyn of the Institute of Tropical Medicine, Antwerp which were an acute-phase serum (no. 718), **cell-culture materials and brains from suckling mice which had already been inoculated with the serum**
- Three laboratories engaged in preliminary studies on the aetiological agent reported the isolation of a "virus" **which was morphologically similar to Marburg "virus"**

- “Virus isolation” from the original human material was attempted in:
 - Culture preparations of Vero cells
 - Suckling mice inoculated intraperitoneally (i.p.) and intracerebrally (i.c.)
 - Young guinea pigs (200-250g) inoculated i.p.
- In inoculated guinea pigs, a febrile illness lasted 4-5 days during which the guinea pigs failed to thrive and **looked ill**
- 1 of the 12 guinea pigs inoculated with original material died on the 12th day after inoculation **while the other 11 guinea pigs slowly recovered**
- Small pieces of liver from a guinea pig killed 5 days after inoculation were fixed in 1% osmium tetroxide and structures **strikingly similar to those seen in the livers of guinea pigs and monkeys infected experimentally with Marburg “virus”** were seen
- The **brains from suckling mice that became sick after being inoculated with acute-phase serum by Professor Pattyn** in Antwerp were reinoculated into four litters of suckling mice
- The mice began dying on the 5th day and were all dead by the 9th day **yet this mouse passage material had not yet been further investigated**
- In cell culture experiments involving Vero cells, **partial cytopathic effect** was seen under low-power microscopical examination
- This effect did not progress to complete destruction of the cell sheet and **could be attributed to a toxic effect of the specimens inoculated**
- There was, however, a **distinct colour change** in the medium **of three** of these cultures and by the 6th or 7th day after inoculation they became **noticeably more acid** than the control cultures (*which were not defined*)
- Electron microscopy of infected guinea pig livers and cell-culture material **revealed structures with a striking resemblance to Marburg “virus”**
- The **disease and lesions produced** in guinea pigs by the new agents **resemble those in guinea pigs inoculated with early passage levels of Marburg “virus”**
- The researchers stated that they had **no positive evidence to suggest** that the “viruses isolated” from northern Zaire and southern Sudan were **related serologically to the Marburg “virus” strain isolated in 1967**
- *In other words, they had no positive evidence to suggest the “virus,” which was identical to Marburg in every other way, was not the exact same “virus” either as they just assumed their new “isolates” were different*
- The researchers admitted that **although their evidence was slight, it suggested** that both outbreaks were caused by “viruses” which are related if not identical

Preliminary Communications

ISOLATION AND PARTIAL CHARACTERISATION OF A NEW VIRUS CAUSING ACUTE HÆMORRHAGIC FEVER IN ZAIRE

K. M. JOHNSON
J. V. LANGE

P. A. WEBB
F. A. MURPHY

*Virology Division, Center for Disease Control,
Atlanta, Georgia 30333, U.S.A.*

- Mr E. T. W. Bowen (Microbiological Research Establishment, Porton Down) sent an aliquot of an acute blood specimen from a patient in Zaire (no. 718, patient M.E.) to the Center for Disease Control, Atlanta, for additional study
- This specimen, and all subsequent acute specimens, **were inoculated into Vero (African green monkey) cells**
- Three days later a **distinct cytopathic change** (focal rounding and refractility) was evident, and an aliquot of **supernatant fluid was removed for negative contrast electron microscopy**
- In EM, large numbers of filamentous "virus" particles were seen which were approximately 100 nm in diameter **and varied in length from 300 nm to more than 1500 nm**
- **In all details, these particles were indistinguishable from Marburg "virus" particles** studied in 1967 (isolates from Germany) and 1975 (isolate from South Africa)
- Vero cells infected with the same "isolate" from Zaire were examined also by thin-section electron microscopy **and the "virus" particles in these sections were identical to those observed in the 1967 and 1975 "isolates"**
- Although preservation of liver tissue was poor upon examination, large numbers of filamentous "virus" particles and inclusion bodies (masses of tubules) were found **which were indistinguishable from those in Marburg "virus-infected" human and guineapig livers studied in 1967 and 1975**
- An antigenic difference between this isolate and Marburg '67 was demonstrated **by indirect immunofluorescence (I.F.A.)**
- **With the exception of a weak reaction to Marburg antigen at a 1/4 dilution of the Zaire convalescent serum**, the new isolate was distinct from Marburg "virus"

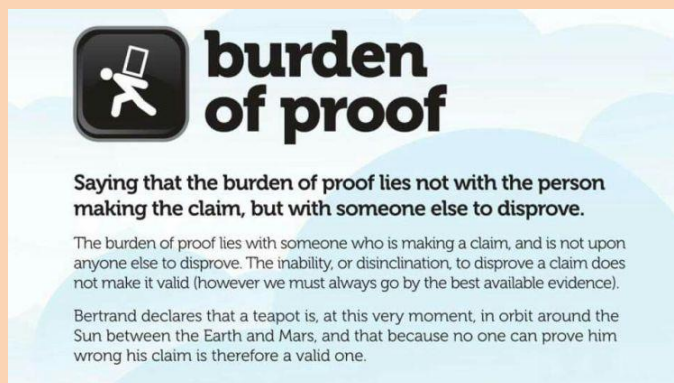


When looking to either prove or disprove a positive claim, such as the existence of an Ebola "virus" as was the case in this particular instance, one must always look to the original foundational papers and see if the scientific evidence supporting the claim actually exists. **Tracing the origin of the Ebola "virus" to its roots led me to three different papers by three different teams of researchers in three different parts of the world. It should come as no surprise to anyone who understands the pseudoscientific methods virology employs that, at no point in time, do any of the papers submitted adhere to the scientific method.** This is absolutely essential in order for a study to be considered scientific. **Nowhere in any of the papers is an Ebola "virus" ever properly purified and isolated directly from the fluids of a sick host, thus there is no independent variable for the researchers to vary and manipulate in order to determine cause and effect. Without a valid independent variable of purified/isolated particles, there can be no true pathogenicity studies utilizing a natural route of exposure, thus there can be no pathogenic claim. Without proper controls, there is no way to determine what other confounding factors could also potentially cause the effect one is searching for through experimentation, thus making the results obtained invalid and meaningless.**

What these pseudoscientific researchers do instead is attempt to skirt around the scientific method by presenting manufactured indirect evidence as a stand-in for the real deal. They are con men selling counterfeit results. **Instead of properly purified and isolated "virus," we get unpurified cell cultured soup mixed with many toxic ingredients such as Vero cells, medium 199, and 7.5% calf serum. Instead of observing the "virus," we get the non-specific cell death known as the cytopathogenic effect blamed on an invisible pathogen which can be a result of many things other than a "virus" such as changing the medium halfway through to one more suitable to producing the desired cytopathic effect. Instead of visualizing nothing but the particles claimed to be the new "virus," we get EM images of unpurified and non-isolated filamentous particles ranging wildly in shape and size which were previously associated with the Marburg "virus."** We get

histopathological lab results showing the same findings obtained through investigation with the Marburg “virus” a decade before. We get non-specific indirect antibody results used to claim that the “virus” is a new “virus” within the same family as the Marburg “virus” even though the other indirect findings should have led to the conclusion that they had either uncovered the same “virus” or that the results in both cases were fraudulent. In other words, **the researchers found the exact same particles in EM images associated with cases of the exact same symptoms of disease attributed to Marburg (and many other diseases) yet as the antibody results did not align, rather than question the credibility of the previous Marburg findings, the researchers decided that what they had was a new “virus” instead.** Anything to keep the story alive.

However, **if one goes back to investigate the Marburg “virus,” one would see the exact same pseudoscientific practices were employed where no “virus” was ever purified and isolated and the same particles belonging to rabies were identified instead as Marburg. If one looks into the rabies “virus,” one will see this same pattern play out, and so on and so forth as this trail is traced back as far as it can go.** The same cycle of deception persists where the same symptoms of disease are given a new name associated with similar and/or identical particles based on indirect and contradictory antibody results. **What one will never find is direct proof for the existence of any “virus” which adheres to the scientific method¹.** All one will ever find is pseudoscientific fiction passed off as scientific fact.



With all of that being said, let this stand as my response to anyone claiming that we must intentionally infect ourselves first in order to say that there is no scientific evidence for the existence of any “virus.” This is asinine and absolutely backwards. The burden of proof is on anyone making the positive claim that a “virus” exists and they must provide the evidence supporting their claim in order to justify it. This logically requires that the scientific evidence for the existence of the Ebola “virus” is contained within the original research papers. I’ve done the hard work for these people and tracked down the original

¹ **More clearly, the existence of a virus which is the unique (or main) causative factor for a specific disease**

studies used to make the case for an Ebola "virus." I've shown that, **at no point in time, was the scientific method ever adhered to nor were any particles assumed to be Ebola ever properly purified and isolated nor proven pathogenic in a natural way.** If anyone disagrees with this assessment, I have a proposal. Please show me in the three papers I shared above where the scientific method was applied and adhered to from the very beginning. **Show me where the researchers purified and isolated the "virus" directly from the sick host. Show me that the particles in the EM images are of only the "virus" and nothing else. Show me that these purified and isolated particles can make a suitable host sick in a natural way and not through injections of unpurified toxins directly into the brain. Show me where the results are reproduced and replicated on a large scale utilizing the proper controls as should be expected of any scientific endeavor.** If any person claiming the existence of an Ebola "virus" can show me where in these papers it was scientifically proven that the Ebola "virus" exists by adherence to the scientific method, then I will go to Africa to intentionally infect myself *on my own dime*. Granted, at that point they will have proven their case thus making my trip to Africa an expensive yet pointless and potentially dangerous endeavor for me. However, if they can not show that this evidence is contained within these three papers, they will concede that the scientific evidence proving the existence of an Ebola "virus" is nowhere to be found and I will look forward to a *free first class trip* to a safari in Africa.

Any takers?



Share this:

- [X](#)
- [Facebook](#)
-

Like this:

[The Ebola "Virus" Part 2](#)

[ViroLI Egv Boosters: The Hanta and Ebola Testing Trap](#)

[The Marburg "Virus" Precursor to Ebola](#)

Published September 26, 2022 By [Mike Stone](#)
Categorized as [Ebola](#)

32 comments



1. PC

September 26, 2022 at 1:36 pm

Thanks , another great article.

I also notice the cognitive dissonance in so many., the contradictions and the logical fallacies .

We all have been indoctrinated in a false biology but many seem to not be able to get out of their programming .

From Dr Lanka's interview.:

https://media2-production.mightynetworks.com/asset/45921073/The_politics_of_fear_Fear_is_the_bioweapon.pdf?_gl=1*ideh1q*_ga*MTIzMTkzOTQ4OC4xNjI1NzM1Otcw*_ga_T49FMYQ9FZ*MTY2NDE5OTA1Mi4xMzA3LjEuMTY2NDE5OTEzMC4wLjAuMA..

"What they see as Ebola in Africa one has to imagine the conditions

eg. the Kongo conflict or where they look for **Lithium .**

Those who survive have huge severe conflicts and have diarrhoea in the healing phase.

Has a friend with **doctors without borders , the Swiss team ,they had 100% cure with Ebola.**

German team gave toxic treatments with riboflavin and medication.

So the cheap treatments were effective while the expensive one caused death.

Same now with medication for the stigmatised ' Covid' patients – it can be deadly as it is actually rat poisoning.

How is ebola defined? – diarrhoea for days.

The Swiss estimated how much water is lost and replenished the water via iv drip . They had zero death . It was even published. All survived.

—
The solution is simple. (to the human predicament)

If one understands biology , one knows that there can not be a pathogen .

We know what causes disease.

There are no symptoms with ' covid' or cancer that cannot be explained via the body-soul biology.

Majority believe in a 'virus' , a terror biology, maybe an 'inherited' substance from mummy or daddy , or smoked the wrong thing and suddenly have a terror organisation that has the only scope to kill one and is stronger than one, and it is also claimed it 'travels ' within the body.(metastasis)

This idea of the 'C' , the name cancer should not even be mentioned . It is voodoo and voodoo functions.

All you need is a few photos of kids that were shot years ago in another location and to add a scary narrative.

This is how fear functions .These pictures , people perceive as reality.

Fear works , the bioweapon is fear.

The photos and the false narrative, this combination becomes the spotlight, it hypnotises people .

And it is so powerful , one can make so much money out of fear. **The biotech industry made 7.3 billion net last year .."**

Loading...

[Reply](#)



1. [Mike Stone](#)

September 26, 2022 at 2:15 pm

Fear is the real virus.

Loading...

[Reply](#)



2. barni cutica

September 27, 2022 at 2:15 am

Rabie is not a virus! Is a BACTERIA! Is NEVER EVER possible to create a VACCINE against a VIRUS!!! So the "anti COVID vaccine " IS THE GREAT MEDICAL FRAUD IN THEASE UNIVERSES!!!

Loading...

[Reply](#)



1. [Mike Stone](#)

September 27, 2022 at 12:05 pm

Rabies is said to be a "virus," not a bacteria. I did a fee articles on rabies here that you may find interesting:

<https://viroliegy.com/category/rabies/>

I agree that the "Covid" vaccine, like all vaccines, are medical fraud. They must all be stopped.

Loading...

[Reply](#)



3. PC

September 27, 2022 at 2:06 pm

Ebola: the new fake outbreak by Jon Rappoport

The virus is the cover story

March 2, 2021

" David Rasnick, PhD, announced this shocking finding:

"I have examined in detail the literature on isolation and Ems [EM: electron microscope pictures] of both Ebola and Marburg viruses. I have not found any convincing evidence that Ebola virus (and for that matter Marburg) has been isolated from humans. There is certainly no confirmatory evidence of human isolation."

The real reasons for the "Ebola outbreak" include, but are not limited to: industrial pollution; organophosphate pesticides (causes bleeding); vast overuse of antibiotics (causes bleeding); severe and debilitating nutritional deficiencies (which can cause bleeding); starvation; drastic electrolyte loss; chronic diarrhea; grinding poverty; war; stolen farm land; vaccination campaigns (in people whose immune systems are compromised, vaccines can easily wipe out their last shreds of health).

Irrelevant or non-existent viruses function as cover stories to conceal actual and inconvenient causes of illness, such as industrial pollution, ag pesticides, GMO food, fracking chemicals, radiation, etc.

The medical cartel and its government allies move a step closer to being able to mandate all vaccines for the population, with no exemptions permitted.

<https://blog.nomorefakenews.com/>

The Ebola Lie Exposed!

Published on October 24, 2014

1. No "Ebola" virus has ever been isolated!

2. Barbarous animal testing

3. Epidemics always started in hospitals

4. People were more likely to be killed by the "prevention measures" and the "treatments"

5. Coercive "treatment" carried out by the WHO gestapo-style

The German Medical Review 118 of 29 June 2000 (Ärzte-Zeitung) had already admitted that vaccine doses with 100-1,000 higher concentrations than what is used in Europe had been given and had led to numerous deaths of African babies in the nineties.

Dr. Stefan Lanka commented on these "trials" naming them for what they were (or still are?) – secret genetic experiments on African people: "When you are poisoned with such a lethal dosis, your liver stops creating the globulins needed for blood-clotting. You then start bleeding internally and externally, it is called hemorrhagic fever..." and of course they are blaming it once more on a virus!"

<https://drsircus.com/general/ebola-lie-exposed/>

Loading...

[Reply](#)



Mike

4.

[September 27, 2022 at 9:32 pm](#)

Thank you Mr. Mike Stone for another great walk-through tour of the fictional (read Psych-War) "Land Of Absurdia" aka "The Science/Virology"!

Loading...

[Reply](#)



Mike Stone

1.

[September 28, 2022 at 12:34 am](#)

You are very welcome Mike! ☐

Loading...

[Reply](#)



Tom

5.

[September 28, 2022 at 12:15 am](#)

The symptoms almost looks like some kind of fentynal od or other drug od. Poor Africa, always the headwaters for the next monster disease or virus. I do not think Ebola exists as a virus. It's another relabeled health difficulty brought on by lack of vitamins and minerals and invading toxins.

Loading...

[Reply](#)



Mike Stone

1.

[September 28, 2022 at 12:36 am](#)

I'm sure the symptoms are brought on by various factors. They are all related to the same detox process. I am breaking down the WHO's 1978 report currently. It is amazing the amount of toxic drugs injected into the patients. It's no wonder they did not survive.

Loading...

[Reply](#)



6. Rich

September 28, 2022 at 1:16 pm

Reading this, I realized another internal contradiction of virology (forgive me, if this has been documented elsewhere).

They say it is known that many viruses are zoonotic, and many have a "natural reservoir/host", which carries the virus, but does not get sick from it (e.g. bats for Coronaviruses and Ebola). These are basically "asymptomatic carriers", so likely they just "detected" it in some specimens through PCR.

Now, if you know that many animals do not suffer the same disease i.e. symptoms from these viruses... why would they use animals to show pathogenicity? Why would they even expect mice, hamsters and so on to get sick when they inject them? (Injection ofc is another issue, since it's not a natural way in infection). Why would they expect these animals to show similar symptoms?

As with so many things in virology, it just doesn't make sense on their own terms.

Loading...

[Reply](#)



1. [Mike Stone](#)

September 28, 2022 at 3:19 pm

I wonder how these reservoir animal spread the "virus" if they are asymptomatic. Ebola is said not to spread asymptotically. Granted, they can change their fiction at any time. □

Loading...

[Reply](#)



7. Jeffrey Strahl

September 30, 2022 at 5:52 pm

Thanks for another excellent article, Mike. It was just perfect to send a FB friend who frantically sent me a video about how coming shots include codes or "Marburg and Ebola" viruses. □

Loading...

[Reply](#)



1. [Mike Stone](#)

September 30, 2022 at 7:06 pm

Thanks Jeffrey! When looking to the history of Ebola, it is amazing how anyone fell for there being a new "virus." The problem is, no one ever looks to the evidence presented to see what it actually shows. If they did, they would realize it tells a much different story than what we were sold.

Loading...

[Reply](#)



1. Jeffrey Strahl

September 30, 2022 at 7:42 pm

And it's mind blowing, Mike, how trenchant anti-vaxxers can be about their virus hugging. One attendee at a group lunch yesterday insisted she got "Covid," because she felt something she never had before, and tested positive. She had no idea what the test measures. **She also went to the "bio-weapon" argument.** She said she was aware that neither the two alleged ingredients of the bio-weapon,, HIV and SARS, had ever been isolated, but insisted it must have been some sort of virus. I asked her where and when contagion theory, germ/virus theory, had been proven. No idea, but "I'm not a science person." SMH.

Loading...



2. [Mike Stone](#)

September 30, 2022 at 8:58 pm

It's sad how much control some have given over their brains. People have no confidence in their own ability to learn, understand, and grow. They are stuck in neutral.

Loading...



3. [Jeffrey Strahl](#)

September 30, 2022 at 9:27 pm

This is one result of the "education" system, which divides up people into two groups, one channeled into the sciences (where they are also taught to stay in their specific lanes) and one which is told (implicitly) they have no prayer of ever understanding science. Will be writing an article which focuses on this similarity between 9/11 and the "virus" affair soon.

Loading...



4. [Mike Stone](#)

October 2, 2022 at 1:04 am

Awesome! I can't wait to read it! ☐

Loading...



8. [George](#)

October 1, 2022 at 2:05 pm

Yes, on the surface, Ebola looks terrifying, but scratch the surface and you can overcome the fear and see the truth. All of the details you've provided bar the escape back into fear. Fear drives people into religion rather than into the truth. As you know, the truth sets you free. All of virology looks like a religion to me—all of it—including the COVID scam, for which I offer my opinion.

They created the SARS-CoV-2 virus on a computer. This is not in dispute among virologists. When a person understands the ramifications of this fact in conjunction with why the germ theory of disease is called a theory, then he is free to ask, exactly why does a person believe in COVID-19? The only logical conclusion must be that the person is acknowledging the legitimacy of a new religion. If this is the situation, then the next reasonable question should be, has the believer become a devotee and proceeded to practice its rituals, like excessive hand washing, social distancing, and mask wearing? Has he listened to the lustral incantations on the evening news to monitor the growth and progress of his religion?

Additionally, has the proselyte progressed far enough in the COVID faith to the point where he presents himself for periodic confession and testing when he believes he's been in contact with something unclean or with a person defiled by the disease? And, furthermore, has he received communion with the injection of one or more COVID-19 vaccines?

If so, then his fate rests with the gods having power over the disease—the principal dieties being Pfizer, Moderna, and AstraZeneca. If the believer is accepted by one of the COVID gods, then the promise of deliverance from the scourge of the disease awaits him; if not, then he will have to endure their wrath until such time as he is sufficiently punished for his impiety.

Of course, all of this is an illusion, except for the part about the injections.

Loading...

[Reply](#)



1. [Mike Stone](#)

[October 2, 2022 at 1:06 am](#)

Belief in “viruses” parallels religion perfectly, even the use of fear to keep people from questioning anything they have been told.

Loading...

[Reply](#)



9. [George](#)

[October 2, 2022 at 9:35 am](#)

Science replaced by consensus

California Bill Barring Docs From Telling COVID Lies Signed Into Law
— Law won't stop docs who spread misinfo on social media; only during direct patient care

“California Governor Gavin Newsom signed legislation on Friday that gives the state some ammunition against physicians who spread lies about COVID in the context of direct patient care, although it won't apply to those who spread such misinformation on social media.

It is said to be the first such law in the nation.”

<https://www.medpagetoday.com/special-reports/exclusives/101008>

Loading...

[Reply](#)

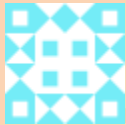


1. [Dude in Japan](#)

[October 2, 2022 at 11:04 pm](#)

Newsom is only governor right now because of similar shenanigans as the 2020 resident biden theft.

[Reply](#)



10. [Dude in Japan](#)

[October 2, 2022 at 11:02 pm](#)

“Dr. Piot is currently the Director of the London School of Hygiene and Tropical Medicine.”

^is it just me or does the name of that school sounds AWFULLY familiar?

[Reply](#)



1. [Mike Stone](#)

October 3, 2022 at 3:48 pm

Definitely rings a bell for sure ☐

[Reply](#)



1. mike

October 3, 2022 at 7:42 pm

Yeah, I've heard they have a huge tip-jar that has a big "but".

11. Pingback: [The Ebola "Virus" Part 2 – ViroLI.Egy](#)



12. Brian

October 17, 2022 at 2:58 am

Anyone read Ken Alibek's "Biohazard" (1999)?

He ran Biopreparat, the USSR's massively funded undercover bioweapons production program, which incidently, was just a small part of the overall state-run apparatus of biowarfare R&D . Yes, they weaponized all sorts of pathogens, not the least of which were viral. Are we to believe that the funding and patriotic passion for these programs arose from a Lysenko-like mass hypnosis, or was their virology not a lie? Who is Koch if it kills??

Just asking.

[Reply](#)

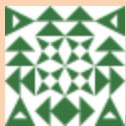


1. [Mike Stone](#)

October 17, 2022 at 5:22 pm

I'm not sure I understand your question. Are you asking whether "viral" bioweapons exist and can kill?

[Reply](#)



1. mike

October 18, 2022 at 6:59 pm

Spy vs Spy narrative....(rinse and repeat)

13. Pingback: [L'INVENTION DU « VIRUS EBOLA » – JEMINFORMETV.COM](#)

14. Pingback: [EN — LARRY ROMANOFF: Biological Warfare in Action — Chapter 14 – EBOLA](#)

15. Pingback: [CN — LARRY ROMANOFF: 生物战在行动 — 第14章——埃博拉病毒](#)

The Ebola "Virus" Part 2



In [part 1](#) of this breakdown of the fraud that is the Ebola "virus," I looked at the three main publications from three different teams of researchers which were submitted to *The Lancet* in 1977 as proof for the existence of a new "virus" causing haemorrhagic fever in 1976. The three teams, including one based at the CDC in Atlanta, coordinated with the WHO in order to associate the new "virus" with the non-specific symptoms of disease occurring in Zaire. They did so even though every piece of information acquired through their investigations pointed to the Marburg "virus," which itself was based on the acquisition of fraudulent indirect evidence in 1967.

As the WHO was intimately involved in the coordination of these three teams and the determination of the Ebola "virus" as the causative agent, I thought it would be fun to go through their own recounting of the events that led to this conclusion. In 1978, the WHO was kind enough to release a 23-page report that served as a summary of the information that was acquired during the investigation into the outbreak of haemorrhagic fever in a hospital in Zaire. As the report is rather long, I will provide the relevant highlights. In order to make it easier to breakdown and emphasize the pertinent information, I am doing things a little differently this time around. Instead of presenting the highlights as one continuous text, the provided sections from the report will be broken up by commentary with additional information inserted along the way. There is still the usual summary at the end as well as a link to download the entire report

for anyone to read if they so desire. So with that being said, let's jump in and see what we can uncover about this "virus" directly from the WHO:

Ebola haemorrhagic fever in Zaire, 1976

"Between 1 September and 24 October 1976, 318 cases of acute viral haemorrhagic fever occurred in northern Zaire. The outbreak was centred in the Bumba Zone of the Equateur Region and most of the cases were recorded within a radius of 70 km of Yambuku, although a few patients sought medical attention in Bumba, Abumombazi, and the capital city of Kinshasa, where individual secondary and tertiary cases occurred. **There were 280 deaths, and only 38 serologically confirmed survivors.**

I want to start by immediately pointing out in this following section, we will see that the common denominator seen in the vast majority of these cases of haemorrhagic fever was the injection of drugs like chloroquine, an anti-malaria drug, at a hospital in Zaire. While I will discuss the use of chloroquine a little later, note that the initial patient was given this injection as he was presumed to have malaria based on his symptoms. After the injection, his symptoms went into clinical remission yet he once again came down with a fever 5 days after his treatment. Spoiler alert: we will later find out that the patient experienced gastrointestinal bleeding after receiving chloroquine and ultimately died on September 8th, 1976:

The index case in this outbreak had onset of symptoms on 1 September 1976, five days **after receiving an injection of chloroquine for presumptive malaria** at the outpatient clinic at Yambuku Mission Hospital (YMH). **He had a clinical remission of his malaria symptoms.** Within one week several other persons **who had received injections at YMH** also suffered from Ebola haemorrhagic fever, and almost all subsequent cases **had either received injections at the hospital** or had had close contact with another case. Most of these occurred during the first four weeks of the epidemic, after which time the hospital was closed, 11 of the 17 staff members having died of the disease. **All ages and both sexes were affected, but women 15-29 years of age had the highest incidence of disease, a phenomenon strongly related to attendance at prenatal and outpatient clinics at the hospital where they received injections.** The overall secondary attack rate was about 5%, although it ranged to 20% among close relatives such as spouses, parent or child, and brother or sister.

Note above that pregnant women were among the highest incidence of the symptoms of disease said to be Ebola. Remember this piece of information from the WHO in part 1?

“It can be difficult to clinically distinguish EVD from other infectious diseases such as malaria, typhoid fever and meningitis. **Many symptoms of pregnancy and Ebola disease are also quite similar.**”

https://www.who.int/news-room/fact-sheets/detail/ebola-virus-disease?gclid=Cj0KCQjwj7CZBhDHARIsAPPWv3cNnRDMQ6A8_meGwLE7XzuMOX1WvF97TctCB1A3nu1AfVv0MxnTwi4aAimkEALw_wcB

EBOLA

- Hiccups
- Difficulty Swallowing

OVERLAPPING SYMPTOMS

- Fever
- Bleeding
- Headache
- Joint and muscle pain
- Weakness and fatigue
- Abdominal pain/right upper quadrant pain
- Difficulty breathing
- Vomiting
- Diarrhea

COMPLICATED LABOR

- Blurred vision
- Convulsions

Hiccups is a symptom of Ebola?!?!

According to the WHO, pregnancy symptoms mimic Ebola. Strangely enough, pregnant women seemingly had the highest incidence of the disease in 1976 and were given injections (presumably of chloroquine but this is not directly stated) in the prenatal and outpatient clinics. What a coincidence!

Active surveillance disclosed that cases occurred in **55 of some 550 villages** which were examined house-by-house. The disease was hitherto unknown to the people of the affected region. **Intensive search for cases in the area of north-eastern Zaire between the Bumba Zone and the Sudan frontier near Nzara and Maridi failed to detect definite evidence of a link between an epidemic of the disease in that country and the outbreak near Bumba. Nevertheless it was established that people can and do make the trip between Nzara and Bumba in not more than four days: thus it was regarded as quite possible that an infected person had travelled from Sudan to Yambuku and transferred the virus to a needle of the hospital while receiving an injection at the outpatient clinic.**

In this next section, **the WHO points out that the symptoms of Ebola are non-specific.** They tried to add other conditions under the Ebola brand such as hepatitis, pancreatitis, and disseminated intravascular coagulation (a blood clotting condition leading to massive bleeding associated with inflammation, infection, and cancer) yet the lab results were inconclusive:

Both the incubation period, and the duration of the clinical disease averaged about one week. **After 3-4 days of non-specific symptoms and signs,** patients typically experienced progressively severe sore throat, developed a maculopapular rash, had intractable abdominal pain, and **began to bleed from multiple sites, principally the gastrointestinal tract. Although laboratory determinations were limited and not conclusive,** it was concluded that pathogenesis of the disease included non-icteric hepatitis and possibly acute pancreatitis as well as disseminated intravascular coagulation.

As I showed in part 1, **the particles claimed to be the Ebola “virus” were admitted to be morphologically identical to those associated with the Marburg “virus” yet the WHO and their counterparts at the CDC stated that these “viruses” were immunologically different based on non-specific indirect antibody results.** Interestingly, the “virus” was only said to be “isolated” in 8 of 10 cell cultures performed and the particles were only said to be found in the liver samples of three cases:

This syndrome was caused by a virus morphologically similar to Marburg virus, but immunologically distinct. It was named Ebola virus. **The agent was isolated from the blood of 8 of 10 suspected cases using Vero cell cultures.** Titrations of serial specimens obtained from one patient disclosed persistent viraemia of $10^{6.5}$ - $10^{4.5}$ infectious units from the third day of illness until death on the eighth day. **Ebola virus particles were found in formalin fixed liver specimens from three cases. Survivors of infection were found to have indirect fluorescent antibodies to Ebola virus** in titres of 1:64-1:256 within three weeks after onset of disease and these serum titres persisted with only slight decrease for a period of 4 months.

A total of 201 units (200-300 ml each) of plasma containing Ebola virus antibodies in titres of at least 1:64 were obtained and frozen. Two of these units were used to treat a laboratory worker infected with Ebola virus. **This person recovered, which suggests that the antibodies may have helped therapeutically.**

Note in this next section, **the WHO admits that the transmission of the "virus" ended when stopping the injections at the hospital.** How odd. □

Virus transmission was interrupted by stopping injections and by isolation of patients in their villages. Use of protective clothing and respirators, strict isolation of patients, and careful disposal of potentially contaminated excreta and fomites will almost certainly prevent future major outbreaks. **The virus is probably rarely transmitted by infectious aerosols, although infection via large droplets remains a possibility.**

We also learn that Ebola antibodies were found in five people who were never sick and had no history of contact with any infected individuals. What this ultimately means is that the WHO was strategically setting the stage so that they have the excuse ready for asymptomatic cases of Ebola when the so-called "specific" antibodies are found in people that they are not supposed to be found in:

Only limited ecological investigations were made, since the epidemiology of the outbreak strongly suggested that the virus had been imported into Bumba Zone. Ebola virus was not recovered from representative samples of bedbugs or of rodents (*Rattus rattus* and *Mastomys* spp.) having more or less close contact with humans. **Ebola virus antibodies were found, however, in five persons who were not ill and had not had contact with the "infected" villages or the Yambuku hospital during the epidemic.** If these findings can be confirmed by an independent method of testing, they would suggest that the virus is in fact endemic to the region and should lead to further effort to uncover a viral reservoir in Zaire."

If we are to believe the findings as presented by the WHO, the Ebola “virus” originated from an infected person traveling from Sudan to Zaire. This unidentified person received treatment at the Yambuku hospital by way of a parenteral injection. The same needle was not sterilized and was then reused on another unsuspecting patient, thus **resulting in the spread of the disease from patient-to-patient by way of the continuous use of the same unsterilized needles.**



In this next section, the WHO plants the seed for the narrative to follow. It is claimed that only 5 syringes and needles were used for every patient throughout the day. While the WHO claims the needles were not sterilized, they state that the needles were rinsed in pans of warm water and/or boiled.

“Five syringes and needles were issued to the nursing staff each morning for use at the outpatient department, the prenatal clinic, and the inpatient wards. These syringes and needles were apparently not sterilized between their use on different patients but rinsed in a pan of warm water. At the end of the day they were sometimes boiled. The surgical theatre had its own ample supply of instruments, syringes, and needles, which were kept separately and auto-claved after use.

Contrary to what the WHO says, the use of heat is a common form of sterilization in hospitals:

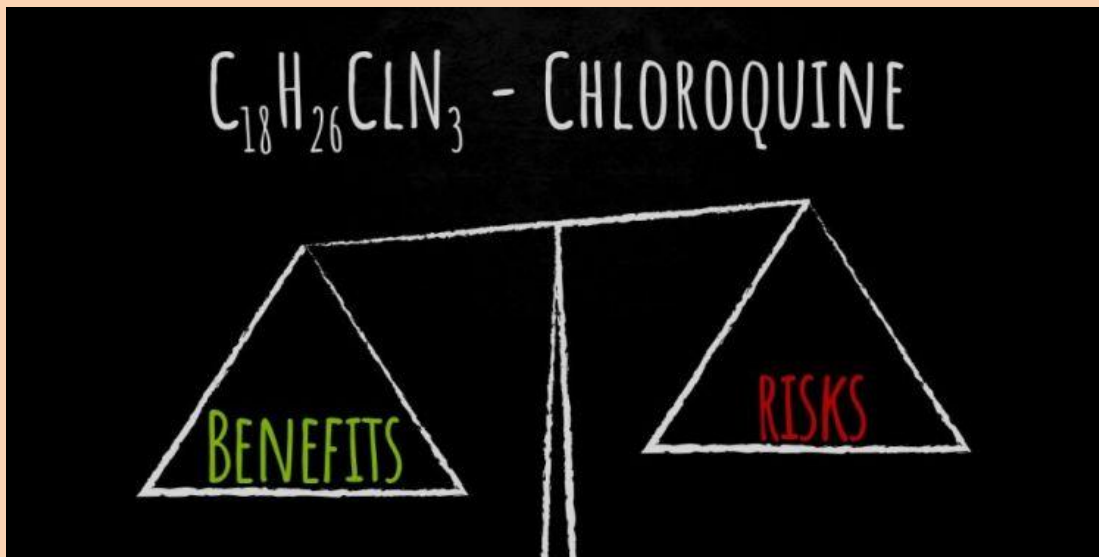
“Sterilization is the process of killing harmful microorganisms and bacteria on an object by subjecting them to an environment that they can’t endure.

Sterilization can be performed via two main routes: **heat application** and chemical means.

Heat is the most common form of sterilization and is used in most hospitals and doctors’ offices via an autoclave.”

<https://study.com/academy/lesson/how-to-sterilize-a-needle.html>

The WHO also states that the surgery department used syringes and needles that are autoclaved after use. Thus, we are supposed to believe that the hospital in Zaire apparently had different protocols for sterilizing syringes/needles between outpatient, inpatient, and the prenatal ward versus the surgery department. While possible, is it likely? Does it even matter? As we learned earlier, the WHO stated that “viral” transmission stopped as injections at the hospital stopped. They did not say injections using unsterilized needles. What the WHO wants us to believe is that the “virus” transmission stopped due to the “virus” not being transferred by injections using unsterilized needles being passed between the patients. However, as is always the case, there is a much more plausible and likely scenario.



The index patient was injected with chloroquine after being diagnosed with malaria. In fact, his gastrointestinal bleeding did not occur until after the use of chloroquine. This anti-malaria drug is a well-known for its toxic and serious side effects. For a full listing of the reported side effects you can click [here](#) but I want to highlight a few of the relevant ones:

- **back, leg, or stomach pains**

- black, tarry stools
- blood in the urine or stools
- fever
- skin rash, hives, or itching
- sore throat
- **unusual bleeding or bruising**
- upper right abdominal or stomach pain
- vomiting

Now reread this section from earlier in the WHO's report:

"After 3-4 days of non-specific symptoms and signs, patients typically experienced progressively **severe sore throat**, developed a maculopapular **rash**, had intractable **abdominal pain**, and began to **bleed from multiple sites**, principally the **gastrointestinal tract**."

Looks like the symptoms for severe Ebola line up perfectly with the side effects from chloroquine injection. Go figure. ☐♂

Interestingly, chloroquine has been studied as a drug to treat Ebola patients yet in animals models, it led to worsening of the conditions and death:

"However, while chloroquine inhibited Ebola virus replication in vitro, **it caused rapid worsening of Ebola infection in guinea pigs and made no difference to mortality in mice or hamsters**."

More about chloroquine and hydroxychloroquine

"When the same dose (90 mg/kg) of chloroquine was given to hamsters challenged with MA EBOV, the study had to be terminated on day 2 after treatment. **Nearly all the treated animals, in both the MA EBOV and the mock-challenged groups, died of acute toxicity after administration of chloroquine intraperitoneally, typically within 30 min after treatment (Figure 2, panel B)**."

https://wwwnc.cdc.gov/eid/article/21/6/15-0176_article

Thus, we must ask ourselves what makes more sense logically. **Was it the "unsterilized" needles spreading a new "virus" or the side effects of injections of toxic drugs which resulted in the symptoms claimed to be Ebola?**



Lethal injection.

The following section below details what a probable, possible, and proven case of Ebola is. According to the WHO:

- **Probable:** living in endemic area, had received an injection or been around a probable or proven case and died after experiencing 2 or more symptoms
- **Proven:** the “virus” was either “isolated” or demonstrated by electron microscopy or they had antibody titres within three weeks of symptoms
- **Possible:** a person with a headache or fever with or without other symptoms who had contact with a probable or proven case

Interestingly, **the WHO notes that possible cases were treated with antimalarial drugs, antibiotics, and antipyretics to exclude other diseases common to the area.**

In other words, if they weren't a probable/proven case before treatment, they most likely were afterwards:

“A probable case of Ebola haemorrhagic fever was a person living in the epidemic area who died after one or more days **with two or more of the following symptoms and signs: headache, fever, abdominal pain, nausea and/or vomiting, and bleeding.** The patient must have, within the three preceding weeks, **received an injection** or had contact with a probable or a proven case, **the illness not having been otherwise diagnosed on clinical grounds.** A proven case was a person from whom Ebola virus was **isolated or demonstrated by electron microscopy or who had an indirect fluorescent antibody (IFA) titre of at least 1:64 to Ebola virus within three weeks after onset of symptoms.** **An Ebola virus infection was deemed to have occurred in persons who had a similar IFA antibody titre, but had not been ill during the period 30 August to 15 November 1976.**

A possible case was a person with headache and/or fever for at least 24 hours, with or without other signs and symptoms, who had contact with a probable or a proven case within the previous three weeks. These patients were treated with antimalarial drugs, antibiotics, and antipyretics to exclude other diseases common to the area. Persons reporting such symptoms retrospectively were bled and their sera were tested for Ebola virus antibodies. Also any case of fever with bleeding reported to

the Ministry of Health from any part of Zaire, whatever the clinical outcome, was regarded as a possible case, and every effort was made to establish a diagnosis by virological or histopathological means.”



What is interesting about this next section is how the surveillance teams set out to find cases of the new disease and how they apparently educated the villagers about a disease that they were supposedly still trying to study and understand themselves. The teams were said to have been educated on the differential diagnosis of Ebola from other diseases:

“The objectives of surveillance teams were to find past and active cases of Ebola haemorrhagic fever, to detect possible convalescent cases, to educate the public as to the nature of and means of preventing the disease, and to establish beyond question the termination of the outbreak. Ten special active surveillance teams were recruited and trained. Each consisted of four persons; a team leader (physician or nurse), two nurses, and a chauffeur. **The subjects covered during training were the differential diagnosis of Ebola haemorrhagic fever, its epidemiology (including possible modes of transmission), the means of protecting personnel, and methods for obtaining family census data and recording probable and possible cases.** The teams were provided with standard forms, a written schedule, and detailed maps showing the villages they were to cover during a two-week period. Each team was assigned a four-wheel-drive vehicle, some of which had radios, and was provided with food, water, gowns, caps, gloves, boots, respirators, and equipment for obtaining blood samples. **Chloroquine, tetracycline, aspirin, and a drug against intestinal parasites were all supplied in tablet form**ⁱ. A physician supervised five teams by frequent field visits and administrative reviews.”

According to the Medical Dictionary, differential diagnosis is defined as:

1. A list of conditions that may cause a **particular clinical sign or symptom.**
2. The arrival at a diagnosis by means of **comparing the similarities and differences in various clinical signs.**

<https://medical-dictionary.thefreedictionary.com/differential+diagnosis>

Differential diagnosis of an acute, severe, short-incubation (<21 days), febrile illness from sub-Saharan Africa includes:

- Malaria (especially falciparum malaria)
- Typhoid fever*
- Other bacterial enteritides*
- Leptospirosis
- Rickettsial infections
- Plague*
- Viral hepatitis*
- Dengue
- Chikungunya
- Yellow fever
- Rift Valley Fever
- Lassa fever*
- Marburg hemorrhagic fever*
- Severe influenza*
- African trypanosomiasis
- Bacterial pneumonia
- Meningococemia*
- EVD*

"Many of these diseases **present non-specifically**, often with fever and malaise. In advanced cases, hemorrhage is common, **but may be present in only about 50% of EVD cases**. There have been at least 20 suspected cases of EVD in the U.S.; however, **only about**

one-fifth of these cases met criteria for testing. Nevertheless, these suspected cases drain limited healthcare resources because of the heightened concern for EVD. **Some of the patients ultimately had malaria and influenza.**" <https://westjem.com/perspective/ebola-virus-disease-essential-public-health-principles-clinicians.html>

In order to diagnose a condition, there must be differences in the clinical signs and symptoms. This creates a bit of a problem as the **symptoms of Ebola mimic many other more common diseases including influenza, malaria, typhoid fever, meningitis, yellow fever, and even pregnancy**. There are no obvious differences in symptomology which is why the CDC and the WHO both state that diagnosis based on symptoms alone is difficult (i.e. impossible) and requires indirect laboratory methods to confirm infection. **The CDC states that diagnosis of Ebola requires use of PCR in order to "confirm" a case** which was not available to researchers in 1976 as PCR was not invented until 1983. In the WHO's August 2014 ***Ebola and Marburg virus disease epidemics: preparedness, alert, control, and evaluation***, it is stated that cases must be laboratory confirmed, reiterating that it is done either by **way of PCR or by non-specific IgM antibody tests:**

"LABORATORY-CONFIRMED CASES:

Any suspected or probable cases with a positive laboratory result. Laboratory-confirmed cases **must test positive for the virus antigen, either by detection of virus RNA by reverse transcriptase-polymerase chain reaction (RT-PCR), or by detection of IgM antibodies directed against Marburg or Ebola.**"

There is absolutely no way any of these teams would have been able to differentially diagnose anyone based on clinical symptoms alone. In fact, **the researchers in 1976 had 15 possible cases of haemorrhagic fever that mimicked Ebola:**

“Fifteen **possible cases** of haemorrhagic fever occurring outside the main epidemic area were investigated from Kinshasa. **Ebola haemorrhagic fever was ruled out in each instance on clinical, virological, or pathological grounds.** Final diagnoses included **typhoid fever, viral hepatitis, amoebiasis, acute pulmonary oedema, and carbon monoxide poisoning.**”

They ultimately decided these 15 cases of haemorrhagic fever were not real cases of Ebola as the results of previously established indirect methods led to diagnoses such as typhoid fever, “viral” hepatitis, amoebiasis, acute pulmonary oedema, and carbon monoxide poisoning. The only method to “diagnose” an Ebola case was either through elimination based on these previously established indirect methods for other diseases associated with the same symptoms or by way of non-specific indirect antibody results. This was the circular way in which they can claim a person experiencing a set of symptoms is an Ebola patient in one case while someone else with the same symptoms is a malaria patient in the other. They love to ignore the fact that, **in order for any of these tests to be accurate, not only must the “virus” in question be purified and isolated first in order to calibrate and validate the tests, disease prevalence must also be known first. Disease prevalence can only be determined by clinical diagnosis through differentiating symptoms between these diseases which is an admitted impossibility,** thus the case results in 1976 on up to today are utterly meaningless.

Origin and course of the epidemic



As I previously spoiled, **we find out here that the initial Ebola patient was diagnosed with malaria, given a chloroquine injection, and had his symptoms subside for a**

few days only to have them come back worse than before. The patient ultimately died of gastrointestinal bleeding a few days later:

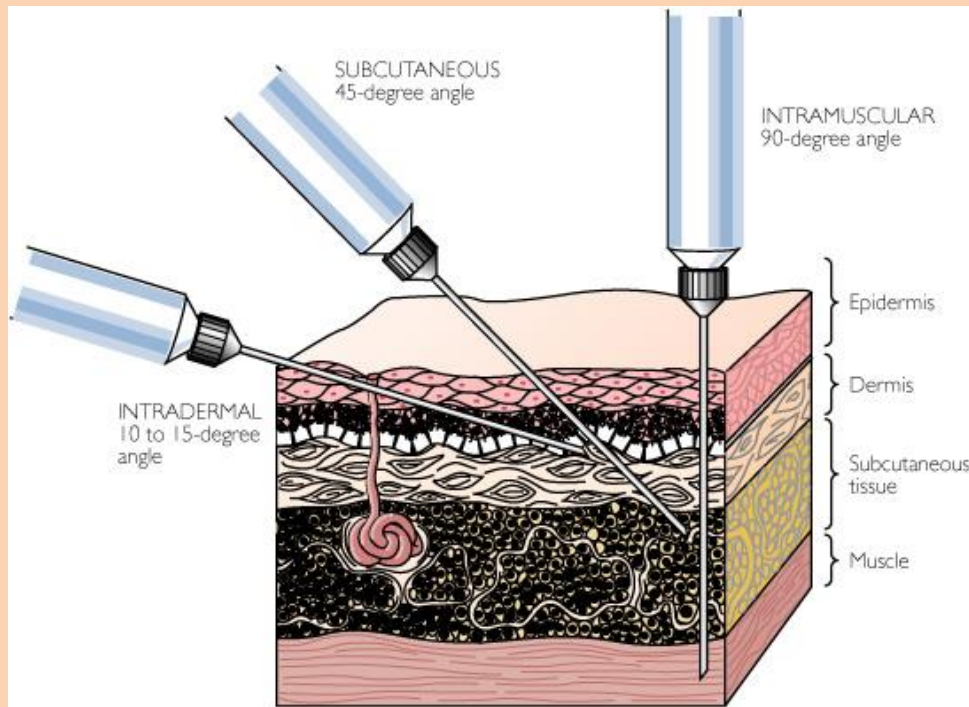
"The first known case, a 44-year-old male instructor at the Mission School, presented himself to the outpatient clinic at **Yambuku Mission Hospital (YMH)** on 26 August 1976 **with a febrile illness thought to be malaria**. This man had toured the Mobaye-Bongo zone in the northern Equateur Region by automobile from 10 to 22 August with 6 other Mission workers. The group visited some of the larger towns (Abumombazi, Yakoma, Katokoli, Wapinda) along the road from Yambuku to Badolit, but never arrived at that village because a bridge had been washed away a few kilometres east of the town. On 22 August, fresh and smoked antelope and monkey meat were purchased on the road about 50 km north of Yambuku. The patient and his family ate stewed antelope on his return, but not the monkey meat. **He was given chloroquine by parenteral injection on 26 August**. His fever resolved rapidly and he was afebrile until 1 September when he again had fever to 39.2° C. **Other symptoms and signs ensued and he was admitted to YMH on 5 September with gastrointestinal bleeding. He died on 8 September.**

It is also noted **that 9 other cases of haemorrhagic fever occurred within the first week and all of them had been treated for other diseases at the hospital. None of the initial diagnoses for these 9 cases were recorded.**

At least 9 other cases occurred during the first week of September, all among persons who had received treatment for other diseases at the outpatient clinic at YMH. Names of persons treated at the outpatient clinic and specific diagnoses were not recorded. Thus, it was impossible to determine whether persons with fever had visited YMH in late August. It was of interest, however, that a man about 30 years of age had been admitted to the medical ward on 28 August suffering from "dysentery and epistaxis", a diagnosis not otherwise listed in the preceding eight months. This man, listed as resident in Yandongi, the capital village of the collectivity some 7 km from Yambuku, was taken from the hospital two days later. He turned out to be a person completely unknown to the residents and authorities of Yandongi.

However, it was stated that **parenteral injection was the principal mode of administration of nearly all medicines:**

Case histories quickly suggested that YMH was a major source of dissemination of Ebola haemorrhagic fever. **It was learned that parenteral injection was the principal mode of administration of nearly all medicines."**



For those unfamiliar, parenteral injections are given through the skin usually in one of five ways: subcutaneous (into the fat), intraperitoneal (in the stomach), intravenous (in the veins), intradermal (under the skin), and intramuscular (in the muscle). This process bypasses the skin and mucous membranes leading to some notable drawbacks for this mode of treatment such as:

1. Drug administration by these routes is irreversible and **poses more risks than the other routes**
2. It is an invasive route of drug administration and thus, it can cause fear, pain, **tissue damage, and/or infections.**
3. Injections have limitations for the delivery of protein products, particularly those that require sustained levels.
4. It is **generally riskier.**
5. **The preparation to be injected has to be sterile.**
6. Drug administered by parenteral routes with the exception of intra-arterial route might still be eliminated by first-pass metabolism in liver prior to distribution to the rest of the body.

7. Help is always needed to administer a parenteral dosage form.

<https://www.pharmapproach.com/parenteral-route-of-drug-administration-advantages-and-disadvantages/>

This affirms that the **medical interventions utilizing risky injections with known toxic side effects were the most likely source of disease, including symptoms such as gastrointestinal bleeding.** Of course, in the eyes of the WHO, it couldn't be that the injections of toxic medications were capable of producing said symptoms and were the likely culprit of disease as it had to be a new "virus." However, how they could possibly conclude this after these next admissions is beyond me:

"All ages and both sexes were affected (Table 3) but females slightly predominated. Age/sex attack rates, using the Yandongi collectivity as the population denominator, showed that adult females had the highest attack rate. **Much of this excess illness was associated with receipt of parenteral injections at YMH or one of its clinics.** The distribution of disease by age group and type of transmission was essentially equal for both sexes **except for injection-associated illness** among persons 15-29 years old. Females comprised 22 of 24 such cases in the 21-village study.

The single common risk factor in comparison with matched family and village controls for 85 of 288 cases where the means of transmission was determined, was receipt of one or more injections at YMH. Injections received away from YMH were very unusual. Other factors **such as previous case-contact**, exposure to food, water, hospital buildings, domestic and wild animals, or travel within three months prior to onset, **were not associated with this type of transmission.** An additional 149 persons acquired the disease following contact with patients, usually in their home villages, **and 43 cases had a history of both patient contact and receipt of injection within three weeks prior to onset of illness. Seventeen persons who lived outside Yambuku had contact at YMH and may have received injections there without reporting this fact to their family.**

Most of the cases related to injection occurred during the first 4 weeks of the epidemic (Fig. 4). **Indeed, it seems likely that closure of YMH was the single event of greatest importance in the eventual termination of the outbreak.**

Several parameters were compared for **persons acquiring infection by contact and injection, respectively.** Although no statistically significant differences were found in terms of duration of symptoms and signs of illness (Table 4), **no person whose contact was exclusively parenteral injection survived the disease."**

According to the WHO, the excess illness seen was associated with the injections. The disease in women 15-29 years old was higher for those who had been injected during pregnancy. They called these "injection-associated illnesses." The single common denominator amongst all cases was the receipt of one or more injections of medications capable of producing the symptoms associated with the disease. **There were no survivors amongst those who received the injections and after stopping these treatments, the outbreak ended. Even after summarizing these events, the invasive injection and the toxic medications were seemingly not a concern for the WHO.** As is always the case, all other much more logical potential causes were pushed aside for the illogical invisible enemy.

"Five consecutive generations of transmission of Ebola haemorrhagic fever were documented in one instance. **No sporadic, apparently spontaneous, probable cases were recorded.** When "family" was defined as all persons living in contiguous housing and sharing common eating facilities, **secondary attack rates never exceeded 8 % (Table 6).**"

"In December 1976 and January 1977, sera were solicited from as many people as possible; a total of 236 were obtained. **Three persons, 2 of them in clinically noninfected households, who had not had symptoms during or since the epidemic, were found to have Ebola virus IFA titres of at least 1:64.** All 3 had experienced contact with fatal cases."

Serological and ecological studies



The test says Ebola "Virus" on it so it must be specific. □

In these next few highlights, we find that the antibody measurements, said to be specific and used for diagnosis, are not so specific and were in doubt. The WHO even stated that they were awaiting development of a type-specific method for final interpretation of the antibody results. It is also shown that **there were positive antibody reactions found**

within healthy people who were not recently sick, had no contact with any probable or proven cases, and had no history of visit to the hospital. In other words, **the WHO claimed to have found asymptomatic cases of Ebola using antibody tests which they admit produced doubtful results.**

"Serum specimens were obtained in November and December 1976 and January 1977 from 984 persons resident in 48 of the 55 towns and villages reporting probable cases of the disease. These individuals were classed as clinically ill, not ill but in contact with a case, or neither ill nor in contact. More than half of the subjects were resident in 8 villages, each having more than 5 probable cases. These persons were bled during rapid survey excursions, taking the entire family as the unit of study. The composition of these groups by age, sex, and epidemiological characteristic is given in Table 7 together with the number and category of persons having Ebola virus IFA titres of at least 1:64. Data from Yamolembia I are included. Thirty-eight positives were found. Twenty (16.5%) of 121 ill persons were confirmed as having had Ebola haemorrhagic fever, and 10 (2.5%) of 404 persons in contact with cases also had such antibodies. **There were 4 antibody-positive persons who admitted neither illness nor contact with patients. These people were questioned a second time and bled again, and confirmed to have Ebola IFA antibodies.** Antibodies were found also in sera of 4 people whose history was not clear and who could not be found a second time for confirmatory study.

In a further effort to document either concurrent asymptomatic infection or possible past infection with Ebola virus, 442 persons were bled in 4 neighbouring villages that had had no fatal cases of the disease. Sera from 5 persons 8-48 years old contained IFA antibodies in titres of 1:64. **None of these people were sick, had had contact with persons in other villages, or had visited YMH during the epidemic.**

Sera from 58 persons in various exposure categories had anti-Ebola IFA titres of 1:4-1:32. **The specificity of these reactions was doubted** when it was found that samples from 4 of 200 San Blas Indians from Panama also had such "antibodies" for Ebola but not Marburg virus. **Final interpretation of these data awaits development of another method for measurement of type-specific antibodies to these agents."**

The asymptomatic infection excuse is still in use today to cover up finding so-called "specific" antibody results in healthy people. From a 2014 *Lancet* study:

"Evidence suggests that **many Ebola infections are asymptomatic,** a factor overlooked by recent outbreak summaries and projections. Particularly, results from one post-Ebola outbreak serosurvey **showed that 71% of seropositive individuals did not have the**

disease; another study reported that **46% of asymptomatic close contacts of patients with Ebola were seropositive.**"

[https://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(14\)61839-0/fulltext](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(14)61839-0/fulltext)

Hospitalized patients

Here we find that, in 3 cases of EBV at the Ngaliema Hospital, the patients all presented with similar symptoms in the initial stages including fever, headache, anorexia, vomiting, a rash, haemorrhage, and a severe sore throat. However, after a period of days, the symptoms varied between the three patients. This may just have something to do with the fact that each patient was subjected to different toxic treatment cocktails which are outlined in the section below. Not surprisingly, all three succumbed to their ailments after treatment. It was determined that they all suffered **hypovolaemic shock, which is a condition where there is sudden and severe blood loss that leads to the heart being unable to pump enough blood throughout the body, eventually resulting in multiple organ failure. One possible cause of this shock is damage to the stomach. What could possibly lead to such an outcome in these patients? Perhaps trauma to the gastrointestinal organs by way of multiple injections of cocktails of toxic medications known to damage the stomach?:**

"Early symptoms and signs in all 3 patients at Ngaliema Hospital included fever, headache, anorexia, and vomiting. A morbilliform rash appeared on the anterior trunk in each of these patients on day 5 or 6, spread to the back and limbs, then faded within 48 hours. Haemorrhage and severe sore throat began between the fourth and seventh days of illness.

One patient had oral and conjunctival petechiae beginning on day 4, haematemesis and melaena from day 4, gingival bleeding on day 7, and bleeding from injection sites on day 8. Another had melaena only, beginning on the sixth day of illness. The third patient had a single episode of haematemesis on day 7 followed by melaena and ecchymosis on the next day. Progressive glossitis and pharyngitis beginning on day 3 were noted in one patient who developed severe erythema and oedema of the soft palate and pharynx leading to pronounced dysphagia. All three patients were febrile throughout the course of illness, with temperatures frequently above 39°C. Two patients had terminal tachycardia. One patient died on day 7 and two on the eighth day of illness.

Clinical laboratory tests were done on the first patient, but only a few measurements were carried out on the other two cases to avoid undue exposure of hospital laboratory staff to the virus. Leucocyte counts on the first patient were 7600 and 8900/mm³ on days 5 and 7, respectively. Platelets on days 4, 6, and 7 were 162,000, 150,000 and 150,000/mm³; these were days when frank haemorrhage occurred. During

this time, serum SGOT rose from 90 to greater than 200 units/ml and the SGPT increased from 40 to more than 200 units/ml. Serum bilirubin rose from 25.6 $\mu\text{mol/l}$ on day 5 to 59.8 $\mu\text{mol/l}$ on day 7. Partial thromboplastin time (PTT) was 47 seconds on the fifth day. This patient produced only 200 ml of urine on the seventh day and none during the next day when she died. The second patient, on whom no laboratory tests were done, became anuric during the last 2 days of life.

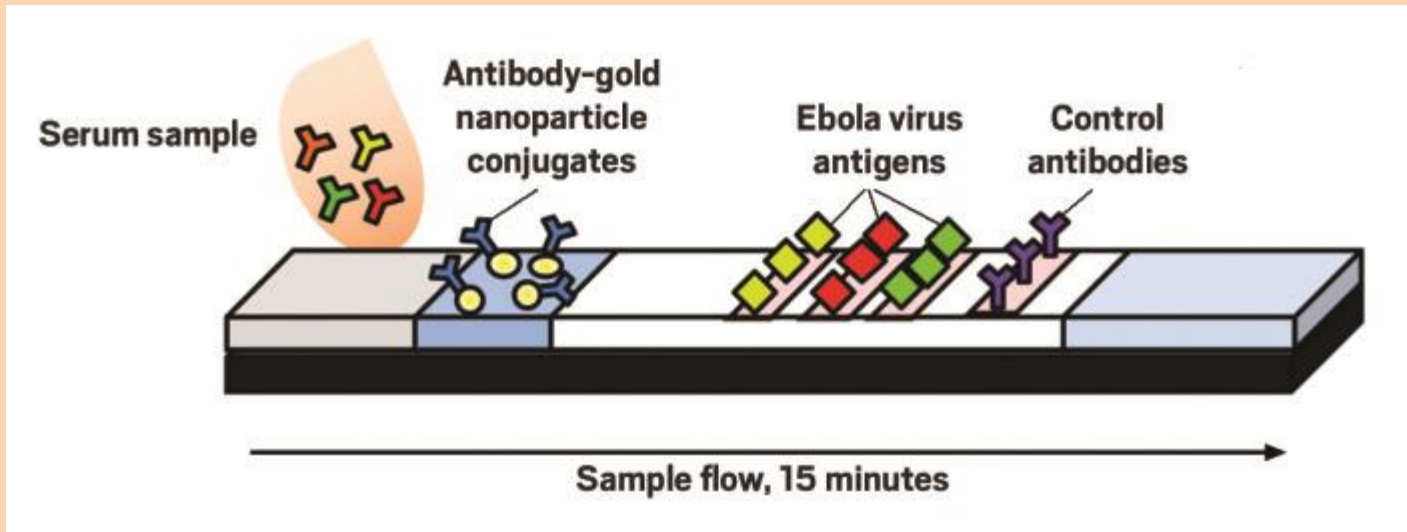
The third patient had white blood cell counts of 9,400 and 12,300/ mm^3 on days 7 and 8, respectively. Platelet counts on these days were 253,000 and 205,000/ mm^3 , while P17 values were 45 and 50 seconds, respectively. Fibrin degradation products, measured with a commercial kit (Burroughs-Wellcome) were recorded as 1+ and 2+ on days 7 and 8.

The first case was treated with aspirin, antibiotics, corticosteroids, blood transfusion, and intravenous fluids. The second patient received aspirin, hydrocortisone, immunoglobulin, intravenous fluids, and an experimental drug, moroxydine. Enterovioform was given to control diarrhoea but without success. The third patient was treated for unconfirmed malaria during the first 2 days of illness. When the etiological agent of the epidemic was shown to be a Marburg-like virus, she was given, on day 4, 500 ml of Marburg human plasma obtained from a recovered patient in South Africa. This plasma had an IFA titre of 1:32. In anticipation of disseminated intravascular coagulation (DIC), she was given 16,000 units of heparin on day 6 and 30,000 units daily thereafter. Although anticoagulation was unsatisfactory, as shown by the normal PTT on days 7 and 8, she had less clinical bleeding than the other two patients. **On the day prior to death she complained of substernal chest pain and had a tachycardia of 136 with a gallop rhythm.** Digitalization slowed this rate only slightly. Marked oedema of the face and upper limbs was present.

Although no autopsies were performed, it appeared clinically that these patients died of hypovolaemic shock. Evidence for DIC was fragmentary, but this syndrome may well have precipitated the bleeding and shock in all cases. Postmortem liver biopsy in the first case revealed marked focal hepatic-cell necrosis with large intracytoplasmic eosinophilic inclusions. **Marburg virus-like particles were visualized with an electron microscope (2).**

Ebola virus was recovered on day 6 from blood specimens from patient 1 and on days 3 and 6 from patient 2. Quantitative virus assays on blood from the third patient are shown in Table 8. **No IFA antibodies against Ebola or Marburg viruses were present.**

Retrospective field studies



How they want you to believe the antibody tests are made.

If you needed more reason to doubt antibody results, look no further. In this survey of 231 probable cases, 34 had IFA antibodies. 59% of those with antibodies had symptoms thus leaving 41% of those probable cases asymptomatic. Many others who had direct contact with fatal cases reported having had symptoms but no antibodies whatsoever. In other words, **many of those who were sick had no antibodies and nearly half of those who had antibodies were never sick:**

Questionnaire forms were completed on 231 probable cases 1 year of age or older, 34 individuals who were found to have Ebola virus IFA antibodies, and 198 controls. The numbers of responses obtained for each symptom and the percentages responding positively in these groups are shown in Table 9. Fever and headache were almost invariably present. The headache often radiated to the cervical spine and was associated with low-back pain radiating into the legs. Sore throat was often reported in association with a sensation of a "ball" in the throat. Chest pain and pleuritis were uncommon. **Of persons with antibodies, 59% had one or more symptoms, the most prominent being fever, headache, abdominal pain, and arthralgia. Many more persons who had been in contact with fatal cases reported symptoms but had no Ebola virus antibodies.** Illness in antibody-positive individuals was, in general, marked by profound prostration, weight loss, and a convalescent period of 1-3 weeks."

According to the WHO, a urinary protein test, one not in use as a diagnostic today, was utilized early on in the outbreak to diagnose cases. How many of these patients were misdiagnosed based on a faulty test?

"The **only clinical laboratory test done** on patients admitted to Yambuku Hospital **was urinary protein**. This was reported as uniformly positive and **was used as a major diagnostic criterion** by the nursing sisters early in the epidemic.

The WHO admits that the virological studies, arguably the most important part of the (pseudo)scientific evidence needed to understand a new "virus," were limited. This is apparent as they used small sample sizes where "virus isolation" was only attempted in 10 cases and only 4 liver biopsies were performed:

Virological studies were limited. Ebola virus was isolated in African green monkey kidney cells (Vero) from blood specimens in 8 of 10 cases attempted. These specimens were taken 2-13 days after onset of symptoms. Of interest was the simultaneous detection of virus and IFA Ebola antibodies to a titre of 1:32 in one patient. This man was in the 13th and penultimate day of his illness. **Ebola virus particles were also visualized in 3 of 4 postmortem liver biopsies obtained from clinically suspect cases."**

The Ebola "virus" was said to have the highest case mortality rate since rabies. Perhaps the toxic cocktails injected into these patients may have helped bring about the high amounts of fatality?:

"No more dramatic or potentially explosive epidemic of a new acute viral disease has occurred in the world in the past 30 years. **The case mortality rate of Ebola haemorrhagic fever in Zaire of 88% is the highest on record except for rabies infection.** In the circumstances it was not surprising that much desired information was never obtained. **Delays in recognition, notification to international health agencies, and specific diagnosis of the disease contributed greatly to this outcome.** No better example comes to mind to illustrate the need for national disease surveillance and the prompt solicitation of international assistance, nor of the need for the development of international resources, comprising personnel, equipment, transport, communication, and finance, that can be made available in a very few days to cope with such emergencies.

Interestingly, both Ebola and rabies have a connection to dangerous injections. In the case of rabies, until the 1980's, the treatment involved a series of shots in the stomach:

"There has been confusion among the general public because of historical fears and **the painful injections of anti-rabies vaccination (nerve tissue vaccine administered over the abdomen) given in the past."**

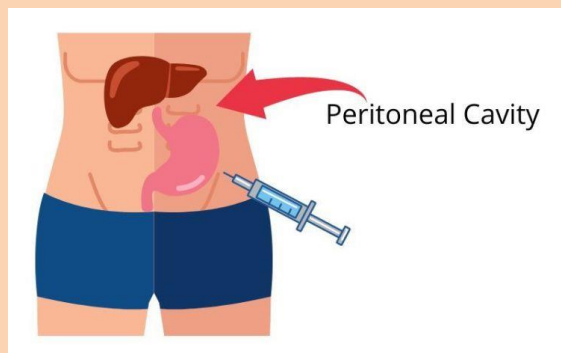
<https://www.google.com/url?sa=t&source=web&rct=j&url=https://www.who.int/docs/default-source/searo/india/health-topic->

[pdf/b5010.pdf%3Fsfvrsn%3D619e77a3_2&ved=2ahUKEwian7aUorb6AhXdkIkEHWPEBOg4ChAWegQIFRAB&usq=AOvVaw2S3jACpOqMEg9qBA6fmZj](https://www.oklahoman.com/story/lifestyle/health-fitness/2013/07/14/whats-it-like-to-get-a-rabies-shot/60899910007/)

In fact, there could be as many as 21 injections into the abdomen for rabies:

“Several years ago, **treatment for rabies included 21 injections into a person’s stomach.** It was extremely painful and involved a long needle. However, since the early 1980s, there’s a much different rabies vaccine to treat humans for rabies exposure.”

<https://www.oklahoman.com/story/lifestyle/health-fitness/2013/07/14/whats-it-like-to-get-a-rabies-shot/60899910007/>



As intraperitoneal injections (in the stomach cavity) are one of the 5 routes used for parenteral treatment, could these Ebola patients in 1976 have been given the toxic drugs in this manner as was done for rabies until the 1980’s? Injections of toxic drugs directly into the stomach would be a possible explanation for an increase in gastrointestinal bleeding. Damage to the stomach by way of trauma is also a risk factor for hypovolaemic shock as suffered by the patients in Ngaliema Hospital. Unfortunately, the WHO does not define the route of parenteral injection so we can only speculate.

These next few sections offer some random pieces of information that poke holes into the WHO’s story. First, you will see the WHO admit that laboratory data was virtually non-existent for this outbreak, yet they confirm that the clinical picture resembled the Marburg “virus.” They thought that the agents “isolated” in Sudan and Zaire were identical but they had yet to perform the (previously admitted to be doubtful) antibody testing to confirm this. **The WHO claimed that viremia (the presence of “virus” in the blood) is a constant feature of the “virus” based on a single study involving the small sample size of just one patient.** However, the WHO then stated that no evidence was obtained for persistent “viral” carriage in the Zaire cases of Ebola infection, a phenomenon documented on two occasions for Marburg “virus,” but warn that this information is based on a small sample size. It seems very clear that the WHO likes to have their cake and eat it too:

Although laboratory data were virtually non-existent, the clinical picture seen in this outbreak resembled illness produced by the related Marburg virus. If anything, the evolution of Ebola haemorrhagic fever appeared to be more inexorable and less variable than Marburg virus infection. Though far from proven, we suspect that acute defibrination syndrome and pancreatitis were major features of the syndrome and severe liver disease was evident.

In contrast to observations made simultaneously in Sudan, the illness in Zaire had fewer respiratory symptoms, a shorter clinical course, and a higher fatality rate (4). Whether this was due to differences in the virulence of the virus per se or to host and ecological variables such as climate (relative humidity) is not known. **At the present time the agents recovered from Sudan and Zaire are thought to be identical, although definitive neutralization tests have not yet been done.**

Viraemia appears to be a constant feature of Ebola virus infection in man. The virus persisted in large amounts in the blood in the single, well studied case. The finding of both virus and antibodies in the blood of another agonal case 13 days after onset of symptoms raises the possibility, regarded as unlikely, that antigen-antibody complexes may contribute to the pathology of infection. **This and a number of other important virological questions can only be pursued for the moment through studies using monkeys.** One of the most pressing is the need for a way to make rapid diagnosis in suspected cases of the disease by searching for cells containing viral antigen. Retrospective specific diagnosis of fatal cases by electron microscopic examination of formalin-fixed liver biopsies appears quite promising and should be attempted in all cases of acute febrile haemorrhagic disease in Africa.

No evidence was obtained for persistent viral carriage in the Zaire cases of Ebola infection, a phenomenon documented on two occasions for Marburg virus (5, 6). But it should be remembered that the number of appropriate, immunosequestered sites sampled was very small. However, semen from one patient infected with a Zaire strain of Ebola virus in the United Kingdom contained virus for more than 2 months after onset of symptoms (3).

In the final highlights, we get the WHO's simplified version of events that reiterates earlier points which should have cast doubt on their conclusions that a new "virus" was the cause of an outbreak of a new disease. First, we get the admittance that the Ebola "virus" had a low rate of secondary person-to-person transmission, meaning it did not easily spread even in cases of close contact with "infected" individuals. They state that the way the new "virus" made it to the hospital will never be known, yet they believed it was brought from the Sudan by man. **The way that it was decided that the "virus" spread was through the contaminated needles and syringes used for injections into the sick patients. Once these injections stopped, so too did the outbreak:**

The Zaire epidemic had all the attributes of a common source outbreak, together with a fortunately low rate of secondary person-to-person transmission. The means by which the virus was introduced into Yambuku Mission Hospital will probably never be precisely known, but it seems possible that it was brought directly from the Sudan by man. **Dissemination of the agent into the villages of the region was principally through contaminated equipment used for parenteral injections.** The epidemic waned when the hospital was closed for want of medical staff. That careful disposal of contaminated excreta and fomites, as well as strict barrier nursing using respirators, could break the chain of transmission was demonstrated during the small outbreak in Kinshasa. Still simpler isolation precautions and a change in the cultural customs at funerals appears to have contributed to the dying out of infection in the villages.

Interestingly, **the WHO admitted that cases brought about by injection were different and more likely to be fatal than those that they claimed were secondary cases acquired without injections. This obviously makes sense when viewed from the standpoint that the non-specific symptoms of disease were the result of the unnatural injection of various toxic medications directly into the patients and not the effect of a new "virus."** Secondary cases were nothing more than looking for similar symptoms in villagers and using fraudulent antibody results to claim the symptoms were caused by the same imaginary "virus:"

Although the data were not always statistically convincing, **we had the strong impression that Ebola haemorrhagic fever acquired by injection differed from that due to contact with another case. The mortality was higher. In one study, secondary transmission rates also were higher from index cases that were parenterally induced.** It may be that increased virus replication and excretion following parenteral infection accounts for all or most of these differences, but other causes were by no means excluded.

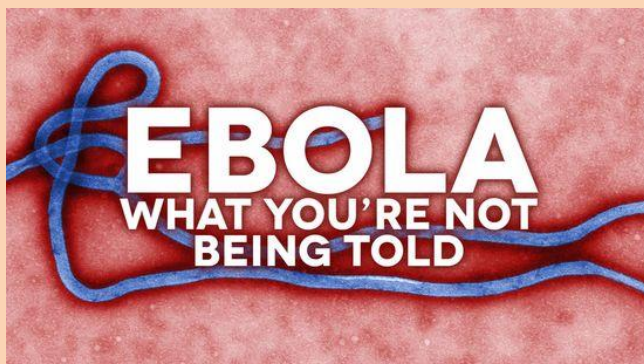
The WHO went on to admit that "neonatal" cases were not definitively elucidated. In other words, they could not explain how the babies acquired the disease from the mother. There was apparently no questioning of the injections of harmful medications into the pregnant women potentially bringing about death and disease in infants. It had to be this magical "virus" somehow passing through the placenta and infecting the unborn child:

The observed "neonatal" cases of the disease were not definitively elucidated. One wishes to know whether Ebola virus can pass through the placenta and infect the fetus, and whether virus is present in human milk and is infectious if ingested.

Once again hammering the final nail in the coffin of the Ebola “virus,” the WHO reminded us that better antibody tests were needed in order to interpret the results. While fraudulent antibody results should never carry any weight as evidence, these findings were the only way the researchers could differentiate Ebola from “Marburg” and many other “viruses,” thus their questionable “accuracy” speaks volumes. The IFA results were essential to the case that Marburg and Ebola were different “viruses” yet the WHO was unsure if these results were correct. They further add that, like the Marburg “virus,” the source of the Ebola “virus” was unknown. This should tell you everything you need to know:

Finally, a better method for measuring Ebola virus antibodies is needed in order to interpret the serological findings reported here. That less than 20% of persons gave a history of acute illness following contact with a fatal case was no surprise. Most of these persons had mild, self-limiting diseases, these being highly endemic in the area. **But if the IFA data are correct, at least 2.5% of persons in contact with fatal cases experienced subclinical infection. In addition, the finding of antibodies in a few individuals in the absence of any known contact with Ebola virus during the epidemic raises the possibility that the agent is in fact endemic in the Yambuku area and is occasionally transmitted to man.** A definitive answer is essential to further ecological exploration of what is now a very mysterious agent. **As in the case of Marburg virus, the source of Ebola virus is completely unknown beyond the simple fact that it is African in origin.”**

[bullwho00439-0113-1](#) [Download](#)



In Summary:

- Between September 1st and October 24th, 1976, 318 cases of (*assumed*) acute “viral” haemorrhagic fever occurred in northern Zaire
- There were 280 deaths, and only 38 **serologically confirmed** (*i.e. non-specific antibody results*) survivors

- The index case in this outbreak had onset of symptoms on September 1st, 1976, five days **after receiving an injection of chloroquine for presumptive malaria** at the outpatient clinic at Yambuku Mission Hospital (YMH).
- He had a **clinical remission** of his malaria symptoms
- Within one week several other persons **who had received injections at YMH** also suffered from Ebola haemorrhagic fever, and almost all subsequent cases had **either received injections at the hospital** or had had close contact with another case
- All ages and both sexes were affected, but women 15-29 years of age had the highest incidence of disease, **a phenomenon strongly related to attendance at prenatal and outpatient clinics at the hospital where they received injections**
- Intensive search for cases in the area of north-eastern Zaire between the Bumba Zone and the Sudan frontier near Nzara and Maridi **failed to detect definite evidence of a link between an epidemic of the disease in that country and the outbreak near Bumba**
- Nevertheless it was established that people can and do make the trip between Nzara and Bumba in not more than four days: **thus it was regarded as quite possible that an infected person had travelled from Sudan to Yambuku and transferred the "virus" to a needle of the hospital while receiving an injection at the outpatient clinic**
- *In other words, they could not establish any link between two outbreaks but still assumed it was possible for a hypothetical scenario where an unknown "infected" individual carried a new "virus" and made their way to the hospital and unknowingly transmitted the new "virus" by way of the same needle being used with other patients*
- **After 3-4 days of non-specific symptoms and signs**, patients typically experienced progressively severe sore throat, developed a maculopapular rash, had intractable abdominal pain, and began to bleed from multiple sites, principally the gastrointestinal tract
- **Although laboratory determinations were limited and not conclusive**, it was **concluded** that pathogenesis of the disease included non-icteric hepatitis and possibly acute pancreatitis as well as disseminated intravascular coagulation
- This syndrome was caused by a "virus" **morphologically similar to Marburg "virus,"** but immunologically distinct (*i.e. everything was identical besides non-specific antibody results*)
- The agent was "isolated" from the blood of 8 of 10 suspected cases **using Vero cell cultures**
- Ebola "virus" particles were found in formalin fixed liver specimens **from three cases**
- Survivors of infection were found to have **indirect fluorescent antibodies** to Ebola "virus"
- A total of 201 units (200-300 ml each) of plasma containing Ebola "virus" antibodies in titres of at least 1:64 were obtained and frozen and used on an "infected" person who recovered, which **suggested** to them that the antibodies may have helped

therapeutically (and the invisible antibodies may have done absolutely nothing while the person recovered despite their use so it amounts to pointless speculation)

- “Virus” transmission **was interrupted by stopping injections** and by isolation of patients in their villages (i.e. the stoppage of injections of chloroquine ended the “viral” spread... □)
- They somehow decided that the “virus” is **probably rarely transmitted** by infectious aerosols, **although infection via large droplets remains a possibility** (yet more evidence less and baseless speculation)
- Ebola “virus” antibodies were found in five persons **who were not ill and had not had contact with the “infected” villages or the Yambuku hospital during the epidemic** (in other words, the antibodies were either non-specific and/or there are asymptomatic Ebola patients walking around...or the more likely scenario that it is all fraudulent)
- **Five syringes and needles** were issued to the nursing staff each morning for use at the outpatient department, the prenatal clinic, and the inpatient wards
- These syringes and needles were **apparently not sterilized** between their use on different patients **but rinsed in a pan of warm water and at the end of the day they were sometimes boiled**
- This led the researchers to conclude it was a “virus” being transmitted from person-to-person due to needle use rather than the contents of the injection
- On a side note, **chloroquine injections, as was given to the patients, are known to cause the exact same symptoms associated with Ebola:**
 - **back, leg, or stomach pains**
 - **black, tarry stools**
 - **blood in the urine or stools**
 - **fever**
 - **skin rash, hives, or itching**
 - **sore throat**
 - **unusual bleeding or bruising**
 - **upper right abdominal or stomach pain**
 - **vomiting**
- A **probable** case of Ebola haemorrhagic fever was a person living in the epidemic area who died after one or more days with **two or more of the following symptoms and signs:** headache, fever, abdominal pain, nausea and/or vomiting, and bleeding
 - The patient must have, within the three preceding weeks, **received an injection** or had contact with a probable or a proven case, **the illness not having been otherwise diagnosed on clinical grounds**

- A **proven** case was a person from whom Ebola "virus" was "**isolated**" or **demonstrated by electron microscopy or who had an indirect fluorescent antibody (IFA) titre** of at least 1:64 to Ebola "virus" within three weeks after onset of symptoms
- An Ebola "virus" infection was deemed to have occurred in persons **who had a similar IFA antibody titre, but had not been ill** during the period August 30th to November 15th, 1976
- A **possible** case was a person with headache and/or fever for at least 24 hours, **with or without other signs and symptoms**, who had contact with a probable or a proven case within the previous three weeks
- These patients were **treated with antimalarial drugs, antibiotics, and antipyretics to exclude other diseases common to the area**
- *In other words, as long as you experienced one or two of the non-specific symptoms and either had an injection or been around those who were deemed probable/proven by way of indirect electron microscopy or non-specific antibody results, you were considered an Ebola patient*
- The objectives of surveillance teams were to find past and active cases of Ebola haemorrhagic fever, to detect possible convalescent cases, **to educate the public as to the nature of and means of preventing the disease**, and to establish beyond question the termination of the outbreak (*how did they educate the public on the nature and means of preventing a "virus" they were still supposedly identifying and studying? □*)
- The subjects covered during training were **the differential diagnosis of Ebola hemorrhagic fever**, its epidemiology (including **possible modes of transmission**), the means of protecting personnel, and methods for obtaining family census data and recording probable and possible cases (*how could they differentially diagnose a disease that mimicked many other diseases sharing the exact same symptoms...and even pregnancy?*)
- **Chloroquine, tetracycline, aspirin, and a drug against intestinal parasites were all supplied in tablet form** (*because if you can't find cases naturally, you might as well create them*)
- Fifteen **possible cases** of haemorrhagic fever occurring outside the main epidemic area were investigated from Kinshasa and **Ebola haemorrhagic fever was ruled out in each instance on clinical, virological, or pathological grounds** (*i.e. the indirect evidence pointed to other assumed causes*)
- Final diagnoses included:
 - Typhoid fever
 - "Viral" hepatitis
 - Amoebiasis
 - Acute pulmonary oedema
 - Carbon monoxide poisoning

- The first patient **was given chloroquine by parenteral injection** on August 26th and his fever resolved rapidly and he was afebrile until 1 September when he again had fever to 39.2° C
- Other symptoms soon followed and he was admitted to YMH on September 5th **with gastrointestinal bleeding** and died on September 8th
- It was learned that **parenteral injection** (*injections into the body by various routes*) was the principal mode of administration of nearly all medicines
- **The single common risk factor** in comparison with matched family and village controls for 85 of 288 cases where the means of transmission was determined, **was receipt of one or more injections at YMH**
- Other factors **such as previous case-contact**, exposure to food, water, hospital buildings, domestic and wild animals, or travel within three months prior to onset, **were not associated with this type of transmission**
- 43 cases had a history of both patient contact **and receipt of injection within three weeks prior to onset of illness**
- Seventeen persons who lived outside Yambuku had contact at YMH and **may have received injections there without reporting this fact to their family**
- **No person whose contact was exclusively parenteral injection survived the disease**
- Indeed, it seems likely that closure of YMH **was the single event of greatest importance in the eventual termination of the outbreak**
- *In other words, the risky and unnatural mode of injection as well as the toxic medications capable of producing the same exact symptoms are all that is needed to explain these cases, not a new invisible "virus"*
- **No sporadic, apparently spontaneous, probable cases** were recorded
- When "family" was defined as all persons living in contiguous housing and sharing common eating facilities, **secondary attack rates never exceeded 8%**
- Efforts were made to document either **concurrent asymptomatic infection** or possible past infection with Ebola "virus"
- Sera from 5 persons 8-48 years old contained IFA antibodies in titres of 1:64 and **none of these people were sick, had had contact with persons in other villages, or had visited YMH during the epidemic** (*i.e. they were asymptomatic*)
- Sera from 58 persons in various exposure categories had anti-Ebola IFA titres of 1:4-1:32 **yet the specificity of these reactions was doubted** when it was found that **samples from 4 of 200 San Blas Indians from Panama also had such "antibodies" for Ebola but not Marburg "virus"**
- Final interpretation of these data **awaited development of another method** for measurement of **type-specific** antibodies to these agents
- *In other words, the antibody results claimed to be specific were not so specific and the antibody results needed to be confirmed with a yet undeveloped "more specific" method*

- Early symptoms and signs in all 3 patients at Ngaliema Hospital included fever, headache, anorexia, and vomiting **yet the remaining symptoms varied amongst them**
- Clinical laboratory tests were done on the first patient, **but only a few measurements were carried out on the other two cases** to avoid undue exposure of hospital laboratory staff to the "virus"
- The **first patient** was treated with aspirin, antibiotics, corticosteroids, blood transfusion, and intravenous fluids
- The **second patient** received aspirin, hydrocortisone, immunoglobulin, intravenous fluids, **an experimental drug**, moroxydin, and enterovioform to control diarrhoea but without success
- The **third patient** was treated for unconfirmed malaria during the first 2 days of illness and when the etiological agent of the epidemic was shown to be a Marburg-like "virus," **she was given, on day 4, 500 ml of Marburg human plasma obtained from a recovered patient in South Africa**
- All three patients **died** after treatment
- Although no autopsies were performed, **it appeared clinically that these patients died of hypovolaemic shock** (*rapid loss of blood that can be caused by internal bleeding in the abdominal organs and digestive tract*)
- **Marburg "virus-like" particles** were visualized with an electron microscope in one case
- **No IFA antibodies** against Ebola or Marburg "viruses" were present in any of the 3 victims
- Questionnaire forms were completed on 231 probable cases 1 year of age or older, **34 individuals who were found to have Ebola "virus" IFA antibodies**, and 198 controls
- Of persons with antibodies, **59% had one or more symptoms**, (*41% apparently had no symptoms*) the most **prominent being fever, headache, abdominal pain, and arthralgia**
- **Many more persons who had been in contact with fatal cases reported symptoms but had no Ebola "virus" antibodies**
- *Here we can see how unreliable antibody results are when 41% had no symptoms of disease and those who had symptoms after contact with fatal cases had no antibodies whatsoever*
- The **only clinical laboratory test** done on patients admitted to Yambuku Hospital was **urinary protein** which was reported as uniformly positive and then **used as a major diagnostic criterion** by the nursing sisters early in the epidemic (*note: this is not a diagnostic used today which leads one to wonder how many early cases were misdiagnosed using a faulty method no longer in use?*)
- Virological studies were said to be **limited**
- Ebola "virus" was "isolated" in **African green monkey kidney cells (Vero)** from blood specimens in **8 of 10** cases attempted.

- Ebola “virus” particles were also visualized in **3 of 4** postmortem liver biopsies obtained from clinically suspect cases
- The **case mortality rate of Ebola haemorrhagic fever in Zaire of 88%** is the highest on record except for rabies infection
- Although **laboratory data were virtually non-existent**, the clinical picture seen in this outbreak **resembled illness produced by the related Marburg “virus”**
- At that time, the agents recovered from Sudan and Zaire were **thought to be identical**, although definitive neutralization tests had not yet been done
- Viraemia appeared to be a constant feature of Ebola “virus” infection in man as **the “virus” persisted in large amounts in the blood in the single, well studied case** (*one case □*)
- The WHO stated that a number of other important virological questions **can only be pursued for the moment through studies using monkeys**
- **No evidence was obtained** for persistent “viral” carriage in the Zaire cases of Ebola infection, **a phenomenon documented on two occasions for Marburg “virus”**
- The Zaire epidemic had all the attributes of a common source outbreak, together with a **fortunately low rate of secondary person-to-person transmission** (*i.e. not that infectious nor transmissible*)
- According to the WHO, the means by which the “virus” was introduced into Yambuku Mission Hospital **will probably never be precisely known**, but it **seemed possible** that it was brought directly from the Sudan by man
- **Dissemination of the agent into the villages of the region was principally through contaminated equipment used for parenteral injections** (*see how they made it about “contaminated” needles rather than the mode of injection with numerous toxins?*)
- The epidemic **waned when the hospital was closed** for want of medical staff (*and thus stopping the injections of toxic drugs*)
- The WHO had the strong impression that Ebola haemorrhagic fever **acquired by injection differed from that due to contact with another case** as the mortality was higher
- The observed “neonatal” cases of the disease **were not definitively elucidated**
- The WHO again admitted that **a better method for measuring Ebola “virus” antibodies was needed** in order to interpret the serological findings reported here (*which is a pretty troubling admittance when the only way they claimed a new “virus” as well as past and present cases was based on antibody results*)
- That **less than 20%** of persons gave a history of acute illness **following contact with a fatal case** was apparently no surprise
- The WHO stated that **if the IFA data are correct**, at least 2.5% of persons in contact with fatal cases experienced subclinical infection (*those antibody results sure seem rather sketchy based on the WHO’s continued hesitancy about their accuracy*)

- In addition, **the finding of antibodies in a few individuals in the absence of any known contact with Ebola "virus"** during the epidemic raised **the possibility that the agent is in fact endemic in the Yambuku area and is occasionally transmitted to man**
- As in the case of Marburg "virus," **the source of Ebola "virus" is completely unknown** beyond the simple fact that it is African in origin



If we are to believe the findings as presented by the WHO, the Ebola "virus" originated from an infected person traveling from Sudan to Zaire. This unidentified person received treatment at the Yambuku hospital by way of a parenteral injection. **The same needle was not sterilized and was then reused on another unsuspecting patient, thus resulting in the spread of the non-specific symptoms of disease from patient-to-patient by way of the continuous use of the same unsterilized needles.** This may make some sort of sense to those who are still under the "viral" spell and are unwilling to dig a little deeper than just scratching the surface. However, for anyone looking at the evidence presented critically and logically, there is a much more reasonable explanation for the apparent outbreak of disease other than a newly discovered Ebola "virus."

The symptoms associated with the Ebola "virus" are non-specific and mimic other diseases common to the area including influenza, malaria, yellow fever, typhoid, and even pregnancy thus the symptoms were not new or unheard of. The original patient was treated for malaria by way of an injection of chloroquine. While his malaria symptoms were said to clinically disappear after treatment, more severe symptoms showed up days later, and the patient eventually succumbed to gastrointestinal bleeding. Chloroquine is a known toxic medication with gastrointestinal problems and unusual bleeding listed as known side effects. The parenteral mode of injection is said to be risky and, if done so through the abdominal cavity, can lead to damage to the organs in the abdomen. **Further patients treated at the hospital for unrelated conditions presenting with similar symptoms, including pregnant women, were also given injections of different medications, and they, too, developed the symptoms associated with severe hemorrhagic fever. Many patients were given cocktails of numerous drugs and antibiotics by way of parenteral injection. Everyone given injections at the hospital eventually died from**

their illnesses. The WHO even noted that those who received the injections had a different presentation of the disease than cases which were unrelated to the injections with the main difference being higher mortality in those who were treated at the hospital. It was observed that once the injections stopped, the transmission of the disease stopped as well. To anyone looking at this logically, it isn't hard to see the connection between the injection of toxic drugs and the associated symptoms of disease. This does not require a new "virus" as an explanation yet the WHO ignored exploring parenteral injections of chloroquine and other toxic (and at least in one case experimental) drugs as a possibility in their investigations.

When this knowledge is combined with the fact that **the researchers never purified nor isolated any "virus" directly from the blood of any sick patient in Zaire** as well as the fact that all of the findings from the cell cultured concoctions they created in the lab pointed to the same indirect evidence associated with the Marburg "virus" discovered less than a decade before, it is simply astonishing that they could conclude there was ever a new "virus" to begin with. **The only evidence that the researchers used to claim that the Ebola "virus" was somehow a new "virus" distinct from Marburg were the Indirect Fluorescent Antibody test results which were admitted by the WHO to be less than ideal.** In fact, the antibody results were all over the place with many people who never had any contact with an Ebola patient and were never sick in any way testing positive for the antibodies said to be specific to the "virus." Meanwhile, many who had symptoms and direct contact with Ebola patients had no antibodies whatsoever. How could the results of such testing tell the researchers that they had a unique "virus?" When one understands the fraud of antibody research and that these theoretical entities have, like "viruses," never been scientifically proven to exist, it is easy to see how the inaccurate results can be used to claim whatever the researchers want them to say. When one also realizes that the results from the antibody testing directly contradicted the Marburg findings thus showing those results to be fraudulent as well, one will understand that the CDC and the WHO were left with no choice but to try and claim a new "virus" related to Marburg was to blame in order to keep the lie about the "accuracy" of the antibody results intact.

It is pretty clear that the entire Ebola affair was one giant cover-up for the side effects brought about by toxic drugs injected parenterally. As the symptoms were non-specific and aligned with many other diseases common to the area, it was easy for the researchers to find people suffering similar symptoms in order to claim that they too were also victims of the newly "discovered" pathogenic "virus." **This pattern of covering up toxic injections with the findings of a novel "virus" was seen a decade before with the Marburg "virus" associated with Polio vaccination experimentation. They also used this same tactic to cover up poisoning by way of chemicals such as in the case with Polio and DDT/lead arsenate.** Once everyone

recognizes this pattern, they will be able to see right through the fraud and then we can all start working together to ensure that these tricks are no longer used to fool the gullible into fear and further poisoning by toxic drugs and injections. Together, we can break this sick cycle once and for all.

Share this:

- [X](#)
- [Facebook](#)
-

Like this:

[The Ebola "Virus" Part 1](#)

[ViroLIEgy Boosters: The Hanta and Ebola Testing Trap](#)

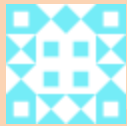
[The Marburg "Virus:" Precursor to Ebola](#)

Published October 3, 2022 By [Mike Stone](#)

Categorized as [Ebola](#)

19 comments

1. Pingback: [The Ebola "Virus" Part 1 – ViroLIEgy](#)



2. [Dude in Japan](#)

[October 4, 2022 at 11:48 am](#)

After reading about the effects of chloroquine in this article, I have a hard time taking people like Simone Gold and Robert Malone (controlled opposition, ESPECIALLY Malone) seriously when they say ANYTHING is "safe and effective(tm)." Even now, on alt-tech sites like Gab, they constantly push these poisons on us and people eat up their lies. I mean I know they are allopaths, so I throw their views out with the bath water anyway but the fact so many people still listen to them and actually BELIEVE what they say is disturbing as hell.

Loading...

[Reply](#)



1. [Mike Stone](#)

[October 4, 2022 at 1:15 pm](#)

I believe the "safe and effective" pharmaceuticals are few and far between, if there even is such a thing. When people push these toxins as cures, I immediately suspect they are up to no good and have ulterior motives.

Loading...

[Reply](#)



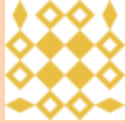
3. [Tom](#)

October 4, 2022 at 2:40 pm

All they need are 8 willing mice and the virologists can prove anything.

Loading...

[Reply](#)



4. [MolecCodicies](#)

October 5, 2022 at 9:55 am

Would love to see a breakdown of the supposed Anthrax pathogen in the future. With germ theory thoroughly demolished I'm now finding the case of the post-9/11 anthrax letters that were traced to Ft. Detrick incredibly enigmatic.

Another phenomenon that has me scratching my head now is infected wounds. I once was quite viciously bit on the hand by a stray cat. Dark lines appeared on my arm over the following 3-4 days, eventually reaching up to my shoulder. A doctor prescribed me antibiotics and told me I would have died if I waited longer for treatment. The lines on my arm cleared up with 48 hours after taking the pills.

As far as I'm aware cats do not possess venom so I'm now left wondering what actually happened to me. Staph infections would seem an important topic to address as well.

Both of these are (seemingly) bacteria-related, rather than "viruses", but I'm sure I'm not the only ex-germ theory believer who would be very interested in hearing an anti-germ theory perspective on anthrax and wound infections.

Loading...

[Reply](#)



1. [Mike Stone](#)

October 6, 2022 at 4:50 pm

Thanks for the response! I definitely plan to do more work with bacteria, hopefully starting with my next post. There is a ton of untapped potential there to explore for sure. I will see what I can uncover. ☐

Loading...

[Reply](#)



2. [Eddie](#)

October 9, 2022 at 3:06 am

Thanks much.

The caption "Hiccups is a symptom of Ebola?!?!?" certainly made me laugh out loud. However, they're not listed as a symptom at the page linked to. Is there another source listing that symptom?

Loading...

[Reply](#)



1. [Mike Stone](#)

October 9, 2022 at 4:00 am

The image is linked to this book:

<http://library.lol/main/A6AA592668A1A8E22592E4C8589C255C>

From the book, it states this:

"In the context of any given Ebola outbreak, clear criteria for ETU admission should be developed, based upon the predominant manifestation of Ebola virus disease occurring at that time. These criteria need to be highly sensitive so as to keep infected individuals isolated from the rest of the community, but this will necessarily make any suspect case definition less specific and may lead to a high rate of ETU admissions among those with illnesses that mimic Ebola.

During the 2013–2015 West African Ebola epidemic, the clinical criteria for admission used in most screening algorithms consisted of:

- **Fever plus at least one clinical symptom for children under 5 years of age, or**
- **Fever plus at least two clinical symptoms for children over 5 years of age, or**
- **Close contact with an Ebola suspect or case within the preceding 3 weeks.**

The clinical symptoms included headache, anorexia, fatigue, musculoskeletal pain, diarrhea, nausea or vomiting, abdominal pain, difficulty swallowing, difficulty breathing, HICCUPS, and unusual or unexplained bleeding (World Health Organization 2016a)."

I also found this:

The Unlikely Clue That Led Doctors in West Africa to Ebola: Hiccups

"So many devastating, horrifying stories have come out of the ongoing Ebola crisis ravaging West Africa. This one, however, is one of the more surprising nuggets to emerge. In a new piece for Vanity Fair, journalist Jeffrey E. Stern describes the unlikely way researchers identified the disease as it first started to spread across Guinea — all thanks to a single, odd symptom: HICCUPS."

<https://www.thecut.com/2014/09/how-hiccups-helped-doctors-identify-ebola.html>

So as crazy as it seems, hiccups is a symptom of Ebola. □

Loading...



3. ClariFire

October 19, 2022 at 3:10 am

Premature wound sealing is the main problem that leads to "infected wounds." When allowed to drain properly there shouldn't be an issue. I suspect that IF you prematurely seal, you must sterilize.

Loading...

[Reply](#)



5. Eddie

October 9, 2022 at 4:42 am

Ha ha ha ha. Reminds me of Sebastien Powell's recent review of the Hellenesque literature relating to Chicken Pox.

But this quote gets to something I've been curious about (yeah, I know, trying to suss the logic of virologists is akin to taking up residence in Wonderland): "My suspicion is that this would be related to the way the virus affects that body."

Shouldn't virologists be able to know, from looking at their EM images, what symptoms will be induced by the "infection" displayed before them? In other words, shouldn't "CPE" look markedly different for any given virus (or at least any given virus family)?

And while we're at it: Just as a brick cannot be seen from space but the Great Wall Of China can be, shouldn't a viral mass, as it fills up a cell, be able to be seen in a light microscope? Like, it should be apparent, should it not, when the cell is e.g. 5%/10%/50%/80% full of viral particles? And shouldn't the subsequent explosion be able to be witnessed as well? And shouldn't such images be included in isolation papers?

Just some idle wondering of a Saturday night...

Thanks again for your yeoman work with this site.

Loading...

[Reply](#)



1. [Mike Stone](#)

October 10, 2022 at 7:35 pm

You bet! Thanks for reading! ☐

Loading...

[Reply](#)



6. [Eddie](#)

October 9, 2022 at 2:45 pm

"We can handle Ebola safely in the hospital setting and provide the best care to the patient."

<https://www.zerohedge.com/medical/ebola-back-us-screen-passengers-uganda>

Loading...

[Reply](#)



7. [superfooder](#)

October 13, 2022 at 12:54 am

Mike, awesome as always.

A suggestion: further up, you sometimes have one paragraph in white, then another paragraph in red, then white, then red. It's confusing to me which white paragraph goes with which red paragraph, whether above it or below it.

Example:

(In red)

...symptoms but had no Ebola virus antibodies. Illness in antibody-positive individuals was, in general, marked by profound prostration, weight loss, and a convalescent period of 1-3 weeks."

(In white)

According to the WHO, a urinary protein test, one not in use as a diagnostic today, was utilized early on in the outbreak to diagnose cases. How many of these patients were misdiagnosed based on a faulty test?

(In red)

"The only clinical laboratory test done on patients admitted to Yambuku Hospital was urinary protein. This was reported as uniformly positive and was used as a major diagnostic criterion by the nursing sisters early in the epidemic."

Maybe, you can somehow parenthesize the two that go together, which might make them easier to follow.

Loading...

[Reply](#)



1. [Mike Stone](#)

October 13, 2022 at 8:31 pm

Thanks! I'm sorry but I'm a little confused. I don't normally use red. Most of the time I highlight quoted sections in blue, purple, and tan. Are you reading online or on the WP Reader or somewhere else?

Reply



[superfooder](#)

1.

October 13, 2022 at 8:46 pm

You're right.

They are blue or tan highlights vs unhighlighted.

I checked on PC, iPhone and iPad.

Not a big thing, but thinking of the future with millions of posts historically.

8. Pingback: [„Virus“ ebola – 1. část - Resetheus z.s.](#)



[PC](#)

9.

March 28, 2023 at 6:59 am

The nonsenses posted by Mercola.

https://articles.mercola.com/sites/articles/archive/2023/03/28/ebola-lab-leak.aspx?ui=351b973b51dd8d94b40d43b54c99780f794c82daa03fe9c71f963a4ce91bf507&sd=20101212&cid_source=dnl&cid_medium=email&cid_content=art1HL&cid=20230328_HL2&cid=DM1366259&bid=1757073894

Reply



[Cliff Lind Hjulskov](#)

10.

June 12, 2026 at 6:55 pm

In the Shadow of a Virus an Angel is Dancing you, Mike Stone, I am extremely grateful to you for your thoroughness and insight. That people who believe in injecting foreign proteins and various toxic substances behind the barrier we have developed from nature is quite incomprehensible, and they show this alone by the fact that no one has wanted to do studies with vaccinated versus unvaccinated people. It is enough for me to say stop and think. As you write "they are up to no good and have ultimate ulterior motives", and I agree with you, I wrote at the beginning of the COVID plandemic: "How does 'science' make sense in treating healthy people to 'protect' the weak?"

You may read it here if you find the time.

Thank you, Mike. Your work is life-saving for the planet and its inhabitants.

<https://drive.google.com/file/d/1j8eggdidnMWu6O0PvDgbvYpPeIDB9jvC/view?usp=sharing>

Reply



[Mike Stone](#)

1.

June 13, 2026 at 6:10 pm

Thank you so much for the kind words and support, Cliff! I greatly appreciate it. ☐

i Google AI:

is aspirin an anticoagulant

No, aspirin is not an anticoagulant. While both are types of "blood thinners," they work in fundamentally different ways: [\[1, 2, 3, 4\]](#)

- **Antiplatelet (Aspirin):** Aspirin stops blood cell fragments called platelets from clumping together to form a clot. It is typically used to prevent heart attacks and strokes. [\[1, 2\]](#)
 - **Anticoagulant:** These medications (like warfarin, apixaban, or heparin) slow down the clotting process by targeting specific proteins in the blood (the "clotting cascade"). They are often used to treat or prevent deep vein thrombosis (DVT) or strokes in patients with atrial fibrillation. [\[1, 2, 3, 4\]](#)
- For a full breakdown of how these medications differ and what they are used for, you can check out the [Cleveland Clinic Health Essentials](#) guide on blood thinners.

This is for informational purposes only. For medical advice or diagnosis, consult a professional. AI responses may include mistakes. [Learn more](#)

6 sites

- Is aspirin an anticoagulant? - Walgreens The Thread
No, aspirin is not an anticoagulant. Anticoagulants are medicines that delay the clotting of blood. They are used to help reduce t...

Walgreens The Thread

- Is Aspirin a Blood Thinner? What You Should Know - GoodRx
GoodRx is NOT insurance. So how does aspirin thin blood? Aspirin is an antiplatelet medication. This means that aspirin blocks blo...

GoodRx

- Is Aspirin a Blood Thinner? - Cleveland Clinic Health Essentials
Is Aspirin a Blood Thinner? Yes, aspirin is a commonly prescribed blood thinner that can lower cardiovascular disease risk — but i...

Cleveland Clinic Health Essentials

Show all

You said: can cloroquine cause bleeding?

Yes, chloroquine can cause unusual bleeding and bruising, though it is a rare side effect. Unlike aspirin, chloroquine does not directly thin the blood, but it can occasionally suppress the bone marrow where blood cells are produced. [\[1, 2, 3, 4\]](#)

How Chloroquine Causes Bleeding

According to the [Mayo Clinic drug guide](#), chloroquine can cause serious blood disorders. [\[1, 2\]](#)

- **Thrombocytopenia:** **Chloroquine can cause a drastic drop in blood platelets (the cells responsible for clotting). When platelets are too low, minor injuries can cause severe bleeding.** [\[1, 2, 3, 4, 5\]](#)
- **Bone Marrow Suppression:** In rare cases, the drug causes bone marrow failure or pancytopenia (a severe drop in red blood cells, white blood cells, and platelets combined). [\[1\]](#)

Signs of Bleeding to Watch For

If you or someone you know is taking chloroquine, seek medical attention right away if you notice any of these warning signs: [\[1\]](#)

- **Skin changes:** Frequent, unexplained bruising or tiny red spots on the skin (petechiae).
- **Bodily fluids:** Blood in the urine, dark/tarry stools, or regular nosebleeds.
- **Oral bleeding:** Bleeding gums when brushing teeth or eating. [\[1, 2, 3, 4\]](#)

Drug Interactions

The risk of bleeding increases significantly if chloroquine is taken alongside actual blood thinners, aspirin, or Nonsteroidal Anti-inflammatory Drugs (NSAIDs) like ibuprofen. [\[1, 2, 3\]](#)

Are you **currently taking chloroquine**, or looking into its side effects for a **specific medical condition**? Knowing if you are pairing it with **other medications** can help me provide more tailored information.

This is for informational purposes only. For med

Summaries 1: page 22 on

Summaries 2: page 60 on