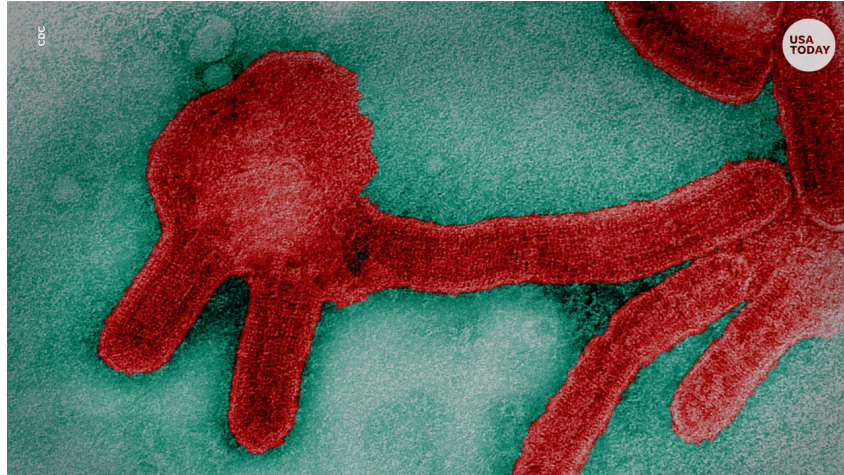


VIROLIEGY < [HTTPS://VIROLIEGY.COM/](https://viroliegy.com/)>

Exposing the lies of Germ Theory and virology using their own sources.

The Marburg “Virus:” Precursor to Ebola



“

Most of the known viral diseases were excluded and the infectious agent was shown to be a hitherto unknown virus with many peculiar characteristics: it infects guinea pigs but not adult mice and is larger than known viruses and of different shape.

Preface to *Marburg Virus Disease* by G. A. MARTINI • R. SIEGERT

If you've been paying attention to the mainstream media for some reason, you're probably well aware of the re-emergence of many different “viruses” seemingly sprouting up around the world over the last few months. We've seen the oddly accurate prediction of the monkeypox in May 2022, the

threats of obscure "viruses" such as nipah/langya, hantavirus, strawberry hepatitis A, the tomato flu, and the apparent reappearance of **polio**. < <https://viroliogy.com/category/polio/>> One of the other lesser known "viruses" currently making the rounds in the media is known as the Marburg "virus." While it may not be as scary sounding to those who are unfamiliar with it, the "virus" has a close relative whose name may be enough to strike fear into the hearts of the ignorant. For those who do not know, the Marburg "virus" is the original incarnation of the Ebola "virus." My guess is you may be more familiar with the latter of the two.



In recent weeks, there have been reports of the Marburg "virus" making its presence felt in Ghana, a place which has never reported a case of the deadly "virus" before. Sadly, not only did Ghana report its first Marburg case early July 2022, they also reported their first deaths associated with the disease:

"An outbreak of Marburg fever has been detected in Ghana, whereas West Africa had been free of cases except for one case in Guinea in 2021. Currently, 98 people are considered contacts and are in isolation. No cases of Marburg fever have yet been detected among these contact cases.

So far two **unrelated** men have had Marburg fever. Presenting symptoms **such as diarrhea, fever, nausea and vomiting**, the two men, aged 26 and 51, have both died."

<https://unric.org/en/marburg-virus-disease-origins-and-symptoms/> < <https://unric.org/en/marburg-virus-disease-origins-and-symptoms/>>

If you listen to the mainstream media, you may be convinced to believe that the Marburg "virus" is a "highly contagious, highly virulent" and deadly

disease ready to burst forth and become the next epidemic in need of fearing:

“

The life-threatening virus is highly contagious, and has no known cure or approved vaccine.

<https://www.usatoday.com/story/news/health/2022/07/28/marburg-virus-disease-outbreak-symptoms-treatment/10173445002/> <
<https://www.usatoday.com/story/news/health/2022/07/28/marburg-virus-disease-outbreak-symptoms-treatment/10173445002/>>

“

The WHO calls the disease “epidemic-prone,” meaning that it can spread easily between people if not prevented.

<https://www.healthline.com/health-news/could-the-marburg-virus-start-another-outbreak-what-we-know> <
<https://www.healthline.com/health-news/could-the-marburg-virus-start-another-outbreak-what-we-know>>

“

Marburg virus disease is a highly virulent disease that causes haemorrhagic fever, with a fatality ratio of up to 88%. It is in the same family as the virus that causes Ebola virus disease.

https://www.who.int/health-topics/marburg-virus-disease#tab=tab_1 <
https://www.who.int/health-topics/marburg-virus-disease#tab=tab_1>

“

Although MVD is uncommon, MARV has the potential to cause epidemics with significant case fatality rates.

<https://www.ecdc.europa.eu/en/infectious-disease-topics/z-disease-list/ebola-virus-disease/facts/factsheet-about-marburg-virus> <
<https://www.ecdc.europa.eu/en/infectious-disease-topics/z-disease-list/ebola-virus-disease/facts/factsheet-about-marburg-virus>>

However, looking into the Marburg “virus” outbreaks tells an entirely different story. This is only the second reported outbreak of the Marburg “virus” in West Africa, a place where it was unheard of before. The first appearance in this region of what is considered a highly contagious “virus” said to spread through contact with bodily fluids was a single case reported in Guinea last year. Similar to the recent **[monkeypox outbreak](https://viroliogy.com/category/monkeypox/)**, <
<https://viroliogy.com/category/monkeypox/>> the patients diagnosed with Marburg in Ghana had no relation to each other, no travel history to an endemic country, nor any contact with any animals said to carry the “virus.” Both were farmers who reported non-specific symptoms such as diarrhea, fever, nausea and vomiting. Sadly, they both succumbed to their

symptoms after treatment at the hospital. The final toll of this outbreak of the "highly contagious virus" was limited to four people.

How did a "highly contagious virus," which has never been seen in West Africa before, make its way to that part of the continent when those infected had no history of travel to an area where the "virus" is said to be endemic? How did this deadly "virus" only infect one person in Guinea in 2021 and only 4 in Ghana in 2022 if it is highly virulent and contagious? In fact, in the vast majority of its history, the Marburg "virus" has shown that it is anything but "highly contagious," with the highest outbreak occurring in Angola in 2004 with only 252 cases. If we are to go by the **CDC's own data**, <https://www.cdc.gov/vhf/marburg/outbreaks/chronology.html> there have only been 475 total cases of this "highly contagious virus" worldwide since its discovery in 1967. Those statistics do not seem to jibe with the definition of a "epidemic-prone highly contagious, highly virulent virus."

So what is the story behind the Marburg "virus?" When and how did this dangerous pathogen burst onto the scene? What new symptoms of disease originally prompted the search for a novel etiological agent? What methods, if any, were employed in order to purify, isolate, and characterize the particles assumed to be the "virus?" Let's see what we can uncover about the first "virus" discovered in the *Filoviridae* family.

According to the CDC, the Marburg "virus," was discovered in 1967 after an outbreak of symptoms commonly associated with hemorrhagic fever occurred among laboratory workers conducting research who had come in contact with African green monkeys. It is one of seven species of "filovirus," with the other six belonging to separate versions of the Ebola "virus." The symptoms of the disease associated with the Marburg "virus" are non-specific and overlap with many different diseases, such as malaria, typhoid fever, or dengue and/or any of the "viral" hemorrhagic fevers that may be endemic to a specific area. This obviously makes clinical diagnosis difficult (i.e. impossible) due to the similarities between the diseases. In other words, there is nothing new nor specific regarding the Marburg "virus" as it is just another in a long chain of names given to the same symptoms of disease:

"Marburg virus disease (MVD) is a rare but severe hemorrhagic fever which affects both people and non-human primates. MVD is caused by the Marburg virus, a genetically unique zoonotic (or, animal-borne) RNA virus of the filovirus family. The six species of Ebola virus are the only other known members of the filovirus family.

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

"Clinical diagnosis of Marburg virus disease (MVD) can be difficult. Many of the signs and symptoms of MVD **are similar to other infectious diseases (such as malaria, typhoid fever, or dengue) or viral hemorrhagic fevers** that may be endemic in the area (such as Lassa fever or Ebola). This is especially true if only a single case is involved."

<https://www.cdc.gov/vhf/marburg/index.html> <

<https://www.cdc.gov/vhf/marburg/index.html>>

Introduction



- Marburg virus disease (MVD) (formerly known as Marburg hemorrhagic fever) was first identified in 1967 during epidemics in Marburg and Frankfurt in Germany and Belgrade in the former Yugoslavia from importation of infected monkeys from Uganda.
- MVD is a severe and highly fatal disease caused by a virus from the same family as the one that causes Ebola virus disease. These viruses are among the most virulent pathogens known to infect humans. Both diseases are rare, but have a capacity to cause dramatic outbreaks with high fatality.

We can learn a lot about the "discovery" of this new "filovirus" from the writings of Werner Slenczka, a man who self-identifies as an expert in "filoviruses" as he was intimately involved with their origin. Dr. Slenczka is an associate professor at the Institute of Virology who helped identify the filament-like particles claimed to be the "virus" by way of **electron microscopy.** < <https://virology.com/category/electron-microscope-images/>> He wrote a few papers detailing what occurred throughout the "discovery" process and offered some rather interesting revelations.

The first excerpts presented here are from a paper he published on the Marburg "virus" in 2007, 40 years after it was first identified. According to Slenczka, the first people "infected" with the "virus" in August of 1967 were

treated in their own homes for up to 10 days. The symptoms were not alarming at first but progressively became worse as undisclosed treatments were administered. It was initially thought that the patients were suffering from typhoid fever or dysentery. As the symptoms became worse, the patients were admitted to the hospital. Of the 7 patients who became hemorrhagic, 5 eventually succumbed to their illness, some as soon as one day after admission. Cases of the new "virus" as well as estimates for the incubation period were eventually determined retrospectively. Interestingly, one of the patients with severe illness who was never hospitalized recovered completely on his own. Contrary to the claims of the mainstream media today, it was eventually decided at the time that the "virus" was *not highly contagious* as there were only a few secondary cases, no tertiary infections, and no new cases after the initial "outbreak."

In mid-September of 1967, experiments with Guinea pigs were started in order to determine the cause of the disease. The researchers serially passaged toxic goo between the Guinea pigs and created "similar" symptoms to the human disease after later passages, thus concluding that they had obtained a filterable "virus," even though they were unable to obtain any electron microscopy images of the "virus." Guinea pig spleen and liver tissues were examined by Slenczka using indirect fluorescence antibody testing and it was found that there were intracytoplasmic intrusions in the Guinea pigs, but not the humans. This method was used to select the blood of the "infected" animals which were then used for electron microscopy examination using a newly developed technique by the lead researcher. They subsequently "discovered" the "virus" in the animals (but not the humans) in November of 1967, nearly 3 months after the initial cases of the disease:

Forty Years of Marburg Virus

"In early August 1967, patients with unusual symptoms indicating an infectious disease were admitted to the university hospitals in Marburg and Frankfurt. **The first patients were treated in their homes for up to 10 days, even though the illness was described as beginning suddenly with extreme malaise, myalgia, headache, and a rapid increase in temperature to as high as 39°C or more.** Although the clinical symptoms were not very alarming during the first 3–4 days, additional symptoms and signs appeared at the end of the first week. **Gastrointestinal symptoms, such as nausea, vomiting, and diarrhea, indicated to health care practitioners that the diagnosis might be dysentery or typhoid fever.** The patients

were therefore admitted to a hospital. At admission, most patients were observed to have conjunctivitis, exanthema, and enanthema, but shigellae or salmonellae were not found. During the second week after onset of disease, patient temperatures fell to 38°C, and petechiae and more-severe signs of hemorrhagic diathesis were recorded for ~25% of patients. As indicated by transaminase levels, liver destruction reached its maximum at days 7 and 8 after onset of disease. Leukopenia with the appearance of immature polymorphonuclear leukocytes and thrombocytopenia (<10,000 cells/mm³) were detected. Patients were bleeding from all body orifices and from needle punctures. When the outcome was fatal, death occurred during the second week after onset of disease, at day 9 on average (range, day 7–16). **In some cases, patients died from severe hemorrhagic shock on the day after hospital admission.** Severe hemorrhagic signs, as seen in ~25% of patients, were a signum mali ominis. All patients who died had hemorrhaging. Of 7 patients with manifest hemorrhages, 5 succumbed to the disease. Orchitis, a typical late-stage symptom, appeared in the third week after onset of disease or even at relapse during the fifth week. Mental confusion and paraesthesias were indicative of cerebral involvement. **Relapses with hepatitis, orchitis, and uveitis with virus persisting in semen and in the anterior eye chamber were typical during the convalescent phase of both Marburg virus (MARV) and Ebola virus (EBOV) infections.** In 1 case, a patient transmitted infection to his wife 120 days after onset of his disease, most probably by sexual intercourse. Virus was detectable in seminal fluid.

The incubation time of MARV disease could only be estimated retrospectively, after the source of infection and the date of exposure were known. Incubation ranged from 5 to 9 days, with an average of 8 days. The ratio of primary to secondary infections was 21:3 in Marburg, 4:2 in Frankfurt, and 1:1 in Belgrade. Three cases of secondary infection resulted from inadvertent needle-stick inoculations; in 1 case, a pathology technician cut himself on the forearm with a knife during a postmortem examination. **Airborne transmission between humans did not occur, as indicated, for example, by the instance of a young man who slept in the same bed with his brother only a couple of days before he died; the brother did not develop disease and was seronegative for MARV 6 months later. One of the patients had been severely ill at the time of the outbreak but, for unknown reasons, was not hospitalized. He recovered and, 15 years later, maintained that he had had MARV**

disease. At that time, he underwent serological testing and was found to be seropositive for MARV antibody by IFA and ELISA. He had been exposed to monkey kidney cell cultures, which were used for the production of poliomyelitis vaccine. At 6 months after the outbreak, blood specimens were obtained from 120 persons who had been in close contact with patients or with infectious material but who had not developed disease. The specimens were tested for MARV antibody by complement fixation test, IFA, and ELISA and were found to be seronegative. Therefore, there was no indication of clinically inapparent infection."

"By mid September, it had become evident that the agent exhibited a low contagiousness. Only a few cases of secondary infection and no cases of tertiary infection had occurred, and no new cases had occurred during the previous 2 weeks. Therefore, Rudolf Siegert ([figure 1](#)) resumed experiments with guinea pigs, together with a Chinese colleague, Hsin Lu Shu. **They found that the agent could be passed among guinea pigs and exhibited pathogenicity that increased from passage to passage.** At the third passage, the animals fell ill with fever, hepatitis, and hemorrhagic disease that closely resembled human disease, and they died within 10 days after inoculation, with a marked drop in temperature. **However, all efforts to determine the etiological agent by light or electron microscopy failed. Opportunistic bacterial infections were a major problem.**

At this time, specimens of human and guinea pig convalescent serum were available, and some of the serum specimens were tagged with fluorescein for direct IFA. Three weeks later, W.S. ([figure 1](#)) detected intracytoplasmic inclusions in the tissues of infected guinea pigs, by IFA. Animals that had infected cells in the liver and spleen were selected for further studies using electronmicroscopy. Blood specimens from these animals were inactivated with formalin and sent to Dietrich Peters at the Bernhard Nocht Institute in Hamburg. Formalinized plasma was spun directly onto electron microscope (EM) grids, by means of a new technique developed by Gerhard Müller, and negative staining was done. **By these methods, MARV was identified on 20 November, < 3 months after the outbreak had begun ([figure 2](#)).**"

It needs to be noted that, according to Slenczka's account of the events, no "virus" was ever properly purified and isolated at any point during the investigation into the potential cause of disease afflicting the laboratory workers in 1967. In fact, the initial evidence for the existence of the Marburg "virus" essentially boiled down to experimentally making Guinea pigs sick through serial passaging of unnatural injections of blood/tissues into their stomachs, examining their spleens and livers using **non-specific antibodies** < <https://virology.com/category/antibodies/> > to find non-specific inclusions, and then taking electron microscopy images of the sickened animals blood and claiming random particles seen in the unpurified and contaminated samples are the "virus" in question. Eventually, after many unsuccessful attempts of trial and error failures, it was claimed that the "virus" could be propagated in cell culture (without CPE) using the Guinea pig (not human) materials.

For a better understanding of the fraudulent methods used to propagandize the public on a new deadly "virus," let's break down the main three focus areas (animal experiments, EM imaging, and cell culture) even further and see what the evidence shows.

1. Animal Experimentation



“

“Experimental infection apparently results in 100% fatality. If this situation occurs in nature, obviously there could not be any serologic positives. However, if the experimental route of inoculation differs from that occurring in nature, it is possible that 100% fatality will not develop.”

https://link.springer.com/chapter/10.1007/978-3-662-01593-3_22 <
https://link.springer.com/chapter/10.1007/978-3-662-01593-3_22>

The first piece of evidence used for the existence of a new “virus” was the creation of “similar” symptoms in Guinea pigs by continually injecting them with serially passaged diseased fluids/tissues. What these injections actually contained is difficult to determine as the original study is in German and the translation is not clear. The study, lead by Rudolf Siegert, mentioned doing successive passages with whole blood, plasma or organ material yet what the exact methods were beyond that remain obscure due to a faulty translation.

Fortunately Rudolf Siegert collected much of the Marburg research into a book in 1971. Oddly, he did not include his original paper ***On the etiology of an unknown human infection originating from monkeys*** <
<https://pubmed.ncbi.nlm.nih.gov/4294540/>> yet he did provide a study explaining the procedures used for passaging the “virus” in Guinea pigs which furnished some insight into the kind of experiments that were ultimately carried out. What we can see in the highlights from the included paper below is that the clotted blood was haemolyzed with sterile distilled water and the organs triturated using a cold mortar. A phosphate buffer diluent containing 10 p. 100 normal rabbit serum was added to the blood mixture which was used to inject mice intracerebrally (in the brain) and intraperitoneally (in the stomach) as well as Guinea pigs intraperitoneally. The researchers admitted that all “isolation” attempts were negative except for in Guinea pigs as, after injecting the Guinea pigs in the stomach with blood samples, organ suspensions, and Cercopithecus organ pools,

the animals developed a febrile (fever) reaction 4 to 6 days after inoculation. Clinical symptoms in the animals included loss of appetite and weight, "bloated face," and enlargement of the testes. The researchers concluded that from the human and monkey material, an organism was "isolated" and transmitted through four to six passages in Guinea pigs even though no "virus" was ever purified, isolated, and visualized:

Passage of Marburg Virus in Guinea Pigs

"Clotted blood was haemolyzed with sterile distilled water and the organs triturated using a cold mortar **and phosphate buffer diluent containing 10 p. 100 normal rabbit serum added** to give a final concentration of about 20 percent by volume."

"Suckling mice were inoculated **intracerebrally** (ic) and **intraperitoneally** (ip) with 0.02 ml.

Guinea pigs (200-300 g) **were inoculated ip** with 4 ml. For further passaging, whole blood harvested by cardiac puncture **was injected ip.**"

Results

All isolations attempts **have been negative** except in guinea pig.

Guinea pig Passages

Guinea pigs injected by ip route **with blood samples** of patient H. F. (HF 1 and HF 2) **organ suspensions** of patient P. S. and **Cercopithecus organ pools** M 10 and M 14 consistently developed a febrile reaction 4 to 6 days after inoculation.

The febrile stage lasted 3 to 7 days.

Whole blood taken during this febrile stage has been **successfully passaged ip in guinea pigs through 3 to 6 passages (Figs. 1 to 5)**. The incubation period was shortened to 2-3 days and some guinea pigs died from 7 to 17 days after inoculation. **Clinical symptoms in the animals were: loss of appetite and weight, "bloated face" and**

enlargement of the testes. At autopsy, we found splenomegaly and lung consolidation.

Fever reactions in guinea pigs were produced using early blood samples from the preceding passage. In one instance (Fig. 4, passage 4), fever reaction followed inoculation of blood taken at the 11th day. This fact confirms observation that infection seems to produce a long lasting viraemia."

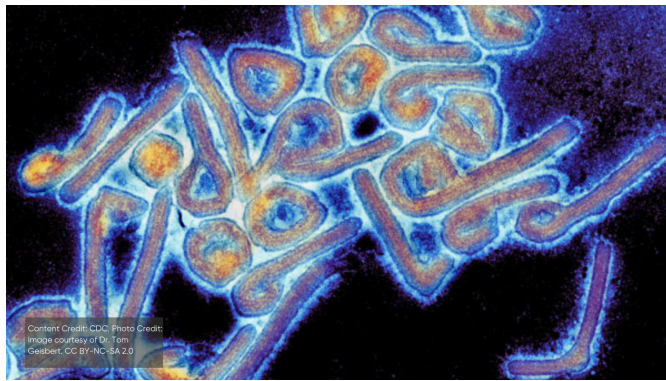
Conclusions

"The results reported above show that from human and monkey material, **an organism has been isolated and transmitted through four to six passages in guinea pigs.**"

DOI: 10.1007/978-3-662-01593-3_16

As can be seen from the above study, the researchers are conflating the experimental creation of disease in Guinea pigs through serial passaging and unnatural routes of infection with the "isolation" of a "virus." At no point was a "virus" ever **purified and isolated** <<https://viroliegy.com/category/purification-isolation/>> from the fluids of a sick human, a monkey, nor a Guinea pig. Beyond the non-specific fever, weight loss, and loss of appetite (occurring after injections of toxic unpurified goo into the stomach), none of the symptoms experienced by the Guinea pigs (bloated face, enlarged testes) related to the human condition whatsoever. In fact, Rudolf Siebert and Werner Slenczka later admitted in their paper **Laboratory Diagnosis and Pathogenesis**, that regarding the pathogenesis of the disease in man, "*apparently the virulence of the Marburg virus differs in monkey, man, and guinea pig.*" Yet it was based on these experimental results that it was determined a new pathogenic "virus" was hiding in the fluids of the sickened Guinea pigs which were then used for electron microscopy imaging in order to try and find the new "virus."

2. Electron Microscopy Images



“

The second possibility of making a rapid diagnosis lies in the direct demonstration of the virus with the electron microscope. This may succeed if the agent is directly centrifuged from serum or plasma on a carrier, according to Peters and Muller [7]. The characteristic morphology leaves no doubt as to the diagnosis. However, we have no experience enabling us to answer the question concerning the probability that the agent may be overlooked with this method. It is for this reason that attempts to isolate the virus in guinea pigs and in various cell culture systems should follow in any case.

https://link.springer.com/chapter/10.1007/978-3-662-01593-3_22 <
https://link.springer.com/chapter/10.1007/978-3-662-01593-3_22>

While the researchers failed in their initial attempts to identify the “virus” by electron microscopy after sickening the Guinea pigs, they eventually determined through direct and indirect antibody staining results that they could find the “virus” in the livers and spleens of these animals. While

Rudolf Siegert's study did not offer much insight into how they determined that the particles observed were the actual "virus" they were looking for, he did offer some interesting comments about their morphology which fortunately were not lost in translation:

On the etiology of an unknown human infection originating from monkeys

"The results described show that "Marburg Virus" is not identical, **but it is morphologically closely related to the virus vesicular stomatitis (1, 2, 6, 9, 11, 12), the Coval (1, 3) Egtved- (13) and finally the rabies virus (8, 10).** A fundamental difference from these viruses seems to have the particular tendency to be growth in length."

DOI: 10.1055/s-0028-1106144

According to Seigert, the particles he observed looked morphologically like the vesicular stomatitis, coval, egtved, and **rabies "viruses."** <https://viroliogy.com/category/rabies/> The main difference was in the length of the particles. However, does the length of the particles really distinguish these "viruses" from each other all that much? Could these all be random pieces from the same long strands broken apart or even created from the procedures used to capture the images? Presented below are the images of the Marburg "virus" supplied by Seigert which show some longer particles mixed with other smaller particles which resemble the bullet shape of the aforementioned "viruses" listed in his paper. The third image included is one provided by Slenczka said to be from the original "isolate" in 1967 while the colorized pictures are images taken from recent 2022 articles on the Marburg "virus" cases in Ghana:



Abb. 5. Erreger aus Meerschweinchenblut (Mee 81), 4. Passage, Stamm „L“; bei 100 000 g aus Plasma direkt auf Objektträger sedimentiert. Negativkontrastiert. Vergrößerung 90 000fach.

imgflip.com

2332

Zur Arbeit Siegert u. a. (Seite 2341—2343)

Zur Ätiologie einer unbekannt, von Affen ausgegangenen menschlichen Infektionskrankheit



Abb. 6. Wie Abb. 5, besonders lange Form. Vergrößerung 60 000 fach.

imgflip.com

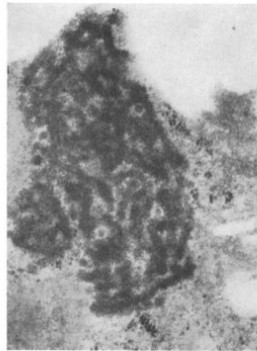
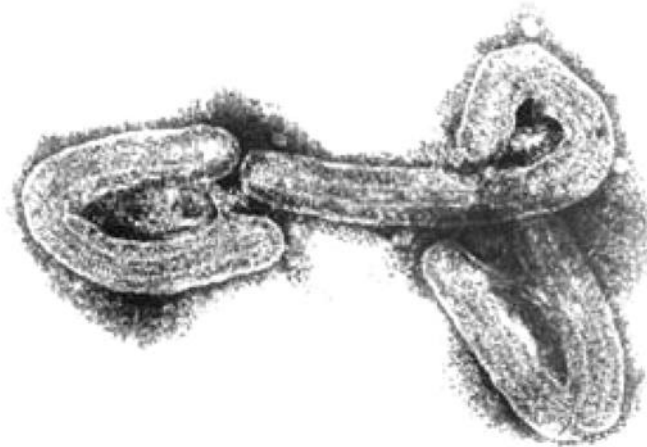
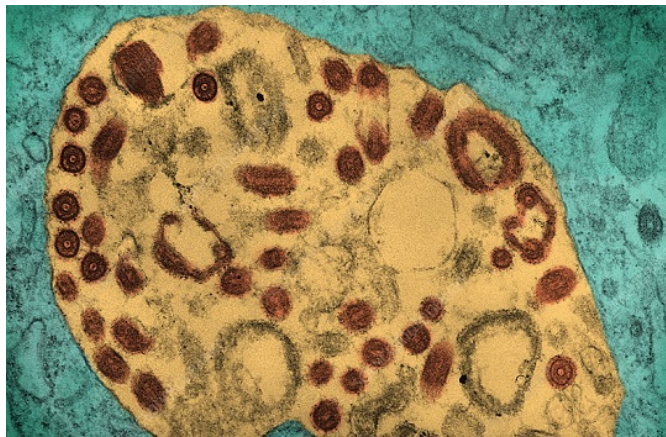


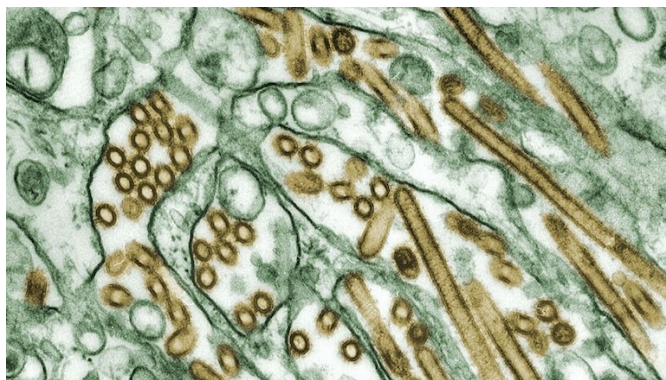
Abb. 7. Schnitt durch infizierte Meerschweinchenleber (Mee 85), 7. Passage, Stamm „H + F“. Erreger-Aggregat an Zellperipherie. Kontrastiert mit Uranylacetat und Bleicitrat. Vergrößerung 35 000fach.





<https://www.bmj.com/content/378/bmj.o1797> <

<https://www.bmj.com/content/378/bmj.o1797>>



<https://www.indiatoday.in/amp/information/story/what-is-marburg-virus-197711-2022-07-20> <

<https://www.indiatoday.in/amp/information/story/what-is-marburg-virus-197711-2022-07-20>>

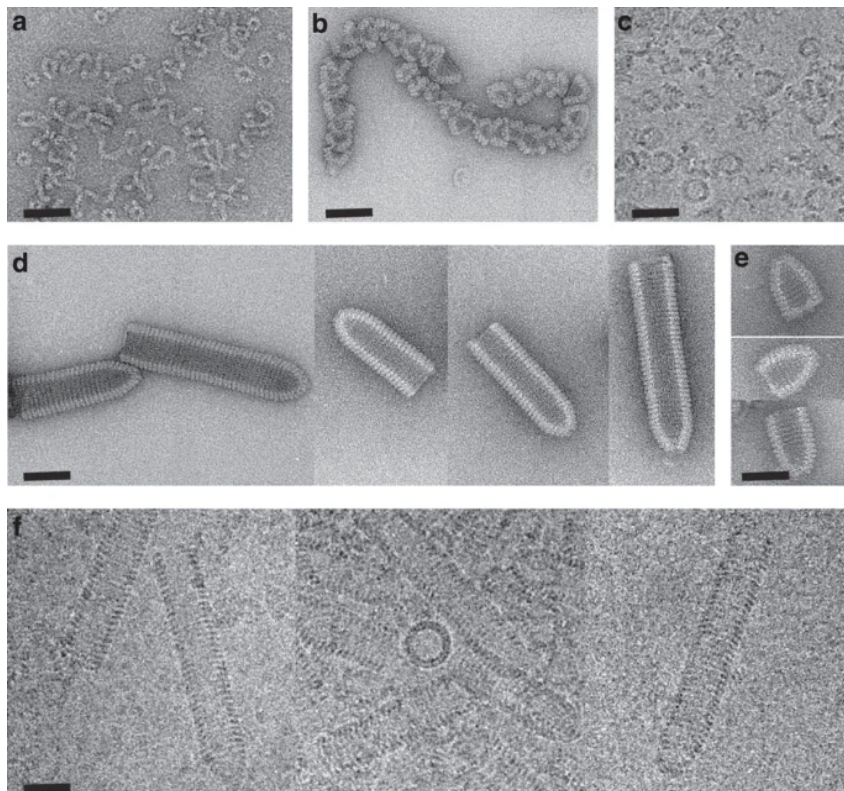
As can be seen, the Marburg "virus" comes in different shapes and sizes, from long filament particles to smaller bullet-like particles to even those resembling "corona-like" structures. It seems that the image of the "virus" heavily depends upon the way the picture is taken/cropped as well as how the particles are stained and colorized. Below are images of the "viruses" said by Seigert to be morphologically similar to the Marburg "virus."

Vesicular Stomatitis

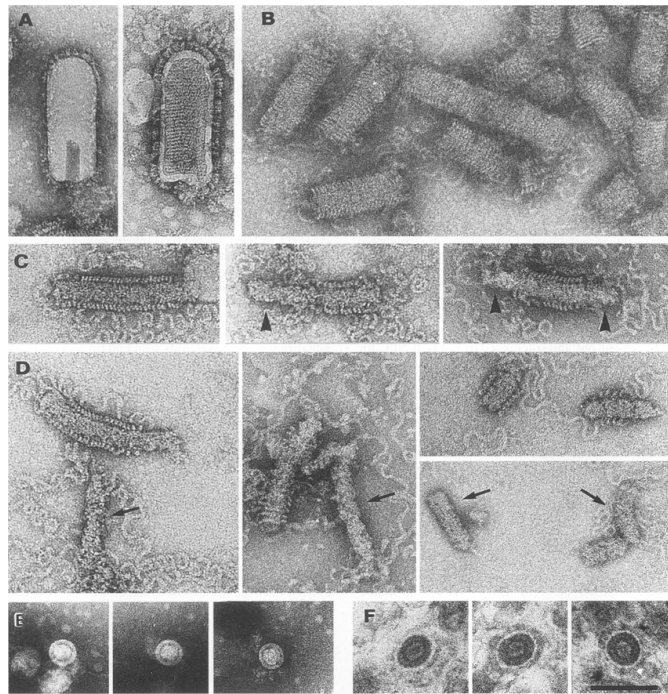
In these images, we can see the similar bullet-like shape in the vesicular stomatitis "virus" as seen in the particles claimed to be the rabies "virus." Interestingly, the particles appear to vary in size from the smaller bullets to the much more elongated forms resembling the Marburg "virus." This "unwinding" was described in the third image as most likely a result of the negative staining procedures done to visualize the "virus" particles.



<https://www.utmb.edu/virusimages/VI/vesicular-stomatitis-indiana-virus> <
<https://www.utmb.edu/virusimages/VI/vesicular-stomatitis-indiana-virus>>



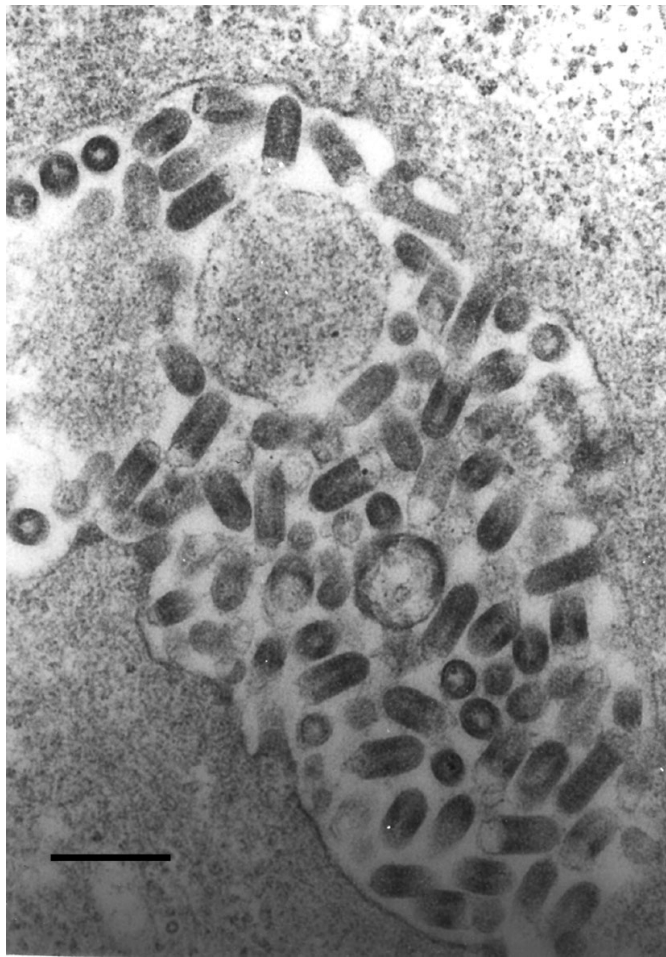
<https://www.nature.com/articles/ncomms2435> <
<https://www.nature.com/articles/ncomms2435>>



“The **difference in degree of unwinding is probably caused by intrinsic variations in the negative staining procedure** due to differences in local hydrophilicity of the support film, sample concentration, room temperature, and humidity.” https://www.researchgate.net/figure/EM-of-VSV-structure-Negative-staining-was-done-with-I-N-SST-A-Intact-VSV-The_fig1_14969735 < https://www.researchgate.net/figure/EM-of-VSV-structure-Negative-staining-was-done-with-I-N-SST-A-Intact-VSV-The_fig1_14969735>

Viral Haemorrhagic Septicemia Virus (VHSV)

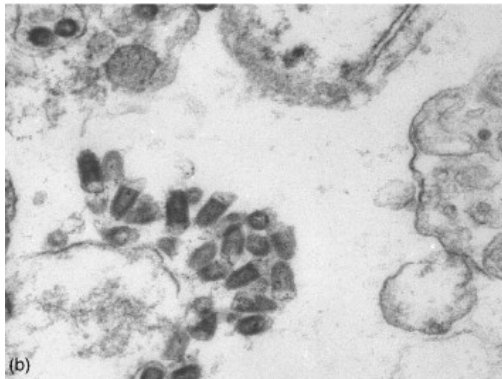
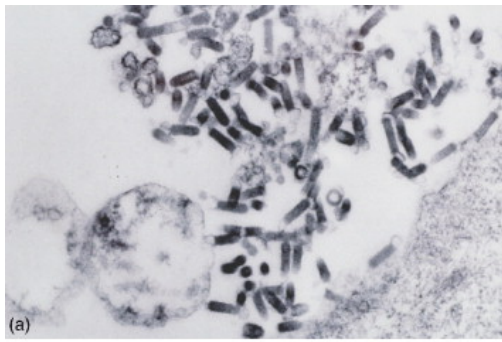
The VHSV is a fish “virus” that also supposedly shares the exact same shape as the rabies “virus” and is morphologically similar to the Marburg “virus.” As can be seen, the shapes of the particles once again range from small bullet-like entities to longer filaments, perhaps caused once again by the negative staining procedures.



https://www.researchgate.net/publication/6080687_Mortality_event_in_freshwater_drum_Aplodin

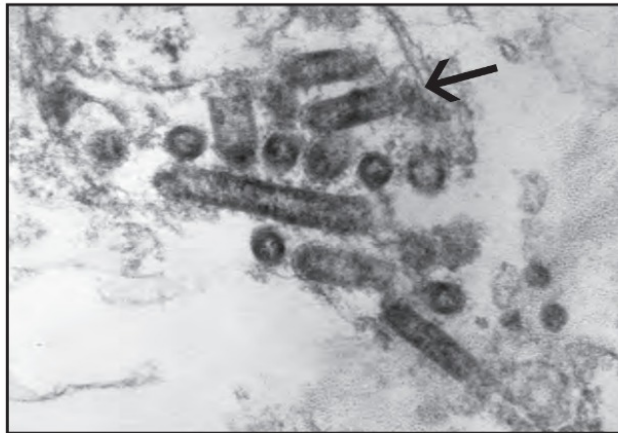
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https://www.researchgate.net/publication/6080687_Mortality_event_in_freshwater_drum_Aplodin



<https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/viral-hemorrhagic-septicemia-virus> <

<https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/viral-hemorrhagic-septicemia-virus>>



Electron micrograph of VHSV particles (arrow) in a cultured EPC fish cell, X 56,500

imgflip.com

https://www.adfg.alaska.gov/static/species/disease/pdfs/fishdiseases/north_american_viral_her

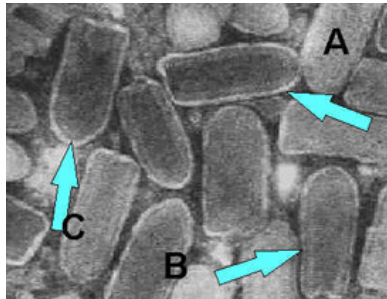
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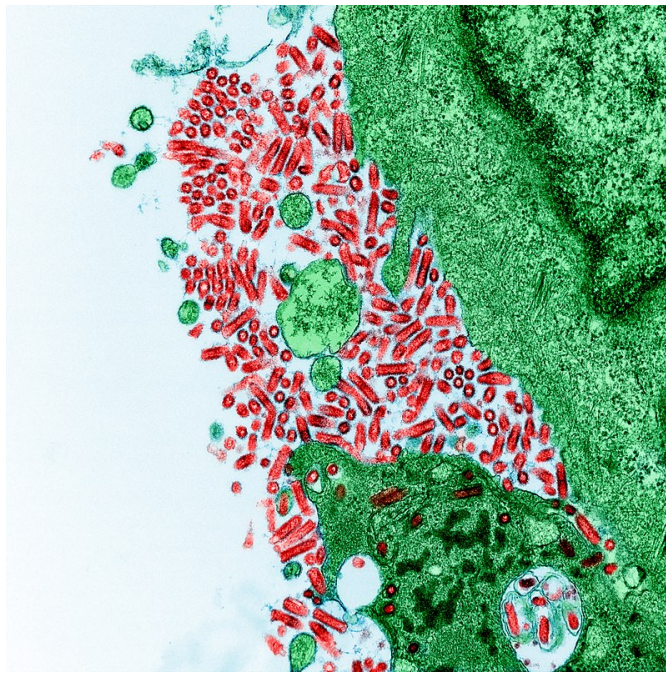
Rabies

Even the rabies "virus," said to be the bullet-like particles seen in the TEM images, show various shapes and sizes depending on the staining and colorization. Some appear to be long Marburg-like filament forms that

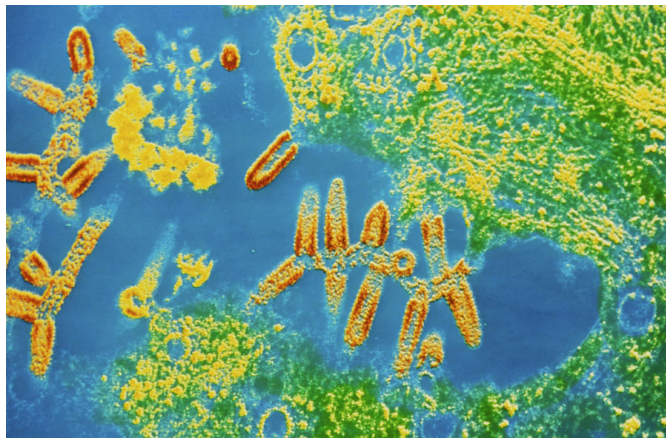
happen to be present in a U-shape upon imaging. Interestingly enough, up to the early 1980's, the Marburg "virus" was classified as a rhabdovirus due to the similarities between the morphology of the "viruses." It was even originally given the moniker *Rhabdovirus simiae*.



https://www.cdc.gov/rabies/diagnosis/electron_microscopy.html <
https://www.cdc.gov/rabies/diagnosis/electron_microscopy.html>



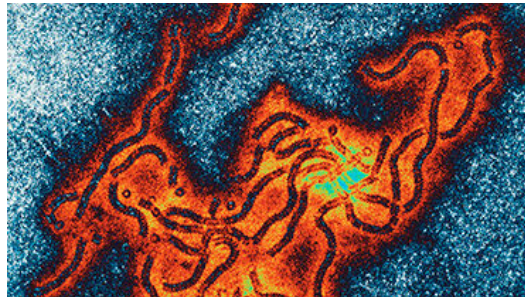
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https://en.m.wikipedia.org/wiki/Rabies_virus>



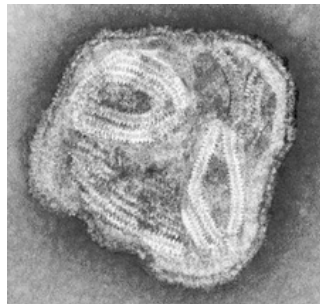
<https://fineartamerica.com/featured/tem-of-rabies-viruses-cnriscience-photo-library.html> < <https://fineartamerica.com/featured/tem-of-rabies-viruses-cnriscience-photo-library.html> >

While it is easy to see the similarities between the Marburg “virus” and the other “viruses” Seigert highlighted in his study, he failed to mention that we can find similar looking filament particles associated with many other “viruses” as well:

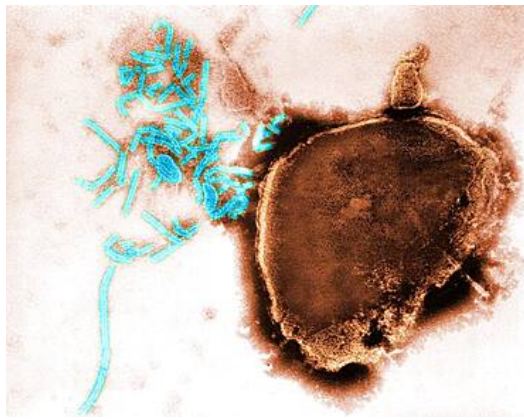
Mumps



<https://www.ecdc.europa.eu/en/immunisation-vaccines/facts/vaccine-preventable-diseases> < <https://www.ecdc.europa.eu/en/immunisation-vaccines/facts/vaccine-preventable-diseases> >



<http://www.virology.uct.ac.za/vir/teaching/linda-stannard/paramyxovirus> < <http://www.virology.uct.ac.za/vir/teaching/linda-stannard/paramyxovirus> >



<https://www.robertharding.com/index.php?>

[lang=en&page=search&s=mononegavirales&smode=0&zoom=1&display=5&sortby=1&bgcolor=white](https://www.robertharding.com/index.php?lang=en&page=search&s=mononegavirales&smode=0&zoom=1&display=5&sortby=1&bgcolor=white)

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Measles

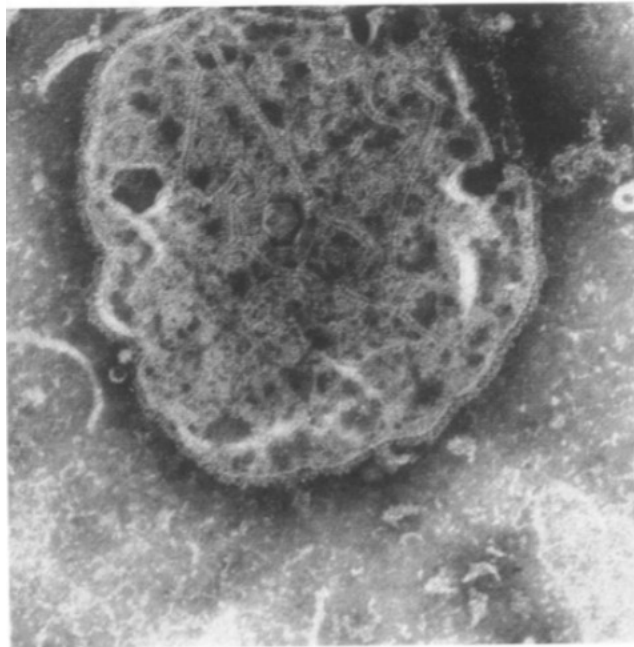


FIG. 1. *Measles particle negatively stained with phosphotungstic acid. The internal components (nucleo-capsid) and the external surface projections are evident. $\times 70,000$.*

imgflip.com

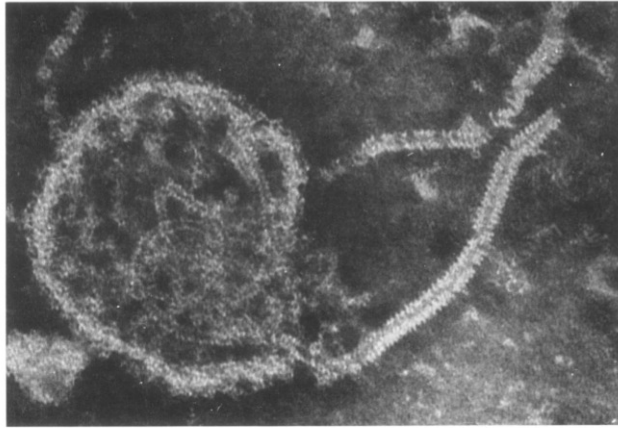
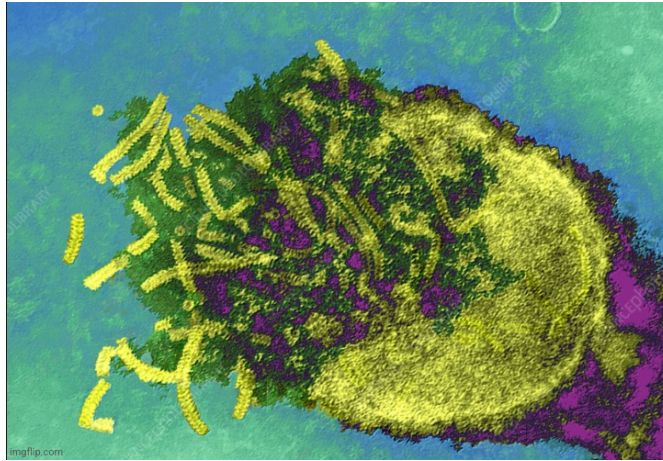


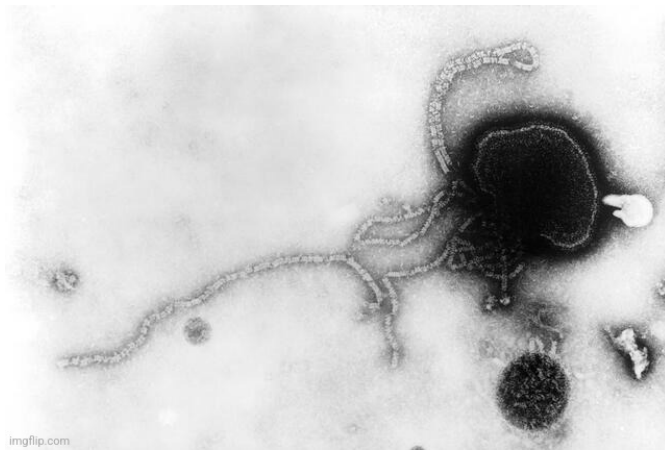
FIG. 2. Disrupted measles particle stained with phosphotungstic acid. The helical nucleocapsid is released partially from the viral particle. $\times 222,100$.

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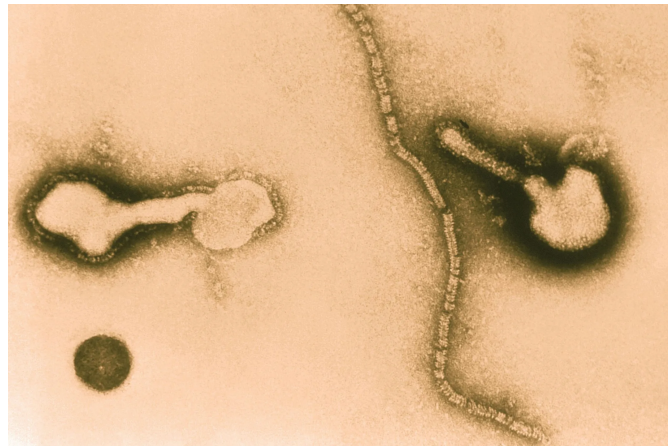


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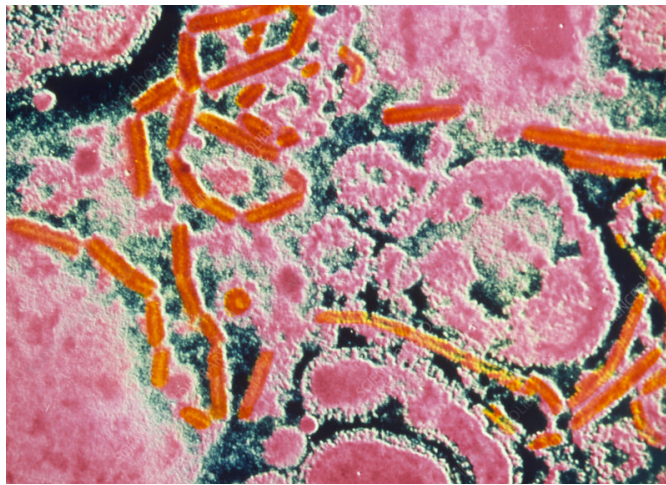
Parainfluenza



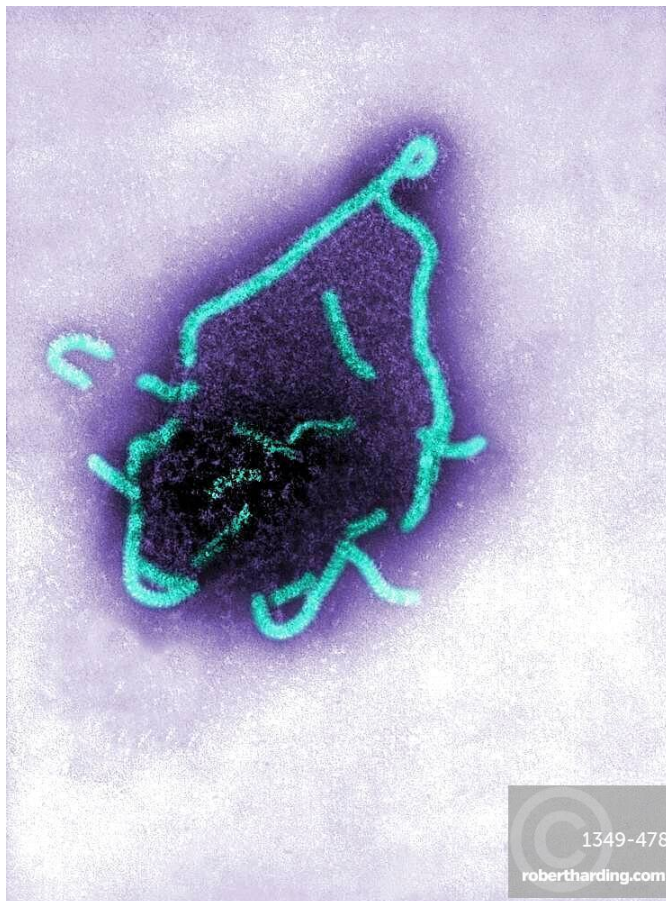
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<https://pharmaceutical-journal.com/article/news/existing-drugs-are-effective-against-human-parainfluenza-virus> < <https://pharmaceutical-journal.com/article/news/existing-drugs-are-effective-against-human-parainfluenza-virus> >



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It can be seen that the images of these particles claimed by Seigert to be of different "viruses" definitely share similar morphology (i.e. form and

structure). It is clear that the staining and colorization of the particles as well as how they are zoomed in and presented definitely influences how they appear in the images. It is even stated in one study that the negative staining procedures have an impact on how the particles form. It is evident, when including images of filament forms of other "viruses," that this is yet another case where the same particles (seen in various stages of formation/degradation) are being cropped from an unpurified sea of different shapes and structures and focused on as the representative culprit in numerous diseases.

As the electron microscopy images play such a pivotal role in the proof supplied for the Marburg "virus," it is important to gain some greater insight into the electron microscopy process used by Werner Slenczka and Co. in order to identify the Marburg "virus." For this we can now turn our attention to a 2017 article he wrote describing how his "filovirus" research began. In this account, we find out that the blood samples taken from the Guinea pigs were mixed with glutaraldehyde and formaldehyde before imaging. Slenczka admitted that searching for an unknown pathogenic agent in EM can be fatiguing and frustrating as he explained that many contaminants can be found within the sample. This was the case with the Marburg "virus" as numerous microbial contaminants were present in the sample (thus it was unpurified and not isolated) which made the interpretation of the data difficult. As stated before, the spleens and livers of the Guinea pigs were used for EM imaging utilizing direct and indirect antibody fluorescence, and the samples were air-dried and fixed with ice-cold acetone. Since they did not know if the unknown agent would be killed by acetone, Slenczka claimed that they handled the slides with extreme caution. It then took them 3 weeks of hard work to get the initial results. Slenczka stated that what he saw in his slides resembled the Negri bodies said to be specific to the rabies "virus" yet somehow this finding told him that this effect was the cause of a new "virus." He used his initial findings to mark any Guinea pig sample with these inclusions as ones with the "virus" in need of further analysis by EM.

The blood samples which were selected for further analysis were once again subjected to glutaraldehyde and formaldehyde and studied. Dr. D. Peters, along with his technician, spent more than a day looking through the samples with no results. On the second day, Dr. Peters left the search for a lunch break and handed the reigns to Dr. G. Müller. Once Dr. Peters arrived back from his lunch break, he was informed by Dr. Müller that the "virus" had been found (in less than an hour) as they had observed unknown particles of a different morphology and structure to any that had been seen before. Obviously, this is not the case as these same particles have been observed with various "viruses" as detailed previously.

Ultimately, the entire "isolation" process did not involve the purification and isolation of any "virus" directly from human fluids and instead relied entirely on assumptions based on indirect evidence obtained from animal, antigen, and EM studies using unpurified and non-isolated material from Guinea pigs rather than humans.

Interestingly, as the laboratory researchers who were initially "infected" with the "virus" were conducting polio vaccine research using monkey kidney cell cultures, it was assumed that the "virus" came from the imported monkeys. However, according to Slenczka, none of the monkeys at any of the laboratories ever showed any signs of disease at any location. He also stated that the exact lethality data of the incriminated monkeys was never communicated. Thus, it was left up to mere assumptions that some persistently infected monkeys from "Monkey island" which were caged for research may have been the source for importing Marburg "virus" to Europe. According to Slenczka, it was known that a large number of monkeys from the same source in Uganda were transported to Sweden, Japan, Czechoslovakia, Italy, Switzerland, and England at the same time for the purpose of preparing cell cultures. However, no outbreaks were reported at any of these locations. Thus, Slenczka was forced to admit that his hypothesis, just like his "virus," was solely based on assumptions:

Filovirus Research: How it Began

"These experiments were a step in the right direction, but the agent remained unidentified. To facilitate identification, **blood from infected animals, taken at the climax of the disease, was mixed with glutaraldehyde and formaldehyde to inactivate and preserve the unknown agent** and was sent to the electron microscopy (EM) laboratories at the University of Marburg and the Bernhard Nocht Institute for Tropical Medicine in Hamburg, Germany for analysis: Dr. D. Peters, head of the Virology Department at the Bernhard Nocht Institute, was a renowned virological electron microscopist and highly experienced in analyzing viral structures.

Of course, **EM-based search for an unknown pathogen in biological materials can be extremely fatiguing** and--in case of a negative result--very frustrating.

In the case of the guinea pig material, an additional obstacle became evident soon. **A serious complication, often encountered in the search for unknown pathogens, is contamination by organisms unrelated to the disease. These “pick-up” contaminants may interfere with the etiological agent or may even cause disease themselves.** Contamination may occur as a result of a preexisting infection. **During passage to new animals, contaminants might be transferred with a higher efficiency than the unknown etiological agent.** The risk of cultivating a contaminating agent may be reduced by using animals from an SPF (specific pathogen free) breed. **In 1967, SPF guinea pigs could not be afforded in Marburg. Instead, the animals were purchased from local breeding stations which did not control for infections. Therefore, in the experiments carried out by Drs. Siegert and Shu, it happened that microbial contaminations, including pseudomonads, pasteuriae, and in some cases paramyxoviruses were present in the guinea pigs. Although it was quite clear that these well-known organisms were not the etiological pathogen, the presence of these contaminants complicated data interpretation.”**

“Organs from infected and noninfected guinea pigs, especially livers and spleens, were used to make imprint preparations on microscopic slides, **which were then air-dried and fixed with ice-cold acetone. Since we did not know if the unknown agent would be killed by acetone, we handled these slides with extreme caution. It took 3 weeks of hard work before we had the first results.** I found brilliantly fluorescent cytoplasmic inclusions in liver cells from an infected guinea pig. Since all the controls were negative, I was sure I had found antigenic structures of the unknown pathogen (Fig. 4). **At this time, it was not yet possible to tell whether these inclusions, which resembled the Negri bodies found in rabies virus-infected cells (Goldwasser et al. 1959), were indicative of a viral or bacterial infection.** However, it was clear that I had detected something that nobody had seen before; structures of an unknown agent causing a deadly disease (Slenczka et al. 1968).

Using this assay, it was now possible to identify those animals which were infected with this agent to select material for EM investigations. Once again, guinea pig blood treated with glutaraldehyde and formaldehyde was sent to the Bernhard Nocht Institute in Hamburg

for EM analysis. **Dr. D. Peters, together with a technician, analyzed negative stained material for more than a day but did not observe anything reminiscent of a viral structure. On the second day of his search, Dr. Peters left the laboratory for a lunch break and handed the specimen to his coworker, Dr. G. Müller, asking him to continue the search. In less than an hour, Dr. Müller had succeeded in finding viral particles that, due to their sizes and unique morphologies, were identified as the products of an unknown virus (Fig. 5).** When Dr. Peters returned from his lunch break, Müller showed him the new virus. **It is not clear why Dr. Peters had not found the viral particles when he examined the samples.** The most probable explanation seems to be that the particles had spontaneously sedimented to the bottom of the tube and Dr. Peters took material from the top only."

"The researchers who deserve credit for isolating and identifying Marburg virus are Walter Mannheim, University of Marburg **for successful transmission to guinea pigs**, Werner Slenczka, University of Marburg for **detecting and identifying the Marburg virus antigen** by immunofluorescence analysis, and Gerhard Müller, Bernhard Nocht Institute for **identifying the virus by EM**. Walter Mannheim, a bacteriologist, **was uninterested in co-authoring publications despite his involvement in the virus isolation.**"

"The most intriguing question regarding these monkeys is their state of health. Where and at what time did they acquire the virus? **Why did they not show signs of disease at any location;** not when they were in Entebbe, not upon their arrival in London, Frankfurt, Marburg, or Belgrade, and not when they were finally euthanized? The lethality of imported NHPs was about 5% at that time. An increase in lethality of imported NHPs should certainly have raised suspicion. **Exact lethality data of the incriminated monkeys were never communicated.** There can be no doubt that the animals were inspected carefully before they were used."

"Assuming that some persistently infected monkeys from "Monkey island" could have been the source for importing Marburg virus to Europe might help to explain some peculiarities of this outbreak. It is known that a large number of monkeys from the same source in Uganda were transported to Sweden, Japan, Czechoslovakia, Italy, Switzerland, and England at the same time and for the same

purpose: to prepare cell cultures. But no outbreaks were reported at any of these locations. It is possible that when the shipments to Germany and to Yugoslavia were assembled, there were not enough monkeys left at the collecting station and therefore, that animals from "Monkey island" were used to supplement the shipment. Among the animals captured from "Monkey island" were possibly some which had survived an infection with Marburg virus but appeared to be healthy. This might explain why Marburg virus was exclusively transported with shipments to locations in Germany and in Yugoslavia.

Admittedly, the above-formulated hypothesis is based on assumptions. But it offers an intriguing explanation addressing many of the open questions regarding this outbreak that, until now, have remained unanswered."

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3. Cell Culture

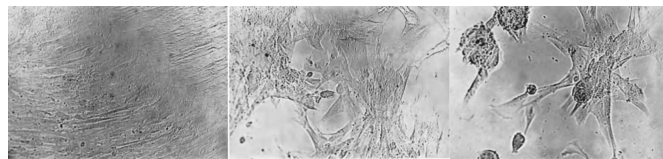


Fig. 1. Uninfected ELF cells

Fig. 2. Beginning CPE

Fig. 3. Maximal CPE

“

In cell cultures cytopathic alterations are absent. However, as early as the third day cytoplasmic antigen inclusions may be recognized, which increase considerably in number and size until the 7th day.

https://link.springer.com/chapter/10.1007/978-3-662-01593-3_22 <
https://link.springer.com/chapter/10.1007/978-3-662-01593-3_22>

Beyond the issues related to the interpretation of contaminated and unpurified EM images and the lack of disease in the monkeys assumed to have been carriers of the “virus,” early attempts to propagate the “virus” in **cell cultures** < <https://viroliegy.com/category/cell-culture/>> failed miserably. Many different cell lines were used and conflicting results were often obtained. Many times, the researchers claimed that the “virus” successfully replicated in the cells even though the required **cytopathogenic effect (CPE)** < <https://viroliegy.com/2021/09/04/creating-the-cytopathic-effect/>> was never observed. This is the effect which virologists use to claim that the structural changes observed in a host cell during culturing are the result from “viral” infection and replication. As one **source** < <https://cytosmart.com/resources/resources/virus-induced-cytopathic-effect-cpe-examples-and-readouts>> stated, the CPE is a way of “seeing” and *indirectly* measuring a “viral” infection by looking at the damage a “virus” causes to a cell. This damage is a measurement that is widely used in virology labs all over the world in order to determine indirectly the presence of a “virus.” Without observing such an effect, the culture should theoretically show that no “virus” is present in the sample. However, as this effect is not always observed, virologists found a way around the lack of being able to produce this effect in all cultures by claiming that some “viruses” do not produce CPE at all while others only do so in certain cell lines. They state that even without the observance of CPE, the cultures are still successful despite the lack of any structural

changes denoting the highjacking of the cell by the "virus" and the subsequent replication process.

The below highlights are from a 1971 paper which summarized the attempts to cultivate the Marburg "virus" and explained the confusing and contradictory results. The authors concluded that while the "virus" could be propagated in culture, the general rule was that no gross cytopathic changes could be observed:

Cultivation of the Marburg Virus (*Rhabdovirus simiae*) in Cell Cultures

"At the time when the causative agent of Marburg haemorrhagic fever was still unknown, attempts were made to isolate the agent from patients. For isolation purposes laboratory animals and many cell systems were employed **but all initial trials in cell cultures were unsuccessful** (SIEGERT et al., 1967; MAY and KNOTHE, 1968; SIEGERT et al., 1968). **Later when the virus was isolated in guinea pigs** (SIEGERT et al., 1967; SMITH et al., 1967; KUNZ et al., 1968; MAY and KNOTHE, 1968; KISSLING et al., 1968), **many investigators tried to propagate Marburg virus in various cell cultures.** Presently, much data are available which will be summarized in this report."

In primary cells of Cercopithecus kidney, the species with which the agent was imported to Germany, SIEGERT et al. (1968) found that the virus replicated without cytopathic effect (CPE). They also demonstrated the virus specific antigen by means of the immunofluorescent method. **On the contrary HAAS et al. (1968) observed gross CPE in primary Cercopithecus kidney cells.** These cells were only less sensitive to the virus than subcutaneously infected Cercopithecus monkeys.

In primary Rhesus monkey kidney cells, the virus replicated **but without CPE** (HOFMANN and KUNZ, 1968). Primary cells of human origin also propagated the virus. MAY and KNOTHE (1968) **could not observe CPE** in human leucocytes **but reported slight CPE** in primary human amnion cells (MAY et al., 1968).

We could demonstrate replication of the virus in primary guinea pig embryo fibroblasts **but no CPE was seen** (HOFMANN and KUNZ, 1968).

In chick embryo cells, **we found a very slight virus replication** (HOFMANN and KUNZ, 1968), while other investigators **did not observe propagation at all** (SMITH et al., 1967; MAY and KNOTHE, 1968)."

"Established cell lines were also investigated for propagation of Marburg virus. At first cells derived from monkeys were tested. **Although we found that permanent Cercopithecus kidney cells (strain GMK-AH) produced high titers of virus-1 ml of culture fluid contained 10^6 infective doses for guinea pigs-we could not demonstrate any CPE. KISSLING et al. (1968) were more successful; they observed CPE in their cultures in the second day p.i., which was in total about the 4th-5th day.**

In the VERO cell line, the virus also propagated. **No CPE was observed by SIEGERT et al. (1968) and only slight CPE by KISSLING et al. (1968).**

From cells of Rhesus monkey origin, heart cells (strain CMH) **allowed only slight virus growth** (HOFMANN and KUNZ, 1968), while in kidney cells (strain LLC-MK2) **no virus replication was demonstrable** (SMITH et al., 1967).

In the studies of SMITH et al. (1967) the Marburg virus propagated in L cells (mouse embryo cells) **without CPE; we could not demonstrate any virus growth in those cells.**

Heart cells derived from guinea pigs also allowed virus replication **without CPE** (KISSLING et al., 1968).

The first reports of Marburg virus-induced CPE in cell cultures were by ZLOTNIK et al. (1968). They had found that the virus was adaptable to BHK21 cells. In the first passage typical inclusion bodies similar to those found in guinea pig liver, were seen in infected cells after 13 days and CPE appeared about the 23rd day. After a few passages, inclusion bodies as well as CPE appeared

earlier. In our laboratory BHK21 cultures showed only slight changes which appeared very late (HOFMANN and KUNZ, 1968). KISSLING et al. (1968) tested two strains of BHK21. One, the WI 2 strain, behaved as the strain in our laboratory, but the other was highly susceptible. Cytopathic effect was observed about the 2nd-5th day after infection.


In contrast to 3 other teams of investigators (SIEGERT et al., 1968; SMITH et al., 1967; MAY and KNOTHE, 1968), we could propagate Marburg virus quite well in our HeLa strain. One ml of culture fluid contained 10⁴ infective particles for guinea pigs, **however we found no CPE** (HOFMANN and KUNZ, 1968).

Other strains deriving from human sources were also tested for Marburg virus induced CPE. KISSLING et al. (1968) **propagated the virus in foreskin fibroblasts** **In the first passage of virus CPE was demonstrable but could not be reproduced in serial passages.** In U cells, a stable cell line of human amnion, **the agent also replicated but without CPE** (HOFMANN and KUNZ, unpublished).

In our laboratory we had previously tested many cell systems, but we were unable to detect a cell line in which Marburg virus propagates with CPE. Finally we came across the ELF (embryonal human lung fibroblasts) cell strain, in which CPE appears about the 3rd day and reaches its maximum about the 5th day after infection. Cytopathic effect begins in focal areas and consists of spindling and later on of clumping of cells. Finally the foci become confluent (see Figs. 1-3). **It must be mentioned that, although changes are severe, they are never complete and eventually healthy cells may grow in and repair the lesions."**


"Summarizing the sometimes conflicting results obtained by the various investigators, it can be stated that cells deriving from mammals such as monkey, guinea pig, and hamster and human cells are susceptible to the virus. Often high titers were produced by the cells **although as a rule no gross cytopathic changes could be observed."**

There is a new
deadly Virus in town called




MARBURG


Some Signs and Symptoms of the Virus




Unexplained bleeding through the eyes, nose, gums, ears, anus and skin.




Vomit with blood



Joint and muscle pain




High fever



Headache

How to be Safe from the Virus

1. Wear protective materials and wash your hand with soap under running water when you come into contact with an infected person.
2. Avoid eating bush meat because you may not know the source.
3. **Do not handle** bodies of people suspected to have died of Marburg. Report the death to the approved authority.



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Fear Propaganda #1

In Summary:

- Marburg "virus" disease (MVD) is said to be a rare but **severe hemorrhagic fever** which affects both people and non-human primates
- It is one of seven belonging to the *Filoviridae* group, **with the other six species of Ebola "virus"** the only other known members of the "filovirus" family
- Marburg "virus" was **first recognized in 1967, when outbreaks of hemorrhagic fever occurred simultaneously in laboratories** in Marburg and Frankfurt, Germany and in Belgrade, Yugoslavia (now Serbia)
- The first people "infected" had been **exposed to Ugandan imported African green monkeys** or their tissues while conducting research
- Clinical diagnosis of Marburg "virus" disease (MVD) **can be difficult as many of the signs and symptoms of MVD are similar to other infectious diseases** (such as malaria, typhoid fever, or dengue) or "viral" hemorrhagic fevers that may be endemic in the area (such as Lassa fever or Ebola)
- The first patients were **treated in their homes for up to 10 days**, even though the illness was described as beginning suddenly with extreme malaise, myalgia, headache, and a rapid increase in temperature to as high as 39°C or more
- Although **the clinical symptoms were not very alarming** during the first 3–4 days, additional symptoms and signs appeared at the end of

the first week

- Gastrointestinal symptoms, such as nausea, vomiting, and diarrhea, **indicated to health care practitioners that the diagnosis might be dysentery or typhoid fever**
- In some cases, patients died from severe hemorrhagic shock on the day **after hospital admission**
- *Note that there is zero information on what treatments the patients underwent before and after admission to the hospital which could have worsened their clinical symptoms*
- Airborne transmission between humans **did not occur**, as indicated, for example, by the instance of a young man who slept in the same bed with his brother only a couple of days before he died; **the brother did not develop disease** and was seronegative for MARV 6 months later
- One of the patients had been **severely ill** at the time of the outbreak but, for unknown reasons, **was not hospitalized and recovered** and, 15 years later, maintained that he had had MARV disease (*full recovery from severe illness without hospital admission/treatment... 🤔*)
- By mid September, it had become evident that the agent **exhibited a low contagiousness**
- Only a **few cases** of secondary infection and **no cases** of tertiary infection had occurred, **and no new cases had occurred during the previous 2 weeks**
- Rudolf Siebert performed experiments with Guinea pigs and found that **the agent could be passed among Guinea pigs** and exhibited pathogenicity that increased from passage to passage (*i.e. the more they injected Guinea pigs with cultured diseased goo and tissues, the more they became sick*)
- However, **all efforts** to determine the etiological agent by light or electron microscopy **failed** and opportunistic bacterial infections **were a major problem**
- *In other words, they claimed there was a "virus" inside the toxic goo serially passaged and injected into Guinea pigs but they could not find it*
- After this failure, specimens of human and Guinea pig convalescent serum were available, **and some of the serum specimens were tagged with fluorescein for direct IFA**
- Three weeks later, W.S. **detected intracytoplasmic inclusions** in the tissues of infected guinea pigs, by IFA
- Formalinized plasma was spun directly onto electron microscope (EM) grids, **by means of a new technique developed by Gerhard Müller**, and negative staining was done
- By these methods, the Marburg "virus" was identified on November 20, 1967, **almost 3 months after the outbreak had begun**
- For the Guinea pig experiments, clotted blood was haemolyzed with sterile distilled water and the organs triturated using a cold mortar **and phosphate buffer diluent containing 10 p. 100 normal rabbit serum added** to give a final concentration of about 20 percent by volume
- Suckling mice were inoculated **intracerebrally (IC)** and **intrapitoneally (IP)** with 0.02 ml while Guinea pigs (200-300 g) were **inoculated IP** with 4 ml
- For further passaging, whole blood harvested by cardiac puncture **was injected IP**
- All isolations attempts **were negative** except in Guinea pigs

- Guinea pigs injected by IP route **with blood samples** of patient H. F. (HF 1 and HF 2) **organ suspensions** of patient P. S. and **Cercopithecus organ pools** M 10 and M 14 consistently developed a febrile reaction (*i.e. fever*) 4 to 6 days after inoculation
- Whole blood taken during this febrile stage had been successfully **passaged IP** in Guinea pigs **through 3 to 6 passages**
- The incubation period was shortened to 2-3 days and **some Guinea pigs died** from 7 to 17 days after inoculation
- Clinical symptoms in the animals were:
 1. Loss of appetite and weight
 2. "Bloated face"
 3. Enlargement of the testes
- The researchers felt that the results reported showed that from human and monkey material, **an organism had been "isolated" and transmitted through four to six passages in Guinea pigs**
- *In other words, injecting diseased tissues and fluids successively into Guinea pigs and causing symptoms equalled "isolating a virus"*
- According to the electron microscopy imaging search for the "virus," Rudolf Siebert described the particles seen as **morphologically closely related** to the vesicular stomatitis "virus," the Coval, Egtved, and the rabies "virus"
- A fundamental difference from these "viruses" seemed to have the particular tendency **to grow in length**
- In order to identify the "virus" particles in EM, blood from infected animals, taken at the climax of the disease, **was mixed with glutaraldehyde and formaldehyde** to inactivate and preserve the unknown agent
- Werner Slenczka admitted that EM-based search for an unknown pathogen in biological materials **can be extremely fatiguing** (*which would not be the case in a purified/isolated preparation*)
- A serious complication, often encountered in the search for unknown pathogens, **is contamination by organisms unrelated to the disease**
- These "pick-up" contaminants **may interfere with the etiological agent or may even cause disease themselves**
- During passage to new animals, **contaminants might be transferred with a higher efficiency** than the unknown etiological agent
- In 1967, special pathogen-free guinea pigs **could not be afforded** in Marburg so the animals were purchased from local breeding stations **which did not control for infections**
- Therefore, in the experiments carried out by Drs. Siebert and Shu, it happened that **microbial contaminations**, including pseudomonads, pasteurellae, **and in some cases "paramyxoviruses" were present in the guinea pigs**
- The presence of these contaminants **complicated data interpretation**
- Samples were air-dried and fixed with ice-cold acetone yet, **as they did not know if the unknown agent would be killed by acetone**, they handled these slides with extreme caution
- It took **3 weeks of hard work** before they had the first results
- Slenczka found inclusions within the samples **which resembled the Negri bodies found in rabies "virus-infected" cells**
- *Somehow, finding the same inclusions as seen in rabies led Slenczka to believe that this was the result of a new "virus"*

- Dr. D. Peters, together with a technician, analyzed negative stained material for more than a day **but did not observe anything reminiscent of a "viral" structure**
- On the second day of his search, Dr. Peters left the laboratory for a lunch break and handed the specimen to his coworker, Dr. G. Müller, asking him to continue the search and in less than an hour, Dr. Müller had succeeded in finding "viral" particles that, **due to their sizes and unique morphologies**, were identified as the products of an unknown "virus"
- *Remember that it was admitted that the Marburg "virus" resembled many other "viruses," including rabies, morphologically*
- It is **not clear why Dr. Peters had not found the "viral" particles** when he examined the samples
- According to Slenczka, the researchers who deserve credit for "isolating" and identifying Marburg "virus" are:
 1. Walter Mannheim, University of Marburg for **successful transmission to guinea pigs** (he was uninterested in co-authoring publications despite his involvement in the "virus" isolation)
 2. Werner Slenczka, University of Marburg **for detecting and identifying the Marburg "virus" antigen** by immunofluorescence analysis
 3. Gerhard Müller, Bernhard Nocht Institute **for identifying the "virus" by EM**
- *Note that the entire "isolation" process did not involve the purification and isolation of any "virus" directly from human fluids and instead relied entirely on assumptions based on indirect evidence obtained from animal, antigen, and EM studies using unpurified and non-isolated material from Guinea pigs*
- As the laboratory workers who were sickened were **working on polio research using monkey kidney cell cultures**, it was *assumed* that they had become infected from the imported monkeys
- However, **none of the monkeys** showed signs of disease at any location
- Exact lethality data of the incriminated monkeys **was never communicated**
- Slenczka **assumed** that some persistently infected monkeys from "Monkey island" could have been the source for importing Marburg "virus" to Europe
- It was known that a large number of monkeys from the same source in Uganda were transported to Sweden, Japan, Czechoslovakia, Italy, Switzerland, and England **at the same time and for the same purpose: to prepare cell cultures**
- However, **no outbreaks were reported** at any of these locations
- Slenczka admitted that his formulated hypothesis **was based on assumptions**
- As stated before, the attempts to isolate a "virus" **from human patients failed**
- For isolation purposes laboratory animals and many cell systems were employed **but all initial trials in cell cultures were unsuccessful**
- It wasn't until when the "virus" was "isolated" **in guinea pigs** that further attempts to culture a "virus" were made
- Many investigators tried to propagate Marburg "virus" **in various cell cultures**

- In primary cells of Cercopithecus kidney, the species with which the agent was imported to Germany, Seigert et al. (1968) found that the **"virus" replicated without cytopathic effect (CPE)**
- On the contrary Haas et al. (1968) **observed gross CPE** in primary Cercopithecus kidney cells
- In primary Rhesus monkey kidney cells, the "virus" replicated **but without CPE** (Hofmann and Kunz, 1968)
- Primary cells of human origin also propagated the "virus" yet May and Knothe (1968) **could not observe CPE** in human leucocytes but reported **slight CPE** in primary human amnion cells (May et al., 1968)
- Hofmann and Kunz could demonstrate replication of the "virus" in primary guinea pig embryo fibroblasts **but no CPE was seen**
- In chick embryo cells, Hofmann and Kunz found a **very slight "virus" replication while other investigators did not observe propagation at all** (Smith et al., 1967; May and Knothe, 1968)
- Although Hofmann and Kunz found that permanent Cercopithecus kidney cells (strain GMK-AHL) produced high titers of "virus," **they could not demonstrate any CPE**
- Kissling et al. (1968) were more successful; **they observed CPE in their cultures in the second day p.i., which was in total about the 4th-5th day**
- In the VERO cell line, the "virus" also propagated **while no CPE was observed** by Siegert et al. (1968) and **only slight CPE** by Kissling et al. (1968)
- From cells of Rhesus monkey origin, heart cells (strain CMH) allowed **only slight "virus" growth** (Hofmann and Kunz, 1968), while in kidney cells (strain LLC-MK2) **no "virus" replication was demonstrable** (Smith et al., 1967)
- In the studies of Smith et al. (1967) the Marburg "virus" propagated in L cells (mouse embryo cells) **without CPE** yet Hofmann and Kunz **could not demonstrate any "virus" growth in those cells**
- Heart cells derived from guinea pigs also **allowed "virus" replication without CPE** (Kissling et al., 1968)
- The first reports of Marburg **"virus-induced" CPE** (*interesting phrase here as CPE is only supposed to be "virus-induced"*) in cell cultures were by Zlotnik et al. (1968) who found that the "virus" was adaptable to BHK21 cells
- In the first passage **typical inclusion bodies similar to those found in guinea pig liver**, were seen in infected cells after 13 days and CPE appeared **about the 23rd day**
- **After a few passages**, inclusion bodies as well as CPE **appeared earlier** (*showing that the passaging caused the deterioration of the cells and the creation of the CPE, not the "virus"*)
- **In contrast to 3 other teams of investigators** (Siegert et al., 1968; Smitg et al., 1967; May and Knothe, 1968), Hofmann and Kunz claimed that they could propagate Marburg "virus" quite well in HeLa strain **however no CPE was observed**
- Other strains deriving from human sources were also **tested for Marburg "virus" induced CPE** as Kissling et al. (1968) propagated the "virus" in foreskin fibroblasts
- In the first passage of "virus" CPE was demonstrable **but could not be reproduced in serial passages**
- In U cells, a stable cell line of human amnion, the agent also replicated **but without CPE** (Hofmann and Kunz, unpublished)
- Hofmann and Kunz had previously tested many cell systems, **but were unable to detect a cell line in which Marburg "virus"**

- propagates with CPE** until they came across the ELF (embryonal human lung fibroblasts) cell strain, in which CPE appears about the 3rd day and reaches its maximum about the 5th day after infection
- However, they admitted that although changes were severe, **they were never complete and eventually healthy cells may grow in and repair the lesions**
 - **Summarizing the sometimes conflicting results** obtained by the various investigators, it was stated that cells deriving from mammals such as monkey, guinea pig, and hamster and human cells were susceptible to the "virus" as often high titers were produced by the cells, **although as a rule no gross cytopathic changes could be observed**

GOOD LIFE
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Simple facts about Marburg

What is Marburg Virus Disease?

It is a killer disease caused by the Marburg virus. It spreads quickly from person to person and kills in a short time, but can be prevented.

How is Marburg Spread?

It is spread through:

- Direct contact with wounds, body fluids like blood, vomitus, stool and urine of a person suffering from Marburg virus disease.
- Unsterilised injection, contaminated linen, body clothing or beddings.
- Using skin piercing instruments that have been used by an infected person .
- Direct physical handling of persons who have died of the Marburg virus.

What are the signs and symptoms of a person suffering from Marburg?

- Headache and vomiting of blood
- Joint and muscle pains
- Bleeding through the body openings i.e. eyes, ears, anus and skin
- Decreased urine

What should be done to someone suspected to be suffering from Marburg?

- Patients should be handled with care, avoiding contact with their body fluids e.g. blood, saliva or sweat.
- The person should be taken to the nearest health facility immediately for proper assessment and management.
- The clothing and beddings of the person should be disinfected with household bleach to avoid infecting others at home.

How long does Marburg virus disease live in the environment and what is the risk of transmission on objects, such as a package or other materials?

Marburg virus may survive in dried blood for several days. The risk of transmission from objects such as packages and other materials is low. The main risk of spread is from direct contact with ill individuals or body fluids (e.g., blood, saliva, urine) from individuals who have symptoms. The most important thing is to disinfect blood /fluid spills or other objects at once.

What is the treatment for Marburg virus disease?

No specific treatment for this disease is available. Patients should receive supportive care, including careful fluid and electrolyte management and treatment of other infections that may arise as a result of being sick with Marburg virus disease. Where medical resources exist, intensive care, including mechanical devices to support breathing, blood pressure support, and replacement of blood and clotting factors, may be necessary.

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Fear Propaganda # 2

We are currently living in a time where novel "viruses" and old classics are seemingly breaking out in various parts of the world. Each of these outbreaks are presented by the media as potential epidemics/pandemics waiting to happen, if not now then definitely in the near future. We are being continually primed for this eventuality. What is interesting is that most of these diseases share the same non-specific symptoms including a flu-like illness and some form of rash. The diseases seem to be presenting in patients in atypical ways in non-endemic countries where the patients have no history of travel to known hotspots nor contact with any animals

assumed to be carriers. As clinical diagnosis is impossible due to the overlapping symptoms, fraudulent PCR tests are being used to generate cases.

The Marburg "virus" is the latest in this "viral" merry-go-round to make an appearance. Listening to the MSM, this "highly contagious virus" has the potential to explode into the world unless it is prevented immediately, even though there are no known treatments nor vaccine which are said to be successful against it. The recent "outbreak" in Ghana infected four people while the previous "outbreak" in Guinea in 2021 infected just one. To date, there have been a grand total of 475 cases of the Marburg "virus" worldwide since its "discovery" in 1967. Is this really the "highly contagious, virulent, and lethal virus" we have been sold?

Looking into the history of the "virus" paints an entirely different picture than the one sold by the MSM. Not only was the initial outbreak among polio vaccine researchers considered not alarming from the start, the patients were treated at home for up to 10 days. Those who were eventually admitted to the hospital were the ones who ultimately succumbed as quickly as one day after admission. One patient, who was considered severely ill, never sought hospital treatment and made a full recovery. Without knowing the exact treatments given to the patients prior to and immediately after admission to the hospital, one must wonder whether the treatments caused the severity of the symptoms seen in those who ultimately succumbed to the disease.

The discovery process for the novel "virus" creates an even more convincing case that there was never any new pathogen detected. First of all, no "virus" was ever purified nor isolated directly from the fluids of a sick human nor monkey. The only evidence used to claim the existence of the Marburg "virus" comes entirely from Guinea pig experiments where unpurified goo and tissues were injected and serially passaged in the animals. After experimentally sickening the animals, non-specific antibody testing was performed in order to find Negri-like inclusions (said to be specific to rabies but shown repeatedly not to be) in the spleens and livers of the Guinea pigs, and then blood samples were sent for electron microscopy imaging in order to find the "virus." The hunt for the "virus" particles was long and the initial experienced researcher was unable to find anything after a day-and-a-half of searching. It wasn't until he left for lunch that his less-experienced lab assistant was able to find the "virus" after under an hour of looking. The filament-like particles were chosen as the representative for the Marburg "virus" due to their uniqueness and length. However, as admitted by lead researcher Rudolf Siebert, the particles were morphologically similar to many "viruses," including the

rabies "virus" associated with the aforementioned Negri bodies. We can also find these filament-like forms associated with other "viruses" such as mumps, measles, parainfluenza, and many others.

According to Werner Slenczka, a man intimately involved in generating these EM images, the samples were contaminated by many microbial agents and even "paramyxoviruses" that made the interpretation difficult. He stated that the search for the "viral" particles was fatiguing and often frustrating. This confirms that the images used as evidence for the existence of the Marburg "virus" came from unpurified contaminated samples and thus there is no way to be able to state that the particles associated with the Marburg cases are in fact a "virus" at all. Slenczka also admitted that the monkeys assumed to be the source of the "virus" were never sick at any point and were used in other labs which never experienced any outbreaks whatsoever.

Even more interesting is that the attempts to "isolate" a "virus" were admitted to be failures. Early attempts to propagate the "virus" in cell cultures repeatedly ended in the lack of evidence of any "virus." However, this did not stop researchers from trying numerous cell lines for culturing until they got the results they wanted to see. After successive trial-and-error resulting in confusing, conflicting, and contradictory results, it was decided that, while the Marburg "virus" could propagate in cell cultures, it did so without producing the cytopathogenic effect (CPE), the very criteria used by virologists to indirectly "see" that the "virus" has highjacked the cell in order to replicate.

What we are left with regarding the Marburg "virus" are non-specific symptoms of disease associated with non-specific Negri-like inclusions in tissues and particles claimed to be unique "viral" structures that are in fact morphologically similar to many other "viral" particles. We also have a "virus" that is said to propagate in cell culture while not producing the very effect looked for to determine a "virus" is present within the sample. In other words, the Marburg "virus" is yet another in a long line of names given to the same symptoms of disease represented by the same random particles. It is just another fraudulent theatrical production put forward by the pseudoscience known as virology.

48 comments



mike