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The Ebola virus: a review of progress and development in research

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ABSTRACT

The Ebola virus was identified in the year 1976 and has caused periodic outbreaks in West African countries. The disease has a case fatality rate up to 90%. Ebola has been classified as a biosafety level four pathogen and there is no currently approved vaccine or treatment for the virus. However, remarkable progress has been demonstrated by researchers in understanding the pathogenicity of the Ebola virus. Several animal models have been cultivated to develop diagnostics, vaccines and therapeutic drugs.

1. Introduction

For the past decade researches have been conducted in laboratories to better understand the biology and potential therapies of Ebola virus (EBOV)[1]. However, field based research in high risk populations such as impoverished villages much progress has not been accomplished. For instance, there have been outbreaks in the Democratic Republic of Congo in 2007, 2008 and in Uganda in 2007[2].

The EBOV belongs to the Filoviridae family[3] which affects both human and non-human primates (NHPs), causing severe hemorrhagic fever syndrome. The disease is characterized with symptoms and signs of fever, focal necrosis of the liver, kidney and spleen bleeding diathesis, fulminant shock resulting in death with a mortality rate reaching 90%[4,5]. The first two outbreak of the EBOV included illnesses such as fever, headache, vomiting and diarrhea. Nonetheless, during the early diagnosis of the EBOV, hemorrhagic manifestations were the most prominent features seen in patients who died[6]. The Filoviridae consist of three general names known as EBOV, Marburg virus (MARV) and Cuevavirus[7]. The disease is also considered to be a category A agent and potential bio-weapon agent[8].

The first outbreak of an unknown infectious disease (Marburg disease) was reported in Germany and Yugoslavia in the year 1967. An estimated 31 persons were affected in which 7 persons died. Eventually, a new strand of the virus was extracted from a patient and was traced back to velvet monkey imported from Uganda. The disease was named the 'Marburg disease' because it was located in the West German town of Marburg[9].

In 1976, an occurrence of hemorrhagic fever started to spread rapidly in Sudan and Zaire with tremendous level of deaths. Specimens were isolated from patients and tested which revealed that the virus resembled the MARV but had different reactive

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properties[9].

This paper aims to review various researches done, developments and progress made concerning the EBOV over the past several years.

2. Epidemiology

The EBOV has a case fatality rate of 30% to 90% and increased frequency in the African region due to weaker health infrastructure and services. The EBOV is sub-divided into five species: Zaire ebolavirus (ZEBOV), Sudan ebolavirus (SEBOV), Tai forest ebolavirus, Bundibugyo ebolavirus (BDBV), and Reston ebolavirus (REBOV)[10]. After the first case of the virus was discovered in 1967 in Germany, it appeared in Africa, two neighboring locations: Sudan and Zaire (SEBOV and ZEBOV), now in the Democratic Republic of the Congo[11]. Finally, it was named the EBOV after a small river located in the northwestern region of the Democratic Republic of Congo[12]. The third strain of the virus was discovered in 1994 and it was called the Cote d'Ivoire EBOV which was noted in the Tai forest[13]. The fourth strain of the virus was found in the equatorial Africa and it was called the BDBV[14]. Additionally, the last virus species was discovered in the Philippines and it was named the REBOV. The EBOV continues to be a plague for the occupants of West Africa, with increasing number of outbreaks seen in 2000[5]. The ZEBOV, SEBOV and BDBV has caused the most tremendous outbreak in sub-Saharan Africa. There have been outbreaks of the EBOV in countries such as Uganda, Sudan, Gabon, Democratic Republic of Congo and the Republic of Congo[15].Moreover, the emergence of the REBOV found in pigs raises public health concerns and food safety in the Philippines and can become a major problem in the near future[5]. The first few cases of the EBOV in Zaire occurred among factory workers and the reservoir animal host was unknown[6]. Eventually, an experiment conducted in the regions of Gabon and the Republic of Congo, suggested that fruit bats are believed to be the reservoir for EBOV[16]. And it is transferred to other hosts such as humans and gorillas[17]. Additional host of the virus are small rodents, duikers, NHPs and shrews. The current outbreak in Guinea, Liberia Sierra Leone and Nigeria showed that the greatest mode of contracting the virus is human to human transmission[18].

The virus is highly contagious which is transmitted to individuals in direct contact with bodily fluids from an infected person[19]. The risk of transmission is highest during the latent stage of the disease but the level of transmission decreases during the early stages even if there is a high risk exposure[6]. Persons that are at the greatest risk for infection of the EBOV during an outbreak are, scientists[20], health care workers, relatives and those in close contact with ill individuals and deceased patients. Basic hygienic practices can be cultivated in the prevention of the EBOV such as regular washing of hands and changing of attire before and after getting in contact with these animals. Moreover, the consumption of sick animals should be avoided[18].

Looking at the 2014 EBOV disease (EVD) outbreak in West Africa, as of September 14, 2014, a total of 4 507 probable and confirmed cases, including 2 296 deaths from EVD (Zaire species) had been reported from five countries in West Africa: Guinea, Liberia, Nigeria, Senegal and Sierra Leone. The World Health Organization Ebola Response Team analyzed a detailed subset of data on 3 343 confirmed and 667 probable Ebola cases collected from the five countries and found out that the majority of the patients are 15-44 years of age with 49% male. The case fatality rate was estimated at 70.8% (95% *CI*, 69-73) among persons with known clinical outcome of infection. The course of infection, including signs and symptoms, incubation period (11.4 d) and serial interval (15.3 d), is similar to that reported in previous outbreaks of EVD. Assuming no change in the control measures for this epidemic, the team projected that by November 2, 2014, the cumulative reported numbers of confirmed and probable cases will be 5 740 in Guinea, 9 890 in Liberia and 5 000 in Sierra Leone, exceeding 20 000 in total[21].

In the 2014 outbreak, the World Health Organization conducted a virological analysis to determine if there was any linkage between the EBOV in West Africa and the Democratic Republic of Congo. The epidemiological investigation and results concluded that the outbreaks in the Democratic Republic of Congo were completely separate and independent event from the cases reported in West Africa. The finding reassures investigators that the virus has not spread from West to Central Africa[22]. However, investigators have isolated 99 EBOV genomes from infected patients in Sierra Leone. Upon examination of the specimens, investigators concluded that there is rapid mutation of the virus which could have implication for the development of diagnostics, vaccines, and therapies of the EBOV. It was observed that the sequence of the virus has changed since the start of the outbreak and the researchers have not found any additional zoonotic sources of the virus in the outbreak strains. Additionally, it was mentioned that the EBOV can affect approximately 20 000 persons before it is contained[23]. Nonetheless, the typical symptoms seen in patients with the EBOV can be mistaken for other infectious diseases that are more common[2].

3. Transmission epidemiology

A published article in 1995 reported that two control NHPs were infected with the ZEBOV without direct contact with