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# Lassa Fever in West Africa: A Clinical and Epidemiological Review

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## Authors' contributions

This work was carried out in collaboration between all authors. Author DJO designed the study, performed the statistical analysis, wrote the protocol, and wrote the first draft of the manuscript. Authors DJO and GIA managed the analyses of the study. Authors DJO and POE managed the literature searches. All authors read and approved the final manuscript.

#### Article Information

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**Review Article** 

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# ABSTRACT

Since 1969, when the first case of Lassa fever was documented in Lassa town, Nigeria, Lassa fever has assumed endemic proportion with yearly outbreaks in West African countries of Nigeria, Sierra Leone, Guinea, and Liberia; with devastating public health implications. Despite the progress made in recent times in understanding the replication pattern of Lassa fever and the breakthroughs in the pathogenesis and molecular epidemiology of Lassa virus, as well as the development of the appropriate technologies for early diagnosis of this highly virulent and contagious viral hemorrhagic disease, control of Lassa fever outbreaks has remained elusive in the West African sub-region.

This review article aims to provide the healthcare workers and other stakeholders with the needed comprehensive knowledge about Lassa virus, and the disease it causes, with emphasis on the control and preventive measures. The data for this review was gotten from analysis of research articles and surveillance reports on Lassa fever, using search engines such as Pub Med, Google Scholar, and department of health websites.

Ineffective control measures and non-availability of resources for early diagnosis and prompt treatment of cases were identified to be responsible for the frequent outbreaks and high case fatality rates. We made a case for international collaboration in the development of effective and affordable vaccine for Lassa fever.

Keywords: Lassa virus; Lassa fever; West Africa; endemic; epidemics; prevention; Lassa fever vaccine.

# **1. INTRODUCTION**

Lassa fever is an acute viral zoonotic illness caused by Lassa virus, an arenavirus known to be responsible for a severe hemorrhagic fever characterized by fever, muscle aches, sore throat, nausea, vomiting, and chest and abdominal pain. The virus exhibits persistent, asymptomatic infection, with profuse urinary virus excretion in Mastomys natalensis, the ubiquitous and highly commensal rodent host [1]. Lassa virus is transmitted by contact with excretions or secretions (including feces and urine) of infected rats accessing food items and water inside human residences and other centers with human activities. Other possible routes of transmission of Lassa fever such as broken skin or mucus membrane directly exposed to infectious material have also been suggested by other investigators. Epidemics arising from human-to-human transmission have equally been established in healthcare institutions in West Africa. Lassa fever is endemic in West Africa, with 300,000 to 500,000 cases and 5000 deaths occurring yearly across Nigeria, Sierra Leone, Guinea, and Liberia. However, between 70-80% of Lassa virus infections remain asymptomatic, mild or self-limiting and in most cases may pass unnoticed [1-4].

In spite of the progress made in recent years in understanding the etiopathogenesis and molecular epidemiology of Lassa fever, as well as the development of the state-of-the-art technologies for early diagnosis of this lifethreatening disease, West African sub-region has continued to experience frequent community and nosocomial outbreaks, with high morbidity and mortality rates and serious economic burden.

There is no available vaccine for Lassa fever. Hence, control measures targeted at reducing rodent-to-human contact and human-to-human transmission; and early identification of infected individuals and prompt treatment are focal points in curtailing the yearly epidemics. The objective of this article, therefore, is to provide a critical overview of Lassa virus infection in the West African sub-region with consideration of the origin of the virus, its virology and pathogenesis, epidemiology and clinical presentation, treatment, prevention and control, as well as the efforts in vaccine development.

# 2. METHODS

The data for this review was gotten from analysis of research articles and surveillance reports on Lassa fever, using search engines such as Pub Med, Google Scholar, and department of health websites. We searched the published medical literature up to August, 2017 for relevant articles written in English, using the keywords: "Lassa virus", "Lassa fever", "West Africa", "endemic", "epidemics", "prevention", and "Lassa fever vaccine". All abstracts found were screened by one reviewer. Those potentially eligible were read in full by two reviewers independently and subsequently discussed during a consensus meeting. Reference lists of each of the publications were cross checked to retrieve relevant publications that had not been identified during the initial search. The exclusion criterion was non-English articles.

## 2.1 Historical Perspectives

There are documented cases of deaths from undiagnosed clinical entities resembling Lassa fever between 1920 and 1950, in Nigeria, Sierra Leone, and other West African countries [5]. However, Lassa fever virus was first detected (1969) and reported (1970) by Frame and his colleagues in Lassa town (from where the virus derived its name) located in Borno State, North East geopolitical zone of Nigeria [5]. The first victim of Lassa virus infection is an American Missionary working in the area who later died of complications arising from the illness. That same outbreak had a nosocomial spread to Jos, Plateau State, North Central Nigeria (1969-1970) [4]. Following its historical discovery in Nigeria, epidemics of Lassa fever have been reported in other West African countries. It has also been transported across the borders of American and European countries.

## 2.2 Epidemiology

Lassa fever is endemic in several West African countries (Fig. 1) including Nigeria, Sierra Leone, Guinea, and Liberia [6]. Every year, between 300,000 - 500,000 people in this region are affected, resulting in over 5,000 deaths annually [4,7]. In endemic situation, the overall case fatality rate (CFR) of Lassa fever is estimated to be in the range of 1-10 percent. However, during epidemic outbreak, the CFR of Lassa virus may be up to 50 percent while higher rate has been recorded in severe cases [8]. The high degree of seroprevalence of Lassa virus - specific antibodies in the general population residing in the endemic regions, although highly variable depending on the geographical location (from 7% in Guinea and 15-20% in Sierra Leone and Liberia; to over 20% in Nigeria) [3,9], indicates that most infections are mild or possibly even asymptomatic and do not result in hospitalization. Nosocomial outbreaks are however, associated with higher mortality rates ranging from 36% to 65% [10,11]. There is no age, gender or racial predilection.

Serological evidence of Lassa fever has also been found in Mali, Senegal and Central African Republic. Sporadic imported cases have been reported in the United States of America, Europe, and Asia, and laboratory infection has occurred among health workers in the USA during handling of infected specimens [12,13].

## 2.3 Risk Factors for Transmission

Lassa fever outbreaks in endemic areas are invariably fuelled by every activity or factor that promotes increased contact between man and rodent. They include poor sanitation, crowding, deforestation, rodent hunting, bush burning, civil unrest, and agricultural activities such as rice cultivation that provide food supplies for rodents [14]. Rural dwellers in West Africa are at risk of Lassa fever because of proximity to animal reservoir, open construction of African villages, the practice of drying grains by road sides or outside homes and unprotected grain storage within homes. All these factors are known to facilitate increased rodent-man contact or contamination of food sources by infected rodent secretions [15].

It has been shown that immunosuppression arising from certain underlying communicable or non-communicable diseases, chemotherapy as well as pregnancy (especially if infection occurs during the third trimester) can enhance the acquisition and establishment of Lassa fever, and may aggravate mortality rate pushing it up to about 80 percent [16]. Infection during pregnancy can lead to fetal death (because the virus has high affinity for placenta and other highly vascularized tissues), abortion, including loss of newborn (in 90% of cases) or maternal



Fig. 1. Lassa fever distribution map (https://web.stanford.edu/group/virus/arena/2005/LassaFeverVirus.htm)

death [17,18]. Serious congenital defects or anomalies are common sequelae in children born with Lassa fever infection.

## 2.4 Mechanism of Transmission

Lassa fever is a zoonotic disease and the Lassa virus exhibits persistent, asymptomatic infection with profuse urinary virus excretion in the ubiquitous rodent vector, Mastomys natalensis [3]. There is new evidence that Lassa virus is also hosted by other rodent species: the African wood mouse Hylomyscus pamfi in Nigeria, and the Guinea multimammate mouse Mastomys erythroleucus in both Nigeria and Guinea [19]. Lassa fever is transmitted to humans via contact with an infected rodent, or through inhalation of air contaminated with infected rat's excretions or secretions such as feces, urine or nasal discharges (aerosols) [8]. Lassa virus infection can also be acquired through broken skin or mucous membrane directly exposed to infectious material or item [3,16].

Nosocomial acquisition of Lassa virus infection is mainly through contact with infected patient, exposed hospital workers or unscreened infected blood [16]. Similarly, direct contact with infected semen, or vaginal fluids including consumption of infected breast milk have been suggested as possible modes of transmission of Lassa fever [4].

# 2.5 Virology

Lassa virus, an Arenaviridae, is categorized under the group known as 'Old World Arenaviruses' on the basis of their antigenic and molecular properties [20,21]. The group consists of Lassa virus and Lymphocytic choriomeningitis virus (LCMV). Lassa virus is characterized by high genetic variability hence there was initial difficulty regarding the design of primers for Polymerase Chain Reaction (PCR) in molecular studies of the virus. Consequently, some Lassa virus strains were believed to escape detection by PCR during the early studies [22]. Other members of the Old World African Arenaviruses that share similar properties and closely related to Lassa virus include Ippy, Mobala, and Mopeia viruses. However, these strains of Arenaviruses have not yet been associated with any human disease [23,24].

Lassa virus is an enveloped, single-stranded, bisegmented RNA virus (Figs. 2 and 3). It is a rapidly replicating virus and has inherent ability to temporarily control its own replication. This attribute preferentially allows the spike proteins component of the virus to be produced last during replication, and therefore, delay the recognition of the virus by the host's immune system. Consequently, the process is believed to aid the virus virulence and evasion of the host's defense mechanism [25,26].

Lassa virus genome nucleotide studies have revealed the existence of four lineages of the virus; three members (lineage I-III) of which are found in Nigeria while the fourth (lineage IV) was traced to other parts of West Africa including Liberia, Guinea and Sierra Leone. There is no documentation of any difference in the case fatality ratio among the different lineages [21,27,28].

## 2.6 Pathogenesis

Following acquisition of the virus through contact with infected rodent urine, saliva, respiratory secretion, or blood; Lassa fever infection is initiated in the victim. While the main targets of Lassa virus once inside the host are the antigenpresenting cells, Lassa virus infects almost every

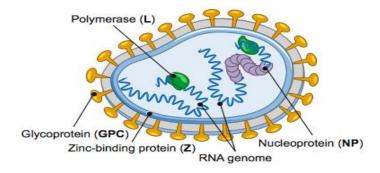


Fig. 2. Lassa virion (http://www.cell.com/doi/story/10.1016/pic.2015.08.11.2271)

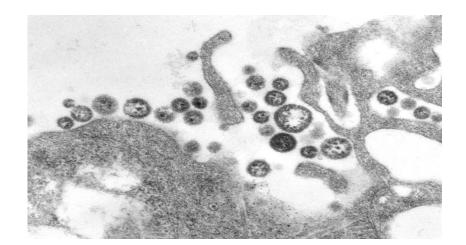


Fig. 3. Transmission electron microscopic (TEM) image depicted numbers of Lassa virus virions adjacent to some cell debris (https://phil.cdc.gov/phil/details.asp?pid=8700)

tissue in human body leading to multi-systemic dysfunction, and can suppress host's innate interferon (IFN) response by inhibiting the translocation of interferon regulatory factor-3 (IRF-3) [26,29]. In addition, Lassa virus characteristically exhibits exonuclease activity to only double-stranded RNAs (ds RNAs), which often blocks IFN responses. This is achieved through digestion of pathogen associated molecular pattern (PAMP), which enables the virus to evade host's immune responses [4].

The blood vessels are always the most affected and the virus multiplies in cells of the reticuloendothelial system causing capillary lesions. Hemorrhage may be present in the intestine, liver, myocardium, lungs and the brain [3,30].

Patients with subclinical infections to Lassa fever pass unnoticed. However, this group, along with survivors of the acute Lassa fever illness, stands the risk of developing sensorineural hearing loss of different degrees in future. The hearing loss is usually bilateral and can affect all frequencies of hearing [31], and according to WHO about 25% of patients exposed to Lassa virus are affected [2]. The pathogenesis of this loss is believed to follow an hearing immunological reaction between the circulating Lassa virus antibodies and the basal cell membrane /outer hair cells of the cochlear [31]. Other neurological complications sometimes associated with survivors of Lassa fever are seizures, gait disturbances, tremors and encephalitis [32].

#### 2.7 Clinical Features

In majority of Lassa fever virus infections (approximately 80%) in endemic areas, the symptoms are mild and are undiagnosed. Mild symptoms include slight fever, general malaise and weakness, and headache. In 20% of infected individuals, however, disease may progress to more serious symptoms including hemorrhaging (in gums, eyes, or nose, as examples), respiratory distress, repeated vomiting, facial swelling, pain in the chest, back, and abdomen, and shock with significant fatality (CFR, 40-50%) especially during epidemic outbreaks [14,33]. Generally, incubation period ranges from 6 to 21 days [18,22]. The typical case progression can be divided into three main stages.

Stage 1: Prodromal Illness/Acute Stage: At this stage, the onset of the disease mimics malaria or typhoid fever. First, it begins with respiratory flusymptoms (non-specific illness) like characterized by headache, myalgia, febrile illness, cough, and pharyngitis. Other symptoms include tremors, chest pain, insomnia, and sometimes rashes coupled with gastrointestinal symptoms including diarrhea and vomiting. These earlv symptoms often appear indistinguishable from other bacterial, viral or parasitic infections [31].

Stage 2: Hemorrhagic Stage: This stage involves internal hemorrhage whereby victim bleeds from inside through nostrils, mouth and other orifices resembling that of Ebola. This may lead to organ failure and death [2,31].

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Stage 3: Neurologic Complications: This constitutes part of the late Stage of the illness neurological complications manifesting as including encephalopathy or encephalitis [30]. The most common complication of Lassa fever is deafness. Various degrees of deafness occur in approximately one-third of infections, and in many cases hearing loss is permanent. As far as is known, severity of the disease does not affect this complication; deafness may develop in mild as well as in severe cases. The virus can be detected in the urine of infected patient for 3-9 weeks and in semen for up to three months [2].

Death may occur within two weeks after symptom onset due to multi-organ failure. Approximately 15-20% of patients hospitalized for Lassa fever die from the illness. However, only 1% of all Lassa virus infections result in death [33]. The death rates for women in the third trimester of pregnancy are particularly high. Spontaneous abortion is a serious complication of infection with an estimated 95% mortality in fetuses of infected pregnant mothers [33].

# 2.8 Diagnosis

Lassa fever has emerged as one of the most prevalent viral hemorrhagic fevers in West Africa. However, in most Lassa fever endemic areas of the region, there are serious challenges regarding the laboratory diagnosis and confirmation of the disease due to inadequate facility and low capacity [34].

Because the symptoms of Lassa fever are so varied and nonspecific, clinical diagnosis is often difficult. Lassa fever is most often diagnosed by using enzyme-linked immunosorbent assays (ELISA), which detect IgM and IgG antibodies as well as Lassa antigen (88% sensitivity and 90% specificity for the presence of Lassa fever) [4]. Reverse transcription-polymerase chain reaction (RT-PCR) can be used in the early stage of disease [35]. The virus itself may be cultured in 7 to 10 days, [33] but this procedure should only be done in a high containment laboratory with good laboratory practices. Immunohistochemistry, performed on formalin fixed tissue specimens, can be used to make a post mortem diagnosis [33].

Regardless of the method adopted, Lassa fever will require a biosafety level 4 – equivalent containment during laboratory diagnosis to prevent the acquisition and spread of the disease in the laboratory and hospital environment [4].

# 2.9 Real-time RT-PCR Assay for Lassa Fever

In a recent study on the molecular confirmation of Lassa fever imported into Ghana. real-time RT-PCR assay was performed using the ABI 7300/7500 Real Time PCR System (Life Technology Holdings, Singapore) for the detection of Lassa virus with primers specific for regions of the small (S) RNA gene segment. The reagent used in the preparation of the master mix for the 45-cycle RT-PCR was the Power SYBR® RNA-to-CT™ 1-Step Green Kit (Life Technologies, Carlsbad, California, United States) with the sense primer 36E2 and the antisense primer LVS-339-rev (Table 1). The amplification protocol was as follows: 48 °C for 30 minutes; 95 °C for 10 minutes; and 45 cycles of 95 °C for 15 seconds, 60 °C for 1 minute. At the end of each cycle, fluorescence was measured at 60 °C [36].

Table 1. Sample reverse transcription-PCR test assays (culled from Bonney JHK, et al. Molecular confirmation of Lassa fever imported into Ghana. Afr J Lab Med. 2016;5(1):288)

| Virus | Reagents; Primers and probes (sequence 5' – 3')                    | Target region |
|-------|--|---------------|
| Lassa | OneStep RT-PCR kit (Qiagen);                                       | S/GPC         |
|       | 36E2, ACC GGG GAT CCT AGG CAT TT (10 μM);                          | gene          |
|       | LVS-339-rev, GTT CTT TGT GCA GGA MAG GGG CAT KGT CAT (10 µM)       | -             |
| Lassa | AgPath-ID One-Step RT-PCR kit (Ambion);                            | L-gene        |
|       | LVL 3359D_Y+, AGA ATC AGT GAA AGG GAA AGC AAY TC (10 µM);          | -             |
|       | LVL 3359G_Y+, AGA ATT AGT GAA AGG GAG AGT AAY TC (10 $\mu$ M);     |               |
|       | LVL 3754A_R-, CAC ATC ATT GGT CCC CAT TTA CTA TGR TC (10 $\mu$ M); |               |
|       | LVL 3754D_R-, CAC ATC ATT GGT CCC CAT TTA CTG TGR TC (10 µM)       |               |
| Lassa | Power SYBR® Green RNA-to-CT™ 1-Step Kit (Life Technologies);       | S/GPC         |
|       | 36E2, ACC GGG GAT CCT AGG CAT TT (10 μM);                          | gene          |
|       | LVS-339-rev, GTT CTT TGT GCA GGA MAG GGG CAT KGT CAT (10 µM)       | -             |

## 2.10 Differential Diagnosis

Lassa hemorrhagic fever must be differentiated from other febrile diseases like Ebola, Marburg hemorrhagic fever, malaria, diphtheria, legionellosis, yellow fever, Congo hemorrhagic fever, etc. It is very imperative that clinical assessment be combined with specific laboratory diagnosis to adequately identify the Lassa fever virus in order to commence early treatment which is paramount to the survival of infected individuals [3].

## 2.11 Treatment

Similar to other severe hemorrhagic fevers, supportive treatment is the cornerstone of clinical management of Lassa fever. The main goal is volume resuscitation, accounting for third spacing, diarrhea and vomiting, while avoiding volume overload due to the risk of pulmonary edema. Other goals are electrolyte balance and respiratory support [33].

The only pharmacologic agent with a proven therapeutic effect in individuals with Lassa hemorrhagic fever is the broad-spectrum nucleoside (guanosine) analogue, ribavirin and it is considered the drug of choice for the treatment of the disease. The exact mechanism of action of ribavirin against Lassa virus has not been fully elucidated, but the drug appears to be incorporated into the RNA strand, leading to lethal mutagenesis of progeny genomes [6,37,38].

Benefit was even more significant in higher risk patients with high level viremia and increased liver function tests, and when given within 6 days from symptoms onset. Both oral and intravenous ribavirin were beneficial, with the latter showing a stronger effect in higher risk cases. The recommended intravenous dose is 2.4 g loading dose, followed by 1 g every 6 hours for 10 days, for average weight adults [39]. The side effect profile of ribavirin is as shown in Table 2.

Convalescent plasma, although beneficial in some animal experiments, has failed clinical studies, probably due to lack of neutralizing antibodies. Various experimental treatments were evaluated for Lassa virus, including antivirals, molecules targeting host cells, and immunomodulators. None is currently clinically proven or approved for treatment [40,41].

| Table 2. Adverse | effects associated with |
|------------------|-------------------------|
| ribavirin        | use in humans           |

| Drug form, adverse effect  |  |  |  |
|--|--|--|--|
| Intravenous form   |  |  |  |
| Hemolytic anemia (usually mild to moderate   |  |  |  |
| and reversible)  |  |  |  |
| Rigors with rapid infusion   |  |  |  |
| Oral form <sup>a</sup>   |  |  |  |
| Anemia   |  |  |  |
| Nausea and vomiting  |  |  |  |
| Metallic taste   |  |  |  |
| Dry mouth  |  |  |  |
| Myalgia  |  |  |  |
| Fatigue  |  |  |  |
| Diarrhea   |  |  |  |
| Abdominal pain   |  |  |  |
| Headache   |  |  |  |
| Jaundice   |  |  |  |
| Skin rash  |  |  |  |
| Tachycardia  |  |  |  |
| Thrombocytosis   |  |  |  |
| Elevated lipase level  |  |  |  |
| Neurological perturbations (including mood   |  |  |  |
| and sleep disturbances) <sup>a</sup> Most adverse effects associated with oral ribavirin |  |  |  |
| occur only after prolonged use (weeks to months),  |  |  |  |
| although mild decreases in red blood cell counts have                                    |  |  |  |
| been reported after only 10 days of administration,                                      |  |  |  |
| especially in children   |  |  |  |
| (Adopted from Bausch DG, et al. Review of the  |  |  |  |
| Literature and Proposed Guidelines for the Use of Oral                                   |  |  |  |
| Ribavirin as Postexposure Prophylaxis for Lassa  |  |  |  |
| Fever. Clin Infect Dis. Academic Press, New York;<br>2010:51(12):1435–41)                |  |  |  |
| 2010,01(12).1400-41)   |  |  |  |

## 2.12 Prevention

General measures include institution of policies, task force, committees for surveillance, prevention and control of Lassa fever at national and state levels. Health education/sensitization of the public and health workers about the disease, transmission, manifestations and prevention [15].

Control of rodents by avoiding bush burning, setting traps in and around homes to reduce rat population, blockage of all rat hideouts, and avoidance of contact with rats such as rat hunting for consumption.

Individual and community preventive strategies include keeping good and healthy personal hygiene, cleaning of homes and surrounding environment, and waste should be emptied far away from homes. Spreading of food where rats can have access to it, e.g. by the road side, should be avoided. Storage of foodstuff and water should be done in rat proof containers [15].

Hospital based prevention should focus on adherence to infection control measures, isolation of infected patients, barrier nursing of infected patients, and use of personal protective equipment when caring for patients or working with secretions of infected patients [15].

## 2.13 Post Exposure Prophylaxis

Post exposure prophylaxis (PEP) with ribavirin is recommended for Lassa fever exclusively in the event of a definitive high-risk exposure.

The following are considered high-risk exposures:

- 1. Penetration of skin by a contaminated sharp instrument (e.g., needle stick injury),
- Contamination of mucous membranes or broken skin with blood or bodily secretions (e.g., blood splashing in the eyes or mouth)
- Participation in emergency procedures (e.g., resuscitation after cardiac arrest, intubation, or suctioning) without use of appropriate personal protective equipment
- prolonged (i.e., for hours) and continuous contact in an enclosed space without use of appropriate personal protective equipment [42].

In estimating the risk of infection, it is important to realize that titers of Lassa virus in blood and bodily secretions correlate with disease severity. The most infectious patients are those with severe clinical conditions, usually late in the course of illness. In all cases, the exposure to the index case must be during the period of acute febrile illness. There is no evidence of Lassa virus transmission during the incubation period or after recovery, with the exception of sexual transmission, in which transmission rarely occurs because of delayed clearance (3months after acute infection) of Lassa virus from the gonads [15,43].

When ribavirin PEP is indicated, oral regimen of a 35-mg/kg loading dose (maximum dose, 2.5 g) followed by 15 mg/kg (maximum dose, 1 g) 3 times a day for 10 days is recommended [42]. Patients who develop manifestations of Lassa fever within 21 days of starting oral ribavirin PEP should be immediately tested for Lassa fever by the most sensitive method, usually reverse transcription-PCR, and treatment should be switched to the intravenous form unless the disease can be readily excluded [42,44].

## 2.14 Vaccine

Although the prevention of human contact with the Mastomys rodents is an essential factor in the control of Lassa fever, widespread prevention of such contact is presently impractical in the endemic regions of West Africa, so provision of a vaccine for community and hospital use is an imperative public health need because vaccination is the most viable control measure [45]. Lassa virus vaccine development is hampered by high cost of biocontainment requirement, the absence of appropriate small animal models, genetic diversity of Lassa virus species, and by high HIV prevalence in Lassa fever endemic areas [46]. A previous report had indicated that a vaccinia virus expressing the Lassa virus glycoprotein protected four nonhuman primates against lethal challenge with Lassa virus [3]. This was followed by the finding that single administration of a vaccine expressing the full-length Lassa virus glycoprotein affords protection against Lassa fever in primates, with or without expression of the nucleoprotein [3,47].

The challenge, however, is to overcome the scientific, political and economic obstacles in producing a human use vaccine candidate. It is well established that the glycoprotein confers protection but its duration is unknown and if the nucleoprotein is also included there may be a better duration of protection but it is unclear whether the nucleoprotein as a vaccine may possibly enhance the infection. Two lead Replication Competent (RC) vaccine candidates, reassortant ML29 and recombinant VSV/LASV. have been successfully tested in non-human primates and have been recommended by international vaccine experts for rapid clinical development. Both platforms have powerful molecular tools to further secure safety, improve immunogenicity, and cross-protection. These platforms are well positioned to design multivalent vaccines to protect against all Lassa virus strains found in West Africa [46,48]. Applications of new vaccine technologies give hope that there may soon be a vaccine in clinical trials, however, the difficulty of conducting trials in endemic areas of the West African sub-region and lack of political will remain serious problems [3,49,50].

#### **3. RECOMMENDATIONS**

At the community level, a continuing education of the populace on the dangers of the disease, its modes of transmission and presentation, and the need to seek medical treatment early should be intensified. Awareness campaigns and advocacy on clean and safe environment to promote prevention within the endemic areas are necessary. Abrogation of practices that might enhance contact with the Mastomys rodents should be encouraged.

The secondary level of control should involve setting up operational diagnostic and treatment centers for Lassa fever within the West African sub-region to enhance prompt diagnosis, therapy, and containment of the illness. The health personnel that work in such centers should have access to personal protective equipment, paid enhanced salaries and their lives insured. Well designed and funded researches should be instituted in this field to facilitate ameliorating breakthroughs. Finally, the ultimate aim should be towards producing an effective and safe Lassa fever vaccine that could be incorporated into the national programs on immunization for children and adults [32].

#### 4. CONCLUSION

Lassa fever is a very important zoonotic disease with high endemicity in West Africa. It has yearly outbreaks in this region with high morbidity and mortality, and serious public health impacts. Lassa fever remains a threat, not only to West Africa, but to the world especially in the face of intercontinental heightened travels and increasing globalization. Unfortunately, efforts to control Lassa fever in resource scarce West Africa have been hampered by inadequate research programs and poor political will in sustaining the activities of the international donor agencies.

Proactive measures to control this menace should target adequate education of health care professionals, public health campaigns, and advocacy. It is also necessary to focus on increasing the number of infectious disease control centers with state-of-the-arts diagnostic and research laboratory services, as well as treatment facilities across the West African subregion.

For example, in 2004, the WHO, Tulane University, the Mano River Union country (Guinea, Sierra Leone, and Liberia) governments, and various collaborators and funders established the Mano River Union Lassa Fever Network (MRU-LFN) to assist in the development of national and regional prevention and control strategies for Lassa fever, including the enhancement of laboratory capacity, and training in laboratory diagnosis, clinical management, and infection and environmental control. More international collaborations are needed to conduct further researches into the development of effective and affordable Lassa fever vaccine for West African region. Until then, outbreaks of Lassa fever will continue to ravage and decimate the West African sub-region on yearly basis.

## CONSENT

It is not applicable.

#### ETHICAL APPROVAL

It is not applicable.

#### **COMPETING INTERESTS**

Authors have declared that no competing interests exist.

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