Nigerian Journal of Mathematics and Applications
Volume 32 (II); (2022); 241 258 Printed by Unilorin press
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Special Issue in Honor of Prof. J. A. Gbadeyan's Retirement

A Mathematical Model on Marburg Virus Disease

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Abstract

This research discusses the management of Marburg virus disease (MVD) using a mathematical model. The formulated model consists of ve (5) mutually exclusive compartments. The local and global stability of the DFE of the formulated model is established. Subsequently, it is shown that the e ective management of the disease depends greatly on the incubation period and exposure to the infection. The more the incubation period, the greater the chances of disease incidence reduction.

1. Introduction

1.1. Background. Viral haemorrhagic fevers (VHF) are a group of diseases caused by several distinct families of viruses with multi-organ involvement, predominantly a ecting the cardiovascular system. The spectrum of disease caused by these viruses are quite varied and ranges from asymptomatic infection at one end, to life threatening bleeding from di erent bodily sites at the other extreme. In addition to being zoonotic infections, they are also ribonucleic acid (RNA) viruses known to have the capacity to change at high rate over time and with RNA viruses been the most common cause of emerging disease (diseases from newly identi ed and previously unknown infectious agents) in human population, they are of public health importance. Marburg virus like Ebola virus both belong to the Filoviridae virus family which has envelope made of lipid with each virion containing one single stranded, negative-sense RNA. Marburg virus disease (MVD), formerly known as Marburg haemorrhagic fever is a highly virulent disease with case fatality rate ranging from 24% - 88%. The virus is transmitted to people from fruit bats and spreads among people through human-to-human transmission.

Received: 05/11/2022, Accepted: 02/12/2022, Revised: 12/12/2022. Corresponding author.
2015 Mathematics Subject Classi cation. 00A71 & 03C98.
Keywords and phrases. Marburg virus disease, Mathematical model, Stability analysis
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1.2. History. In early August of 1967, some laboratory workers in Marburg an der Lahn and Frankfurt am Main, west Germany as well as those in Belgrade, Serbia (formerly Yugoslavia) presented with unusual symptoms suspected to be caused by infectious agent of unknown origin. The symptoms were initially unalarming necessitating home-based treatment in Germany. However, by the second week of presentation, haemorrhagic tendency had evolved in some patients warranting admission in university hospitals in Marburg and Frankfurt [1]. Retrospective studies traced the source of infection to African green monkey imported from Uganda to these three locations where the monkeys were been dissected to isolate their kidney cells for the purpose of culturing poliomyelitis vaccine strains [2].
In Germany, all primary cases had either direct contact with grivet monkeys imported from a single Ugandan primate exporter via London, UK, or direct contact with grivet-derived tissues. The primary cases in Marburg involved personnel of a poliomyelitis vaccine manufacturer during activities aimed at the establishment of primary grivet cell cultures using tissues from the imported Ugandan grivets, most likely infected with Marburg virus prior to exportation [3]. Frankfurt outbreak primarily a ected laboratory personnels working at a West German government facility responsible for the safety testing of poliomyelitis vaccines (Paul Ehrlich Institute), them being infected during handling of tissues derived from the imported Ugandan grivets. Following primary infections, secondary nosocomial infections occurred during treatment of the primary case at university hospital and during pathological examination of patients.
Belgrade outbreak was also linked to the imported Ugandan grivet monkeys. The Institute of Virology, Vaccines and Sera of Belgrade received three shipments of nonhuman primates from Uganda via a german dealer and during quarantine of these animals, an unusually high number of them died prompting the institute to perform necropsies on two of the deceased grivets. Sadly however, the veterinarian that performed the necropsies became infected despite donning full complement of personal protective equipment (PPE). After removing his PPE, he held the grivet blood contaminated petri dish hence, becoming infected. His wife who cared for him during the initial phase of his illness at the couple's home had contact with a disposed soiled linen and a blood-soaked cotton gauze pad used during blood sample collection from the veterinarian [3].

1.3. Epidemiology. There was uncertainty as to the origin of the virus, especially as the incriminated African monkeys transited through animal house in London en-route Germany and Yugoslavia plus the virus had not been isolated in the these monkeys. It would take another 8 years for Africa to record its rst reported case of Marburg virus infection in South Africa. The index case was a young Australian man who had toured Rhodesia (Zimbabwe) having contact with wild animals in 1975 with his female companion. On return to South Africa, he had symptoms similar to those of 1967 outbreak in Europe and later died of the infection. His travel companion as well as the nurse that attended to him tested positive to a strain of Marburg virus closely related to the one isolated in 1967.
Since the rst reported South African case of 1975, other African region have had recorded outbreak like the 1980 and 1987 sporadic cases reported in Kenya as well as the outbreak that a ected 154 people in Democratic Republic of Congo between 1998 { 1999. West Africa recorded its rst outbreak in Angola in 2004-2005, killing 227 of 252 a ected persons.
The reservoir host of Marburg virus is the African fruit bat (Rousettus aegyptiacus), this is further con rmed by Towner et al. who worked on at least 10 species of bats [4]. Marburg virus is more prevalent in open, dry areas of eastern and south-central Africa with almost all of the primary infections of natural Marburg outbreaks linked to human entry into

caves inhabited by bats (e.g., cave visitors, mine workers) [2].
Human contract the virus via exposure to an infected animal: either a reservoir host (several bat species) [9] or a spill-over host like non-human primate as described in the rst outbreak of 1967. Following transmission to human, spread of the virus between individuals is the result of direct contact (e.g via broken skin or mucous membrane) with blood or other body uids (saliva, sweat, stool, urine, tears, and breast milk) from infected patients, and with surfaces and materials (e.g. bedding, clothing) contaminated with these uids. Risk of infection include miners, tourist, unprotected contact with infected bat faeces or aerosols, contact with equipment and other objects contaminated with infectious blood or tissues, administration of medical care to infected individuals, household members of carrier, handling of corpses without use of proper protection, etc.

1.4. Clinical manifestation. The interval from infection to onset of symptoms (i.e. incubation period) varies from 2 to 21 days (typically 5-10 days). Onset of symptoms is usually sudden and marked by fever, chills, headache, and myalgia [5]. The course of Marburg virus disease has conventionally been broken down into three phases namely: an initial generalization phase, an early organ phase, and either a late organ phase or convalescence phase depending upon disease outcome [6].
Generalization phase, been the rst stage of symptoms last through rst four/ ve days of disease onset beginning with u-like illness with a characteristic high fever (typically 39{40^{0}C), severe headache, chills, myalgia, prostration, and malaise. For many patients (50%{75%), this is succeeded by gastrointestinal symptoms like anorexia, abdominal pain, severe nausea, vomiting, and watery diarrhea. Because many of the signs and symptoms at this early stage are similar to some diseases (such as malaria and typhoid fever) endemic to many African countries, Marburg virus disease diagnosis is often di cult at this stage, especially if only a single case is involved. One of the distinguishing features is the appearance enanthem and development of dysphasia, and pharyngitis in addition to lymphadenopathy, leukopenia, and thrombocytopenia however, these do not appear until about four to ve days post infection [6].
In early Organ Phase (Day Five to Thirteen), many of the earlier signs and symptoms persist in addition to neurological symptoms and heamorrhagic tendencies. Neurologic symptom is characterized by encephalitis, confusion, delirium, irritability, and aggression. Patients can also develop dyspnea and abnormal vascular permeability, particularly conjunctival injection and edema. Lately in this phase, more than 75% of patients present with some form of clear haemorrhagic manifestation such as petechiae, mucosal bleeding, melena, bloody diarrhea, hematemesis, and ecchymoses with multiple organs dysfunction (including the pancreas, kidney, and liver) occurring later.
Beyond this stage (now late organ/convalescence phase from day thirteen), patients either die from the disease or enter a prolonged phase of recuperation. Typical symptoms include restlessness, confusion, dementia, convulsions, reduced circulation due to severe dehydration, metabolic disturbances, severe di use coagulopathy, multi-organ failure, shock, and coma. Fatalities typically occur 8{16 days following the onset of symptoms, with death usually due to resulting shock and multiple organ failure. Non-fatal cases are typi ed by an extensive convalescent period during which myalgia, exhaustion, sweating, peeling of the skin at the sites of rash, partial amnesia, and secondary infections are all common.

1.5. Diagnosis. Despite the challenge posed by the similarity in clinical symptomatology of MVD and other infectious diseases, early diagnosis and institution of containment measure is of utmost importance both for the patient and for public health safety. Laboratory diagnosis can be achieved in two ways: measurement of host-speci c immune response to

infection and detection of viral particle in infected individual [7]. Host-speci c immune response to infection can be elucidated via antibody-capture enzyme-linked immunosorbent assay (ELISA) using IgM-capture ELISA and IgG-capture ELISA in early and late disease course respectively. The virus can be visualized in the centrifuged serum of patients with high level of viraemia as well as thin section of infected tissue using direct electron microscopic.
Immunohistochemistry on formalin xed material as well as immuno uorescence on impression smear of tissues can also be used to demonstrate viral particle.
Virus isolation using vero cell culture can also be undertaken on the blood, organ tissues, throat washings, urine or semen of infected patient. Reverse-transcriptase polymerase chain reaction (RT-PCR) assay is also a used as a con rmatory test.

1.6. Prevention and Control. Currently there are no vaccines or antiviral treatments approved for the disease therefore, management of patient is mainly symptomatic and supportive. Once a patient is suspected to have MVD or a con rmed case, barrier-nursing techniques should be used to prevent direct physical contact with the patient. These precautions include wearing of protective gowns, gloves and masks, placing the infected individual in strict isolation, sterilization or proper disposal of needles, equipment, and patient excretions.
Although a rare disease, when it occurs, it has the potential to spread to other people, especially health care sta and family members who care for the patient. Therefore, increasing awareness in communities and among health-care providers of the clinical symptoms of patients with MVD is critical. Better awareness can lead to earlier and stronger precautions against the spread of the disease in both family members and health-care providers. During visits in mines or caves inhabited by fruit bat colonies, people should wear gloves and other appropriate protective clothing (including masks) while partnership between veterinarian and clinician should be strengthened to reduce zoonotic disease burden [9]. Samples taken from humans and animals for investigation of MVD should be handled by trained sta and processed in suitably equipped laboratories while contacts of infected persons should be monitored for 21 days. Corpse attributable to MVD should be buried in a safe and digni ed manner, as direct contact with the corpse could lead to contracting the infection [8].

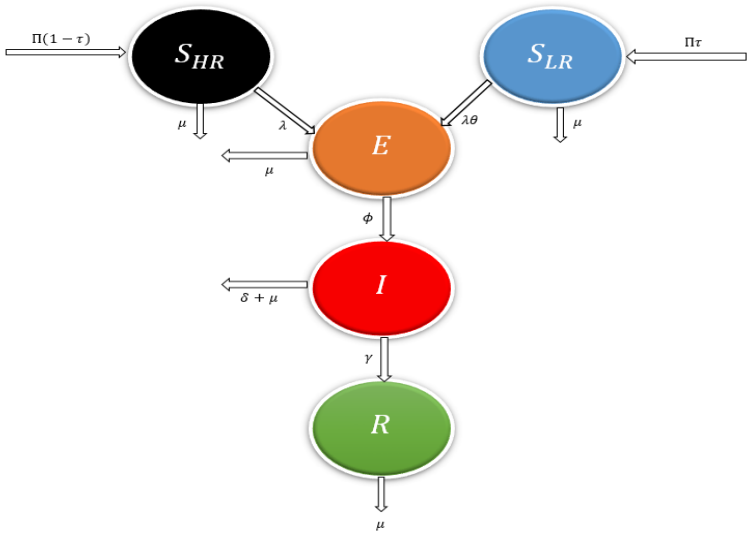
2. Model Formulation

2.1. Introduction. This research considers Marburg virus disease, a disease dating back to 1967. The model is formulated using the famous SEIR approach. All together, the model has ve (5) compartments, as the susceptible class S has two subdivisions. This subdivision is factored due to the risk of exposure level to the infection. Family of the patient diagnosed of MVD, the medical team managing the patient, hunters that had close contact with African fruit bats (including their uids and faeces), which is the reservoir host of the infection and veterinarians who have had contacts with non-human primates that may be carrying the infection are among the high risk individuals tagged as S_{HR}. The remaining individuals in the susceptible class is tagged S_{LR} which is the division of susceptible individuals who are of low risk to contracting the infection. Individuals who are still asymptomatic of the infection are in the exposed (E) compartment while the symptomatic individuals are housed in the infectious (I) compartments. Individuals who have recovered from the infection are kept in the recovered (R) compartment.
Individuals are recruited into the susceptible class (S) at the rate . Since the S class has been divided into S_{HR} and S_{LR}; people move into the S_{LR} compartment at the rate

while the remaining individuals who are at high risk move into the S_{HR} compartment at the rate (1). Individuals who contract the infection do so at the rate after an e ective contact with the carrier (human or animal). However, individuals in the S_{LR} class are assumed to contract the infection at a lesser rate due to limited contact with the infected carrier, hence, a modi cation parameter is introduced to slow down the rate at which the S_{LR} individuals contract the infection. After exposure and contracting the infection, individuals progress to the symptomatic stage at the rate and get recovered by progressing to the recovered class at the rate. Death due to the infection and nature are designated as and respectively. The interaction between these compartments is diagrammatically represented in Figure 1 and mathematically represented as (2.2). is mathematically de ned in (2.3). At any point in time, the total population $N(t)$ is

(2.1) $$N(t) = S_{HR}(t) + S_{LR}(t) + E(t) + I(t) + R(t)$$

with homogeneous mixture of the population assumed.



Figures 1: The Marburg virus disease model

(2.2)
$$\begin{cases} \frac{dS_{HR}}{dt} = (1 \quad) \quad (\quad + \quad) S_{HR} \\ \frac{dS_{LR}}{dt} = \quad (\quad + \quad) S_{LR} \\ \frac{dE}{dt} = (S_{HR} + S_{LR}) \quad (\quad + \quad) E \\ \frac{dI}{dt} = E \quad (\quad + \quad + \quad) I \\ \frac{dR}{dt} = I \quad R \end{cases}$$

(2.3) $$= \frac{I}{N}$$

2.2. Positivity and Boundedness of Solution.

Theorem 2.1. Let $S_{HR}(t) \quad 0$; $S_{LR}(t) \quad 0$; $E(t) \quad 0$; $I(t) \quad 0$; $R(t) \quad 0$; then the solutions S_{HR}; S_{LR}; E; I and R are positive for all $t \quad 0$. The region is positively invariant and attracts all positive solutions.

Proof. Using the given initial conditions, it is easy to show that the solutions are positive. However, assuming by contradiction that there exists another initial time t_{s1}: $S_{HR}(t_{s1}) = 0$; $S'_{HR}(t_{s1}) < 0$ and $S_{HR}(t) > 0; S_{LR}(t) > 0; E(t) > 0; I(t) > 0; R_(t) > 0$ for $0 < t < t_{s1}$ or there exists t_{s2} : $S_{LR}(t_{s2}) = 0$; $S'_{LR}(t_{s2}) < 0$ and $S_{HR}(t) > 0; S_{LR}(t) > 0; E(t) > 0; I(t) > 0; R_(t) > 0$ for $0 < t < t_{s2}$ or there exists $t_e : E(t_e) = 0$; $E'(t_e) < 0$ and $S_{HR}(t) > 0; S_{LR}(t) > 0; E(t) > 0; I(t) > 0; R_(t) > 0$ for $0 < t < t_e$ or there exists t_i : $I(t_i) = 0$; $I'(t_i) < 0$ and $S_{HR}(t) > 0; S_{LR}(t) > 0; E(t) > 0; I(t) > 0; R_(t) > 0$ for $0 < t < t_i$ or there exists t_r : $E(t_e) = 0$; $E'(t_e) < 0$ and $S_{HR}(t) > 0; S_{LR}(t) > 0; E(t) > 0; I(t) > 0; R_(t) > 0$ for $0 < t < t_r$. In the first case, when $S_{HR}(t_{s1}) = 0$;

$$S'_{HR}(ts_1) = (1 \quad) > 0 \quad (6 \; 1) \tag{}$$

which is a contradiction to the claim that $S'_{HR}(ts_1) < 0$, meaning S_{HR} remains positive. Following the same approach,

$$\begin{aligned} S'_{LR}(ts_2) &= \quad > 0 \\ E'(t_e) &= \quad {}_1(S_{HR} + S_{LR}) > 0 \\ I'(t_i) &= \quad E > 0 \\ R'(t_r) &= \quad I > 0 \end{aligned} \tag{2.4}$$

which shows that $S_{LR}; E; I$ and R all remain positive. Also from (2.2),

$$\frac{dN}{dt} = \qquad N$$

$$=) \quad \frac{dN}{dt} = \qquad I \quad N \qquad N$$

Since $N(t) \quad I(t)$; then

$$(\quad + \quad)N(t) \quad \frac{dN}{dt} \qquad N$$

which implies that is bounded and attracts all positive solution.

3. Stability Analysis

3.1. Introduction. In this section, the establishment of the basic reproduction number (R_0) is done. This R_0 is subsequently used to establish the local stability of the formulated model before showing that it is globally asymptotically stable. Setting the left hand side (LHS) of (2.2) to 0 gives the disease free equilibrium (DFE), which is as expressed in (3.1).

$$f S_{HR}; S_{LR}; E; I; R g = \frac{(1 \quad)}{\qquad}; \text{—}; 0; 0; 0 \tag{3.1}$$

3.2. Basic Reproduction Number (R_0). To establish the basic reproduction (R_0), the next generation matrix as discussed in [10, 11] and subsequently used in [12, 13] is used. Following the notations used in [10], then F and M matrices for the new infection terms and the remaining transition terms are respectively given by

$$F = \begin{matrix} (S_{HR} + S_{LR}) \\ 0 \end{matrix} \tag{3.2}$$

$$V = \begin{matrix} (\quad + \quad)E \\ (\quad + \quad + \quad)I \quad E \end{matrix} \tag{3.3}$$

(3.4) $$\Rightarrow \quad F = \begin{matrix} 0 & \frac{(S_{HR} + S_{LR})}{N} \\ 0 & 0 \end{matrix}$$

and

(3.5) $$\Rightarrow \quad V = \begin{matrix} (\quad + \quad) & 0 \\ & (\quad + \quad + \quad) \end{matrix} \quad :$$

Hence,

(3.6) $$FV^{0} = \frac{1}{(\quad + \quad)(\quad + \quad + \quad)} \begin{matrix} 0 & \frac{[\quad (1 \quad)+ \quad]}{} \\ 0 & 0 \end{matrix} + \begin{matrix} & 0 \\ (\quad + \quad + \quad) & \end{matrix}$$

(3.7) $$) \quad R_0 = \frac{[1 \quad + \quad]}{(\quad + \quad)(\quad + \quad + \quad)} :$$

The R_0 (equation (3.7)) as established above represents the number of secondary infection to be expected from a primarily recorded case.

3.3. Local Stability Analysis. By Theorem 2 of van den Driessche and Watmough (2002) [10], the following Lemma 3.1 is presented.

Lemma 3.1. The DFE of the model (3) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable otherwise.

Proof. In establishing the local stability of the model under consideration, the Jacobian matrix of (2.2) is used and the resulting expression is given as (3.8)

(3.8) $$\begin{matrix} 0 & & & & & & 1 \\ \B & \frac{I}{N} + & 0 & 0 & \frac{S_{HR}}{N} & 0 & \C \\ \B & 0 & \frac{I}{N} + & 0 & \frac{S_{LR}}{N} & 0 & \C \\ \B & \frac{I}{N} & \frac{I}{N} & (\quad + \quad) & \frac{S_{HR} + S_{LR}}{N} & 0 & \C \\ @ & 0 & 0 & & (\quad + \quad + \quad) & 0 & A \\ & 0 & 0 & 0 & & & \end{matrix} ;$$

evaluating (3.8) at the DFE (equation (3.1)), gives (3.9)

(3.9) $$\begin{matrix} 0 & & & & & 1 \\ \B & 0 & 0 & (1 \quad) & 0 & \C \\ \B & 0 & 0 & & 0 & \C \\ \B & 0 & 0 \quad (\quad + \quad) & [(1 \quad + \quad)] & 0 & \C \\ @ & 0 & 0 & (\quad + \quad + \quad) & 0 & A \\ & 0 & 0 \quad 0 & & & \end{matrix} :$$

The Eigen values of (3.9) are found to be

$$_1 = \ _2 = \ _3 =$$

and

(3.10) $$(\quad + \quad) \quad \frac{(\quad (1 \quad)+ \quad)}{(\quad + \quad + \quad)} \quad :$$

(3.11) $$\Rightarrow \quad ^2 + (\quad + \quad + 2 \quad + \quad) \ + (\quad + \quad)(\quad + \quad + \quad) \quad (1 \quad + \quad) = 0$$

(3.12) $$\Rightarrow \quad ^2 + (\quad + \quad + 2 \quad + \quad) \ + (\quad + \quad)(\quad + \quad + \quad)(1 \quad R_0) = 0$$

(3.13)
$$\Rightarrow \quad {}_{4;5} = \frac{(+ + 2 +) \quad p \overline{(+ + 2 +)^2 \quad 4(+)(+ +)(1 \quad R_0)}}{2}$$

(3.14)
$$\Rightarrow \quad {}_{4;5} < 0 \ () \quad R_0 < 1$$

Having all the Eigen values to be negative implies stability. Hence, stability of the DFE is being established. When $R_0 < 1$; the implication is that the epidemic cannot be contained.

3.4. Global Stability Analysis. In this section, the use of the global stability theorem discussed by [15] is employed. The e ciency of this method has been demonstrated in [16], [17] and [13] among other researches.
Considering the model as given in (2.2), let $X = (S_{HR}; S_{LR}; R)$ and $Z = (E; I)$ such that

(3.15)
$$\frac{dX}{dt} = F(X; 0) \quad \text{and} \quad \frac{dZ}{dt} = G(X; Z)$$

$F(X; 0)$ represents the right hand sides of $\frac{dS_{HR}}{dt}; \frac{dS_{LR}}{dt}$ and $\frac{dR}{dt}$, where $E = I = 0$; and also, $G(X; Z)$ is the right hand sides of both $\frac{dE}{dt}$ and $\frac{dI}{dt}$.
Hence, $\frac{dX}{dt} = F(X; 0)$

(3.16)
$$\Rightarrow \quad \begin{cases} \frac{dS_{HR}}{dt} = (1 \quad) \quad S_{HR} \\ \frac{dS_{LR}}{dt} = \quad S_{LR} \\ \frac{dR}{dt} = I \quad R \end{cases}$$

Let $X = (S_{HR}; S_{LR}; R) = f\frac{(1 \quad)}{}; —; 0g$ (DFE). Next is to show that X is globally stable.

Solving (3.16) gives

(3.17)
$$\begin{cases} S_{HR} = \quad {}_0^{R_t} e^{\ 1}(1 \quad)d \ {}_1 + S_{HR}(0) \ e^{\ t} \\ S_{LR} = \quad {}_0^{R_t} e^{\ 2} \quad d \ {}_2 + S_{LR}(0) \ e^{\ t} \\ R = \quad {}_0^{R_t} e^{\ 3} d \ {}_3 + R(0) \ e^{\ t} \end{cases}$$

Since $\frac{dS_{HR}}{dt} = (1 \quad) \quad S_{HR}$; then

$$\frac{dS_{HR}}{dt} + S_{HR} = (1 \quad)$$

$$\Rightarrow \quad e^t \ \frac{dS_{HR}(t)}{dt} + S_{HR}(t) = (1 \quad)e^t$$

$$\Rightarrow \quad \frac{d}{dt} \ e^t S_{HR}(t) = (1 \quad)e^t$$

$$\Rightarrow \quad e^t S_{HR}(t) = \int (1 \quad)e^t \, dt$$

$$e^t S_{HR}(t) = \frac{(1 \quad)}{} e^t + C$$

$$=) \quad S_{HR}(t) = \frac{(1 \quad)}{} + Ce^{\quad t}$$

at $t = 0$;

$$=) \quad S_{HR}(0) = \frac{(1 \quad)}{} + C$$

$$=) \quad S_{HR}(t) = \frac{(1 \quad)}{} + S_{HR}(0) \quad \frac{(1 \quad)}{} \quad e^{\quad t}$$

as $t \; ! \; 1$;

$$=) \quad \lim_{t!1} S_{HR}(t) = \frac{(1 \quad)}{}:$$

Similarly,

$$\lim_{t!1} S_{LR}(t) = \text{—} \quad \& \quad \lim_{t!1} R(t) = 0:$$

Next is to make sure that $G(X; Z)$ satisfy the following condition;
i. $G(X; 0) = 0$;
ii. $G(X; Z) = D_z(X \; ; 0)Z \quad \hat{G}(X; Z)$; $\hat{G} \quad 0$:
$(X \; ; 0) =) \quad (\frac{(1 \quad)}{}; \text{—}; 0; 0; 0) \quad \& \quad D_z G$
$(X \; ; 0) =) \quad (\frac{(1 \quad)}{}; \text{—}; 0; 0; 0)$ and $D_z G(X \; ; 0)$ represents the Jacobian of $G(X; Z)$ with respect to E and I, evaluated at $(X \; ; 0)$

$$(3.18) \qquad \begin{matrix} (\; + \;) \; \frac{}{N} (S_{HR} + S_{LR}) \\ (\; + \; + \;) \end{matrix} =) \qquad \begin{matrix} (\; + \;) \; \text{—} ((1 \quad) + \quad) \\ (\; + \; + \;) \end{matrix}$$

It is true in (3.18) that the non-diagonal elements are always positive because 1. This con rms the correctness of $D_z(X \; ; 0)Z$. Next is to show that $\hat{G} \quad 0$.

$$\hat{G}(X; Z) = \begin{matrix} \frac{I}{N} (S_{HR} + S_{LR}) \\ 0 \end{matrix} \qquad \begin{matrix} \frac{I}{N} (S_{HR} + S_{LR}) \\ 0 \end{matrix}$$

$$(3.19) \qquad I \quad \frac{S_{HR}}{N} \quad \frac{S_{HR}}{N} + \frac{S_{LR}}{N} \quad \frac{S_{LR}}{N}$$

$$(3.20) \qquad =) \quad I \quad \frac{S_{HR}}{N} \quad 1 \quad \frac{S_{HR} N}{S_{HR} N} + \frac{S_{LR}}{N} \quad 1 \quad \frac{S_{LR} N}{S \; LRN} \quad ;$$

where $S_{HR} = \frac{(1 \quad)}{}$; $S_{LR} = \text{—}$; and $N = \text{–}$:
Since $S_{HR} \quad S_{HR}$ and $S_{LR} \quad S_{LR}$; then $S_{HR} \quad N$ and $S_{LR} \quad N$:
Hence at equilibrium,

$$1 \quad \frac{S_{HR} N}{S_{HR} N} > 0 \quad \text{and} \quad 1 \quad \frac{S_{LR} N}{S_{LR} N} > 0:$$

Thus, $\hat{G}(X; Z) \quad 0$: Therefore, the DFE is globally asymptotically stable.

Discussion of Results. This section presents the numerical results gotten after simulating the model using the values de ned in Table 1. Figure 2 shows the dynamics of the infection for all the considered groups. With an initial population of $f S_{HR}; S_{LR}; E; I; R g = f 345; 234; 122; 10; 0g$, the S_{HR} population rose to over 120,000 (Figure 2a, $= 0:56$) within 7 years after which it started declining, while it goes as high as 400,000 within the same period in Figure 2b ($= 0:01$). Around the time of its decline, individuals who have contracted the infection but still are asymptomatic begin to rise [from 122 initial population to over 100,000 (Figure 2a) and to over 1,200,000 in Figure 2b]. This shows when the infection is not properly managed, it could turn to a major epidemic whose impact would be highly disastrous as there is no identi ed medication for its cure yet. Also, it implies that the impact of natural death rate is signi cant, the more people die, the less the number of persons to contract the infection.

S/No	Parameter	Meaning	Value	Source
1		Recruitment into the susceptible class	123456	Assumed
2		Infection rate	0:35 100	Assumed
3		Modi cation parameter for the low risk susceptible group	0:25	Assumed
4		Natural mortality rate	0:56	Assumed
5		MVD induced death rate	0:5	[14]
6		Progression to full blown infection rate	$\frac{1}{12}$	[5]
7		Recovery rate	0:2	[14]
8		Fraction of the low risk susceptible individuals	0:4	Assumed

Table 1. Table of values

The incubation period ( ) is the rate at which individuals who have contracted the infection but are asymptomatic progress to the infected compartment. Its e ect is measured using the 2 days minimum asymptomatic period as the initial value and 21 days maximum period as terminal value. Varying the parameter between these range on both the exposed (E) and infected (I) shows reasonable delay in the period of infection and eventual reduction in the number of exposed and as well infected persons (Figure 3). When the the exposed individual graduate to the infected compartment immediately (2 days), it shows the infected person has a compromised immune system and is prone to developing other health challenges, hence, the commensurate behaviour between the exposed and infected compartment with $= \frac{1}{2}$. However, when the manifestation of symptoms gets delayed till around 12 days, there is signi cant reduction in the number of fully symptomatic individuals as shown in Figure 3. The infection is almost annihilated when the delay in incubation period is 21 days (See Figure 3b). The same pattern is maintained in the recovery class (Figure 6a), as more persons get infected, more persons get recovered. When there are no people with infection, there shall be no recovery.

On the contrast, the modi cation parameter shall be e ective when it is small. Its impact is measured in Figure 4 and Figure 6b. If only 25% of the low risk susceptible individuals get exposed, it means consequential reduction in the number of infected and recovered as compared to 50% or 75%. Hence, it is expected as shown in those Figures that the rate at which low risk susceptible class (S_{LR}) contracts the MVD is lower than that of S_{HR}. As for the fraction of high and low risk susceptible individuals ( ), lesser value of gives more infection. This is reasonable as it implies lesser number of people is in the low risk susceptible class. When the value goes higher, it implies there are more

people in the S_{LR} compartment and consequential e ective reduction in the number of exposed and infected individuals.

Figures 2a: Population dynamics with $= 0:56$

Figures 2b: Population dynamics with $= 0:01$

Figures 2a and 2b are for dynamics of the Marburg virus disease.