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A Mathematical Model for the Control of Ebola Virus Disease with Vaccination Effect

Agbata Benedict Celestine[1,†,*], Yahaya Jibrin Danjuma[1,‡], Odeh Jane Ojowu[2,‖], Ekwoba Lucky[3,§], Emmanuel Olorunfemi Senewo[1,¶], Onoja Kingsley Anselem[4,✠], Agbebaku Dennis Ferdinand[5,*,II], Obeng-Denteh William[6,т]

[1]Department of Mathematics, Faculty of Science and Mathematics, Universitas Diponegoro, Semarang, 50275, Indonesia
[2]Department of Mathematics, Benue State University, Makurdi, Nigeria
[3]Department of Pure and Industrial Chemistry, Prince Abubakar Audu (Formerly, Kogi State) University, Anyigba, Nigeria
[4]Department of Pharmaceutical and Medicinal Chemistry, Nnamdi Azikiwe University, Awka, Nigeria
[5]Department of Mathematics, Faculty of Physical Sciences, University of Nigeria, Nsukka, Nigeria
[6]Department of Mathematics, College of Science, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana

[†]*agbatacelestine92@gmail.com,* [II]*dennis.agbebaku@unn.edu.ng,*

**Corresponding author*

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Abstract: Ebola Virus Disease (EVD) remains a major global health threat, marked by periodic outbreaks with severe mortality and socioeconomic consequences. In this study, a comprehensive mathematical model to analyze the transmission dynamics of EVD, explicitly incorporating key epidemiological factors such as vaccination, treatment efficacy, and human contact rates. The model stratifies the total human population into seven compartments: Susceptible (S), Vaccinated (V), Exposed (E), Infected (I), Hospitalized (H), Deceased (D), and Recovered (R) is formulated. Using the next-generation matrix method, the basic reproduction number (R_0) is derived to assess the potential for disease spread. Stability analysis demonstrates that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable otherwise. Numerical simulations and sensitivity analyses are conducted to explore the model's dynamics under various intervention scenarios. The findings highlight the crucial role of high vaccination coverage and effective treatment in significantly reducing EVD incidence and prevalence. Sensitivity analysis identifies the contact rate as a critical driver of transmission, indicating that minimizing contact with infectious individuals substantially lowers outbreak magnitude. Furthermore, the study determines threshold values for vaccination and treatment effectiveness that must be achieved to ensure outbreak containment. The model emphasizes the necessity of integrated control strategies that combine vaccination, timely treatment, and public health behaviour modifications. These results offer actionable insights for policymakers and health authorities aiming to design effective response plans. The study recommends prioritizing sustained vaccination campaigns, strengthening healthcare infrastructure, and implementing public awareness programs to enhance community compliance and preparedness against future EVD outbreaks.

Keywords: Ebola virus disease; Mathematical modeling; Vaccination; Treatment efficacy; Sensitivity analysis; Epidemic control; Public health interventions; Outbreak mitigation;
AMS Math Codes: xxxxx; xxxxx.

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1. Introduction

Ebola virus disease (EVD), also known as Ebola hemorrhagic fever, is a severe illness caused by the Ebola virus, a member of the Filoviridae family [1]. The disease was first discovered in 1976 during outbreaks in Sudan and the Democratic Republic of Congo (DRC). Since then, sporadic outbreaks have occurred primarily in Central and West Africa, with the largest outbreak recorded in West Africa between 2014 and 2016. EVD is characterized by a very high fatality rate, ranging from $25\,\%$ to $90\,\%$, depending on the outbreak and access to healthcare [2]. The Ebola virus can be is transmitted to humans through contact with infected animals, such as fruit bats, monkeys, or apes, found in the African rainforest. Once the virus enters the human population, it spreads through direct contact with bodily fluids (blood, feces, saliva, urine, sweat, vomit, breast milk) of infected individuals or contaminated surfaces and materials. Preventing Ebola transmission requires implementing strict infection control measures, including: Personal protective equipment (PPE) for healthcare workers, Safe burial practices to prevent exposure to infected corpses, Quarantine and isolation of infected individuals, Contact tracing and monitoring of individuals exposed to Ebola patients, Public health education campaigns to raise awareness about the disease and its prevention [1]. The signs and symptoms of Ebola virus disease typically appear 2 to 21 days after exposure and include: Fever, headache, Severe Weakness, Muscle pain, Fatigue. Diarrhea, Vomiting, Abdominal pain, Unexplained bleeding or bruising (hemorrhage). There is currently no specific antiviral treatment for Ebola virus disease [1, 2]. Supportive care, including hydration, pain management, and treatment of complications, such as secondary infections and organ failure, is crucial. Experimental therapeutics and vaccines are being developed and tested, but their efficacy and availability remain limited. The Ebola virus spreads through direct contact with bodily fluids of infected individuals, including blood, saliva, sweat, urine, feces, vomit, and breast milk. Transmission can also occur through contact with contaminated surfaces, materials, or medical equipment. Several experimental vaccines have been developed to prevent Ebola virus infection. The most widely used vaccine is the rVSV-ZEBOV vaccine, which has shown efficacy in clinical trials during the West African Ebola outbreak. The vaccine is based on a live, attenuated vesicular stomatitis virus (VSV) expressing the Ebola virus glycoprotein [2, 3]. It has been shown to be safe and effective in preventing Ebola virus infection and is being used in outbreak response and vaccination campaigns in affected regions. Ebola virus disease remains a significant public health concern in Africa, particularly in regions where outbreaks occur sporadically. Factors such as inadequate healthcare infrastructure, poor infection control practices, and limited access to resources exacerbate the impact of Ebola outbreaks in affected communities. Prompt detection, response, and vaccination efforts are essential for containing outbreaks and preventing further transmission [2]. Several authors have studied mathematical modelling of infectious diseases. [10] proposed a generalized epizootic model of Ebola virus disease (EVD) in bat population by considering the environment contamination. They also investigated the stability analysis. [29] presented a novel soliton-based SIR (Susceptible-Infectious-Recovered) model to investigate the spatiotemporal spread of infectious diseases in nomadic populations. The authors incorporated mobility patterns typical of nomadic groups into a reaction-diffusion framework, allowing for spatially dynamic interactions. The model effectively captured wave-like disease propagation influenced by periodic movement. Through numerical simulations, it was demonstrated that soliton solutions could represent stable traveling infection waves, offering new perspectives in understanding disease persistence and control in transient communities. This approach provided a promising
direction for epidemiological studies involving populations with high mobility and low access to healthcare infrastructure. [30] developed a deterministic mathematical model to assess the dynamics of alcohol addiction within a population, incorporating key compartments such as susceptibles, moderate users, addicted individuals, and those in treatment. They examined the model's equilibrium states and used the Routh-Hurwitz criteria to determine local stability. A threshold parameter, similar to the basic reproduction number in epidemiology, was derived to assess addiction persistence. Numerical simulations illustrated how treatment and awareness campaigns significantly reduced addiction prevalence. The model emphasized the importance of early intervention and effective rehabilitation programs in mitigating long-term societal impacts of alcohol abuse. [31] investigated the bifurcation structures of a discrete-time epidemic model that incorporated vaccination and demographic processes such as births and deaths. The study focused on the existence of co-dimension one (e.g., saddle-node and transcritical) and codimension two bifurcations, which signify qualitative shifts in the dynamics as parameters vary. Using center manifold theory and bifurcation analysis, the authors showed how varying vaccination rates and infection probabilities led to rich dynamical behavior, including

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periodic outbreaks and complex transitions. The findings underscored the importance of parameter sensitivity in public health planning and in predicting critical transitions in disease prevalence.

Other relevant studies include [4,5,9,11,13]. The primary aim of this study is to develop and analyze a mathematical model that captures the transmission dynamics of Ebola Virus Disease (EVD) in a human population, with the goal of identifying effective control strategies. The specific objectives are to incorporate key epidemiological factors such as vaccination, treatment, contact rate, hospitalization, and burial practices into the model; to derive and analyze the basic reproduction number (R0) to understand disease thresholds; to perform sensitivity analysis to identify the most influential parameters affecting disease spread; and to conduct numerical simulations to evaluate the effectiveness of various intervention strategies on disease reduction and eventual eradication. The novelty of this study lies in its comprehensive and realistic modeling approach, which extends beyond traditional compartmental models by including a deceased but unburied compartment a critical and often overlooked route of Ebola transmission. Additionally, the model integrates the interplay between vaccination efficacy, treatment effectiveness, and behavioral interventions, offering a more holistic view of epidemic control. By combining analytical derivations, sensitivity analysis, and numerical simulations, the study provides a robust framework for evaluating threshold-dependent outcomes and optimizing resource allocation in public health responses. This integrated approach contributes new insights into how multiple, simultaneous interventions can synergistically suppress and eliminate Ebola outbreaks.

1.1. Ebola Virus Disease (EVD) in Africa

Ebola virus disease (EVD), a severe and often fatal illness, has posed significant challenges to healthcare systems and communities across Africa [23]. Since its discovery in 1976, Ebola outbreaks have occurred sporadically in various African countries, with notable prevalence in regions of Central and West Africa [22]. The disease is characterized by high mortality rates, rapid transmission within communities and healthcare settings, and profound social and economic consequences for affected populations. This discussion will explore the impact of Ebola virus disease on Africa, including its transmission dynamics, effects on healthcare systems, social and economic ramifications, response strategies, and efforts to enhance resilience and preparedness.

Outbreaks: Ebola outbreaks have occurred sporadically in Africa since the virus was first identified in 1976 [22]. The largest outbreak in history began in West Africa in 2014, primarily affecting Guinea, Liberia, and Sierra Leone [24]. This outbreak highlighted the challenges in controlling the spread of the virus, including weak healthcare infrastructure, limited resources, and social factors such as distrust of authorities and traditional burial practices that facilitate transmission.

Transmission: Ebola is transmitted through direct contact with bodily fluids of infected individuals or contact with contaminated surfaces or materials [22]. The virus can spread rapidly within communities, healthcare settings, and during funeral rituals where there is close contact with infected individuals or their bodily fluids. The high case fatality rate of Ebola, which can be up to $90\,\%$, contributes to the severity of outbreaks [22].

Impact on Healthcare Systems: Ebola outbreaks place significant strain on healthcare systems in affected countries. Hospitals and clinics become overwhelmed with cases, leading to shortages of medical supplies, healthcare workers, and hospital beds [23]. The diversion of resources and personnel to manage Ebola can disrupt routine healthcare services, exacerbating other health problems in the community.

Social and Economic Impact: Ebola outbreaks have profound social and economic consequences. Fear and stigma surrounding the disease can lead to social ostracization of survivors and affected communities, hindering efforts to control the outbreak [22]. Economic activities may also be disrupted, particularly in heavily affected areas, due to travel restrictions, trade disruptions, and decreased productivity.

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Response and Prevention: International organizations, governments, and local communities collaborate to respond to Ebola outbreaks through various measures [22]. These include case identification and isolation, contact tracing, community engagement and education, safe burial practices, and vaccination campaigns. Additionally, research efforts focus on developing effective treatments and vaccines to combat the disease [22].

Resilience and Preparedness: Despite the challenges posed by Ebola outbreaks, African countries and the international community have made strides in building resilience and preparedness to respond to future outbreaks [24]. This includes strengthening healthcare systems, improving disease surveillance and laboratory capacity, training healthcare workers, and enhancing community awareness and engagement.

Prevalence of Ebola in Africa: Africa has experienced numerous Ebola outbreaks, predominantly in Central and West Africa, where the virus is endemic [22]. The Democratic Republic of the Congo (DRC) and neighboring countries in the Central African region have faced recurring outbreaks of Ebola, posing ongoing challenges to public health systems and regional stability [25].

Mortality Rate in Africa: The mortality rate of Ebola in Africa has varied across outbreaks but can be as high as $90\,\%$ in some cases [22]. Mortality rates are influenced by factors such as access to healthcare, the timing of treatment initiation, the virulence of the virus strain, and the effectiveness of public health interventions.

2. Model Formulation

The total human population is stratified into seven epidemiological compartments to reflect the various stages of Ebola Virus Disease (EVD) progression and control measures. These compartments include: Susceptible individuals (S), Vaccinated individuals (V), Exposed individuals (E),. Infected individuals (I),. Hospitalized individuals (H). Deceased but unburied individuals (D), . Recovered individuals (R). The model assumes a constant recruitment rate of susceptible individuals at a rate denoted by Λ Susceptible individuals are vaccinated at a rate α_1, moving them into the vaccinated compartment. Due to the possibility of vaccine failure, vaccinated individuals may still become exposed to the virus at a rate α_2 after contact with infectious persons. Additionally, unvaccinated susceptible individuals become exposed through effective contact with infected individuals at a rate λ. Once exposed, individuals progress to the infectious stage at a rate ω_1, ω_2, representing the average incubation period. Infected individuals are hospitalized at a rate ψ_1, depending on healthcare accessibility. Infected individuals may die due to the disease at a rate ψ_2, while hospitalized individuals may also die due to disease complications at a rate ψ_2,. These deceased individuals accumulate in the unburied deceased compartment, from which they are buried at a rate ψ_3, an important public health intervention given the infectious nature of EVD corpses. Hospitalized individuals recover and move to the recovered class at a rate μ. A natural (non-disease-related) death rate ψ_4 is assumed to act uniformly across all compartments.

Susceptible (S): This compartment represents individuals who are susceptible to contracting Ebola virus disease. Susceptible individuals have not been infected with the virus and can become infected upon exposure to infected individuals or contaminated materials.

Vaccinated (V): The vaccinated compartment consists of individuals who have received a vaccine against Ebola virus disease. Vaccination reduces the susceptibility of individuals to infection and can contribute to herd immunity, thereby helping to control the spread of the virus within the population.

Exposed (E): Individuals in the exposed compartment have been infected with the Ebola virus but have not yet developed symptoms. During the incubation period, these individuals are not infectious but can later transition to the infected compartment.

Infected (I): Infected individuals are those who have developed symptoms of Ebola virus disease and are capable of transmitting the virus to others. This compartment represents individuals who are actively contributing to the spread of the disease within the population.

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Variables	Interpretation
$\mathrm{N(t)}$	Total human population
S	Susceptible population
V	Vaccinated population
E	Exposed individuals
I	Infected individuals
H	Hospitalized Individuals
D	Dead and unburied population
R	Recovered individuals
Parameters	Descriptions
Λ	Recruitment rate
α_1	Vaccination rate
λ	Force of infection
α_2	Rate of exposure due to vaccine failure
α_3	Rate at of infection due to vaccine failure
μ	Natural death rate
ω_1	Rate of infection of exposed humans
ε_1	Disease induced death rate associated with I compartment
ε_2	Disease induced death rate associated with H compartment
ω_2	Hospitalized rate of infected individuals
ψ_1	Death rate of infected individuals

Cuadro 1: Variables and Parameters Used.

Hospitalized (H):The hospitalized compartment includes individuals who have developed severe symptoms of Ebola virus disease and require medical care. Hospitalization is necessary for managing complications and providing supportive treatment to improve patient outcomes.

Deceased (D): Individuals in the deceased compartment have succumbed to Ebola virus disease. This compartment represents the unfortunate outcome of severe cases of the disease and underscores the importance of timely medical intervention and public health measures to prevent fatalities.

Recovered (R): Recovered individuals have successfully cleared the Ebola virus from their system and have developed immunity to subsequent infections. This compartment reflects the resilience of the human immune system and the potential for individuals to overcome the disease with proper medical care and support. Each compartment interacts dynamically within the model, with individuals transitioning between compartments based on specific rates of infection, recovery, hospitalization, and mortality [32]. By simulating these interactions, mathematical models can provide insights into the spread and impact of Ebola virus disease within a population and inform public health strategies for disease control and prevention.

2.1. Variables and Parameters Interpretation

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ψ_2 Death rate of hospitalized individuals
ψ_3 Recovery rate of hospitalized individuals
ψ_4 Rate of burial for the dead and unburied population

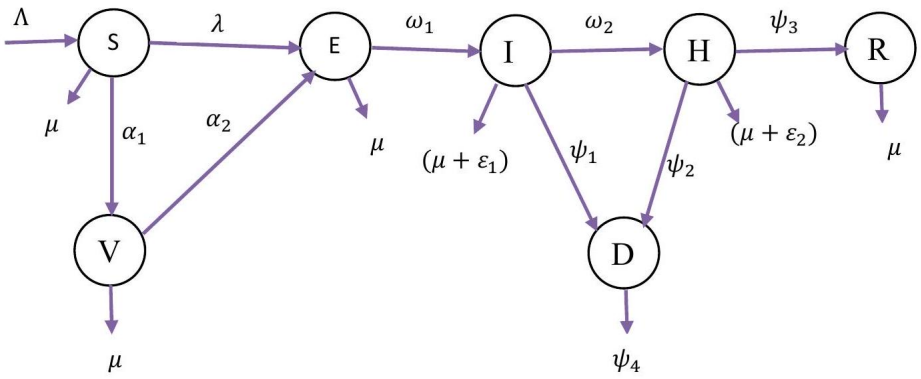

Figura 1: Schematic Diagram for the model

2.2. Model Equations

$$\frac{dS}{dt} = \Lambda - (\lambda + \alpha_1 + \mu)\, S \tag{2.1}$$
$$\frac{dV}{dt} = \alpha_1 S - (\alpha_2 + \mu)\, \mathrm{V} \tag{2.2}$$
$$\frac{dE}{dt} = \lambda S + \alpha_2 V - (\omega_1 + \mu)\, E \tag{2.3}$$
$$\frac{dI}{dt} = \omega_1 E - (\varepsilon_1 + \psi_1 + \omega_2 + \mu)\, I \tag{2.4}$$
$$\frac{dH}{dt} = \omega_2 I - (\psi_2 + \psi_3 + \varepsilon_2 + \mu)\, \mathrm{H} \tag{2.5}$$
$$\frac{dD}{dt} = \psi_1 I + \psi_2 H - \psi_4 D \tag{2.6}$$
$$\frac{dR}{dt} = \psi_3 H - \mu R \tag{2.7}$$

where $\lambda = \frac{\beta(\mathrm{I}+\mathrm{H}+\mathrm{D})}{N}$.

3. Invariant Region of the Model

In mathematical modeling, the invariant region refers to a set of conditions or states within the model that remain constant over time, regardless of initial conditions or parameter variations [21]. In epidemiology, this concept transla-

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tes to a stable set of conditions that characterize the behavior of a disease within a population. Identifying the invariant region is crucial as it provides insights into the long-term behavior of the disease and helps in understanding the underlying mechanisms governing its spread. By delineating the boundaries of this region, researchers can better predict the dynamics of the disease, assess the effectiveness of control measures, and devise strategies for disease management and prevention [20, 21]. Ultimately, the invariant region serves as a cornerstone for informing public health policies and interventions aimed at mitigating the impact of infectious diseases like Ebola.

Theorem 3.1. *The solutions set of the proposed model are feasible whenever $t > 0$, if they enter the invariant region D, which is given by:*

$$D = \left\{ (S, V, E, I, H, D, R) : S > 0, V > 0, E > 0, I > 0, H > 0, D > 0, R > 0, N < \frac{\Lambda}{\mu} \right\}$$

Demostración. The total population of the model is given as

$$N(t) = S + V + E + I + H + D + R$$

The sum of the differential equations is

$$N'(t) = S' + V' + E' + I' + H' + D' + R'$$

On evaluating the algebraic terms, we obtain

$$N'(t) = \Lambda - (S + V + E + I + H + D + R)\mu - (\omega_2 I + \psi_2 H + \psi_4 D)$$

$$N'(t) = \Lambda - \mu N - (\omega_2 I + \psi_2 H + \psi_4 D)$$

$$\frac{dN}{dt} \leq \Lambda - \mu N$$

Solving the differential equation using the integrating factor method, we obtained

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}$$

Applying Birkhoff and Rota's theorem on the inequality, we obtain

$$0 \leq N \leq \frac{\Lambda}{\mu} \text{ as } t \to \infty$$

Thus, D is a positively invariant set with respect to the schematic described by the model so that no solution path leaves through the boundary of region D. Thus, in this region, the model is said to be epidemiologically and mathematically well posed [6, 7]. □

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4. Positivity of Solutions of the Model

In mathematical modeling, ensuring the positivity of solutions is vital for obtaining physically meaningful interpretations, particularly in systems where negative values are not feasible or violate constraints [7]. The positivity of solution is fundamental for ensuring that model predictions accurately reflect the dynamics of infectious diseases within populations. Epidemiological models, such as compartmental models like the SIR (Susceptible-Infectious-Recovered) model, describe how the numbers of susceptible, infectious, and recovered individuals change over time. Positivity constraints ensure that these quantities remain non-negative, as negative values would not make sense in the context of disease spread [6, 26]. Additionally, in models with parameters representing transmission rates, recovery rates, or population sizes, enforcing positivity ensures that these parameters have realistic interpretations and prevent unphysical scenarios, such as negative transmission rates or populations. Techniques such as adding constraints to parameter estimates or using numerical methods that preserve positivity, like non-negative matrix factorization, are employed to maintain the positivity of solutions [26, 27]. By enforcing positivity, mathematical epidemiology models provide valuable insights into disease dynamics, aiding in the development and evaluation of public health interventions.

It is necessary to show that all state variable of the model in are non-negative for all time (t), for the model to be epidemiologically and mathematically feasible in the region D given by [7, 8]:

$$D = \left\{(S, V, E, I, H, D, R) \in R_+^7 : (S + V + E + I + H + D + R) \leq N\right\}$$

This can be done by considering,

$$\left\{(S, V, E, I, H, D, R) \geq 0 \in R_+^7\right\}$$

Lemma 4.1. *Supposed the initial data for the given model (1) be (S, V, E, I, H, D, R) ¿0. Then the solutions (S, V, E, I, H, D, R) of the model (1) are positive for all time $t > 0$*

Demostración. Let $t_1 = \sup\{t > 0 : S > 0, V > 0, E > 0, I > 0, H > 0, D > 0, R > 0 \in [0, t]\}$. Thus $t > 0$.

We have from the first equation that

$$\frac{dS}{dt} = \Lambda - (\lambda + \alpha_1 + \mu) S$$
$$\frac{dS}{dt} \geq -(\lambda + \alpha_1 + \mu) S$$

This can also be written as

$$\int \frac{dS}{S} \geq -\int (\lambda + \alpha_1 + \mu)\, dt$$

We obtained:

$$\ln S \geq -(\lambda + \alpha_1 + \mu) t + C$$
$$S(t) \geq Ce^{-(\lambda+\alpha_1+\mu)t}$$

Applying the initial condition; when $t = 0, S(0) = C$

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Hence, $S(t) \geq S(0)e^{-(\lambda+\alpha_1+\mu)t} \geq 0$

In the same way, it can be shown that $V, E, I, H, D, R > 0$ □

5. The Disease Free Equilibrium of the Model

The steady state where infection does not exist (or absence of the disease), a point where $E = I = H = D = R = 0$ is called the disease-free equilibrium point (DFE) which is given

$$\eta_0 = \{S^*, V^*, E^*, I^*, H^*, D^*, R^* > 0\} = \left\{ \frac{\Lambda}{(\alpha_1+\mu)}, \frac{\alpha_1\Lambda}{\mu\,(\alpha_1+\mu)} 0, 0, 0, 0, 0 \right\}$$

5.1. The Basic Reproduction Number of the Model

The basic reproduction number also called the fundamental reproductive rate of infected persons, R_0, refers to the average number of new infections caused by one Ebola-infected individual in a completely susceptible population throughout their infectious period. This value is determined by employing the next generation operator method on the dynamics system [8]. We calculate the basic reproduction number by using the next generation operator method on the dynamical system

Hence, it follows that

$R_0 = \rho\left(FV^{-1}\right)$ where ρ is the dominant eigenvalue of FV^{-1}

$$F = \begin{bmatrix} 0 & \beta & \beta & \beta \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} P_2 & 0 & 0 & 0 \\ -\omega_1 & q_3 & 0 & 0 \\ 0 & -\omega_2 & P_4 & 0 \\ 0 & -\psi_1 & -\psi_2 & \psi_4 \end{bmatrix},$$

where $\mathrm{P}_1 = \alpha_2 + \mu, P_2 = \omega_1 + \mu, p_3 = \varepsilon_1 + \psi_1 + \omega_2 + \mu, P_4 = \psi_2 + \psi_3 + \varepsilon_2 + \mu$

$$FV^{-1} = \begin{bmatrix} \frac{\omega_1\beta((P_4+\omega_2)\psi_4+\psi_1P_4+\psi_2\omega_2)}{P_2q_3P_4\psi_4} & \frac{((P_4+\omega_2)\psi_4+\psi_1P_4+\psi_2\omega_2)\beta}{P_4q_3\psi_4} & \frac{\beta(\psi_2+\psi_4)}{P_4\psi_4} & \frac{\beta}{\psi_4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \end{bmatrix}$$

$$R_0 = \frac{\beta\omega_1\left(\psi_1P_4 + P_4\psi_4 + \psi_2\omega_2 + \omega_2\psi_4\right)}{P_2q_3P_4\psi_4}$$

5.2. Effects of Reproduction Number of Ebola disease on Public Health Measures

The Basic Reproduction Number (R_0) of Ebola, which represents the average number of secondary infections caused by one infected individual in a susceptible population, greatly influences public health measures.

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1. Higher R_0 : If the R_0 of Ebola is high, it indicates that the virus is highly transmissible. In such cases, more aggressive measures are required to contain the spread. These measures may include strict quarantine, contact tracing, isolation of cases, and community education to promote hygiene and safe burial practices. The goal is to reduce the number of secondary infections and prevent widespread outbreaks [16, 17].
2. Lower R_0 : A lower R_0 suggests that the virus is less transmissible. In this scenario, public health measures may still be necessary but may not need to be as stringent. However, it is crucial to remain vigilant and maintain surveillance to detect and respond to any potential outbreaks promptly [17].
3. Vaccine Development: The R_0 of Ebola also influences vaccine development efforts. A higher R_0 may prompt more urgent research and development of vaccines to prevent transmission. Conversely, a lower R_0 may reduce the urgency but not eliminate the need for vaccines, especially in endemic regions [18].
4. Healthcare Infrastructure: Public health measures must be tailored to the healthcare infrastructure of affected regions. Higher R_0 values may overwhelm healthcare systems, necessitating additional resources such as medical personnel, treatment facilities, and supplies. Lower R_0 values may still strain healthcare systems but to a lesser extent [17, 18].

5.3. Local Asymptotic Stability of the DFE of the Model

Local asymptotic stability of the disease-free equilibrium (DFE) of a model means that if the number of infected individuals is initially small, the system will return to the DFE over time, and the disease will gradually die out. This type of stability is determined by analyzing the system's behavior near the DFE, often using linearization techniques such as the Jacobian matrix and evaluating its eigenvalues [6]. If all eigenvalues have negative real parts, or if conditions like the Routh-Hurwitz criteria are satisfied, the DFE is considered locally asymptotically stable. Typically, this occurs when the basic reproduction number $R_0 < 1$, indicating that the infection cannot invade the population and will not persist.

Theorem 5.1. *The disease-free equilibrium (DFE) point of the model (1) is locally asymptotically stable (LAS) if $R_0 < 1$, and unstable if $R_0 > 1$.*

Demostración. Applying Jacobian matrix to show the local stability of the disease free equilibrium point

$$J_1 = \begin{bmatrix} -(\alpha_1+\mu) & 0 & 0 & \frac{-\beta S}{N} & \frac{-\beta S}{N} & \frac{-\beta S}{N} & 0 \\ \alpha_1 & -P_1 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta(I+H+D)}{N} & \alpha_2 & -P_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \omega_1 & -q_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & \omega_2 & -P_4 & 0 & 0 \\ 0 & 0 & 0 & \psi_1 & \psi_2 & -\psi_4 & 0 \\ 0 & 0 & 0 & 0 & \psi_3 & 0 & -\mu \end{bmatrix}$$

Therefore the characteristic equation corresponding to $J_1(\eta_0)$ is evaluated as $|J - \lambda I|$ I.e

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$$J_1(\eta_0) = \begin{array}{|c|c|c|c|c|c|c|} \hline & -(\alpha_1+\mu) & 0 & 0 & -\frac{\beta(\alpha_2+\mu)}{(\alpha_1+1)\mu+{\alpha_1}^2+\alpha_2} & -\frac{\beta(\alpha_2+\mu)}{(\alpha_1+1)\mu+{\alpha_1}^2+\alpha_2} & -\frac{\beta(\alpha_2+\mu)}{(\alpha_1+1)\mu+\alpha_1^2+\alpha_2} & 0 \\ \hline & \alpha_1 & -P_1 & 0 & 0 & 0 & 0 & 0 \\ \hline & 0 & \alpha_2 & -P_2 & 0 & 0 & 0 & 0 \\ \hline & 0 & 0 & \omega_1 & -q_3 & 0 & 0 & 0 \\ \hline & 0 & 0 & 0 & \omega_2 & -P_4 & 0 & 0 \\ \hline & 0 & 0 & 0 & \psi_1 & \psi_2 & -\psi_4 & 0 \\ \hline & 0 & 0 & 0 & 0 & \psi_3 & 0 & -\mu \\ \hline \end{array}$$

The characteristics polynomial of $J_1(\eta_0)$ is

$$\lambda^7 + a_1\lambda^6 + a_2\lambda^5 + a_3\lambda^4 + a_4\lambda^3 + a_5\lambda^2 + a_6\lambda + a_7$$

where λ represents eigen value and

$$a_1 = (2\mu + \psi_4 + P_4 + q_3 + P_2 + P_1 + \alpha)$$

$$a_2 = \mu^2 + (2P_1 + 2P_2 + 2q_3 + 2P_4 + 2\psi_4 + \alpha_1)\mu + (P_1 + P_2 + q_3 + P_4 + \psi_4)\alpha_1 + (P_1 + P_2 + q_3 + P_4)\psi_4 + (P_2 + q_3 + P_4)P_1 + (q_3 + P_4)P_2 + q_3P_4$$

$$a_3 = (P_1 + P_2 + q_3 + P_4 + \psi_4)\mu^2 + \begin{pmatrix} (2P_1 + 2P_2 + 2q_3 + 2P_4 + \alpha_1)\psi_4 \\ +(2P_2 + 2q_3 + 2P_4 + \alpha_1)P_1 \\ +(2q_3 + 2P_4 + \alpha_1)P_2 + (2P_4 + \alpha_1)q_3 + \alpha_1P_4 \end{pmatrix}\mu + \begin{pmatrix} (P_2+q_3+P_4+\alpha_1)P_1+(q_3+P_4+\alpha_1)P_2 \\ +(P_4+\alpha_1)q_3+\alpha_1P_4 \end{pmatrix}\psi_4 + ((q_3 + P_4 + \alpha_1)P_2 + (P_4 + \alpha_1)q_3 + \alpha_1P_4)P_1 + ((P_4 + \alpha_1)q_3 + \alpha_1P_4)P_2 + \alpha_1q_3P_4$$

$$a_4 = \frac{\begin{pmatrix} ((2P_2+2q_3+2P_4+1)P_1+(2q_3+2P_4+1)P_2+(2P_4+1)q_3+P_4)\psi_4 \\ +\begin{pmatrix}(2q_3+2P_4+1)P_2\\+(2P_4+1)q_3+P_4\end{pmatrix}P_1+((2P_4+1)q_3+P_4)P_2+q_3P_4 \end{pmatrix}\alpha_1}{(\alpha_1+1)\mu+\alpha_1^2+\alpha_2} + \begin{pmatrix} ((P_1 + P_2 + q_3)P_4 + (P_2 + q_3)P_1 + P_2q_3)\psi_4 \\ +((P_2 + q_3)P_1 + P_2q_3)P_4 + P_1P_2q_3 \end{pmatrix}(\alpha_1 + 1)\mu^3 + \begin{pmatrix} \begin{pmatrix}(2P_2+2q_3+1)P_1+(2q_3+1)P_2+q_3)P_4\\+((2q_3+1)P_2+q_3)P_1+P_2q_3\end{pmatrix}\psi_4 \\ +(((2q_3 + 1)P_2 + q_3)P_1 + P_2q_3)P_4 - \omega_1\beta\alpha_2 + P_1P_2q_3 \end{pmatrix}\alpha_1$$

$$a_5 = \frac{+\begin{pmatrix}(\alpha_2+2P_2+2q_3)P_1+(\alpha_2+2q_3)P_2+\alpha_2q_3)P_4\\+((\alpha_2+2q_3)P_2+\alpha_2q_3)P_1+\alpha_2P_2q_3\end{pmatrix}\psi_4}{(\alpha_1+1)\mu+\alpha_1^2+\alpha_2} + ((((P_2 + q_3)P_1 + P_2q_3)P_4 + P_1P_2q_3)\psi_4 + P_1P_2q_3P_4)(\alpha_1 + 1)\mu^3 + \begin{pmatrix} ((((2P_2 + 2q_3)P_1 + 2P_2q_3)P_4 + 2P_1P_2q_3)\psi_4 + 2P_1P_2q_3P_4)\alpha_1^2 \\ +\begin{pmatrix}(((2q_3+1)P_2+q_3)P_1+P_2q_3)P_4-\omega_1\beta\alpha_2+P_1P_2q_3)\psi_4\\+(-\omega_1\beta\alpha_2+P_1P_2q_3)P_4-\alpha_2\omega_1\beta(\omega_2+\psi_1)\end{pmatrix}\alpha_1 \\ +((((\alpha_2 + 2q_3)P_2 + \alpha_2q_3)P_1 + \alpha_2P_2q_3)P_4 + \alpha_2P_1P_2q_3)\psi_4 + \alpha_2P_1P_2q_3P_4 \end{pmatrix}\mu^2}{(\alpha_1+1)\mu+\alpha_1^2+\alpha_2}$$

$$a_7 = \frac{\begin{pmatrix} +\alpha_1^3P_1P_2q_3P_4\psi_4 + 2\mu\alpha_1^2P_1P_2q_3P_4\psi_4 \\ +\begin{pmatrix}(\mu^2P_1P_2q_3 - \beta\alpha_2^2\omega_1 + \mu P_1P_2q_3)P_4\\-\alpha_2^2\omega_1\omega_2\beta\\-\alpha_2^2\omega_1\omega_2\beta\psi_2\end{pmatrix}\psi_4 \\ +\mu P_1P_2q_3P_4\psi_4(\mu+\alpha_2) + \alpha_1\alpha_2\mu P_1P_2q_3P_4\psi_4(1-R_0) \end{pmatrix}\alpha_1}{\mu\alpha_1+\alpha_1^2+\mu+\alpha_2}$$

Applying the Routh-Hurwitz criterion to the characteristic polynomial as shown in [6], we determine the conditions

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under which the equilibrium is locally asymptotically stable.

$$(1 - R_0) > 0$$
$$\Rightarrow R_0 < 1$$

To assess the local stability of the disease-free equilibrium, we apply the Routh-Hurwitz criterion to the seventh-degree characteristic polynomial derived from the linearization of the model. For a seventh-order polynomial, the Routh-Hurwitz criterion requires that all the first elements of the Routh array be positive. If this condition is met, all the roots of the characteristic equation have negative real parts, ensuring local asymptotic stability. In this study, the Routh-Hurwitz conditions are satisfied when the basic reproduction number $\mathrm{R}0 < 1$, confirming that the diseasefree equilibrium is locally asymptotically stable under this threshold. □

5.4. Global Asymptotic Stability of the Disease Free Equilibrium Point of the Model.

To investigate the global stability of the disease free equilibrium, we use the technique implemented by Castillo-Chavez and song [15].

To do this, we write the equation in the uninfected class as

$$\frac{dX}{dt} = F(X, Z)$$

And we re-write the equation in the infected class as

$$\frac{dz}{dt} = G(X, Z)$$

Where $X = S \in R_+^1$ denotes the uninfected population and

$Z = (E, I, H, D) \in R_+^4$ denotes the infected population

$\varepsilon_0 = (X^*, 0)$ represents the disease free equilibrium of the system, and it globally asymptotically stable if it satisfies the following conditions:

$$H_1 : \frac{dX}{dt} = F\left(X^*, 0\right), X^* \text{ is globally asymptotically stable}$$
$$H_2 : \frac{dZ}{dt} = D_Z G\left(X^*, 0\right) Z - \hat{G}(X, Z)$$

$\hat{G}(X, Z) \geq 0$ for all $(X, Z) \in D$ and where $D_Z G\left(X^*, 0\right)$ is an M- matrix (i.e the diagonal elements are no-negative and it is also the Jacobian of $\hat{G}(X, Z) \geq 0$ evaluated at ($X^*, 0$).

If the system satisfies the above condition, then the theorem below holds.

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Theorem 5.2. *The equilibrium point $\varepsilon_0 = (X^*, 0)$. is globally asymptotically stable if $R_0 \leq 1$*

Demostración.

$$F(X,Z) = [\Lambda - (\lambda + \alpha_1 + \mu)\, S]\,, G(X,Z) = \begin{bmatrix} \lambda S + \alpha_2 V - (\omega_1 + \mu)\, E \\ \omega_1 E - (\varepsilon_1 + \psi_1 + \omega_2 + \mu)\, I \\ \omega_2 I - (\psi_2 + \psi_3 + \varepsilon_2 + \mu)\, \mathrm{H} \\ \psi_1 I + \psi_2 H - \psi_4 D \end{bmatrix}$$

At disease free equilibrium,

$$H_1 : \frac{dS}{dt} = [\Lambda - (\lambda + \alpha_1 + \mu)\, S]$$

$$H_2 : D_Z G\,(X^*, 0)\, Z = \begin{bmatrix} \lambda S + \alpha_2 V - (\omega_1 + \mu)\, E \\ \omega_1 E - (\varepsilon_1 + \psi_1 + \omega_2 + \mu)\, I \\ \omega_2 I - (\psi_2 + \psi_3 + \varepsilon_2 + \mu)\, \mathrm{H} \\ \psi_1 I + \psi_2 H - \psi_4 D \end{bmatrix}$$

$$\hat{G}(X,Z) = D_Z G\,(X^*, 0)\, Z - G(X,Z)$$

$$\hat{G}(X,Z) = \begin{bmatrix} \beta(I + H + D)\left(1 - \frac{S}{N}\right) \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

Clearly, $1 \geq \frac{S}{N}$ this implies that $\hat{G}(X,Z) \geq 0$.

Hence, It implies that, for the Ebola disease model, the infection will eventually be eradicated from the population over time, regardless of the initial number of infected individuals. This means the system will naturally return to a disease-free state, provided that the basic reproduction number $\mathrm{R}0 < 1$. In this case, the control measures captured in the model such as isolation, treatment, and vaccination are sufficient to prevent the spread of Ebola and ensure that future outbreaks cannot sustain themselves within the population. □

6. Endemic Equilibrium Point of the Model

The endemic equilibrium point is the steady state where there is persistence or prevalence of a disease in the population. To obtain the endemic equilibrium we set the RHS of the differential equations in to zero and solve for the state variables.

Thus, at the endemic equilibrium point,

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dH}{dt} = \frac{dD}{dt} = \frac{dR}{dt} = 0.$$

Let $\varepsilon^{**} = (S^{**}, V^{**}, E^{**}, I^{**}, H^{**}, D^{**}, R^{**})$ be the endemic equilibrium point.

We have that,

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$$S^{**} = \frac{\Lambda}{(\lambda^{**} + \alpha_1 + \mu)}$$
$$V^{**} = \frac{\alpha_1 \Lambda}{(\lambda^{**} + \alpha_1 + \mu)(\alpha_2 + \mu)}$$

$$E^{**} = \frac{\lambda\Lambda(\alpha_2+\mu) + \alpha_1\alpha_2(\omega_1+\mu)}{(\lambda^{**}+\alpha_1+\mu)(\omega_1+\mu)(\alpha_2+\mu)}$$
$$I^{**} = \frac{\omega_1}{(\varepsilon_1+\psi_1+\omega_1+\mu)}\left(\frac{\lambda\Lambda(\alpha_2+\mu) + \alpha_1\alpha_2(\omega_1+\mu)}{(\lambda^{**}+\alpha_1+\mu)(\omega_1+\mu)(\alpha_2+\mu)}\right)$$
$$H^{**} = \frac{\omega_1\omega_2}{(\varepsilon_1+\psi_1+\omega_1+\mu)(\varepsilon_2+\psi_2+\psi_3+\mu)}\left(\frac{\lambda\Lambda(\alpha_2+\mu) + \alpha_1\alpha_2(\omega_1+\mu)}{(\lambda^{**}+\alpha_1+\mu)(\omega_1+\mu)(\alpha_2+\mu)}\right)$$
$$D^{**} = \left(\frac{\omega_1(\varepsilon_2+\psi_2+\psi_3+\mu) + \omega_1\omega_2\psi_2}{\psi_4(\varepsilon_1+\psi_1+\omega_1+\mu)(\varepsilon_2+\psi_2+\psi_3+\mu)}\right)\left(\frac{\lambda\Lambda(\alpha_2+\mu) + \alpha_1\alpha_2(\omega_1+\mu)}{(\lambda^{**}+\alpha_1+\mu)(\omega_1+\mu)(\alpha_2+\mu)}\right)$$
$$R^{**} = \frac{\omega_1\omega_2\psi_3}{\mu(\varepsilon_1+\psi_1+\omega_1+\mu)(\varepsilon_2+\psi_2+\psi_3+\mu)}\left(\frac{\lambda\Lambda(\alpha_2+\mu) + \alpha_1\alpha_2(\omega_1+\mu)}{(\lambda^{**}+\alpha_1+\mu)(\omega_1+\mu)(\alpha_2+\mu)}\right)$$

7. Sensitivity Analysis of the Model

Sensitivity analysis is carried out to determine the parameters that enhance the spread as well as control of an infection in a population. The sensitivity index of the reproduction number of the model with respect to any parameter say x is given by:
$\mathfrak{J}_x^{R_0} = \frac{\partial R_0}{\partial x} \times \frac{x}{R_0}$

Given that

$$R_0 = \frac{\beta\omega_1\left((\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_1 + (\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4 + \omega_2\psi_2 + \omega_2\psi_4\right)}{(\omega_1+\mu)(\varepsilon_1+\psi_1+\omega_2+\mu)(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4}$$
$$\Im_\beta^{R_0^h} = 1{,}0000$$
$$\Im_{\omega_1}^{R_0} = \frac{\mu}{\omega_1+\mu} = 0{,}2308$$
$$\Im_0 = \begin{pmatrix} \frac{\beta\omega_1(2\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu+\psi_4)}{(\omega_1+\mu)(\varepsilon_1+\psi_1+\omega_2+\mu)(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4} - \frac{\beta\omega_1((\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_1+(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4+\omega_2\psi_2+\omega_2\psi_4)}{(\omega_1+\mu)(\varepsilon_1+\psi_1+\omega_2+\mu)^2(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4} \\ -\frac{\beta\omega_1((\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_1+(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4+\omega_2\psi_2+\omega_2\psi_4)}{(\omega_1+\mu)(\varepsilon_1+\psi_1+\omega_2+\mu)(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)^2\psi_4} \\ \Im_{\psi_1} \end{pmatrix} A$$
$$= 0{,}3502$$

where $A = \psi_1(\omega_1+\mu)(\varepsilon_1+\psi_1+\omega_2+\mu)(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4$

$$\mathfrak{J}_{\psi_2}^{R_0} = \frac{\omega_2(\mu+\psi_1+\psi_3-\psi_4+\varepsilon_2)\psi_2}{(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)(\psi_1^2+(\mu+\varepsilon_2+\psi_2+\psi_3+\psi_4)\psi_1+(\mu+\varepsilon_2+\psi_2+\psi_3+\omega_2)\psi_4+\omega_2\psi_2)}$$
$$= 0{,}4771$$
$$\mathfrak{J}_{\psi_3}^{R_0} = -\frac{\omega_2(\psi_2+\psi_4)\psi_3}{(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)(\psi_1^2+(\mu+\varepsilon_2+\psi_2+\psi_3+\psi_4)\psi_1+(\mu+\varepsilon_2+\psi_2+\psi_3+\omega_2)\psi_4+\omega_2\psi_2)}$$
$$= 0{,}4507$$
$$\mathfrak{J}_{\psi_4}^{R_0} = \frac{-\psi_1^2+(-\mu-\varepsilon_2-\psi_2-\psi_3)\psi_1-\omega_2\psi_2}{\psi_1^2+(\mu+\varepsilon_2+\psi_2+\psi_3+\psi_4)\psi_1+(\mu+\varepsilon_2+\psi_2+\psi_3+\omega_2)\psi_4+\omega_2\psi_2}$$
$$= -0{,}8486$$

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Parameter	Sensitivity Index	Sensitivity Sign
β	1.0000	+ve
ω_1	0.2308	+ve
ψ_1	0.3502	-ve
ψ_2	0.4771	+ve
ψ_4	0.8486	-ve
ε_1	0.0688	-ve
μ	0.2669	-ve

Cuadro 2: Sensitivity Indies for parameters in R_0

$$\Im_{\varepsilon_1}^{R_0} = -\frac{\varepsilon_1}{\varepsilon_1+\psi_1+\omega_2+\mu} = -0{,}0688$$

$$\Im_{\varepsilon_2}^{R_0} = -\frac{\omega_2\left(\psi_2+\psi_4\right)\varepsilon_2}{\left(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu\right)\left(\psi_1^2+\left(\mu+\varepsilon_2+\psi_2+\psi_3+\psi_4\right)\psi_1+\left(\mu+\varepsilon_2+\psi_2+\psi_3+\omega_2\right)\psi_4+\omega_2\psi_2\right)}$$
$$= -0{,}3731$$

$$\Im_{\mu}^{R_0} = \frac{\left(\begin{array}{c} \frac{\beta\omega_1(\psi_1+\psi_4)}{(\omega_1+\mu)(\varepsilon_1+\psi_1+\omega_2+\mu)(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4} \\ -\frac{\beta\omega_1\left((\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_1+(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4+\omega_2\psi_2+\omega_2\psi_4\right)}{(\omega_1+\mu)^2(\varepsilon_1+\psi_1+\omega_2+\mu)(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4} \\ -\frac{\beta\omega_1\left((\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_1+(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4+\omega_2\psi_2+\omega_2\psi_4\right)}{(\omega_1+\mu)(\varepsilon_1+\psi_1+\omega_2+\mu)(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)^2\psi_4} \end{array}\right)\mu(\omega_1+\mu)(\varepsilon_1+\psi_1+\omega_2+\mu)(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4}{\beta\omega_1\left((\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_1+(\psi_1+\psi_2+\psi_3+\varepsilon_2+\mu)\psi_4+\omega_2\psi_2+\omega_2\psi_4\right)}$$

=-0.2669

8. Sensitivity Analysis and Epidemiological Implications (Figure 2a)

Figure 2a presents a sensitivity bar chart showing the normalized sensitivity indices of various parameters with respect to the basic reproduction number (R_0) a key threshold quantity that determines whether an infectious disease can invade and persist in a population [31]. Parameters with positive sensitivity indices have a direct, amplifying effect on R_0, meaning that increases in these parameters lead to a higher value of R_0, thereby facilitating the transmission of Ebola Virus Disease (EVD). In this context, the contact rate (β) which represents the frequency of potentially infectious interactions between susceptible individuals and infectious individuals or
contaminated surfaces exhibits a strong positive sensitivity index. This indicates that an increase in the contact rate significantly raises R_0, promoting faster and wider spread of the virus. Therefore, any public health intervention aimed at reducing the contact rate such as isolating infected individuals, promoting hand hygiene, using personal protective equipment (PPE), and limiting mass gatherings can substantially reduce the transmission potential of EVD [29, 30]. Conversely, parameters with negative sensitivity indices are inversely related to R_0. An increase in these parameters leads to a reduction in the basic reproduction number, thereby contributing to the control and eventual elimination of the disease. Notably, the vaccination (α_1) rate shows a strong negative sensitivity index, indicating that higher vaccination coverage effectively reduces R_0. This finding highlights the critical importance of implementing widespread and timely vaccination programs as a core strategy to mitigate the spread of EVD. The sensitivity analysis reveals that reducing parameters with positive influence on R_0 (such as contact rate) and enhancing those with negative influence (such as vaccination rate) are essential to controlling the epidemic. Strategic public health efforts focused on minimizing exposure and maximizing immunity through vaccination will not only lower R_0 but also drive the disease toward eradication in the population.

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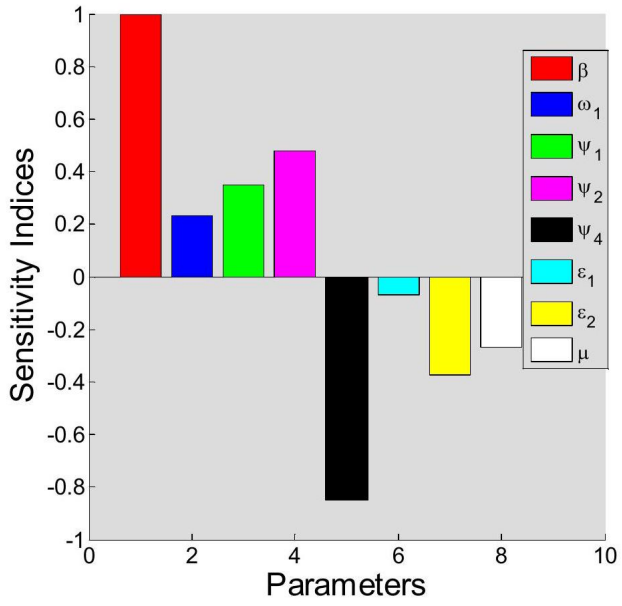

Figura 2: Sensitivity bar chart

9. Numerical Simulations of the Model

Numerical simulation of mathematical models for Ebola disease can have significant effects on public health responses and understanding of the outbreak.

1. Prediction and Forecasting: Mathematical models, when simulated numerically, can provide predictions and forecasts about the spread of Ebola. These simulations allow public health officials to anticipate the trajectory of the outbreak, identify high-risk areas, and allocate resources more effectively [16, 19].
2. Evaluation of Control Measures: Simulation of mathematical models enables the evaluation of different control measures and interventions in a virtual environment before implementing them in real life. This helps policymakers assess the potential impact of interventions such as vaccination campaigns, quarantine measures, and treatment strategies [18].
3. Optimization of Resource Allocation: By simulating different scenarios, mathematical models can help optimize the allocation of limited resources such as healthcare personnel, medical supplies, and treatment facilities. This ensures that resources are allocated where they are most needed to control the outbreak effectively [19].
4. Understanding Transmission Dynamics: Numerical simulation of mathematical models provides insights into the transmission dynamics of Ebola, including factors such as the role of asymptomatic carriers, the effectiveness of contact tracing, and the impact of population mobility. This understanding is crucial for designing targeted interventions [20].
5. Scenario Planning: Simulation allows for scenario planning, where public health officials can explore various hypothetical situations and assess their potential outcomes. This helps in developing contingency plans and preparedness strategies for different scenarios, including worst-case scenarios [19].
6. Communication and Education: Visualizations generated from numerical simulations can aid in communicating complex epidemiological concepts to the public, policymakers, and other stakeholders. This enhances public understanding of the outbreak and the rationale behind public health interventions [20].

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Parameter	Value	Source
Λ	0.202	Assumed
μ	0.03	[12]
α_1	0.25	[12]
λ	0.001	Assumed
ω_1	0.1	[14]
α_2	0.001	Assumed
ε_1	0.5	Assumed
ε_2	0.15	[14]
ω_2	0.80	[15]
ψ_1	0.01	[15]
ψ_2	0.02	Assumed
ψ_3	0.982	[12]
ψ_4	0.0025	Assumed
β	0.027	Assumed

Cuadro 3: Parameter values used for simulations

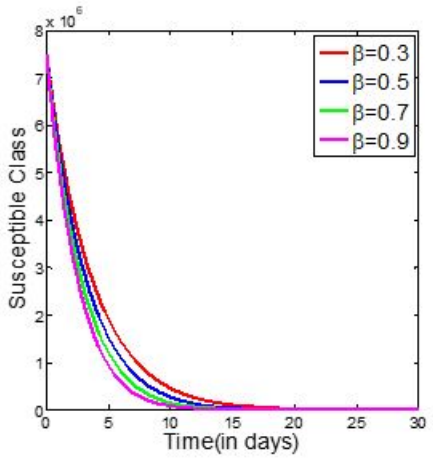

Fig. 3a Graph of susceptible humans against time

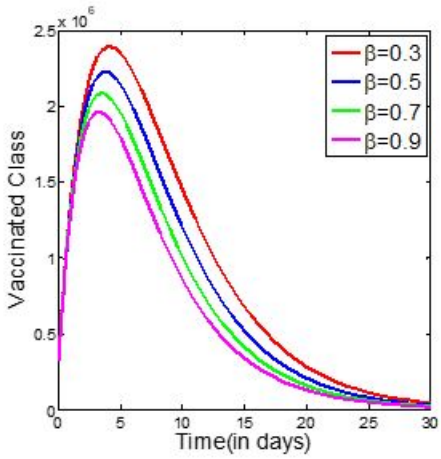

Fig. 3b Graph of vaccinated humans against time

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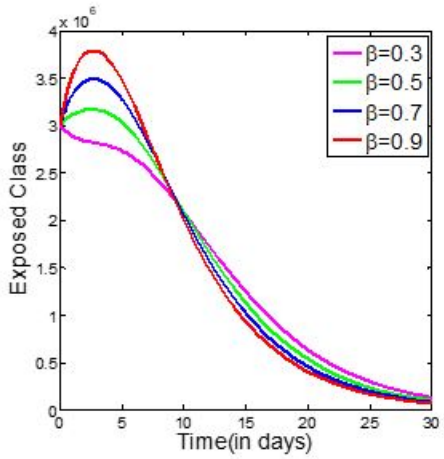

Fig. 3c Graph of exposed humans against time

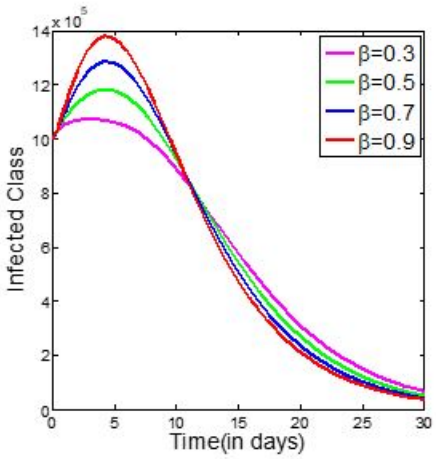

Fig. 3d Graph of infected humans against time

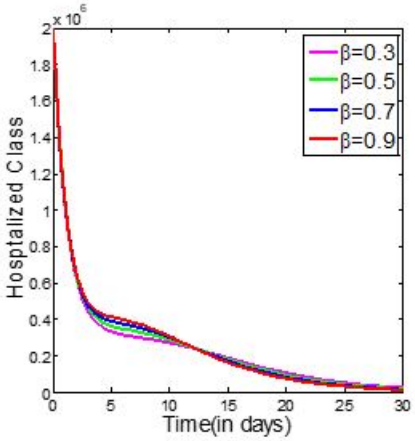

Fig. 3e Graph of hospitalized human against time.

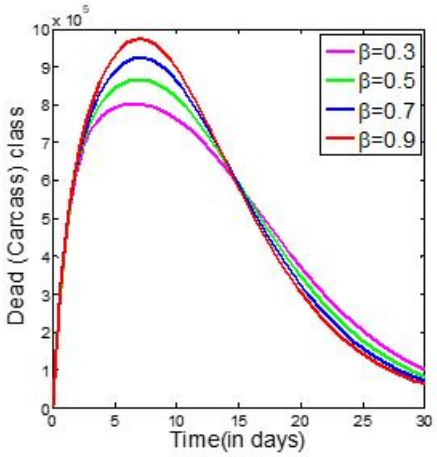

Fig.3f Graph of dead and unburried population

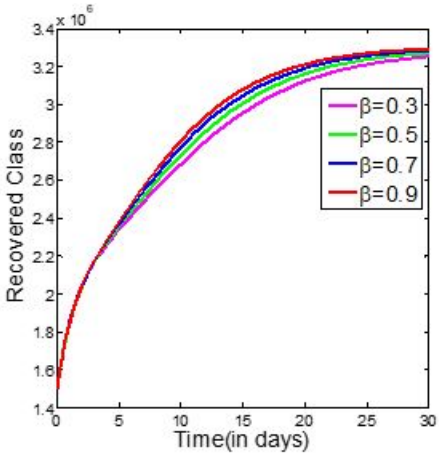

Fig. 3g. Graph of recovered human against time

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9.1. 4.1 Epidemiological Interpretation of Simulation Results (Figures 3a-3g)

Figure 3a illustrates the dynamics of the susceptible population over time under varying contact rates (β). As the contact rate increases, the number of susceptible individuals decreases significantly. This trend is expected, as a higher contact rate increases the likelihood of transmission from infectious individuals to those who are susceptible, thereby reducing the susceptible pool more rapidly [33]. In contrast, Figure 3b shows that the number of vaccinated individuals increases as the contact rate (β) decreases. This suggests the presence of an effective vaccination campaign, which is further supported by the high vaccination rate assumed in the model. A lower contact rate may also imply better compliance with public health measures, such
as seeking vaccination, which leads to a higher number of individuals moving from the susceptible to the vaccinated class. Figure 3c presents the trend of exposed individuals over time. There is an initial rapid increase in the number of exposed individuals, which later declines as the contact rate (β) decreases. This pattern indicates that reduced contact with infectious individuals, likely due to behavioral interventions or public health control measures, effectively curtails the spread of the virus during the incubation period. Similarly, Figure 3d shows the temporal dynamics of the infectious population. An initial rise in the number of infectious individuals is observed, followed by a gradual decline as the contact rate (β) decreases. This decline may be attributed to the combined effects of reduced transmission, increased vaccination coverage (as seen in Figure 3b), and timely hospitalization or isolation of cases.

Figure 3e illustrates the number of hospitalized individuals. As the number of infectious individuals declines, a corresponding decrease in hospitalizations is observed. This reflects the downstream impact of successful interventions in the earlier stages of transmission namely vaccination and reduction in exposure which leads to fewer cases requiring hospitalization. Figure 3f shows the trend in the number of deceased but unburied individuals. Given the high virulence of the Ebola virus, there is an early surge in the number of deaths. However, over time, this number declines sharply and eventually approaches zero. This decline indicates the effectiveness of public health responses such as timely medical intervention, proper case management, and implementation of safe burial practices, which help prevent further infections from contact with corpses. Finally, Figure 3g displays a high recovery rate over time, indicating that with proper medical care and control strategies, a substantial proportion of hospitalized individuals are able to recover. The increasing number of recovered individuals further contributes to the decline in the susceptible and infectious populations.

Generally, the trends observed in Figures 3a to 3g suggest that Ebola Virus Disease (EVD) can be effectively controlled and potentially eradicated from the population through a combination of interventions. These include reducing the contact rate, increasing vaccination coverage, enhancing case detection and treatment, and promoting safe burial practices. The model underscores the importance of integrated control strategies in achieving disease elimination and protecting public health.

4.2 Findings from the Study

1. Impact of Vaccination Rate: High vaccination rates play a significant role in controlling the spread of the Ebola virus. Numerical simulations demonstrate that increasing vaccination coverage substantially reduces the number of new infections, thereby contributing to effective containment of the disease.
2. Effectiveness of Treatment: The implementation of timely and efficient treatment strategies leads to higher recovery rates among infected individuals. The simulations confirm that improved access to medical care significantly limits the progression of the disease and aids in reducing transmission.
3. Role of Contact Rate: Sensitivity analysis underscores the critical role of minimizing contact between susceptible and infectious individuals. A lower contact rate is strongly associated with reduced transmission, highlighting the importance of behavioral interventions such as isolation, hygiene practices, and social distancing.

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4. Threshold Effects: The study identifies specific threshold levels for both vaccination coverage and treatment efficacy. Surpassing these thresholds results in a marked decline
in disease prevalence, thereby strengthening the effectiveness of control measures and moving the system toward disease elimination.
5. Temporal Dynamics: The mathematical model captures the time-dependent behavior of the Ebola outbreak, showing how variations in vaccination rate, treatment effectiveness, and contact rate influence the epidemic's progression over time. This dynamic analysis provides insight into the optimal timing and intensity of interventions.
6. Trade-offs and Synergies: The findings highlight important trade-offs and synergies between different intervention strategies. While both vaccination and treatment independently contribute to disease reduction, their combined application yields a greater overall impact. This suggests that integrated approaches are more effective than isolated efforts.
7. Policy Implications: The results of the study offer valuable guidance for public health policymakers. Emphasis should be placed on expanding vaccination programs, enhancing treatment infrastructure, and enforcing measures to reduce contact with infectious individuals. A coordinated and multifaceted approach is essential for the successful control and eventual eradication of Ebola Virus Disease.

5. Conclusion

This study presented a comprehensive mathematical model for the transmission dynamics of Ebola Virus Disease (EVD), incorporating key epidemiological features such as vaccination, treatment, contact with infected individuals and contaminated surfaces, hospitalization, and safe burial practices. The model stratified the total population into seven distinct compartments Susceptible, Vaccinated, Exposed, Infected, Hospitalized, Deceased (unburied), and Recovered to accurately reflect the natural history of Ebola and the impact of control measures. Through a combination of analytical techniques and numerical simulations, the basic reproduction number (R_0) was derived using the next-generation matrix method. Stability analysis confirmed that the disease-free equilibrium is locally and globally asymptotically stable when $R_0 < 1$, and unstable when $R_0 > 1$, thereby establishing R_0 as a critical threshold parameter for disease persistence. Sensitivity analysis identified the most influential parameters affecting R_0. It was found that the contact rate (β) had a strong positive impact on disease transmission, indicating that reducing contact with infectious individuals is crucial for containment. In contrast, vaccination rate and treatment effectiveness were negatively correlated with R_0, emphasizing their importance in suppressing the outbreak. These findings provide evidence-based insight into the prioritization of public health interventions.

The numerical results demonstrated that increasing vaccination coverage significantly reduces new infections, while effective and timely treatment enhances recovery rates and limits disease progression. Moreover, the simulations showed that combining multiple strategies such as vaccination, treatment, and reduction in contact rate-produces synergistic effects, yielding a greater impact on reducing disease burden than any single intervention alone. The temporal dynamics captured by the model illustrated the evolution of EVD over time and how various intervention measures influence the trajectory of the epidemic. Importantly, the model showed that with sufficient vaccination and treatment efforts, and by minimizing contact with infected individuals and corpses, the disease can be controlled and eventually eradicated from the population. The study underscores the importance of integrated and well-coordinated public health strategies in managing Ebola outbreaks. Policymakers and public health practitioners are encouraged to invest in widespread vaccination campaigns, strengthen healthcare infrastructure
for timely treatment, and enforce strict infection prevention and control (IPC) measures, especially in handling deceased individuals. These combined efforts will not only suppress current outbreaks but also build resilience against future resurgence of Ebola Virus Disease.

Declarations

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Model formulation, Methodology, Simulations. Odeh J. O, Model formulation, Methodology, Conceptualization Methodology, preparation, Model formulation, Analysis. Ekwoba L, Editing, Literature review, writing introduction, preparation,. Senewo E. O Literature review, Conceptualization Methodology, preparation, Analysis, Model formulation. Onoja K. A writing introduction, literature review, editing, preparation, simulations. Agbebaku D. F Analysis, simulations, methodology, supervision, conceptualization. Obeng-Denteh W. Analysis, Methodology, simulations, literature review, editing and supervision

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ORCID

Agbata Benedict Celestine https://orcid.org/0000-0003-3217-7612
Yahaya Jibrin Danjuma https://orcid.org/0000-0002-3701-1585
Odeh Jane Ojowu https://orcid.org/0009-0007-8621-5529
Ekwoba Lucky https://orcid.org/0000-0001-9864-7723
Emmanuel Olorunfemi Senewo https://orcid.org/0009-0000-2188-830X
Onoja Kingsley Anselem https://orcid.org/0009-0003-8058-2089
Agbebaku Dennis Ferdinand https://orcid.org/0000-0002-0709-1748
Obeng-Denteh William https://orcid.org/0000-0002-1865-290X

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