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Susceptibility Inference and Response on Transmission Dynamics of Ebola Virus in Fuzzy Environment

Saravanan Subraja, Murugappan Mullai*, Grienggrai Rajchakit, Govindan Vetrivel and R. Surya

Abstract – This article uses fuzzy parameters to develop a susceptibility inference and response (SIR) model for the Ebola virus. The construction of the SIR model involves considering several aspects, including immunization, therapy, compliance with medical protocols, and Ebola virus load. The parameters representing the infection, mortality, and recovery rates caused by the Ebola virus are expressed as fuzzy numbers. These parameters are then employed as fuzzy parameters in the model. The study of the model uses the generation matrix approach to get the fundamental reproduction number and assess the stability of the equilibrium point inside the model. The findings from the simulation indicate that the variation in the Ebola virus load is associated with disparities in the transmission patterns of the Ebola virus. Also, we compare the impact of the variables of vaccination and following the medical guidelines in reducing the spread of the Ebola virus. Using Matlab software, the numerical simulation for this model is carried out, and the analysis of Ebola virus transmission is investigated in the fuzzy environment.

Keywords— Ebola Virus, Fuzzy Parameter, Immunization, Basic Reproduction Number, Death Rate.

I. INTRODUCTION

The Ebola virus dwells in bats. It infects humans by directly handling or eating bats. The Ebola virus can

infect people by touching contaminated objects or body fluids of infected bats or non-human primates. It is hazardous to humans because it may cause a variety of illnesses and sometimes lead to death. The deaths caused by Ebola are nearly 50%. Compared with past outbreaks, the death rate caused by the Ebola virus has varied from 25% to 90% according to the WHO report, 2023. This virus is spread from wild animals that are already sick to humans. Due to the contact of people who are very close to each other, it spreads from one person to another. Supportive care for rehydration and treatment in the early stages helps to improve the survival of infected people.

Five species of Ebola virus have been found. This disease was first identified in South Sudan and Congo in 1976. Following that, there were 25 instances of Ebola virus epidemics, primarily concentrated in Central Africa. After that, many people in West Africa were affected by this disease from 2014 to 2016, which can be viewed from the systematic work on the Ebola virus disease [1]. This disease's epidemiological diagnosis and clinical manifestation can be learned clearly from [2]. The initial indications of Ebola sickness are fever, fatigue, myalgia, cephalalgia, and throat infections. In addition, symptoms such as vomiting, rashes, diarrhoea, impaired kidney and liver function, and frequent

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internal and external bleeding may occur. We cannot say the symptoms of Ebola disease specifically, and medical diagnosis may be a difficult one. The incubation period of this disease is 2 to 21 days. Let's analyze the history of patients affected by Ebola disease. The disease spreads from contact with dead or sick animals and with a suspected or confirmed Ebola disease patient. The list of the local effects caused by Ebola and its impact on lower child vaccinations is studied [3]. WHO publishes the guidelines and recommendations for the Ebola virus disease and its spread. An overview of the Ebola virus disease and its treatment measures [4] can be observed. Many researchers developed models and analyzed the results of the spread of the Ebola virus. An activity to resist Ebola with three standard disinfectants [5] is experimented with. An analytical verification with a model on the replication and vertical transmission of Ebola from Angolan bats [6] is done. When someone is identified with the Ebola disease, the proper treatment should be given. Intensive care support, oxygenation, antibiotic drugs, and Psychosocial support are critical. Also, renal function, level of electrolyte balance, blood pressure, and rehydration should be checked often. WHO suggested various preventive measures to reduce the spread of the Ebola virus. Some of them are as follows:

1. Reduce contact with infected fruit bats, monkeys, or apes.
2. Gloves and appropriate clothing should be used when handling the animals.
3. Cook the animal products before consumption.
4. Reduce the contact of people with Ebola symptoms and handle those people with safety measures.
5. The burial of the people dead from Ebola disease should be organized carefully.
6. Safe sexual intercourse.
7. Take extra care of the people affected by the Ebola disease.

The SIR model is one of the best models for analyzing the spread of viral diseases. This model includes the fixed population of N individuals into three compartments, which may vary as a function of $S(t)$, $I(t)$, and $R(t)$ due to time where $S(t)$, $I(t)$, and $R(t)$ represent the susceptible, infected and recovered from the disease. The epidemic will end if the infection rate is lower than the recovery. The epidemic will spread if the recovery rate is lower than the infection rate. A design that illustrates the deterministic epidemic model [7] for Ebola virus infection using time-dependent controls is portrayed. W. Chen modeled the Ebola virus based on SIR [8]. The Kenmack-Mckendrick is used for the number of people in a closed community who get a contagious illness over time. Precise numerical values are used as the parameters in current SIR epidemic models, whereas parameter uncertainty and population variability are very probable. Utilizing fuzzy parameters is crucial for enhancing the realism of the model. The paper [9] presents a SIR model for the propagation of COVID-19 that incorporates fuzzy parameters. The fuzzy parametrical approach [10] is featured to analyze the transmission dynamical behavior of an epidemic model. The basic reproduction number [11] is used to analyze the infection by considering the expected number of cases

in a population. Some related math models [12], [13] can be viewed to construct this current model. From the model, we construct a SIR mathematical for the Ebola virus using the fuzzy parameters. We construct the susceptible, infected, and death rate parameters using a SIR model. Depending on the Ebola virus load, the death rate, infected rate, and recovery rate due to the Ebola virus are considered in membership functions. Using the next generation matrix, find the basic reproduction number and then analyze the stability analysis of the SIR model for the Ebola virus by disease-free equilibrium $\mathcal{R}_0(\zeta) < 1$ and the endemic equilibrium $\mathcal{R}_0(\zeta) > 1$. This replication makes assumptions about the parameters of immunization, therapy, adherence to medical guidelines, and Ebola virus load.

II. SIR MODEL FOR EBOLA VIRUS

Examine a SIR model for the Ebola virus (1), (2), and (3) that explains the dynamics of direct transmission, including an interaction between suspected and infected individuals, the transition from infection to recovery, rates of pure births or deaths, the efficiency of vaccinations and treatments, adherence to treatment regimens and fatalities as a result of Ebola infection.

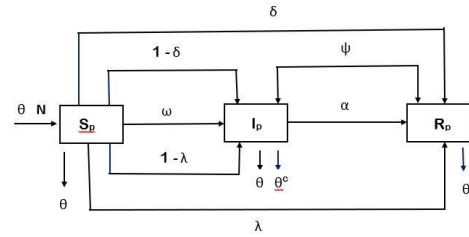


FIGURE 1. The schematic representation of the transmission pathway of the Ebola virus for model SIR.

$$\frac{dS_p}{dt} = \theta - \omega(1 - \delta)(1 - \lambda)S_p I_p - (\theta + \delta + \lambda)S_p \quad (1)$$

$$\frac{dI_p}{dt} = \omega(1 - \delta)(1 - \lambda)S_p I_p - (\theta + \theta^c + \psi + \alpha)I_p \quad (2)$$

$$\frac{dR_p}{dt} = (\psi + \alpha)I_p + (\lambda + \delta)S_p - \theta R_p \quad (3)$$

Here, the rate of a susceptible individual in a total population is represented as S_p . In contrast, I_p is the rate of Infected individuals in a total population, and the rate of Recovered individuals in a total population is represented as R_p . ω is the parameter that represents the Infection rate, and α is the parameter that represents the Recovery rate. The Natural birth or death rate is represented as θ . The parameters representing immunization, treatment effectiveness, and adherence to medical guidelines are δ , ψ and λ respectively. The death rate due to the Ebola virus is represented in the parameter θ^c .

The SIR Model can now be expanded to consider the heterogeneity of the Ebola virus load in each

individual, where people with varying levels of the virus load contribute in different ways to the spread of the Ebola virus.

III. FUZZY SIR MODEL ON EBOLA VIRUS SPREAD

By considering the SIR model for the ebola virus in equations (1) through (3), let ζ represent a person's Ebola virus load. We now consider the heterogeneity in the model by evaluating each person's ability to infect as a function of their Ebola virus load. As a result, the potential of the ebola virus spreading during a contact encounter rises with the Ebola viral load of an individual.

The parameters ω , θ^c and α can be understood as functions of the Ebola virus load by considering the Ebola virus load in each individual. This model, which we will refer to as the fuzzy SIR model (4), (5), and (6) from here on, can be expanded to represented below:

$$\frac{dS_p}{dt} = \theta - \omega(\zeta)(1 - \delta)(1 - \lambda)S_p I_p - (\theta + \delta + \lambda)S_p \quad (4)$$

$$\frac{dI_p}{dt} = \omega(\zeta)(1 - \delta)(1 - \lambda)S_p I_p - (\theta + \theta^c(\zeta) + \psi + \alpha(\zeta))I_p \quad (5)$$

$$\frac{dR_p}{dt} = (\psi + \alpha(\zeta))I_p + (\lambda + \delta)S_p - \theta R_p \quad (6)$$

Let $\omega = \omega(\zeta)$ denote the probability of transfer of the Ebola virus load between a suspected and infected person. When ω is set to a value that is more reasonable than others, it becomes a membership function of fuzzy numbers. The membership function is constructed under the assumption that a minimum Ebola virus load ζ_{min} is required to be transmitted to other individuals, and the possibility of transmission is minimal if an individual has a relatively low number of Ebola viruses. Furthermore, the maximal transmission rate of the Ebola virus equals one at a specific Ebola virus load ζ_0 . It is assumed, however, that a person's total Ebola virus load ζ is constrained by ζ_{max} . We also take into account that immunization and compliance with medical protocols will have an impact on the Ebola virus's transmission rate. Let δ and λ be the metrics that respectively indicate immunization and compliance with medical protocols. The rate of infectious contact (7) in the fuzzy membership function can be found here.

$$\omega(\zeta) = \begin{cases} 0, & \text{if } \zeta \leq \zeta_{min} \\ \frac{(\zeta - \zeta_{min})(1 - \delta)(1 - \lambda)}{\zeta_0 - \zeta_{min}}, & \text{if } \zeta_{min} < \zeta < \zeta_0 \\ (1 - \delta)(1 - \lambda), & \text{if } \zeta_0 < \zeta < \zeta_{max} \end{cases} \quad (7)$$

The graphical representation of the virus load of the infected parameter $\omega(\zeta)$ is given in Fig. 2. Another possibility is to consider the death rate from Ebola virus infection as a fuzzy membership function. The function involves the Ebola virus load rising over time. Hence, we take this function as an increasing function. However, the function might not reach its maximal value of one due to several factors, including the availability of medication, the Ebola-infected person's illness, immunity, etc. Similarly, the rate of death from

Ebola virus infection will change in response to treatment. Hence, it is postulated that the function $\theta^c(\zeta)$ has a maximum value of $(1 - \eta)(1 - \psi) + \theta_0^c$, with $(0 \leq \eta \leq 1)$ and $(0 \leq \psi \leq 1)$, respectively. Thus, the following is how we define a function $\theta^c(\zeta)$ (8) [Fig.3 shows the fuzzy membership function of a death rate parameter].

$$\theta^c(\zeta) =$$

$$\begin{cases} ((1 - \eta) - \theta_0^c)(1 - \psi) \frac{\zeta}{\zeta_0} + \theta_0^c, & \text{if } 0 \leq \zeta < \zeta_0 \\ (1 - \eta)(1 - \psi) + \psi \theta_0^c, & \text{if } \zeta_0 \leq \zeta \end{cases} \quad (8)$$

The graphical representation of the virus-loaded recovery rate parameter $\theta^c(\zeta)$ is given in Fig. 4. Here, ψ is the efficiency of treatment, and θ_0^c ($0 < \theta_0^c < 1$) is the lowest death rate from an Ebola infection. The Ebolavirus load ζ also affects the recovery rate of the ebola virus infection group $\alpha = \alpha(\zeta)$. The duration of the recovery process is according to the Ebola virus load ζ . Then, the function $\alpha(\zeta)$ is a decreasing one. Furthermore, the pace of recuperation is considered as a result of the medicine.

As a result, the fuzzy membership function has the following definition and α_0 is the lowest recovery rate.

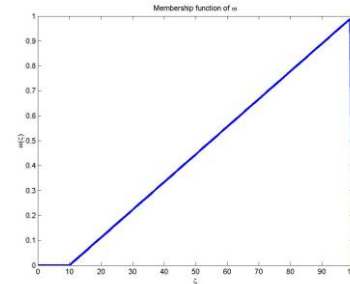


FIGURE 2. Membership function of the rate of Infection.

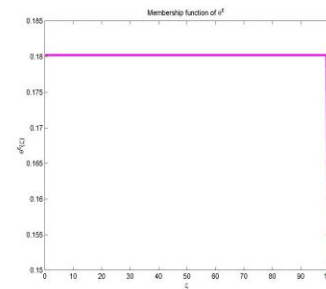


FIGURE 3. Membership function of rate of death.

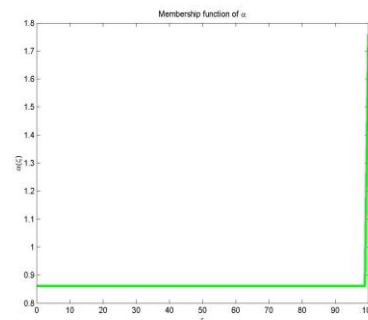


FIGURE 4. Membership fn. of rate of recovery.

Consequently, the definition of the fuzzy membership function of a recovery rate (9) is as follows (Fig.4).

$$\alpha(\zeta) = \begin{cases} (\alpha_0 - 1)(1 - \psi) \frac{\zeta}{\zeta_0} + 1, & \text{if } 0 \leq \zeta < \zeta_0 \\ \alpha_0(1 - \psi) + \psi, & \text{if } \zeta \geq \zeta_0. \end{cases} \quad (9)$$

The infection rate $\omega(\zeta)$, recovery rate $\alpha(\zeta)$ and the death rate $\theta^c(\zeta)$ due to Ebola virus infection are taken as fuzzy parameters in this model. The equation (4) through (6) have the endemic equilibrium point and disease-free equilibrium point. All two equilibrium points must be found by ensuring that $\frac{dS_p}{dt} = 0, \frac{dI_p}{dt} = 0, \frac{dR_p}{dt} = 0$, the respective equations are equal to zero. From this, the equations (4) through (6) becomes (10) through (12):

$$\theta - \omega(\zeta)(1 - \delta)(1 - \lambda)S_p I_p - (\theta + \delta + \lambda)S_p = 0 \quad (10)$$

$$\omega(\zeta)(1 - \delta)(1 - \lambda)S_p I_p - (\theta + \theta^c(\zeta) + \psi + \alpha(\zeta))I_p = 0 \quad (11)$$

$$(\psi + \alpha(\zeta))I_p + (\lambda + \delta)S_p - \theta R_p = 0 \quad (12)$$

Now, we define an equilibrium point for the susceptible rate (S_p), Infected rate (I_p) and Recovery rate (R_p). Here the graphical representations of membership functions of infection, recovery, and death rates in Fig.2, Fig.3, and Fig.4 have been carried out by using Matlab software for the random values of parameters $\zeta, \zeta_0, \zeta_{min}$ and ζ_{max} .

IV. MODEL COMPOSED OF FUZZY SIR WITH DISEASE-FREE EQUILIBRIUM

The regions in which Ebola does not propagate are the state of equilibrium for disease-free settings (13), (14) are (I_p) = (I_p^0) = 0.

Thus, the equation (4) becomes,

$$S_p = S_p^0 = \frac{\theta}{\lambda + \delta + \theta} \quad (13)$$

The equation (6) and equation (13) becomes,

$$R_p = R_p^0 = \frac{\lambda + \delta}{\lambda + \delta + \theta} \quad (14)$$

Thus, equations (4), (5), and (6) according to the disease-free equilibrium points become as (15),

$$D^0 = (S_p^0, I_p^0, R_p^0) = \left(\frac{\theta}{\lambda + \delta + \theta}, 0, \frac{\lambda + \delta}{\lambda + \delta + \theta} \right) \quad (15)$$

V. MODEL OF THE ENDEMIC EQUILIBRIUM USING FUZZY SIR

When a disease may spread under specific conditions, the equilibrium points are commonly referred to as endemic equilibrium points.

These locations are regarded as $S_p = S_p^* \neq 0, I_p = I_p^* \neq 0, R_p = R_p^* \neq 0$. Therefore we can get the following endemic equilibrium points (16), (17) and (18) for the SIR fundamental model from equations (4) through (6), and obtain D' in the form (19),

$$S_p^* = \frac{\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta}{\omega(\zeta)(1 - \lambda)(1 - \delta)} \quad (16)$$

$$I_p^* = \frac{\theta}{\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta} - \frac{\lambda + \delta + \theta}{\omega(\zeta)(1 - \delta)(1 - \lambda)} \quad (17)$$

$$R_p^* = \frac{(\psi + \alpha(\zeta))I_p^* + (\lambda + \delta)S_p^*}{\theta} \quad (18)$$

Thus,

$$D' = (S_p^*, I_p^*, R_p^*) = \left(\frac{\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta}{\omega(\zeta)(1 - \lambda)(1 - \delta)}, \frac{\theta}{\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta} - \frac{\lambda + \delta + \theta}{\omega(\zeta)(1 - \delta)(1 - \lambda)}, \frac{(\psi + \alpha(\zeta))I_p^* + (\lambda + \delta)S_p^*}{\theta} \right) \quad (19)$$

The next-generation matrix approach is used to determine the fundamental reproductive number \mathfrak{R} for equations (1) through (3) [11]. Using Equations ((1) through (3)) as a guide, ascertain \mathfrak{R}_0 .

Let $X = \omega(1 - \delta)(1 - \lambda)S_p I_p$, $Y = (\theta + \theta^c + \psi + \alpha)I_p$, then we obtain $X' = \omega(1 - \delta)(1 - \lambda)S_p$, $Y' = \theta + \theta^c + \psi + \alpha$ and $(Y')^{-1} = \frac{1}{\theta + \theta^c + \psi + \alpha}$. The dominant eigenvalue of $X'(Y')^{-1}$ defines $\mathfrak{R}_0 = \sigma(X'(Y')^{-1})$ (20), which is stated as:

$$\mathfrak{R}_0 = \frac{\omega\theta(1 - \delta)(1 - \lambda)}{(\delta + \lambda + \theta)(\psi + \theta^c + \alpha + \theta)} \quad (20)$$

Thus, from this: $\omega = \omega(\zeta)$, $\theta^c = \theta^c(\zeta)$, $\alpha = \alpha(\zeta)$, then $\mathfrak{R}_0(\zeta)$ (21) is given as:

$$\mathfrak{R}_0(\zeta) = \frac{\omega(\zeta)\theta(1 - \delta)(1 - \lambda)}{(\delta + \lambda + \theta)(\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta)} \quad (21)$$

Here, $\mathfrak{R}_0(\zeta)$ refers to the virus-loaded(ζ) basic reproduction number and $\omega(\zeta)$, $\theta^c(\zeta)$, $\alpha(\zeta)$ are defined as virus-loaded(ζ) fuzzy parameters.

VI. STABILITY ANALYSIS

Theorem 6.1

If $\mathfrak{R}_0(\zeta) < 1$, be the basic reproduction number smaller than 1, then for equations (4) through (6) the disease-free equilibrium point exhibits local asymptotic stability and it becomes unstable when the value of $\mathfrak{R}_0(\zeta) > 1$.

Proof. For the equation (4) through (6), the Jacobian matrix \mathfrak{J} (22) is provided as follows:

$\mathfrak{J} =$

$$\begin{pmatrix} -\omega(\zeta)(1 - \delta)(1 - \lambda)I_p - (\delta + \lambda + \theta) & -\omega(\zeta)(1 - \delta)(1 - \lambda)S_p & 0 \\ \omega(\zeta)(1 - \delta)(1 - \lambda)I_p & \omega(\zeta)(1 - \delta)(1 - \lambda)S_p - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) & 0 \\ 0 & 0 & -\theta \end{pmatrix} \quad (22)$$

By substituting the value of $I_p = 0$, $S_p = \frac{\theta}{\delta + \lambda + \theta}$, we get the Jacobian matrix \mathfrak{J}_0 (23),

$\mathfrak{J}_0 =$

$$\begin{pmatrix} -(\lambda + \delta + \theta) & -\omega(\zeta)(1-\delta)(1-\lambda)\frac{\theta}{\delta+\lambda+\theta} & 0 \\ 0 & \omega(\zeta)(1-\delta)(1-\lambda)S_p^* - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) & 0 \\ 0 & 0 & -\theta \end{pmatrix} \quad (23)$$

The calculation of the eigenvalue for \mathfrak{J}_0 is given by equation (24),

Eigen (\mathfrak{J}_0)

$$= ((\epsilon + \lambda + \delta + \theta)(\epsilon + \theta)(\epsilon + \psi + \theta^c(\zeta) + \lambda(\zeta) + \theta) - \frac{\omega(\zeta)\theta(1-\delta)(1-\lambda)}{\delta+\lambda+\theta}), \quad (24)$$

We get the eigenvalues as (25), (26) and (27),

$$\epsilon_1 = -(\delta + \lambda + \theta) \quad (25)$$

$$\epsilon_2 = -\theta \quad (26)$$

$$\begin{aligned} \epsilon_3 &= -\frac{\omega(\zeta)\theta(1-\delta)(1-\lambda)}{(\delta + \lambda + \theta)} + (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) \\ &= -(\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) \left(-\frac{\omega(\zeta)\theta(1-\delta)(1-\lambda)}{(\delta + \lambda + \theta)(\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta)} + 1 \right) \end{aligned}$$

$$= -(\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) - (-\mathfrak{R}_0(\zeta) + 1) \quad (27)$$

From the theorem, we obtain the following:

If $\mathfrak{R}_0(\zeta) < 1$, then $\epsilon_3 < 0$, and if $\mathfrak{R}_0(\zeta) > 1$, then $\epsilon_3 > 0$.

Theorem 6.2

When the basic reproduction number exceeds 1 i.e $\mathfrak{R}_0(\zeta) > 1$, the endemic equilibrium point forequations (4) through (6) exhibits local asymptotic stability.

Proof. The Jacobian matrix \mathfrak{J}_1 (28) for the equations (4) to (6) and the endemic equilibrium point is given by

$$\mathfrak{J}_1 = \begin{pmatrix} -\omega(\zeta)(1-\delta)(1-\lambda)I_p^* - (\delta + \lambda + \theta) & -\omega(\zeta)(1-\delta)(1-\lambda)S_p^* & 0 \\ \omega(\zeta)(1-\delta)(1-\lambda)I_p^* & \omega(\zeta)(1-\delta)(1-\lambda)S_p^* - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) & 0 \\ 0 & 0 & -\theta \end{pmatrix} \quad (28)$$

By our assumption $j_1 = \omega(\zeta)(1-\delta)(1-\lambda)I_p^* + (\delta + \lambda + \theta)$, $j_2 = \omega(\zeta)(1-\delta)(1-\lambda)S_p^*$, $j_3 = \omega(\zeta)(1-\delta)(1-\lambda)I_p^*$, $j_4 = \omega(\zeta)(1-\delta)(1-\lambda)S_p^* - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta)$.

Thus, (28) is reduced to (29),

$$\mathfrak{J}_1 = \begin{pmatrix} -j_1 & -j_2 & 0 \\ j_3 & j_4 & 0 \\ 0 & 0 & -\theta \end{pmatrix} \quad (29)$$

The roots of $G_1(\epsilon)$ (30) are the eigenvalues of \mathfrak{J}_1 :

$$\begin{aligned} G_1(\epsilon) &= (\epsilon + \theta)[(\epsilon + j_1)(\epsilon - j_4) + j_2j_3] \\ &= (\epsilon + \theta)[\epsilon^2 + (j_1 - j_4)\epsilon - j_1j_4 + j_2j_3] \\ &= (\epsilon + \theta)G_2(\epsilon) \end{aligned} \quad (30)$$

From here, we observe that $\epsilon_1 = -\theta$ is one of the eigenvalues $G_1(\epsilon)$. Through the solution of $G_2(\epsilon) = 0$ we

get the other eigenvalues. By Routh-Hurwitz condition, if equations (31) > 0 and (32) > 0 then $G_2(\epsilon)$ with negative real part has two roots.

$$\begin{aligned} j_1 - j_4 &= [\omega(\zeta)(1-\delta)(1-\lambda)I_p^* + (\delta + \lambda + \theta)] - [\omega(\zeta)(1-\delta)(1-\lambda)S_p^* - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta)] \\ &= [(\mathfrak{R}_0(\zeta) - 1)(\delta + \lambda + \theta) + (\delta + \lambda + \theta)] \\ &\quad - [\omega(\zeta)(1-\delta)(1-\lambda) \\ &\quad \quad \theta - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) \left(\frac{(\mathfrak{R}_0(\zeta) - 1)(\delta + \lambda + \theta)}{\omega(\zeta)(1-\delta)(1-\lambda)} \right) \\ &\quad - \lambda] \left(\frac{(\delta + \lambda + \theta)}{(\delta + \lambda + \theta)} \right) \\ &\quad - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta)] \\ &= \mathfrak{R}_0(\zeta)(\delta + \lambda + \theta) + (1 - \mathfrak{R}_0(\zeta))(\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) \\ &\quad + (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta)(\mathfrak{R}_0(\zeta) - 1) \\ &= \mathfrak{R}_0(\zeta)(\delta + \lambda + \theta) \end{aligned} \quad (31)$$

As of the above discussion, when $(\mathfrak{R}_0(\zeta) > 0)$ then $j_1 - j_4 > 0$.

$$\begin{aligned} j_2j_3 - j_1j_4 &= [(\omega(\zeta)(1-\delta)(1-\lambda)S_p^*)(\omega(\zeta)(1-\delta)(1-\lambda)I_p^*)] \\ &\quad - [\omega(\zeta)(1-\delta)(1-\lambda)I_p^* + (\delta + \lambda + \theta)(\omega(\zeta)(1-\delta)(1-\lambda)S_p^* - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta))] \\ &= \left[\frac{\omega(\zeta)(1-\delta)(1-\lambda)\theta}{(\delta + \lambda + \theta)} - (\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta)(\mathfrak{R}_0(\zeta) - 1) \right] (\mathfrak{R}_0(\zeta) - 1)(\delta + \lambda + \theta) \\ &= (\mathfrak{R}_0(\zeta) - 1)(\delta + \lambda + \theta)(\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta) \end{aligned} \quad (32)$$

From here, we observe that $j_2j_3 - j_1j_4 > 0$ if $\mathfrak{R}_0(\zeta) > 1$. Consequently, we note the equations (4) through (6) are at a bifurcation point when $\mathfrak{R}_0(\zeta) = 1$, and when $\mathfrak{R}_0(\zeta) > 1$, the disease-free equilibrium is stable. Let ζ^* represents the system bifurcation value (34) then ζ^* denotes the equation's solution (33),

$$\begin{aligned} \omega(\zeta)(1-\delta)(1-\lambda)\theta &= (\delta + \lambda + \theta)(\psi + \theta^c(\zeta) + \alpha(\zeta) + \theta), \end{aligned} \quad (33)$$

(i.e.) $\zeta^* =$

$$\frac{\theta((1-\delta)(1-\lambda))^2\zeta_0\zeta_{min} + \zeta_0(\zeta_0 - \zeta_{min})(\delta + \lambda + \theta)(\theta_0^c + 1)}{\theta\zeta_0((1-\delta)(1-\lambda))^2 - ((\delta + \lambda + \theta)(\zeta_0 - \zeta_{min}))((1-\kappa) - \theta_0^c)(1-\psi) + (\alpha_0 - 1)(1-\psi)}$$

where $\zeta^* \leq \zeta_0$. (34)

This allows us to conceptualize ζ^* as an Ebola virus control parameter in the sense that it should be noted that ζ is not greater than ζ^* , if the Ebola virus spreads to people of a particular count.

Corollary 6.3

The equilibrium state without illness and the equilibrium state with disease included in equations

(4) through (6) exhibit local asymptotic stability for $\zeta < \zeta^*$ and $\zeta > \zeta^*$ respectively.

VII. NUMERICAL SIMULATION FOR THE TRANSMISSION OF THE EBOLA VIRUS

The infection, recovery, and death rate parameters i.e. ω, α, θ^c are calculated from the equations (4) through (6). The initial values for N_p, S_p, I_p and R_p which are the key components needed for the numerical simulation, is given below:

TABLE 1. Initial SIR model estimate for the Ebola virus.

Variable	Value
$N_p(0)$	35,842
$S_p(0)$	30,564
$I_p(0)$	6782
$R_p(0)$	5234

TABLE 2. Values of the fuzzy parameters of the fuzzy SIR model for the Ebola virus.

Variable	Value
θ_0^c	2.285×10^{-2}
α_0	7.667×10^{-2}
θ	6.25

By our assumption the values of each parameter and the basic reproduction number of the fuzzy SIR model for the Ebola virus is given below:

TABLE 3. Parameter values of Fuzzy SIR for Ebola Virus.

Replication	η	ζ_{min}	ζ_0	ζ	ψ	δ	λ	\mathfrak{R}_0
1	0.85	20	100	58	0	0	0	6.072×10^{-1}
2	0.85	20	100	42	0	0	0	2.918×10^{-1}
3	0.85	20	100	72	0	0	0	4.327×10^{-1}
4	0.85	20	100	68	50%	0	0	3.549×10^{-1}
5	0.85	20	100	83	60%	0	0	6.228×10^{-1}
6	0.85	20	100	63	40%	0	0	6.222×10^{-1}
7	0.85	20	100	42	0	4%	0	1.851×10^{-2}
8	0.85	20	100	100	0	10%	0	3.851×10^{-2}
9	0.85	20	100	100	0	12%	0	7.341×10^{-1}
10	0.85	20	100	21	0	0	3%	2.713×10^{-4}
11	0.85	20	100	94	0	0	10.5%	1.324×10^{-1}
12	0.85	20	100	76	0	0	12%	6.0932×10^{-2}

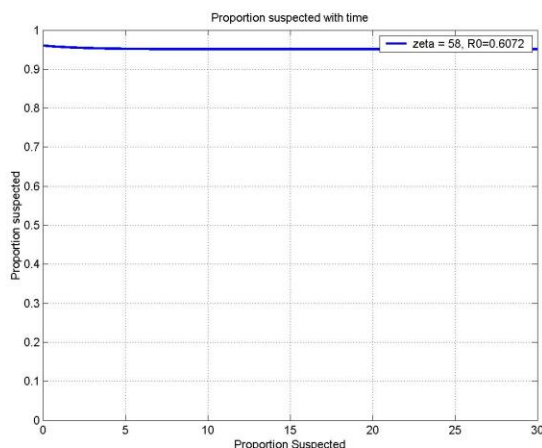


FIGURE 5. The graphical representation of the suspected rate.

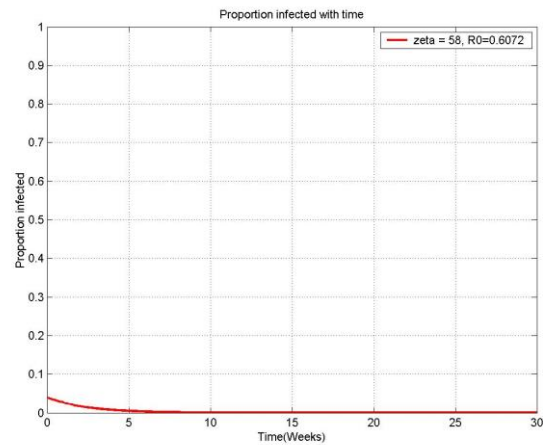


FIGURE 6. The graphical representation of the infected rate.

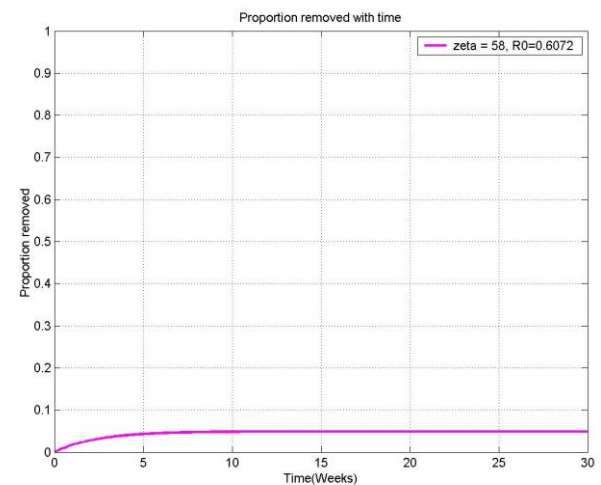


FIGURE 7. The graphical representation of the recovery rate.

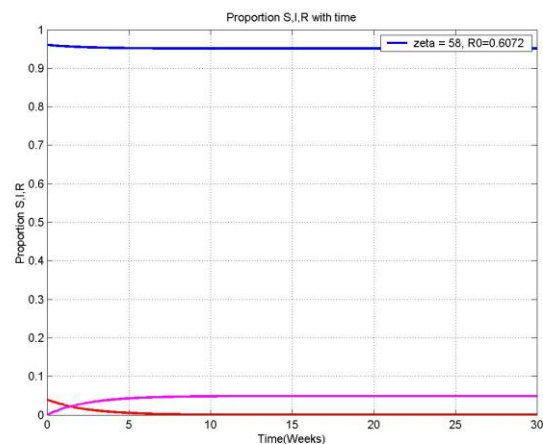


FIGURE 8. The graphical representation of a SIR with time.

Figure 5 shows the fluctuation in the estimated population proportion of a suspected rate for a certain value for ζ . Figure 6, shows the fluctuation in the estimated population proportion infected rate for a certain value for ζ . Figure 7 shows the fluctuation in the estimated population proportion of a recovery rate for a certain value for ζ . Figure 8 shows the combined fluctuation in the estimated population proportion of an SIR for a certain value for ζ .

VIII. CONCLUSION

A mathematical model of the transmission of the Ebola virus has been developed with the help of a SIR model. This investigation considered the characteristics of immunization, therapy, compliance with the medical protocols, and the load of the Ebola virus (ζ). For the sake of this study, the parameters ω, α, θ are considered fuzzy membership functions and denoted as fuzzy parameters. From this model, we observed that the system will be stable when the value of $\mathfrak{R}_0 < 1$, and it becomes unstable when the value of $\mathfrak{R}_0 > 1$. The simulation results indicate that immunization and adherence to health protocols have a significant influence on preventing or not halting the Ebola virus's development. This is the conclusion that can be drawn from the examination findings. Furthermore, treatment does impact slowing or halting the infection rate caused by the Ebola virus; however, this impact is not nearly as significant as the impact of immunization and adherence to health protocols.

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AUTHOR CONTRIBUTIONS

Saravanan Subraja: Conceptualization, Data Curation, Methodology, Validation, Writing – Original Draft Preparation.

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CONFLICT OF INTERESTS

No conflict of interests were disclosed.

ETHICS STATEMENTS

Our publication ethics follow The Committee of Publication Ethics (COPE) guideline.
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REFERENCES

- [1] S.T. Jacob, I. Crozoior, W.A. Fischer et. al, "Ebola virus disease," *Nature Reviews Disease Primers*, vol. 6, no. 13, pp. 1-31, 2020.
DOI: <https://doi.org/10.1038/s41572-020-0147-3>
- [2] M.M. Rooney and M.K. Hinders, "Machine learning for medium access control protocol recognition in

communications networks," *IEEE Access*, vol. 9, pp. 110762–110771, 2021.

- DOI: <http://dx.doi.org/10.1109/ACCESS.2021.3102859>
- [3] U. Das and D. Fielding, "Higher local Ebola incidence causes lower child vaccination rates," *Scientific Reports*, vol. 14, no. 1382, pp. 1-7, 2024.
DOI: <https://doi.org/10.1038/s41598-024-51633-3>
- [4] K. Al-Tameemi and R. Kabakli, "Ebola virus: An overview disease and treatment," *Asian Journal Pharmaceutical and Clinical Research*, vol. 12, no. 10, pp. 57-62, 2019.
DOI: <http://dx.doi.org/10.22159/ajpcr.2019.v12i10.35005>
- [5] H.R. Jonsdottir, D. Zysset, N. Lenz et.al, "Virucidal activity of three standard chemical disinfectants against Ebola virus suspended in tripartite soil and whole blood," *Scientific Reports*, vol. 13, pp. 1-8, 2023.
DOI: <https://doi.org/10.1038/s41598-023-42376-8>
- [6] S.A. Riesle-Sbarbaro, G. Wibbelt, A. Dux et. al, "Selective replication and vertical transmission of Ebola virus in experimentally infected Angolan free-tailed bats," *Nature Communications*, vol. 15, no. 925, pp. 1-15, 2024.
DOI: <https://doi.org/10.1038/s41467-024-45231-0>
- [7] E. Okyere, J.D. Ankamah, A.K. Hunkpe and D. Mensah, "Deterministic epidemic models for Ebola infection with time-dependent controls," *Discrete Dynamics in Nature and Society*, vol. 2020, no. 1, pp. 1-12, 2020.
DOI: <https://doi.org/10.1155/2020/2823816>
- [8] W. Chen, "A mathematical model of Ebola virus based on sir model," *2015 International Conference on Industrial Informatics - Computing Technology, Intelligent Technology, Industrial Information Integration*, pp. 213-216, 2015.
DOI: <https://doi.org/10.1109/ICIIT.2015.135>
- [9] M. Abdy, S. Side, S. Annas, W. Nur and W. Sanusi, "An SIR epidemic model for COVID-19 spread with fuzzy parameters: the case of Indonesia," *Advances in Difference Equations*, vol. 2021, no. 105, pp. 1-17, 2021.
DOI: <https://doi.org/10.1186/s13662-021-03263-6>
- [10] R. Verma, S.P. Tiwari and R.K. Upadhyay, "Dynamical behaviors of fuzzy SIR epidemic model," *Advances in Intelligent Systems and Computing*, vol. 643, Springer, Cham, pp. 482-492, 2018.
DOI: http://dx.doi.org/10.1007/978-3-319-66827-7_45
- [11] P.V.D. Driessche and J. Watmough, "Further notes on the basic reproduction number," *Mathematical Epidemiology: Lecture Notes in Mathematics*, vol. 1945, pp. 159–178, 2008.
DOI: https://doi.org/10.1007/978-3-540-78911-6_6
- [12] M. Mullai, G. Madhan Kumar, G. Rajchakit and G. Vetrivel, "Mathematical modelling on the transmission dynamics of zika virus: transmission dynamics of zika virus," *International Journal on Robotics, Automation and Sciences*, vol. 5, no. 2, pp. 79–84, 2023.
DOI: <https://doi.org/10.33093/ijoras.2023.5.2.9>
- [13] M. Mullai, G. Madhan Kumar, G. Rajchakit and G. Vetrivel, "Transmission dynamics of smoking - a mathematical model: transmission dynamics of smoking," *International Journal on Robotics, Automation and Sciences*, vol. 5, no. 2, pp. 89–93, 2023.
DOI: <https://doi.org/10.33093/ijoras.2023.5.2.11>