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Research Article

Fuzzy SEIR Epidemic Model of Amoebiasis Infection in Human

Saul C. Mpeshe

Department of Mathematical Sciences, University of Iringa, P.O. Box 200 Iringa, Tanzania

Correspondence should be addressed to Saul C. Mpeshe; saul.mpeshe@uoi.ac.tz

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Amoebiasis is a disease caused by the protozoan *Entamoeba histolytica*. It is often asymptomatic but may cause dysentery and invasive extraintestinal infection in humans. To gain an understanding of the disease, a fuzzy SEIR model of *amoebiasis* infection is presented with the fuzziness introduced in the disease transmission rate, the disease-induced death rate, and the rate of recovery from infection. The basic reproduction number $\mathscr{R}_0(v)$ is computed and used to make a comparison with the fuzzy basic reproduction number $\mathscr{R}_0^f$ at different amounts of cysts as well as to analyse the stability of equilibrium points. Stability results indicate that the disease-free equilibrium and the endemic equilibrium are locally and globally asymptotically stable. Further, numerical simulations are carried out to illustrate the analytical results of the fuzzy model system. The result shows that whenever there is an outbreak, the disease is likely to persist in the first month and thereafter it will start to slow down to disease-free equilibrium.

1. Introduction

Amoebiasis is a human infection of the large intestine caused by a protozoan parasite *Entamoeba histolytica* (*E. histolytica*) [1]. It is the most common worldwide cause of mortality from a protozoan after malaria [2–4], causing about 55,000 deaths worldwide in 2010 [5, 6]. *Amoebiasis* has been a major health problem in China, and some parts of Asia, Latin America, and Africa [7].

A brief history of *amoebiasis* leads us to Lambe (or Lambl) in 1859 who reported *amoebae* in the discharge of a patient with dysentery [8]. The name *Entamoeba histolytica* was established in 1903 by Schaudinn due to the parasite ability to cause tissue lysis, and he described it as the causal agent of amoebic dysentery [9].

Transmission of *amoebiasis* occurs through the faecal-oral route, either directly from person-to-person contact or indirectly by eating or drinking faecally contaminated food or water. Sexual transmission by oral-rectal contact is also possible especially among male homosexuals [10–13]. Depending on the immune system of the infected individual and other biological factors which are yet to be known, 90% of the persons infected by *Entamoeba histolytica* develop an asymptomatic *amoebiasis* while 10% of them develop symptoms that identify the *amoebiasis colitis* [14–16].

Due to the resistant character of the cysts of *Entamoeba histolytica*, mature cysts can remain alive for new infection for some weeks in soils, 12 days in a moist and cool environment, and 30 days in water. However, they can stand alive for the temperatures under 50°C and greater than 400°C [14]. The ingestion of one viable cyst may cause an infection [12]. The incubation period varies from a few days to several months or years but is generally 2 to 4 weeks [17, 18].

Modelling approach has played a major role in better understanding the global behaviour of infectious diseases. Modelling of infectious diseases using ordinary or integer-order differential equations has proven to be valuable in analysing the dynamics of various diseases including *amoebiasis* [19, 20]. However, these methods are crisp, deterministic, and precise. Crisp, in this sense, mean dichotomous, that is, yes-or-no-type, or true or false type rather than more-or-less type or anything in between [21]. According to Zimmenmann [21], precision assumes that the parameters of a model represent exactly either our perception of the phenomenon modelled or the features of the

real system that has been modelled and there is no ambiguities.

Many real situations are not crisp and deterministic, and therefore, they cannot be described precisely. The complete description of a real situation will often require more detailed information than a human being could process, and understand. Lack of information and vagueness of the situations may cause the future state of the system not to be known completely [21].

Fuzziness can be found in many areas of daily life, such as in engineering, medicine, meteorology, manufacturing, and others where human judgment, evaluation, reasoning and decision making are important [21–23]. The reasons for such fuzziness is mostly due to our daily communication which uses natural languages, where the meaning of words is very often vague [21, 23]. For example, when we say there is an infection in a certain population, the statement is not necessarily to be true or false. It may be true to some degree, the degree to which an infected person belong to that population. Therefore, the statement is vague and we need to introduce the concept of vagueness to understand the truthness of this statement.

The concept of vagueness is well established through the idea of fuzzy set which involve assigning to each possible individual in the population a value representing its degree of membership. For example, a fuzzy set representing our concept of infection can assign a degree of membership of 1 to a high infection, 0.5 to a medium infection, and 0 to a low infection. Fuzzy sets representing linguistic concepts such as low, medium, and high, are often employed to define states of a variable called a fuzzy variable.

Modelling with fuzzy sets and logic has motivated many researchers in science and social sciences including epidemiology. This is evident from the work by Abdy et al. [24], Li et al. [25], Shi et al. [26], Stiegelmeier and Bressan [27], Shaban et al. [28], Chowdhury et al. [29], and Bhuju et al. [30], just to mention a few. Motivated by their work, a fuzzy model of *amoebiasis* infection is presented in this paper. The paper begins with the definition of some fuzzy concepts followed by the model formulation of a model and a description of some fuzzy parameters used in the model. The feasibility solution, basic reproduction number, fuzzy reproduction number and global stability analysis of the model are also discussed. Numerical simulations of the model are also established to study the behaviour of the disease over a certain time period.

2. The Fuzzy Model of Amoebiasis

2.1. Definitions

Definition 1 (see [21]). A fuzzy set A *in* X is a set of ordered pairs:

$$A = \{(x, \mu_A(x)) | x \in X\}, \tag{1}$$

where X is the universe of discourse, $\mu_A(X)$ is called the membership function or grade of membership (also degree of compatibility or degree of truth) of x in A that maps X to the membership space $M = [0, 1]$, A is nonfuzzy and $\mu_A(X)$ is the memmbership function.

Definition 2 (see [21]). A membership function $\mu_A(X)$ is a mapping from the universe X to the interval $[0, 1]$ denoted by μ: $(X) \longrightarrow [0, 1]$

Definition 3 (see [31]). Let u be a fuzzy subset. The fuzzy integral, or fuzzy expected value of u, FEV$[u]$, is defined by

$$\text{FEV}[u] = \sup_{0 \le \alpha \le 1} \inf\{\alpha, \mu\{u \ge \alpha\}\}, \tag{2}$$

where $\{u \ge \alpha\} = \{x \in \mathbb{R}: u(x) \ge \alpha\}$.

The value of FEV$[u]$ is a real number and it provides a good estimate of mathematics expectancy of the random variable u. It is also a good estimate for the classical expectancy $E(u)$ when the fuzzy subset u is also a random variable [31].

Definition 4 (see [32]). A linguistic variable is a quintuple $(x, T(x), U, G, M)$ in which x is the name of the variable; $T(x)$ is the set of names of linguistic values of x, with each value being a fuzzy variable denoted generically by X and ranging over a universe of discourse U that is associated with the base variable u; G is a syntactic rule for generating the name, X, of values of x; and M is a semantic rule for associating with each X its meaning, $M(X)$, which is a fuzzy subset of U.

2.2. Model Formulation. The model consider a human population subdivided into four compartments namely: susceptible (S), exposed (E), infected (I), and recovered (R). The total human population (N) is therefore given by $N = S + E + I + R$. It is assumed that individuals who have recovered from *amoebiasis* will experience a temporal immunity induced by the disease. The transmission rate (β), recovery rate (γ), and the disease-induced rate (d) are considered to be fuzzy parameters. The parameters and their description as they have been used in the model are given in Table 1. The mode of transmission of *amoebiasis* is presented by Figure 1 with the parameters used in this model described in Table 1.Using the parameters described in Table 1 and the transmission diagram in Figure 1, an SEIR model is formulated using ordinary differential equations as follows:

$$\frac{dS}{dt} = \Lambda - \mu S - \beta IS, \tag{3a}$$

$$\frac{dE}{dt} = \beta IS - (\varepsilon + \mu)E, \tag{3b}$$

$$\frac{dI}{dt} = \varepsilon E - (\mu + d + \gamma)I, \tag{3c}$$

$$\frac{dR}{dt} = \gamma I - \mu R, \tag{3d}$$

where β, γ, and d are fuzzy parameters.

Table 1: Description of parameters used in the model.

Parameter	Description
Λ	Recruitment rate of human
λ	Transmission rate in human
ε	Infectious rate
μ	Natural death rate of human
d	Disease-induced death rate in human
γ	Recovery rate in human

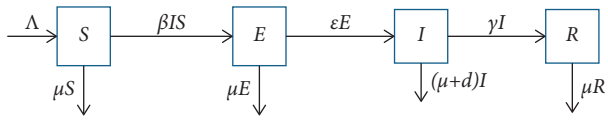

Figure 1: Transmission diagram for Amoebiasis.

2.2.1. Membership Function for β. The transmission rate β depends on the amount of cysts v present in the environment. Using Barros et al. [31, 33] we define the membership function for β as

$$\beta(v) = \begin{cases} 0, & \text{if } v > v_{\min}, \\ \dfrac{v - v_{\min}}{v_m - v_{\min}}, & \text{if } v_{\min} \le v \le v_m, \\ 1, & \text{if } v_m \le v \le v_{\max}, \end{cases} \tag{4}$$

where $v_{\min}$ is the minimum amount of cysts needed for the disease transmission to occur, v_m is the amount of cysts where the transmission of the disease is maximum and equal to 1.

2.2.2. Membership Function for γ. The recovery rate γ depends on the amount of cysts ingested by a human. When the amount of cysts is high it will take a longer time to recover from the disease. Using Verma et al. [34] we define the membership of γ as

$$\gamma(v) = \frac{\gamma_0 - 1}{v_{\max}} v + 1 \quad \text{if } 0 \le v \le v_{\max}, \tag{5}$$

where $0 < \gamma_0 < 1$ is the lowest recovery rate.

2.2.3. Membership Function for Induced-Death Rate d. The disease-induced death rate occurs due to infection of the disease. When the disease transmission is negligible for a lower amount of cysts ingested, there is only natural death because there is no death due to infection. When the amount of cysts is high $v_m < V$ the death will also be high. We, therefore, define the membership of d according to Verma et al. [35] as

$$d(v) = \begin{cases} \dfrac{(1 - d_0)}{v_m} v + d_0, & \text{if } 0 \le v \le v_m, \\ 1, & \text{if } v_m < v, \end{cases} \tag{6}$$

where $0 < d_0 < 1$ is the lowest death rate.

2.2.4. The Amount of Cysts Γ Membership Function. The amount of cysts in the environment is assumed to be different in each population considered which can be seen as a linguistic variable classified as weak, medium and strong. The membership function for the amount of cysts is defined as in Barros et al. [31] as

$$\Gamma(v) = \begin{cases} 0, & \text{if } v < \overline{v} - d, \\ \dfrac{v - \overline{v} + \delta}{\delta}, & \text{if } \overline{v} - d \le v \le \overline{v}, \\ \dfrac{-(v - \overline{v} - \delta)}{\delta}, & \text{if } \overline{v} < v \le \overline{v} + d, \\ 1, & \text{if } v > \overline{v} + d, \end{cases} \tag{7}$$

where $\overline{v}$ is the central value and δ is the dispersion of cyst charge in the population.

2.3. Feasibility of the Model Solution. The feasible region is the positive region $\mathbb{R}^4_+$. Thus, we show that $dX_i/dt \ge 0$ in the region $\mathbb{R}^4_+$. From the model system (3) we see that

$$\frac{dS}{dt}\Big|_{S=0} = \Lambda > 0, \tag{8a}$$

$$\frac{dE}{dt}\Big|_{E=0} = \beta IS \ge 0, \tag{8b}$$

$$\frac{dI}{dt}\Big|_{I=0} = \varepsilon E \ge 0, \tag{8c}$$

$$\frac{dR}{dt}\Big|_{R=0} = \gamma I \ge 0. \tag{8d}$$

Thus, the solution is feasible and positive in the region $\Omega = (S, E, I, R) \ge 0 \in \mathbb{R}^4_+$.

Furthermore,

$$\begin{aligned} \frac{dN}{dt} &= \Lambda - \mu N - dI \ , \\ &\le \Lambda - \mu N. \end{aligned} \tag{9}$$

Solving this differential inequality gives

$$N(t) \le N(0)e^{-\mu t} + \frac{\Lambda}{\mu}\left(1 - e^{-\mu t}\right). \tag{10}$$

As $t \longrightarrow \infty$, we have

$$N(t) \le \frac{\Lambda}{\mu}. \tag{11}$$

Thus, the model solution is bounded and positively invariant in $\mathbb{R}^4_+$.

2.4. Equilibrium Points. The disease-free equilibrium and endemic equilibrium are determined by setting the left-hand side of the model system (2.2) equal to zero. Setting

$E = I = R = 0$ we obtain the disease-free equilibrium of the model as

$$P_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right). \tag{12}$$

For the case $E \neq 0$, $I \neq 0$, $R \neq = 0$, we have the endemic equilibrium $P^* = (S^*, E^*, I^*, R^*)$ where

$$S^* = \frac{(\varepsilon + \mu)(\mu + d + \gamma)}{\beta\varepsilon}, \tag{13a}$$

$$E^* = \frac{\beta\varepsilon\Lambda - \mu(\varepsilon + \mu)(\mu + d + \gamma)}{\beta\varepsilon(\varepsilon + \mu)}, \tag{13b}$$

$$I^* = \frac{\beta\varepsilon\Lambda - \mu(\varepsilon + \mu)(\mu + d + \gamma)}{\beta(\varepsilon + \mu)(\mu + d + \gamma)}, \tag{13c}$$

$$R^* = \frac{\gamma}{\mu} I^* \tag{13d}$$

2.5. The Basic Reproduction Number. For P^* to exist in the feasible region, the condition $0 < S^* < \Lambda/\mu$ or equivalently $\Lambda/\mu 1/S^* \geq 1$ is sufficiently necessary [36]. Now define the basic reproduction number $(\mathcal{R}_0)$ by

$$\mathcal{R}_0 = \frac{\Lambda}{\mu}\frac{1}{S^*}. \tag{14}$$

Then,

$$\mathcal{R}_0 = \frac{\Lambda}{\mu}\frac{\beta\varepsilon}{(\varepsilon + \mu)(\mu + d + \gamma)}. \tag{15}$$

Notice that $\mathcal{R}_0$ consists of β, γ and d which are both functions of the amount of cysts v, hence $\mathcal{R}_0$ is also a function of v and can be denoted as $\mathcal{R}_0(v)$. However, $\mathcal{R}_0(v)$ is not a fuzzy set for its value can be greater than one (1). As more individuals recover from the infection, $\mathcal{R}_0(v)$ is reduced, and hence $0 \leq \gamma_0\mathcal{R}_0(v) \leq 1$ for $\gamma_0 \in (0, 1)$. Therefore, $\gamma_0\mathcal{R}_0(v)$ is fuzzy set and the fuzzy expected value $FEV[\gamma_0\mathcal{R}_0(v)]$ is well defined. The fuzzy basic reproduction number $\mathcal{R}_0^f$ is therefore defined as

$$\mathcal{R}_0^f = \frac{1}{\gamma_0}\mathrm{FEV}[\gamma_0\mathcal{R}_0(v)]. \tag{16}$$

Define

$$\mathrm{FEV}[\gamma_0\mathcal{R}_0(v)] = \sup_{0\leq\alpha\leq 1}\inf[\alpha, k(\alpha)], \tag{17}$$

where $k(\alpha) = \mu[v\colon \gamma_0\mathcal{R}_0(v) \geq \alpha] = \mu(X)$ is a fuzzy measure. Then $\mu(X) = \sup_{v\in X}\Gamma(v)$ and $X \in [v^*, v_m]$ where v^* is the solution to the equation

$$\gamma_0\mathcal{R}_0(v) = \alpha. \tag{18}$$

Thus,

$$k(\alpha) = \mu[v^*, v_m] = \sup_{v^*\leq v\leq v_m}\Gamma(v), \tag{19}$$

where $k(0) = 1$ and $k(1) = \Gamma(v_m)$.

2.6. The Impact of Virus Load on $\mathcal{R}_0^f$. We analyse the impact of v on $\mathcal{R}_0^f$ based on linguistic interpretation of the amount of cysts on whether there is weak, medium or strong amount of cysts to cause infection.

Case 1. Weak Amount of Cysts. $\overline{v} - \delta \leq v_{\min}$ If $\overline{v} - \delta \leq v_{\min}$, then $\beta(v) = 0$ and $\mathcal{R}_0 = 0$. So $\mathrm{FEV}[\gamma_0\mathcal{R}_0(v)] = 0 < \gamma_0$ and hence $\mathcal{R}_0^f < 1$. Therefore, the disease will die out.

Case 2. Medium Amount of Cysts. $\overline{v} - \delta \geq v_{\min}$ and $\overline{v} + \delta \leq v_m$ In this case, then we have $\beta(v) = v - v_{\min}/v_m - v_{\min}$ and $\mathcal{R}_0(v) = \beta(v)\varepsilon\Lambda/\mu(\varepsilon + \mu)(\mu + d(v) + \gamma(v))$. The fuzzy measure $k(\alpha)$ is then given by

$$k(\alpha) = \begin{cases} 1, & \text{if } 0 < \alpha \leq \gamma_0\mathcal{R}_0(\overline{v}), \\ \Gamma(v^*), & \text{if } \gamma_0\mathcal{R}_0(\overline{v}) < \alpha \leq \gamma_0\mathcal{R}_0(\overline{v} + \delta), \\ 0, & \text{if } \gamma_0\mathcal{R}_0(\overline{v} + \delta) < \alpha \leq 1. \end{cases} \tag{20}$$

Thus, if $\delta > 0$, then $k(\alpha)$ is a continuous decreasing function with $k(0) = 1$ and $k(1) = 9$. Hence, $\mathrm{FEV}[\gamma_0\mathcal{R}_0(v)]$ is a fixed point of k and

$$\gamma_0\mathcal{R}_0(\overline{v}) \leq \mathrm{FEV}[\gamma_0\mathcal{R}_0(v)] \leq \gamma_0\mathcal{R}_0(\overline{v} + \delta). \tag{21}$$

It follows therefore

$$\mathcal{R}_0(\overline{v}) \leq \frac{1}{\gamma_0}\mathrm{FEV}[\gamma_0\mathcal{R}_0(v)] \leq \mathcal{R}_0(\overline{v} + \delta), \tag{22}$$

and that

$$\mathcal{R}_0(\overline{v}) \leq \mathcal{R}_0^f \leq \mathcal{R}_0(\overline{v} + \delta). \tag{23}$$

Since $\mathcal{R}_0(\overline{v})$ is also a continuous increasing function, there exist v with $\overline{v} < v < \overline{v} + \delta$ such that $\mathcal{R}_0^f = \mathcal{R}_0(v) > \mathcal{R}_0(\overline{v})$. That is $\mathcal{R}_0^f$ and $\mathcal{R}_0(v)$ coincide and $\mathcal{R}_0^f > \mathcal{R}_0(\overline{v})$. Therefore, the fuzzy average number of secondary infection $\mathcal{R}_0^f$ is higher than the number of infection $\mathcal{R}_0(\overline{v})$ due to the medium amount of cysts.

Case 3. Strong Amount of Cysts. $\overline{v} - \delta \leq v_m$ and $\overline{v} + \delta \leq v_{\max}$ In this case $\beta(v) = 1$ and $\mathcal{R}_0(v) = \varepsilon\Lambda/\mu(\varepsilon + \mu)(\mu + d(v) + \gamma(v))$. The fuzzy measure $k(\alpha)$ is then given by

$$k(\alpha) = \begin{cases} 1, & \text{if } 0 < \alpha \leq \gamma_0\mathcal{R}_0(\overline{v}), \\ \Gamma(v^*), & \text{if } \gamma_0\mathcal{R}_0(\overline{v}) < \alpha \leq \gamma_0\mathcal{R}_0(\overline{v} + \delta), \\ 0, & \text{if } \gamma_0\mathcal{R}_0(\overline{v} + \delta) < \alpha \leq 1. \end{cases} \tag{24}$$

It follows that

$$\gamma_0\mathcal{R}_0(\overline{v}) \leq \mathrm{FEV}[\gamma_0\mathcal{R}_0(v)] \leq \gamma_0\mathcal{R}_0(\overline{v} + \delta), \tag{25}$$

or

$$\mathcal{R}_0(\overline{v}) \leq \mathcal{R}_0^f \leq \mathcal{R}_0(\overline{v} + \delta). \tag{26}$$

Thus, $\mathcal{R}_0^f > 1$ and the disease will persist.

3. Stability Analysis of Equilibrium Points

3.1. Local Stability of the Disease-Free Equilibrium

Theorem 5. *The disease-free equilibrium of the fuzzy amoebiasis model (3) is locally asymptotically stable if $\mathscr{R}_0 < 1$ and unstable if $\mathscr{R}_0 > 1$.*

Proof. We need to show that the Jacobian matrix $J(P_0)$ of the fuzzy model (3) evaluated at the disease-free equilibrium has negative eigenvalues. Further computation shows that $J(P_0)$ of the model (3) at P_0 is

$$J(P_0) = \begin{bmatrix} -\mu & 0 & -\beta S^* & 0 \\ 0 & -(\varepsilon+\mu) & \beta S^* & 0 \\ 0 & \varepsilon & -(\mu+d+\gamma) & 0 \\ 0 & 0 & \gamma & -\mu \end{bmatrix}. \tag{27}$$

From the matrix $J(P_0)$, the diagonal entry $-\mu$ is one of the eigenvalues of $J(P_0)$. Eliminating the column containing $-\mu$ and its corresponding row, we remain with a 2×2 matrix

$$J^*(P_0) = \begin{bmatrix} -(\varepsilon+\mu) & \beta S^* \\ \varepsilon & -(\mu+d+\gamma) \end{bmatrix}. \tag{28}$$

The characteristic equation of $J^*(P_0)$ is

$$\lambda^2 + A\lambda + B = 0, \tag{29}$$

where
$A = (\varepsilon+\mu+\mu+d+\gamma), B = (\varepsilon+\mu)(\mu+d+\gamma)(1-\mathscr{R}_0)$.
Since at disease-free equilibrium $\mathscr{R}_0 < 1$, then B is positive. Using Routh-Hurwitz criteria we conclude that the roots of the quadratic equation are negative real numbers or complex numbers with real parts. Hence, $J(P_0)$ has negative eigenvalues, and thus, the disease-free equilibrium is locally asymptotically stable. □

3.2. Global Stability of the Disease-Free Equilibrium

Definition 6 (Lyapunov function [37]). Let $\mathbf{x} = 0$ be an equilibrium point of $\dot{x}(t) = f(x(t))$. A continuously differentiable function $V(x)$ is a *Lyapunov function* if the following conditions hold:

A1: $V(0) = 0$

A2: $V(x) > 0,\ x \in N\backslash\{0\}$

A3: $\dot{V}(x) < 0, x \in N\backslash\{0\}$

Here $N\backslash\{0\} \in \mathbb{R}^n$ is the neighbourhood of 0 excluding the origin itself.

Theorem 7. *The disease-free equilibrium of the fuzzy amoebiasis model (3) is globally asymptotically stable if $\mathscr{R}_0 < 1$ and unstable if $\mathscr{R}_0 > 1$.*

Proof. Here we investigate the global stability of the disease-free equilibrium by mean of Lyapunov direct method. In this method, we define a positive definite Lyapunov function $V = \omega_1 E + \omega_2 I$. Clearly,

$$\begin{aligned} \frac{dV}{dt} &= \omega_1 \frac{dE}{dt} + \omega_2 \frac{dI}{dt}, \\ &= \omega_1 [\beta IS - (\varepsilon+\mu)E] + \omega_2 [\varepsilon E - (\mu+d+\gamma)I] \\ &\le \omega_1 [\beta IS^* - (\varepsilon+\mu)E] + \omega_2 [\varepsilon E - (\mu+d+\gamma)I]. \end{aligned} \tag{30}$$

If we choose $\omega_1 = \varepsilon$ and $\omega_2 = (\varepsilon+\mu)$, we have

$$\begin{aligned} \frac{dV}{dt} &\le \varepsilon\beta \mathrm{IS}^* - (\varepsilon+\mu)(\mu+d+\gamma)I \\ &\le \left(\frac{\varepsilon\beta S^*}{(\varepsilon+\mu)(\mu+d+\gamma)} - 1 \right)(\varepsilon+\mu)(\mu+d+\gamma)I \\ &\le (\mathscr{R}_0 - 1)(\varepsilon+\mu)(\mu+d+\gamma)I. \end{aligned} \tag{31}$$

Since at disease-free equilibrium $\mathscr{R}_0 < 1$, then $dV/dt \le 0$ and hence, the disease-free equilibrium is globally asymptotically stable. □

3.3. Bifurcation and Fuzzy $\mathscr{R}_0$.

The stability of disease-free equilibrium changes from stable to unstable as $\mathscr{R}_0$ increases through 1. When $\mathscr{R}_0 = 1$, the system obtain a bifurcation at the disease-free equilibrium. Since $\mathscr{R}_0$ depends on the viral load, v, we need to find the value v^* such that $\mathscr{R}_0(v^*) = 1$ and that value is the bifurcation point.

From (15), we have

$$\mathscr{R}_0(v^*) = \frac{\beta(v^*)\varepsilon\Lambda}{\mu(\varepsilon+\mu)(\mu+d(v^*)+\gamma(v^*))}. \tag{32}$$

Also, from (4)–(6), we have

$$\beta(v^*) = \frac{v^* - v_{\min}}{v_m - v_{\min}}, \text{ if } v_{\min} \le v^* \le v_m, \tag{33a}$$

$$\gamma(v^*) = \frac{\gamma_0 - 1}{v_{\max}} v^* + 1, \text{ if } 0 \le v^* \le v_{\max}, \tag{33b}$$

$$d(v^*) = \frac{(1-d_0)}{v_m} v^* + d_0, \text{ if } 0 \le v^* \le v_m. \tag{33c}$$

Using (32) and (29), we can solve for v^* at $\mathscr{R}_0(v^*) = 1$. Further computation shows that v^* exist and is given by

$$v^* = \frac{v_m v_{\max}[\varepsilon\Lambda v_{\min} + \mu(\varepsilon+\mu)(v_m - v_{\min})(\mu+d_0+1)]}{\varepsilon\Lambda v_m v_{\max} + \mu(\varepsilon+\mu)(v_m - v_{\min})[(d_0-1)v_{\max} + (1-\gamma_0)v_m]}. \tag{34}$$

Since there is only one endemic equilibrium, the model exhibit forward bifurcation as shown in Figure 2.

3.4. Global Stability of the Endemic Equilibrium. The local stability of the disease-free equilibrium implies local stability of the endemic equilibrium for the reverse condition [38]. Therefore, we only establish the global stability of the endemic equilibrium.

Theorem 8. *The endemic equilibrium of the fuzzy amoebiasis model (3) is globally asymptotically stable if* $\mathscr{R}_0 > 1$.

Proof. We prove the global stability of the endemic equilibrium via construction of a suitable Lyapunov function of the form

$$V(x_i) = \sum \omega_i \left(x_i - x_i^* - x_i^* \ln \frac{x_i}{x_i^*} \right), \tag{35}$$

where $\omega_i > 0$ is constant to be chosen, x_i is the population of the i^{th} compartment, and x_i^* is the equilibrium point [39].

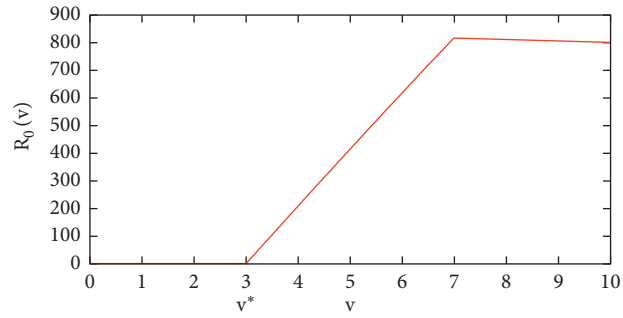

FIGURE 2: Variation of $\mathscr{R}_0(v)$ with $\beta(v)$ and $\gamma(v)$.

Note that the equation containing R in the model system (3) is independent of the other three equations. Therefore, it can be removed from the analysis since the value of R can be obtained when S, E, and I are known. For that case, we now, consider the Lyapunov function

$$V = \omega_1 \left(S - S^* - S^* \ln \frac{S}{S^*} \right) + \omega_2 \left(E - E^* - E^* \ln \frac{E}{E^*} \right) + \omega_3 \left(I - I^* - I^* \ln \frac{I}{I^*} \right). \tag{36}$$

It is easily seen that $V(S, E, I) \geq 0$ and $V(S^*, E^*, I^*) = 0$. The time derivative of V is then given by

$$\frac{dV}{dt} = \omega_1 \left(1 - \frac{S^*}{S} \right) \frac{dS}{dt} + \omega_2 \left(1 - \frac{E^*}{E} \right) \frac{dE}{dt} + \omega_3 \left(1 - \frac{I^*}{I} \right) \frac{dI}{dt}. \tag{37}$$

From the model system (3) we have

$$\begin{aligned} \frac{dV}{dt} = {} & \omega_1 \left(1 - \frac{S^*}{S} \right) [\Lambda - \mu S - \beta SI] + \omega_2 \left(1 - \frac{E^*}{E} \right) [\beta SI - (\varepsilon + \mu) E] \\ & + \omega_3 \left(1 - \frac{I^*}{I} \right) [\varepsilon E - (\mu + d + \gamma) I]. \end{aligned} \tag{38}$$

At endemic equilibrium, $\Lambda = \mu S^* + \beta S^* I^*$, $(\varepsilon + \mu) = \beta S^* I^* / E^*$, and $(\mu + d + \gamma) = \varepsilon E^* / I^*$. Therefore,

$$\frac{dV}{dt} = \omega_1 \left(1 - \frac{S^*}{S} \right) [\mu S^* + \beta S^* I^* - \mu S - \beta SI] + \omega_2 \left(1 - \frac{E^*}{E} \right) \left[\beta SI - \frac{\beta S^* I^*}{E^*} E \right] + \omega_3 \left(1 - \frac{I^*}{I} \right) \left[\varepsilon E - \frac{\varepsilon E^*}{I^*} I \right]. \tag{39}$$

Further simplification gives,

$$\frac{dV}{dt} = -\omega_1 \mu S \left(1 - \frac{S^*}{S} \right)^2 + \omega_1 \left(1 - \frac{S^*}{S} \right) \left(1 - \frac{SI}{S^* I^*} \right) \beta S^* I^* + \omega_2 \left(1 - \frac{E^*}{E} \right) \left(\frac{SI}{S^* I^*} - \frac{E}{E^*} \right) \beta S^* I^* + \omega_3 \left(1 - \frac{I^*}{I} \right) \left(\frac{E}{E^*} - \frac{I}{I^*} \right) \varepsilon E^*, \tag{40}$$

Table 2: Parameters and their description.

Parameter	Description	Value	Source
Λ	Recruitment rate of human	0.0361	[41]
λ	Transmission rate in human	0.00045	Estimated
ε	Infectious rate	1/14	[17, 18].
μ	Natural death rate of human	1/65/365	[41]
d	Disease-induced death rate in human	0.001	Estimated
γ	Recovery rate in human	1/14	[40]

On expanding, we have

$$\frac{dV}{dt} = -\omega_1\mu S\left(1-\frac{S^*}{S}\right)^2 + \omega_1\left(1-\frac{S^*}{S}+\frac{I}{I^*}-\frac{SI}{S^*I^*}\right)\beta S^*I^* + \omega_2\left(1-\frac{E^*}{E}+\frac{SI}{S^*I^*}-\frac{SIE^*}{S^*I^*E}\right)\beta S^*I^* + \omega_3\left(1-\frac{I^*}{I}+\frac{E}{E^*}-\frac{I^*E}{IE^*}\right)\varepsilon E^*, \tag{41}$$

If we choose $\omega_2 = \omega_1, \omega_3 = \omega_1\beta S^*I^*/\varepsilon E^*$, we obtain

$$\begin{aligned}\frac{dV}{dt} &= -\omega_1\mu S\left(1-\frac{S^*}{S}\right)^2 + \omega_1\left(1-\frac{S^*}{S}+\frac{I}{I^*}-\frac{SI}{S^*I^*}\right)\beta S^*I^* \\ &\quad + \omega_1\left(1-\frac{E^*}{E}+\frac{SI}{S^*I^*}-\frac{SIE^*}{S^*I^*E}\right)\beta S^*I^* + \omega_1\left(1-\frac{I^*}{I}+\frac{E}{E^*}-\frac{I^*E}{IE^*}\right)\beta S^*I^*.\end{aligned} \tag{42}$$

Further simplification gives

$$\frac{dV}{dt} = -\omega_1\mu S\left(1-\frac{S^*}{S}\right)^2 + \omega_1\left(3-\frac{S^*}{S}-\frac{I^*E}{IE^*}-\frac{SIE^*}{S^*I^*E}\right)\beta S^*I^*. \tag{43}$$

From the property of arithmetic mean, we have $3 - S^*/S - I^*E/IE^* - SIE^*/S^*I^*E \le 0$, and hence

$$\frac{dV}{dt} = -\omega_1\mu S\left(1-\frac{S^*}{S}\right)^2 + \omega_1\left(3-\frac{S^*}{S}-\frac{I^*E}{IE^*}-\frac{SIE^*}{S^*I^*E}\right)\beta S^*I^* \le 0. \tag{44}$$

Following the LaSalle's invariant principle, it is concluded that the endemic equilibrium is globally asymptotically stable. □

4. Numerical Results and Discussion

In this section, we perform numerical simulation for the fuzzy model system 2.2 to study the persistence of the disease when an outbreak occur. The results of numerical simulations are presented in the form of figures followed by discussion. Where necessary, numerical values are provided.

In numerical simulations, the initial values used to perform simulations are $S = 1000, E = 10, I = 1, R = 1$. Since ingestion of one viable cyst can cause infection then V is taken from 1 to 10. The incubation period is general 2 to 4 weeks [17, 18], but for simulation, we use 2 weeks and hence, the infection rate $\varepsilon = 1/14$. The recovery period for *amebiasis* depends on the severity of the disease. However, the recovery period after medical treatment varies from about 1 to 2 weeks or more [40]. For simulation, we use 2 weeks, and therefore, $\gamma = 1/14$. The parameter values and their source are as given in Table 2.

Figure 3 shows the variations of human subpopulation with time. In Figure 3(a), the parameters β, γ and d are crisp, they do not depend on amount of cysts v present in the environment. In Figure 3(b), the parameters β, γ and d are fuzzy, they depend on amount of cysts v present in the environment. The graphs in Figure 3(a) are the S-curves of the classical SEIR model while the graphs in Figure 3(b) are log-normal curves. Both graphs confirm the theoretical result presented from the stability analysis of the model. That is, *amoebiasis* infection is persistence if $\mathcal{R}_0 > 1$ and it dies

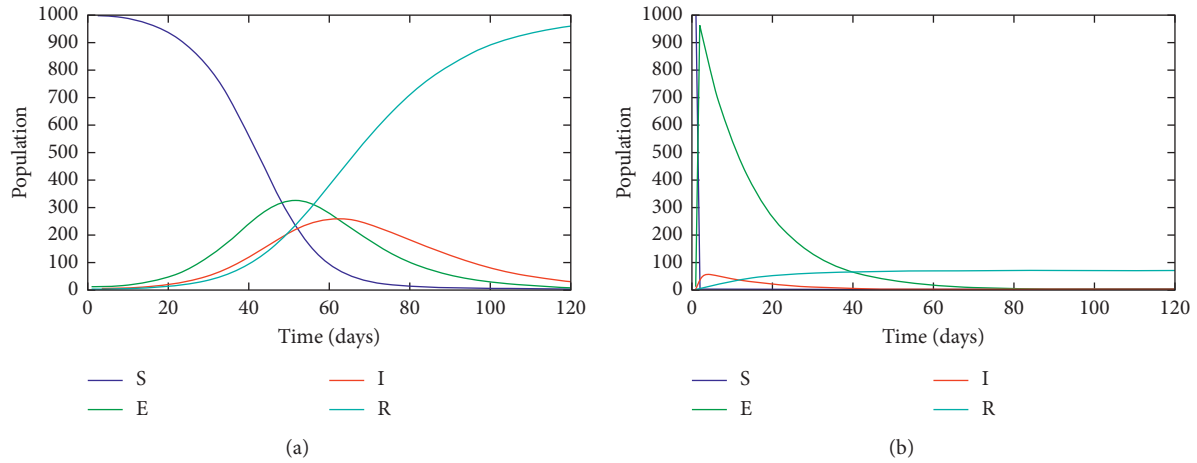

Figure 3: Variations of human subpopulation with time. (a) Crisp β, γ and d. (b) Fuzzy β, γ and d.

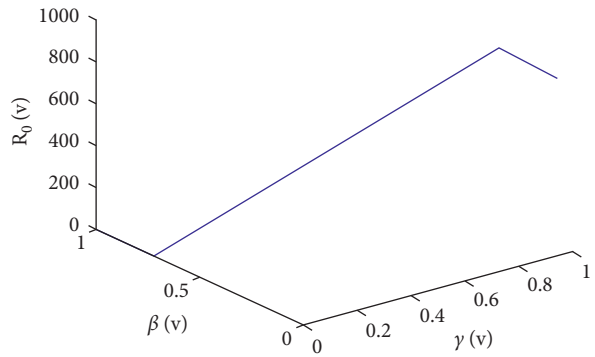

Figure 4: Variation of $\mathscr{R}_0(v)$ with $\beta(v)$ and $\gamma(v)$.

away when $\mathscr{R}_0 < 1$. Thus, applying control measures that reduces $\mathscr{R}_0$ may to manage the transmission of *amoebiasis*.

Figure 4 shows the variations of $\mathscr{R}_0(v)$ with $\beta(v)$ and $\gamma(v)$. When v increases it increase $\beta(v)$ while reducing $\gamma(v)$ which in turn reduces recovery rate and increase $\mathscr{R}_0(v)$. Since $\beta(v)$ and $\gamma(v)$ depends on v, that is, the amount of cyst v ingested by a human, personal protection and hygiene to reduce the amount of cyst ingested is crucial.

Figure 2 shows the variations of $\mathscr{R}_0(v)$ with v. When v increases from 0 to 3, $\mathscr{R}_0(v)$ remains constant, that is, $\mathscr{R}_0(v) = 1$. For $v > 3$, $\mathscr{R}_0(v)$ is greater 1 showing that there is a point of bifurcation $vv^* = 3$ where the equilibrium shift from disease-free equilibrium to endemic equilibrium.

5. Conclusion

Amoebiasis will remain a potentially transmissible protozoan infection worldwide nature of transmission. A fuzzy modelling approach has been used to investigate the dynamics of *amoebiasis* infection in humans. The basic reproduction number $\mathscr{R}_0$ was computed and used to make a comparison with the fuzzy basic reproduction number $\mathscr{R}_0^f$, and study the stability of the disease-free equilibrium and endemic equilibrium. The stability analysis of equilibrium points indicated that both the disease-free equilibrium and endemic equilibrium of the model system are locally and globally asymptotically stable. The stability of the disease-free equilibrium implies that the *amoebiasis* infection can be controlled provided that $\mathscr{R}_0 < 1$. Further analysis indicates that the fuzzy parameters β, γ and d depends on the amount of cyst v ingested by a human. The more cysts ingested the higher the transmission rate and the disease-induced rate, thus, making the recovery to be slow. Effective control mechanisms such as personal prevention and hygiene may help to reduce the number of cysts ingested and hence reduce disease transmission. These results call for a study on the optimal control mechanism to look for the best control strategy for the disease.

Data Availability

No data were used to support this study.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this manuscript.

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