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Research Article

The Mathematical Modelling of Crimean Congo Haemorrhagic Fever Disease: An Approach Based on SIRD Model

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Abstract

In this study, the transmission dynamics of CCHF were investigated through a Susceptible-Infected-Recovered-Dead (SIRD) model formulated as a system of ordinary differential equations and calibrated using epidemiological data collected from Sivas Cumhuriyet University Research Hospital between 2010 and 2024. The model parameters, including the transmission rate β, recovery rate γ, disease-induced mortality rate α and natural mortality rate μ, were estimated from real data. The basic reproductive number (R_0), which is used to determine the potential for an outbreak of disease, was derived using the next generation matrix method. Local stability analyses of disease-free and endemic equilibrium points were performed by examining the eigenvalues of the jacobian matrix using the Routh-Hurwitz criterion. The assessment of global stability was conducted by the construction of an appropriate Lyapunov function. Sensitivity analyses were conducted to ascertain the impact of parameter variation on R_0. Numerical simulations implemented in MATLAB demonstrated the temporal evolution of the epidemic under varying initial conditions and parameter sets. The inclusion of the mortality parameter in the SIR model made the model more realistic and provided realistic results.

Keywords: crimean-congo hemorrhagic fever; sırd model; basic reproduction number (R_0); stability analysis; epidemiology.

Kırım Kongo Kanamalı Ateşi Hastalığının Matematiksel Modellenmesi: SIRD Modeline Dayalı Bir Yaklaşım

Özet

Bu çalışmada, KKKA'nın bulaşma dinamikleri, adi diferansiyel denklemler sistemi olarak formüle edilen ve 2010-2024 yılları arasında Sivas Cumhuriyet Üniversitesi Araştırma Hastanesi'nden toplanan epidemiyolojik veriler kullanılarak, bir Duyarlı-Enfekte-İyileşmiş-Ölü (SIRD) modeli aracılığıyla incelenmiştir. Bulaşma oranı β, iyileşme oranı γ, hastalık kaynaklı ölüm oranı α ve doğal ölüm oranı μ dahil olmak üzere model parametreleri gerçek verilerden tahmin edilmiştir. Hastalık salgını potansiyeli için bir eşik parametresi olarak işlev gören temel üreme sayısı (R_0), yeni nesil matris yöntemi kullanılarak türetilmiştir. Hastalıksız ve endemik denge noktalarının yerel kararlılık analizleri, jacobian matrisinin öz değerlerinin incelenmesi Routh-Hurwitz kriteri yardımıyla yapılmıştır Global kararlılık uygun bir Lyapunov fonksiyonunun oluşturulmasıyla değerlendirilmiştir. Parametre değişiminin R_0 üzerindeki etkisini ölçmek için duyarlılık analizleri yapılmıştır. MATLAB'da uygulanan sayısal simülasyonlar, değişen başlangıç koşulları ve parametre setleri altında salgının zamansal evrimini göstermiştir. Ölüm parametrisinin SIR modeline dahil edilmesi, modeli daha gerçekçi kılmış ve gerçekçi sonuçlar elde edilmesini sağlamıştır.

Anahtar Kelimeler: kırım kongo kanamalı ateşi hastalığı, sırd model, temel üreme sayısı, kararlılık analizi, epidemiyoloji.

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Introduction

Crimean-Congo haemorrhagic fever (CCHF) is an acute tick-borne viral zoonosis caused by a Nairovirus from the Bunyaviridae family. It is characterised by a sudden onset of high fever, muscle pain, dizziness and, in severe cases, haemorrhagic symptoms, shock and multi-organ failure . With a reported case fatality rate ranging from 10% to 40% — and even higher in some outbreaks — CCHF poses a significant threat to public health, particularly in endemic regions such as Turkey, the Balkans, the Middle East, and parts of Africa and Asia. Turkey has reported consistent seasonal outbreaks since the early 2000s, with the majority of cases concentrated in the Central Anatolian and Black Sea regions [1,2,3,4,5].

Transmission primarily occurs through the bite of infected Hyalomma ticks, which act as vectors and reservoirs for the virus. However, secondary human-to-human transmission via contact with infected blood or bodily fluids is also well documented, posing a serious occupational risk to healthcare personnel, slaughterhouse workers, and farmers. Furthermore, climatic and environmental factors such as temperature, humidity and livestock movement patterns can significantly impact tick activity and virus circulation [6,7].

Due to the complex nature of CCHF transmission dynamics, which encompasses both zoonotic and human-to-human pathways, quantitative modelling approaches are crucial for understanding disease spread and designing effective interventions [8,9,10]. Mathematical modelling provides a robust analytical framework for estimating key epidemiological parameters, projecting outbreak trajectories and evaluating control strategies [11,12,13]. In this context, the field of mathematical epidemiology has become increasingly important, particularly with the use of compartmental models such as SIR and SIRD, which simplify the complex processes of infection, recovery and mortality into dynamic systems. Compartmental models based on systems of ordinary differential equations (ODEs) are particularly useful for capturing the temporal dynamics of infectious diseases. The Susceptible-Infected-Recovered-Dead (SIRD) model is one such model, offering a simple yet effective way to incorporate recovery and mortality processes. This is especially important for diseases with a high fatality rate, such as CCHF. While stochastic and spatially explicit models have also been explored, deterministic SIRD models remain widely used due to their simplicity and analytical clarity. More recently, researchers have extended classical models by incorporating fractional-order derivatives, which allow the inclusion of memory effects and non-local dynamics in infectious disease modelling. This fractional approach has been successfully applied to vector-borne diseases such as CCHF, offering improved accuracy in simulating transmission cycles and evaluating the long-term impact of interventions [14,15]. Furthermore, modelling studies have helped identify the most sensitive parameters through sensitivity analysis, enhancing the predictive power and reliability of public health decision-making tools [16,17,18].

Crimean-Congo haemorrhagic fever (CCHF) is a life-threatening zoonotic disease endemic in Turkey. It is transmitted mainly through tick bites and human-to-human contact, and is characterised by a high case fatality rate. The first case was seen in the Kelkit Valley, and the first diagnosis was made in 2003. The disease is more common in provinces such as Tokat,

Sivas, Yozgat, Erzincan and Gümüşhane where the Kelkit Valley passes. Sivas has become an experienced centre that deals with patients coming from these surrounding provinces. This study uses a deterministic *SIRD* model to investigate the transmission dynamics of CCHF in Turkey, with the model parameters being estimated using real-world data obtained from Sivas Cumhuriyet University Research Hospital between 2010 and 2024. The specific objectives are to estimate transmission and recovery rates, calculate the basic reproduction number (R_0) and evaluate the local and global stability of equilibrium points. Additionally, sensitivity analysis is employed to identify the parameters most influential in disease propagation. Numerical simulations are conducted using MATLAB to explore potential outbreak scenarios and inform public health strategies. This integrative approach aims to demonstrate the value of mathematical modelling in supporting data-driven, evidence-based decision-making in the control of emerging infectious diseases such as CCHF.

Model Formulation

Mathematical models are often used to better understand how infectious diseases spread and how they can be prevented. Many models such as SI, SIS, SIR have been developed and analysed in order to obtain better information about the transmission dynamics and control of diseases [19,20,21,22]. In these models, disease-related deaths are ignored. However, disease-related mortality is of great importance in terms of better understanding the dynamic structure of the disease. For this reason, we investigated the SIRD model by adding the *D* death compartment to the SIR model in the literature. When introducing the model, external factors such as migration are ignored. It is also assumed that the population is homogeneous, that individuals react to the disease in the same way, and that they transition from one state to another en masse. Specifically, β is the parameter that shows the rate at which each individual in the population interacts (contacts) with other individuals. The βS contacts made by the susceptible population are with infected individuals at a ratio of I/N. Thus, $\beta SI/N$ corresponds to the incidence rate. Assuming that the transition from the susceptible to the infectious state occurs at the same rate for each individual is necessary in order to perform the calculation and express complex systems mathematically. The same can be said of all assumptions. In our model, we assume that immunity is permanent. Vulnerable individuals fall ill and recover at specific rates. Once they have recovered, they are immunised and cannot contract the disease again. SIRD model is defined as

$$\begin{aligned} \frac{ds}{dt} &= -\frac{\beta SI}{N} \\ \frac{dI}{dt} &= \frac{\beta SI}{N} - \gamma I - \alpha I \\ \frac{dR}{dt} &= \gamma I \\ \frac{dD}{dt} &= \alpha I \ . \end{aligned} \tag{1}$$

The contact rate β, recovery rate γ and mortality parameter α from disease the estimation of parameters will be undertaken, and a comparison will be made between real data and data obtained from the model.

Susceptible (*S*): Individuals susceptible to infection.

Infected (*I*): Individuals who are infected and can transmit the disease

Recovered (*R*): Individuals who recover from the infection and gain immunity.

Dead (*D*): Individuals who have died as a result of the disease.

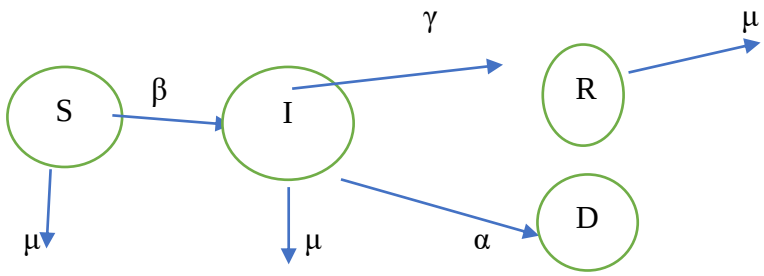

Figure 1. It is a diagram of the mathematical model, where the arrows represent the departures from a compartment at the parameter rates above it.

Including to the model (1) natural birth rate (b) and the natural, non-disease-related mortality rate (μ), we have the following SIRD model:

$$\frac{dS}{dt} = bN - \frac{\beta SI}{N} - \mu S$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I - \mu I - \alpha I \qquad (2)$$

$$\frac{dR}{dt} = \gamma I - \mu R$$

$$\frac{dD}{dt} = \alpha I.$$

The initial conditions are $S(0), I(0), R(0)$ and $D(0) > 0$.

In this model, conditions affecting the population, such as migration, are ignored, and the birth and mortality rates are assumed to be equal ($b = \mu$). Since the total population ($N = S + I + R + D$) is constant, the equation

$$\frac{d}{dt}(S(t) + I(t) + R(t) + D(t)) = 0$$

is satisfied. If the population classes are considered as proportions of the total population in the equation system

$$(s = \frac{S}{N}, i = \frac{I}{N}, r = \frac{R}{N}, d == \frac{D}{N}, n = \frac{N}{N} = 1),$$

the new system obtained is as follows:

$$\frac{ds}{dt} = -\beta si + \mu(1 - s)$$

$$\frac{di}{dt} = \beta si - \gamma i - \mu i - \alpha i \qquad (3)$$

$$\frac{dr}{dt} = \gamma i - \mu r$$

$$\frac{dd}{dt} = \alpha i.$$

In order to show the non-negative of solutions for the system (3), we claim that

$I = \{(S, I, R, D) \in R^4 : 0 \leq S + I + R + D \leq N_0\}$ is the feasibility region of system (3). To prove this claim, we consider the following theorem.

Theorem 1 . The closed set $I=\{(S,I,R,D) \in R^4 : 0 \leq S + I + R + D \leq N_0\}$ is a positive invariant for the model (3) and N_0 is the total population at t = 0.

Proof . Since $N = S + I + R + D$, we have the following differential equation:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dD}{dt}.$$

Using the differential equations for model (3), we get

$$\frac{dN}{dt} = -\beta si + \mu(1 - s) + \beta si - \gamma i - \alpha i - \mu i + \gamma i - \mu r + \alpha i$$

or

$$\frac{dN}{dt} = \mu - \mu N \; . \qquad (4)$$

This equation (4) can be solved using a first-order non-homogeneous linear differential equation. Then we have the solution of equation (4) like that:

$$N(t) = 1 + ce^{-\mu t},$$

and

for t = 0, $N_0 = 1 + c$.

Then we get $c = N_0 - 1$.

Therefore,

$$N(t)=1+(N_0-1)\, e^{-\mu t} .$$

Since $0 \leq e^{-\mu t} \leq 1$ for each $t \geq 0$, then $N \leq 1 + N_0 - 1$, and

$$N \leq N_0 \text{ or } s + i + r + d \leq N_0.$$

Because of $s(t), i(t), r(t), d(t)$ are non-negative, we obtain

$$0 \leq s + i + r + d \leq N_0 \text{ for } t \geq 0.$$

Calculating the basic reproduction number of the model (3)

The basic reproduction number R_0 is an important threshold value in mathematical models. It is represented by R_0. It is defined as the number of secondary cases caused by an infected person during the period of illness. It is important to obtain the parameters in order to calculate the basic reproduction number, which represents the expected number of new cases generated by an infected individual. The basic reproduction number (R_0) plays a fundamental role in quantifying the potential spread of an infectious disease and in assessing the effectiveness of control measures. In zoonotic and vector-borne diseases such as Crimean-Congo Haemorrhagic Fever (CCHF), R_0 is an indicator of a complex biological process involving not only human-human transmission but also tick-human, animal-human and environmental factors. If $R_0 > 1$, the disease tends to spread in the population and has epidemic potential. If $R_0 < 1$, transmission becomes unsustainable and the disease dies out over time. In terms of CCHF, R_0 depends on factors such as tick density, seasonal climatic effects, presence of animal hosts and human exposure level. Therefore, the magnitude of R_0 is not only an epidemiological value but also an important indicator summarising the environmental and biological spreadability of the disease [9]. R_0 can be easily calculated using the following next generation matrix method.

The next generation matrix (NGM) is a mathematical method used to model the spread of infectious diseases. In this study, the NGM is utilised to ascertain the simple reproduction number in the SIRD model. This is then employed to estimate the rate of disease propagation. This matrix is used to estimate the rate of spread of the epidemic, i.e. the simple growth rate, and to determine the measures that can be taken to control the spread of the infection. The NGM method was first proposed by Van den Driessche and Watmough [23,24].

The vector X represents the infected (I) classes, i.e. those who carry the virus, while the vector Y represents the non-infected classes, such as susceptible (S) and recovered (R).

$$\frac{dX}{dt} = F(X,Y) - V(X,Y).$$

Let the vector of new infection rates be denoted by *F*. In other words, *F* represents the transition from *Y* to *X*. Here, *V* represents the vector of all other rates. The recovery rates and death rate are included in the vector *V*. Accordingly,

$$\frac{dY}{dt} = W(X,Y),$$

$$D\mathcal{F}|_{(0,\overline{Y})} = \left[\frac{d\mathcal{F}}{dX} \quad \frac{d\mathcal{F}}{dY}\right]_{(0,\overline{Y})} = \left[\frac{d\mathcal{F}}{dX}\right]_{(0,\overline{Y})}$$

$$D\mathcal{V}|_{(0,\overline{Y})} = \left[\frac{d\mathcal{V}}{dX} \quad \frac{d\mathcal{V}}{dY}\right]_{(0,\overline{Y})} = \left[\frac{d\mathcal{V}}{dX}\right]_{(0,\overline{Y})}$$

$$F = \left[\frac{d\mathcal{F}}{dX}\right]_{(0,\overline{Y})}$$

$$V = \left[\frac{d\mathcal{V}}{dX}\right]_{(0,\overline{Y})}$$

Here, FV^{-1} is called the new generation matrix. The spectral radius of this value helps to calculate the basic growth rate. The spectral radius of FV^{-1} is equal to the dominant characteristic root value of FV^{-1}, which is the maximum characteristic root of FV^{-1}.

According to this

$$\frac{di}{dt} = \beta si - \gamma i - \mu i - \alpha i.$$

Let consider the following *X*, *Y*, *F* and *V* vectors:

$$X = \beta si, \qquad F = \left(\frac{dX}{di}\right) = \beta s,$$

$$Y = \gamma i + \mu i + \alpha i, \qquad V = \left(\frac{dY}{di}\right) = \gamma + \mu + \alpha.$$

Then we get

$$V^{-1} = \frac{1}{\gamma+\mu+\alpha} \text{ and } FV^{-1} = \frac{\beta s}{\gamma+\mu+\alpha}.$$

R_0 is given by the largest of the eigenvalues of the FV^{-1} matrix.

Therefore, it is found as,

$$R_0 = \frac{\beta s}{\gamma+\mu+\alpha} \qquad (5)$$

Now we will get the existence conditions of the equilibrium point for the model (3).

Since the total population ($N = S + I + R + D$) is constant, the equation

$\frac{d}{dt}(S(t) + I(t) + R(t) + D(t)) = 0$ is satisfied.

If the population classes are considered as proportions of the total population in the equation system ($s = \frac{S}{N}, i = \frac{I}{N}, r = \frac{R}{N}, d == \frac{D}{N}, n = \frac{N}{N} = 1, d = 1 - s - i - r$), the new system obtained is as follows:

$$\frac{ds}{dt} = -\beta si + \mu(1 - s)$$

$$\frac{di}{dt} = \beta si - \gamma i - \mu i - \alpha i$$

$$\frac{dr}{dt} = \gamma i - \mu r. \qquad (6)$$

The equilibrium points are in two forms: disease-free and endemic equilibrium point. Accordingly, based on the system (6), disease-free equilibrium point is

$$E_0 = (s, i, r) = (s = 1, i = 0, r = 0).$$

On the other hand, endemic equilibrium point,

$$E_1 = (s^*, i^*, r^*),$$

$$s^* = \frac{\alpha + \gamma + \mu}{\beta}$$

$$i^* = -\frac{\mu(\gamma + \alpha - \beta + \mu)}{(\alpha + \gamma + \mu)\beta}$$

$$r^* = -\frac{\gamma(\gamma + \alpha - \beta + \mu)}{(\alpha + \gamma + \mu)\beta}$$

Lemma 1. For the system (6),

a) E_0= (1,0,0) is an always trivial equilibrium point.

b) If $R_0 > 0$, then $E_1 = (s^*, i^*, r^*)$ is a co-existence equilibrium point.

Stability analysis of the equilibrium point of the model (6)

In this section we analyze stability of the equilibrium point of the system. We use the following Routh-Hurwitz criterion to get the necessary and sufficient conditions**.**

Theorem 2 . Given the polynomial,

$$P(\lambda) = \lambda^{n} + a_1\lambda^{n-1} + a_2\lambda^{n-2} + \ldots + a_{n-1}\lambda + a_n$$

where the coefficients a_i are the real constants, $i = 1,2,3,\ldots,n$, define the n Hurwitz matrices using the coefficient a_i of the characteristics polynomial:

$$H_1 = [a_1],\ H_2 = \begin{bmatrix} a_1 & 1 \\ a_3 & a_2 \end{bmatrix},\ H_3 = \begin{bmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ a_5 & a_4 & a_3 \end{bmatrix},\ldots,\ H_n = \begin{bmatrix} a_1 & 1 & 0 & \ldots & 0 \\ a_3 & a_2 & a_1 & \ldots & 0 \\ a_5 & a_4 & a_3 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & a_n \end{bmatrix}$$

where a_j>0 if $j > n$. All the roots of the polynomial $P(\lambda)$ are negative or have negative reel

part if and only if the determinants of all Hurwitz matrices are positive

$$detH_j > 0 \ \ j = 1; 2; \ldots; n$$

This criterion is called the Routh-Hurwitz criterion.

For $n = 2$, if we use Routh-Hurwitz criterion, it can be said that for the eigenvalues to have a negative real part $\lambda^2 - iz(J)\lambda + det(j) = 0$. The characteristic polynomial obtained from the Jacobian matrix coefficients are positive.

Theorem 3. $\frac{du}{dt} = f(u,v), \frac{dv}{dt} = g(u,v)$ the system contains the equilibrium point (u^*, v^*) of the functions f and g . Suppose that the first-order partial derivatives of some open sets are continuous. Let J be the Jacobian matrix of the equilibrium point

· If $Tr(J) < 0$ and $Det(J) > 0$, the equilibrium point (u^*, v^*) is locally asymptotically stable.

·If $Tr(J) > 0$ or $Det(J) < 0$, the equilibrium point (u^*, v^*) is unstable [24].

Theorem 4. Let $U = \begin{bmatrix} U_1 \\ \vdots \\ U_n \end{bmatrix} \epsilon\, R^n, t \in R$ and $F(U,t)$ being a vector field $((dU)/(dt)) = F(U,t)$
Assume that *U** be the equilibrium point of the system of differential equations and J (*U**) be the jacobian matrix calculated at this equilibrium point. The characteristic equation of a jacobian matrix, *U** is locally asymptotically stable if the equilibrium point satisfies the Routh-Hurwitz criterion, otherwise undecided [24,25,26].

In order to use Theorem 3 and Theorem 4, the equilibrium point's eigenvalues are examined using the jacobian matrix.

$$J = \begin{pmatrix} -i\beta - \mu & -\beta s & 0 \\ i\beta & \beta s - \alpha - \gamma - \mu & 0 \\ 0 & \gamma & -\mu \end{pmatrix},$$

and for E_0= (1,0,0) equilibrium point, we have

$$J(E_0) = \begin{pmatrix} -\mu & -\beta & 0 \\ 0 & \beta - \alpha - \gamma - \mu & 0 \\ 0 & \gamma & -\mu \end{pmatrix}.$$

Then we get the following characteristic equation

$$det(j - \lambda I) = \begin{vmatrix} -\mu - \lambda & -\beta & 0 \\ 0 & \beta - \alpha - \gamma - \mu - \lambda & 0 \\ 0 & \gamma & -\mu - \lambda \end{vmatrix} = 0.$$

The characteristic equation is given by:

$$(\mu + \lambda)^2(\beta - \alpha - \gamma - \mu - \lambda) = 0 \qquad (7)$$

The characteristic equation (7) has following eigenvalues:

$$\lambda_1 = \lambda_2 = -\mu \quad \lambda_3 = \beta - (\alpha + \gamma + \mu).$$

Now, we will show that λ_3 is negative. So we must show that $\beta - (\alpha + \gamma + \mu) < 0$.

We can rewrite this condition as $\beta < (\alpha + \gamma + \mu)$. Since $(\alpha + \gamma + \mu) > 0$, both sides of the inequality can be divided by it:

$$\frac{\beta}{\alpha + \gamma + \mu} < 1.$$

Because of (5), then for $R_0 = \frac{\beta}{(\alpha+\gamma+\mu}$ < 1, it follows that $\lambda_3 < 0$. Therefore, all eigenvalues have negative real parts, and the disease-free equilibrium point

$E_0 = (s, i, r)$= (1,0,0) is locally asymptotically stable when $R_0 < 1$.

The Figure 2 analyses the variation of the basic reproduction number R_0 for Crimean-Congo Haemorrhagic Fever (CCHF) in relation to the transmission rate β, focusing on the local asymptotic stability of the disease-free equilibrium point E_0 = (1, 0, 0). The value of R_0 remains below the epidemic threshold of 1 (dashed line) for all β values considered, indicating that an epidemic cannot occur under current parameter conditions and that the disease-free equilibrium remains locally stable. The figure also serves as a sensitivity analysis, quantitatively showing the effect of β on R_0. Although increases in β initially lead to higher R_0 values, this effect eventually saturates, indicating that other parameters in the system (e.g. recovery or mortality rates) play a limiting role in disease transmission.

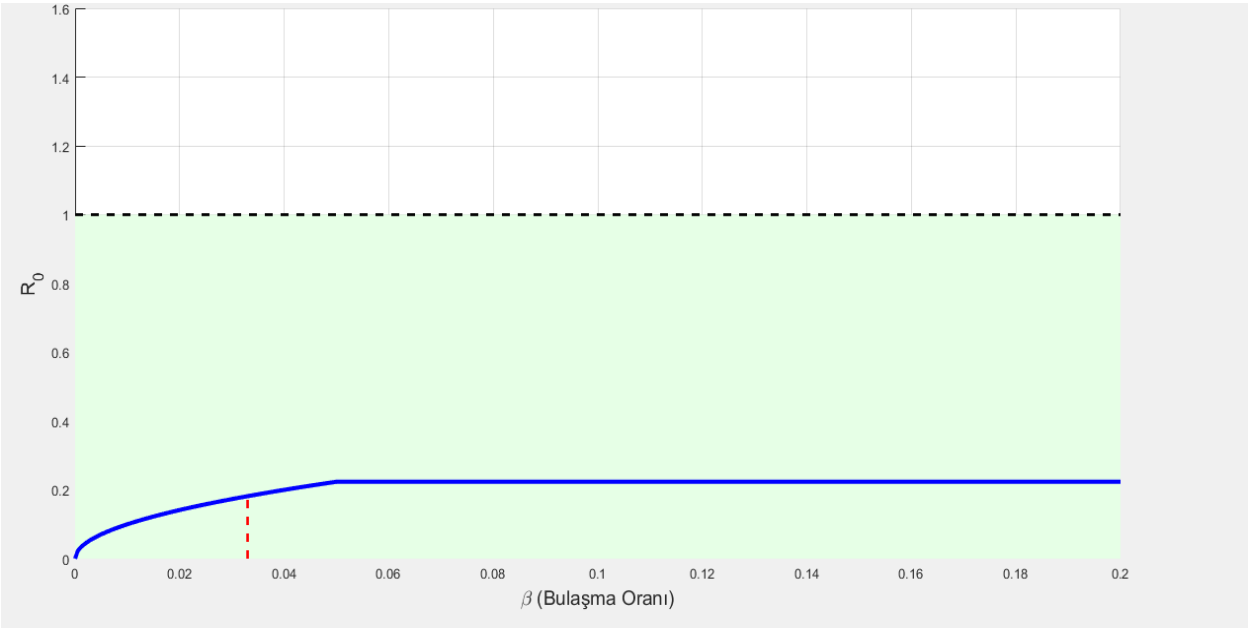

Figure 2. The stability graph of R_0.

Now, let consider the equilibrium point:

$$E_1 = (s^*, i^*, r^*),$$

$$s^* = \frac{\alpha+\gamma+\mu}{\beta} = \frac{1}{R_0}\, i^* = -\frac{\mu(\gamma+\alpha-\beta+\mu)}{(\alpha+\gamma+\mu)\beta} = \frac{\mu(R_0-1)}{\beta},\ r^* = -\frac{\gamma(\gamma+\alpha-\beta+\mu)}{(\alpha+\gamma+\mu)\beta} = \frac{\gamma(R_0-1)}{\beta}.$$

To analyze the stability of the endemic equilibrium, we examine the jacobian matrix evaluated at this point:

$$J = \begin{pmatrix} -\mu R_0 & \frac{-\beta}{R_0} & 0 \\ -\mu(R_0-1) & \frac{\beta}{R_0} - \alpha - \gamma - \mu & 0 \\ 0 & \gamma & -\mu \end{pmatrix} = 0.$$

Then we get the following characteristic equation

$$det(j-\lambda I) = \begin{vmatrix} -\mu R_0 - \lambda & -\beta & 0 \\ \mu(R_0-1) & -\lambda & 0 \\ 0 & \gamma & -\mu-\lambda \end{vmatrix} = 0,$$

$$P(\lambda) = \lambda^3 - (-\mu R_0 - \mu)\lambda^2 + \frac{({R_0}^2\mu^2 + R_0\mu\beta - \mu\beta)\lambda}{R_0} + \frac{\mu(R_0-1)\mu\beta}{R_0} = 0. \qquad (8)$$

The characteristic equation (8) has following eigenvalues:

$$\lambda_1 = -\mu,$$

$$\lambda_2 = -\frac{1}{2}\frac{\mu {R_0}^2 + \sqrt{{R_0}^4\mu^2 - 4{R_0}^2\mu\beta + 4R_0\mu\beta}}{R_0}$$

$$\lambda_3 = \frac{1}{2}\frac{-\mu {R_0}^2 + \sqrt{{R_0}^4\mu^2 - 4{R_0}^2\mu\beta + 4R_0\mu\beta}}{R_0}.$$

It is evident that both λ_1 and λ_2 are negative. Furthermore, for $R_0>1$, λ_3 also becomes negative. Therefore, the endemic equilibrium point

$E_1 = (s^*, i^*, r^*)$ is locally asymptotically stable when $R_0>1$.

Global Stability of the Model (6)

To find the conditions for disease elimination, we analyze the global stability using Lyapunov function.

Theorem 5: If $R_0<1$, $\frac{dL}{dt}<0$ and disease-free equilibrium $E_0 = (1, 0, 0)$, is globally stable.

Proof: To analyze the global stability of the model (6) , we construct a suitable Lyapunov function. Consider the Lyapunov candidate function defined by

$$L(i) = \gamma i.$$

Evaluating this function at the disease-free equilibrium point $E_0 = (1, 0, 0)$, we obtain

$$L(E_0) = L(0) = 0.$$

Next, we examine the time derivative of L to determine whether it is negative definite. Taking the derivative of L with respect to time yields:

$$\frac{dL}{dt} = \gamma\frac{di}{dt}$$

Substituting the differential equation for di/dt from the model (6) :

$$\frac{dL}{dt} = \gamma(\beta si - \gamma i - \mu i - \alpha i).$$

Then we obtain:

$$\frac{dL}{dt} = \gamma(\beta si - \gamma i - \mu i - \alpha i) = \gamma i(\gamma + \mu + \alpha)\left(\frac{\beta s}{\gamma+\mu+\alpha} - 1\right) < 0.$$

This expression is negative when

$$\beta s - (\alpha + \gamma + \mu) < 0 \Rightarrow \frac{\beta}{(\gamma+\mu+\alpha)} - 1 < 0.$$

Since $s \leq 1$, this condition holds globally stable if R_0<1. Therefore, when R_0<1, $\frac{dL}{dt}$<0 and disease-free equilibrium E_0 = (1, 0, 0) is globally stable.

The subsequent graph illustrates the stability graph of R_0.

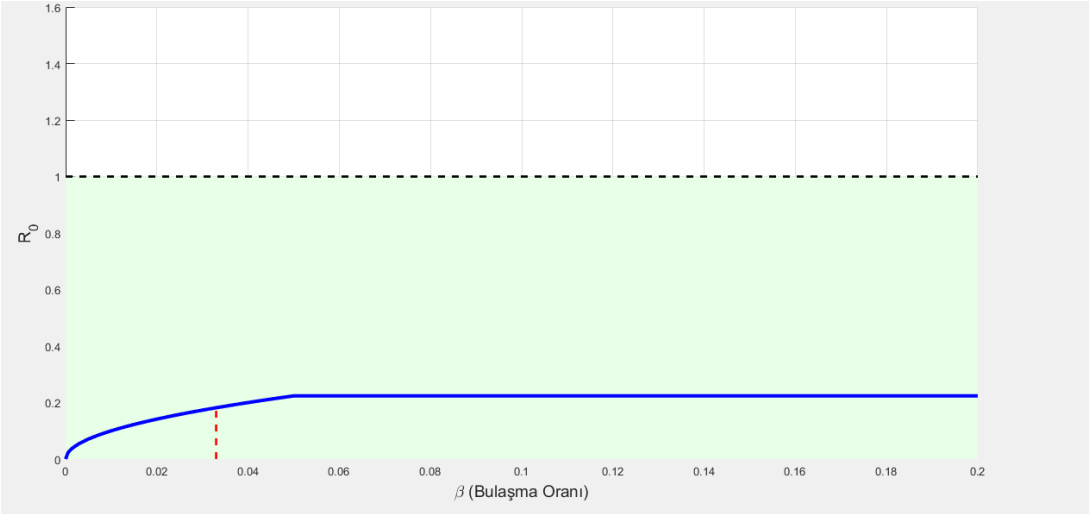

Figure 3. The global stability graph of E_0 equilibrium point via R_0

The Figure 3 shows the variation of the basic reproduction number R_0 as a function of the transmission rate β. The horizontal dashed black line represents the epidemic threshold value $R_0 = 1$. The blue curve remains below this threshold value for all β intervals analysed, indicating the global asymptotic stability of the disease-free equilibrium point E_0 = (1, 0, 0). This graph is also a sensitivity analysis and quantitatively reveals the effect of the parameter β on R_0. As seen in the figure, an increase in β increases R_0; however, this increase stabilises after a certain point. This indicates that other parameters in the system (e.g. recovery or mortality rates) limit the growth of R_0 and the model evolves towards equilibrium.

Findings

The findings obtained in this study are the predictions of the Crimean Congo Hemorrhagic Fever disease, which is widespread in Sivas province and its surroundings as an example from Turkey, obtained with the model (3). Firstly, the model was applied to Crimean-Congo Hemorrhagic Fever, and the estimated values for the following year were obtained. The calculation of errors was conducted using known actual values. In the selection of provinces and epidemics, diseases that may be prevalent according to the migration, climate and geographical conditions of that country were preferred.

The objective of this study is to demonstrate the challenges and potential of epidemiological modelling with limited data sets. The data obtained from Sivas Cumhuriyet University Research Hospital offer a valuable source of information regarding the dynamics of disease propagation.

This information can be utilised by hospital management and public health authorities to develop more effective strategies against future outbreaks. However, the generalizability and reliability of the model's predictions may be affected by the limitations of the dataset, such as the small number of data points. Consequently, the utilisation of more comprehensive and longer-term datasets is recommended for future research endeavours.

A thorough examination of the dataset reveals significant fluctuations in the actual number of patients over the years. Of particular note is the marked disparity between the lowest number of infected individuals documented in 2017 (97) and the highest number recorded in 2022 (942). It is evident that these fluctuations are indicative of the intricacy of the dynamics of disease spread and its sensitivity to external factors.

Conversely, the number of reported deaths has exhibited a more stable upward trend in comparison to the fluctuations seen in the number of infected individuals. This may be indicative of potential increases in disease mortality, enhancements in hospital registration systems, or shifts in demographics.

Table 1 It shows the data on CCHF disease obtained from Sivas Cumhuriyet University Research Hospital between 2010 and 2024.

Year	Total Population (N)	Susceptible (S)	Infected (I)	Recovered (R)	Deceased (D)
2010	642224	641816	408	394	14
2011	627056	626529	527	514	13
2012	623535	623192	343	328	15
2013	623824	623516	308	301	7
2014	623116	622891	225	216	9
2015	618617	618376	241	228	13
2016	621224	621068	156	152	4
2017	621301	621204	97	92	5
2018	646608	646328	280	256	24
2019	638956	638637	319	275	44
2020	635889	635290	599	556	43
2021	636121	635274	847	806	41
2022	634924	633982	942	796	146
2023	650401	650164	237	221	16
2024	637007	636766	241	231	10

Table 2 It shows the estimated values of the number of patients and deaths obtained from the model for Crimean-Congo haemorrhagic fever.

Year	Number of cases	estimated number of cases	Death toll	Estimated number of deaths
2010	408	…….	14	……
2011	527	387	13	14
2012	343	367	15	15
2013	308	348	7	16
2014	225	331	9	17
2015	241	314	13	17
2016	156	298	4	18
2017	97	283	5	19
2018	280	268	24	19
2019	319	255	44	20
2020	599	242	43	20
2021	849	229	41	21
2022	942	218	146	21
2023	239	207	16	22
2024	241	196	10	22

Table 3 It shows that used parameter values

Parameter	**Value**	**Source**
β	0,033	Predicted
γ	0,97	Predicted
α	0,03	Predicted
μ	0,006	Acceptance

Table 4 It shows the data of Sivas Cumhuriyet University Research Hospital

Initial conditions	Value	Source
S(0)	641816	Acceptance
I(0)	408	Sivas Cumhuriyet University Research Hospital
R(0)	394	Sivas Cumhuriyet University Research Hospital
D(0)	14	Sivas Cumhuriyet University Research Hospital

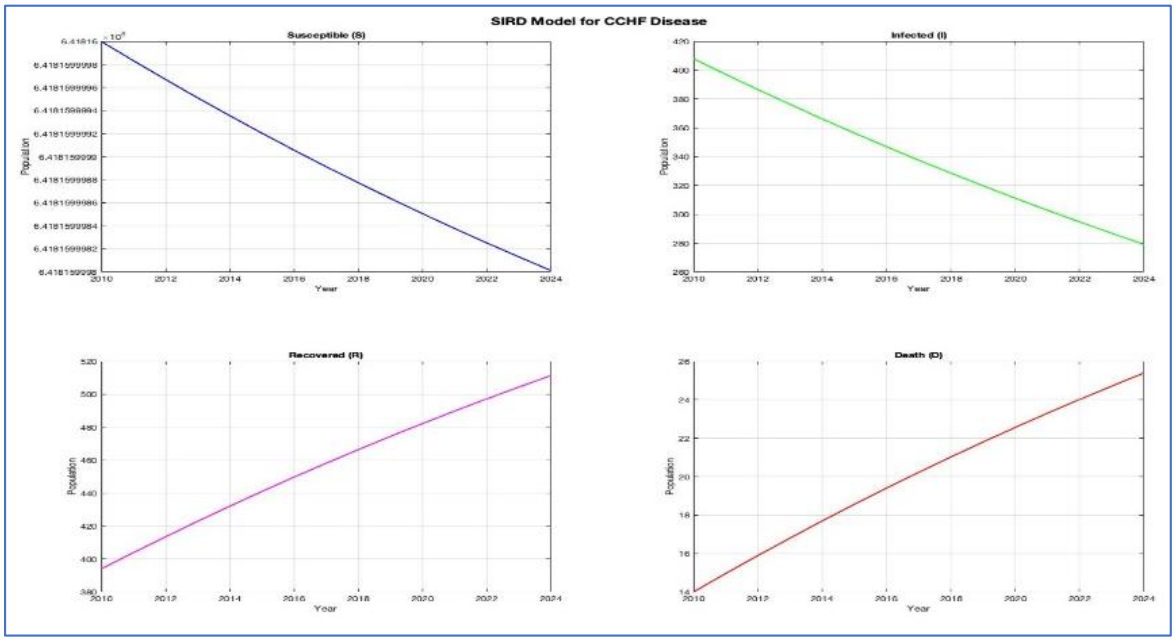

Figure 4. It presents the simulated dynamics of the susceptible (*S*), infected *(I*), recovered (*R*), and disease-induced death (*D*) populations for CCHF using the SIRD model over the period 2010–2024.

In the Figure 4, the number of susceptible individuals is observed to decrease steadily, indicating ongoing transmission within the population. Conversely, the infected population exhibits a decline, thereby indicating the efficacy of the disease's containment. Conversely, the recovered and deceased populations increase over time, reflecting the outcomes of infection. This simulation is designed to shed light on the long-term behaviour of the system and to offer insights into the effectiveness of control measures. Furthermore, the figure provides a sensitivity analysis, illustrating how alterations in model parameters – principally transmission,

recovery, and mortality rates – influence the trajectory of each compartment. The findings emphasise the significance of prompt intervention and the judicious allocation of resources to minimise infections and reduce the mortality rate from diseases.

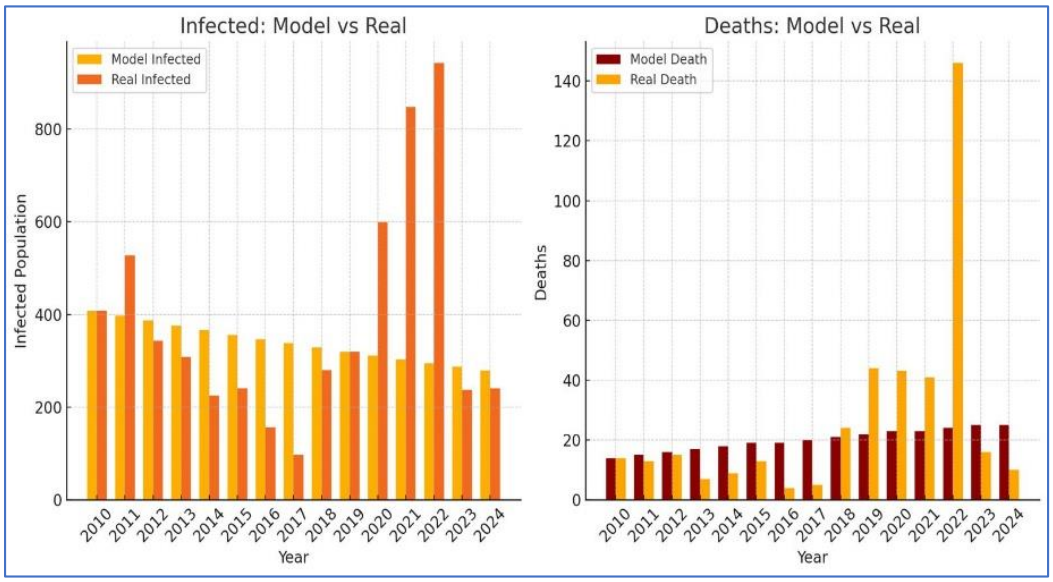

Figure 5. It shows the change in the actual number of patients and deaths and the number of patients and deaths obtained from the model over the years.

Figure 6 shows a numerical simulation of the variation of the basic reproduction number R_0 in response to changes in the transmission rate (β) under different combinations of recovery rate (γ) and mortality from disease (α). This figure allows a sensitivity analysis of how these epidemiological parameters affect the stability of the system. The R_0 is directly proportional to the parameter β and inversely proportional to the parameters γ and α. Of these parameters, β can be controlled and all of them have a positive effect on causality. Therefore, to reduce R_0 it is sufficient to reduce the rate of disease transmission between ticks, animals and humans.

All curves plotted are consistently below the critical threshold $R_0 = 1$ (indicated by the black dashed line), a key condition for disease control. An R_0 value below 1 means that each infected individual transmits the disease to fewer than one person on average, leading to the eventual elimination of the infection from the population. The green shaded area in the graph visually indicates the stable zone where this condition is met.

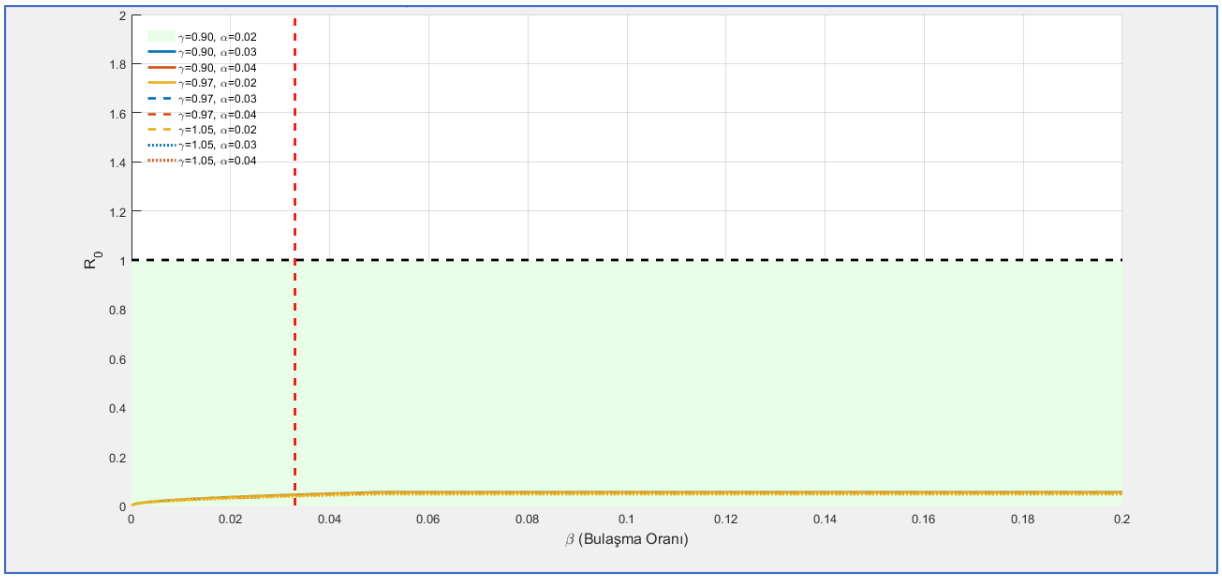

Figure 6. A stability graph of R_0 according to recovery rate (γ) and mortality (α) change.

It has been demonstrated that the $R_0 < 1$ condition is satisfied for all parameter combinations, thereby indicating that the system is stable and the disease will not spread.

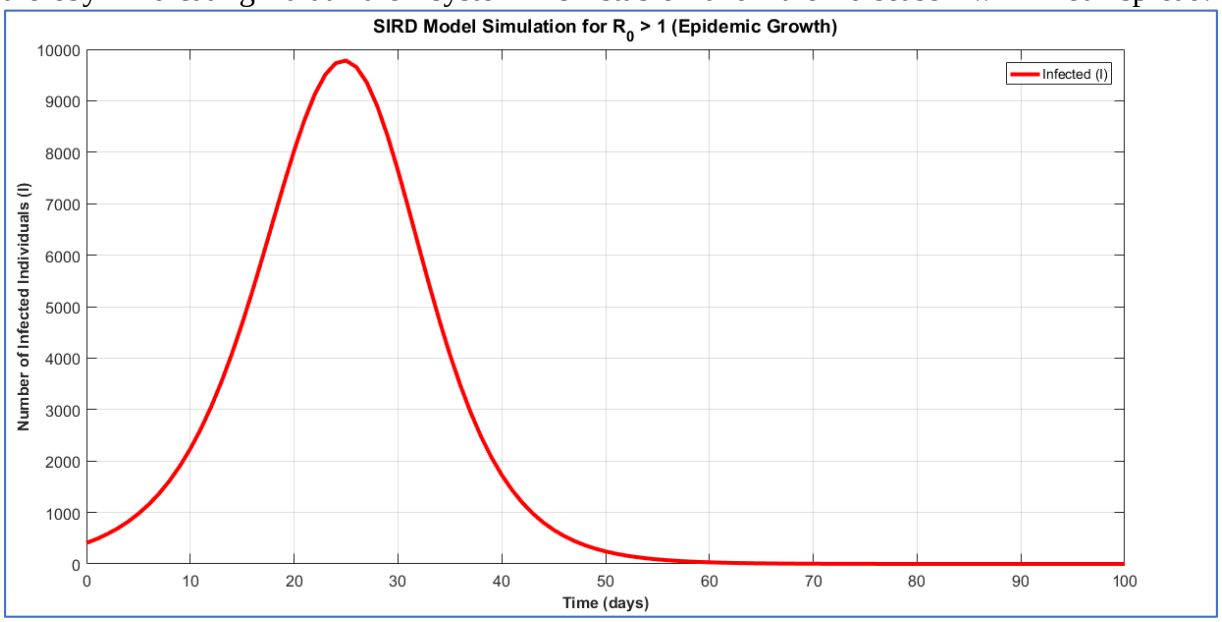

Figure 7. A graph of model (6) for $R_0 > 1$.

Figure 7 shows that, in the model (6) , the number of infected individuals increases rapidly over time and then decreases after reaching the peak. This reveals that the system is initially unstable and that the epidemic spreads, but that over time, the infection is dampened as the number of susceptible individuals is exhausted. When $R_0 > 1$, the system is initially unstable and the epidemic spreads. Over time, however, the infection is dampened as the number of susceptible individuals is exhausted.

Conclusions

In this study, the transmission dynamics of Crimean-Congo Haemorrhagic Fever (CCHF) in Sivas province between 2010 and 2024 were investigated by applying a compartment (3) model. The model is calibrated using real case and mortality data obtained from Sivas Cumhuriyet University Research Hospital to simulate the disease progression under different epidemiological scenarios.

Analysis of both real and simulated data sets revealed year-to-year fluctuations in the number of infections (see Tables 1 and 2). This finding suggests that the spread of CCHF is subject to dynamic biological, ecological and social determinants. In contrast, the number of deaths exhibited a more stable and gradually increasing trajectory, suggesting prolonged exposure to risk factors or potential under-reporting of cases. The model showed a satisfactory degree of accuracy in reproducing these patterns, thus confirming its capacity to capture general trends despite the limitations imposed by the scarcity of available data.

Simulation results (see Figure 4) provide insight into the evolution of susceptible, infected, recovered and dying populations. The number of susceptible individuals has decreased over time, consistent with continued exposure and transmission within the community. Simultaneously, the infected population exhibited a consistent decline, while the recovering and dying populations increased, thus signalling the natural progression of the disease and the effectiveness of current public health interventions. These results underline the importance of maintaining long-term surveillance and ongoing control measures.

Sensitivity analyses are presented in various figures (see Figures 2, 3 and 6) for the basic reproduction number R_0, which is an important indicator of the potential for disease spread. All simulations showed that R_0 remains below the critical threshold of 1 for all combinations of transmission rate (β), recovery rate (γ) and mortality from disease (α). This indicates both local and global asymptotic stability of the disease-free equilibrium. While increases in β initially lead to higher R_0 values, the effect plateaued over time, suggesting the presence of natural or systemic constraints imposed by the dynamics of recovery and mortality.

It is noteworthy that these findings imply that current transmission conditions in the region are insufficient to sustain an outbreak. However, they also emphasise the delicate balance between parameter interactions that can easily be disrupted by environmental change, reduced public health response or zoonotic spread events. Consequently, constant vigilance and proactive prevention strategies remain imperative.

From a broader ecological and public health perspective, the study emphasises the interconnectedness of natural and human systems. As noted in the recommendations, some wildlife species, including partridges, indirectly influence the control of CCHF transmission by contributing to the regulation of tick populations. Maintaining ecological balance is therefore of environmental importance and is also directly beneficial for human health.

Furthermore, the importance of individual protective behaviours cannot be ignored. The use of appropriate clothing in high-risk areas, education on prevention of tick infestation and initiation of early medical intervention have been identified as effective strategies to complement environmental controls. Maintaining animal health, particularly in livestock, which may act as intermediate hosts, also contributes to reducing the burden of human infection.

In conclusion, the findings of this study affirm that mathematical modelling is a valuable tool in understanding vector-borne disease dynamics, even in circumstances where data limitations exist. While the current model provides a robust foundation for analysing CCHF in Sivas Province, future research should incorporate longer-term datasets and additional environmental and demographic variables to enhance its predictive capacity. It is imperative to recognise that a concerted, multidisciplinary strategy — encompassing ecological conservation, public health policy, and individual awareness — is indispensable for curtailing the propagation and ramifications of zoonotic diseases such as CCHF.

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