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Research Article

Solution Dynamics of Climate Finance Models for Mitigating Health and Economic Impacts of Climate-Induced Infectious Disease Outbreaks in Southern Nigeria Using a Semi-Analytical Method

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Abstract. Climate change has heightened the frequency and intensity of infectious disease outbreaks in Southern Nigeria, with profound health and economic implications. Addressing these impacts requires not only epidemiological and environmental understanding but also sustainable financial modeling mechanisms that can allocate, predict, and optimize resource flows for mitigation. This paper presents a mathematical framework for developing a climate finance model that integrates epidemiological and economic variables through a system of coupled differential equations. The *Differential Transform Method* (DTM) is employed to obtain approximate analytical solutions to the nonlinear system, thereby enabling the formulation of dynamic financing policies under uncertain climate conditions. Basic theorems concerning the existence, uniqueness, and convergence of the proposed model are also established. The study provides a foundational basis for adaptive climate financing strategies aimed at reducing the health and economic burdens of climate-induced disease outbreaks.

Keywords. Climate finance, Infectious diseases, Differential Transform Method (DTM), Epidemiological modelling, Southern Nigeria, Economic impact

Mathematics Subject Classification (2020). 34A34, 92D30, 65L05

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1. Introduction

The southern region of Nigeria has received the increased dosages of climate-induced infectious diseases like Malaria, cholera, and Dengue Fever (Baker *et al*. [7]). Modifications in temperature, humidity, and rainfall patterns have expanded vector breeding habitats and assisted transmission intensity (Singh *et al*. [24]). In economic terms, these health impacts have translated into reduced productivity and healthcare costs (Chikezie *et al*. [9]; Agyarko *et al*. [2]).

It has become crucial to build up climate finance-that is, funds to implement climate adaptation and mitigation actions-for the purpose of increasing the resilience of vulnerable communities (Agyarko *et al*. [2]; Modabbernia *et al*. [19]). For instance, "*trans theoretical models*" or "*planning models*" have already been proposed by epidemiological models along with several possible quantitative modeling frameworks of disease dynamics combined with economic and financial systems. In this paper, we propose a differential equation model that embraces the dynamics of disease distribution, environmental factors, and financial actions (Bourouiba [8], Ma *et al*. [15]; Luz and Masoodian [18]). Interestingly, a novel method known as the *Differential Transform Method* (DTM) is introduced and illustrated through a practical approximation in solving the system that simulates financial actions in relation to weather-induced stresses.

1.1 Motivation: The Shift Toward Sustainable Climate Financing in Southern Nigeria

Southern Nigeria is becoming increasingly susceptible to climate-induced health risks, especially infectious disease breakouts like malaria, cholera, and meningitis (Schwerdtle *et al*. [22]; Worsley-Tonks *et al*. [26]; Zanobini *et al*. [29]). Increased temperatures, deteriorating patterns of rainfall, and flood incidents are providing well-suited environments for disease vectors, thus making outbreaks frequent and distributed more widely. These climate-born health threats not only burden the health service delivery system but also undermine economic productivity by diverting labor output, increasing healthcare costs, and decreasing agricultural performance (Neta *et al*. [20]; You *et al*. [28]).

No considerable attention has been given to the incorporation of climate finance into disease prevention and control despite numerous global interventions being introduced to address this issue (Gibb *et al*. [14]; Khoi and Hang [17]; Simon [23]). Funding mechanisms mainly emphasize response rather than preventive, model-based planning that could drive early interventions and resource-efficient allocation. The lack of a firm and robust mathematical and financial framework linkage between climate dynamics, disease transmission, and economic costs has created a significant policy void (Anbalagan and Krishnan [5]; Yeh *et al*. [27]).

The intricate issue of widespread infectious disease and its economic antecedents raises the urgency to develop a climate financial modeling framework capable of quantifying and predicting the financial requirements for mitigating the twin effects of health and economic impacts of climate-induced infectious diseases. This study embarked on developing analytical tools for estimating cost functions and optimal interventions for different climatic scenarios in public health planning and sustainable climate financing in Southern Nigeria.

2. Model Framework and Structure

2.1 Model Formulation

Let the total population $N(t)$ at time t be divided into the standard compartments of a climate-sensitive disease transmission model:

$$N(t) = S(t) + E(t) + I(t) + R(t) \tag{2.1}$$

where $S(t)$, $E(t)$, $I(t)$, and $R(t)$ denote Susceptible population, Exposed population, Infectious population, and Recovered population, respectively (Agbehadji *et al*. [1]; Chinenye *et al*. [10]).

These climate and financial factors are incorporated into the model as follows:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta(T,H)SI - \mu S + \rho R - f(F)S,\\
\frac{dE}{dt} &= \beta(T,H)SI - (\sigma+\mu)E,\\
\frac{dI}{dt} &= \sigma E - (\gamma+\mu+\delta)I - \eta F I,\\
\frac{dR}{dt} &= \gamma I - (\mu+\rho)R,\\
\frac{dF}{dt} &= \xi I - \omega F.
\end{aligned}\right\} \tag{2.2}$$

Here, we make reference to Table 1 as regards the parameters contained in the model:

Table 1. Climate and financial factors parameters

Parameter	Meaning remark
Λ	Recruitment rate
μ	Natural death rate
ρ	Rate of loss of immunity from recovered to susceptible
σ	Progression rate from exposed to infectious
γ	Recovery rate
δ	Disease-induced death rate
$\beta(T,H)$	Transmission rate influenced by climate (temperature T and humidity H)
η	Financial responsiveness coefficient
$F(t)$	Climate finance allocation over time
$f(F)$	Economic mitigation expenditure
ξ	Financial mobilization rate in response to infection
ω	Depletion rate of funds due to spending or inefficiency

This model jointly captures health dynamics (through SEIR compartments) and economic dynamics (through $F(t)$), under climatic perturbations.

2.2 The Model Basic Assumptions

The model for this study is based on the following assumptions, which serve as a logical foundation for the mathematical framework:

I. *Climate–Disease Link:* Disease transmission rates depend on climatic variables such as temperature, rainfall, and humidity.

II. *Homogeneous Population:* Every individual has an equal chance of exposure to infection within the study region.

III. *Dynamic Finance Allocation:* Climate finance varies with disease severity and climate impact over time.

IV. *Economic Cost Function:* Outbreak costs arise from healthcare expenses and productivity losses, both modeled as continuous functions of time.

V. *Climate-Dependent SEIR Model:* Infection and recovery rates depend on temperature and rainfall indices.

VI. *Intervention Effectiveness:* Climate finance interventions continuously reduce transmission rates but with diminishing returns.

VII. *Model Continuity:* All parameters and variables are continuous and differentiable, enabling the use of differential equations and DTM.

VIII. *Closed Population:* Migration effects are negligible; demographic changes stem from births, deaths, and disease mortality.

IX. *Steady Climate Trend:* Short-term climate variations are treated as quasi-stationary within the modeling period.

2.3 Theoretical Foundations

Theorem 2.1 (*Existence and Uniqueness* [11])**.** *Let the following system as contained in* (2.2)*,*

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta(T,H)SI - \mu S + \rho R - f(F)S,\\
\frac{dE}{dt} &= \beta(T,H)SI - (\sigma+\mu)E,\\
\frac{dI}{dt} &= \sigma E - (\gamma+\mu+\delta)I - \eta FI,\\
\frac{dR}{dt} &= \gamma I - (\mu+\rho)R,\\
\frac{dF}{dt} &= \xi I - \omega F,
\end{aligned}\right\} \tag{2.3}$$

be defined on an interval $t \in [0,T]$ *with initial conditions* $S(0),E(0),I(0),R(0),F(0) \geq 0$*. Assume the functions* $\beta(T,H)$*and* $f(F)$*are continuous and bounded on the interval, and all parameters* Λ*,* μ*,* σ*,* γ*,* δ*,* η*,* ξ*,* ω*, and* ρ *are nonnegative constants. Then there exists a unique solution* $(S(t),E(t),I(t),R(t),F(t))$ *on* $[0,T]$*. Moreover, the solution depends continuously on the initial data.*

Proof. Write the system compactly as $X = G(t,X)$ with a state vector $X = (S,E,I,R,F) \in \mathbb{R}^5$. By assumption, $\beta(T,H)$ is continuous in t and bounded, and $f(F)$ is continuous in F. Each component of $G(t,X)$ is therefore continuous in t and continuously differentiable in X on the domain $D = \{X \in \mathbb{R}^5 : X_i \geq 0\}$. In particular, the partial derivatives $\partial G_i/\partial X_j$ exist and are bounded on any compact subset of D. Hence $G(t,X)$ is locally Lipschitz in X uniformly in t on compact time intervals.

By the Picard–Lindelöf theorem, a unique local solution through the given initial condition exists. To extend the solution to the full interval $[0,T]$, we note that the right-hand side satisfies linear growth bounds on compact subsets and that solutions cannot blow up in finite time because all terms that can grow are at most polynomial in the state variables, while inputs and coefficients are bounded. More concretely, summing the equations and using nonnegativity of parameters yields a differential inequality of the form $\frac{d}{dt}X \leq aX + b$ for some constants a and b,

so Gronwall's inequality gives a uniform bound on $\|X(t)\|$ in $[0,T]$. Boundedness prevents finite time blow-up and allows extension of the local solution to the whole interval.

Continuous dependence on initial data follows from standard ODE theory because G is Lipschitz in X. This completes the proof. □

Theorem 2.2 (*Conservation of Total Population*)**.** *Let $N(t) = S(t)+E(t)+I(t)+R(t)$. For the system*

$$\left.\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta(T,H)SI - \mu S + \rho R - f(F)S, \\ \frac{dE}{dt} &= \beta(T,H)SI - (\sigma+\mu)E, \\ \frac{dI}{dt} &= \sigma E - (\gamma+\mu+\delta)I - \eta FI, \\ \frac{dR}{dt} &= \gamma I - (\mu+\rho)R, \end{aligned}\right\} \tag{2.4}$$

the total population satisfies the identity:

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I - f(F)S - \eta FI. \tag{2.5}$$

In particular, if there are no disease- induced deaths and no flows that remove susceptible or infective via the finance terms (that is, $\delta = 0$, $f \equiv 0$, and $\eta = 0$), then

$$\frac{dN}{dt} = \Lambda - \mu N. \tag{2.6}$$

A special subcase of interest is $\Lambda = \mu N$ at the initial time or on a chosen time scale, in which case $N(t)$ is constant.

Proof. Sum the four compartment equations:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}. \tag{2.7}$$

Substitute the right-hand sides and cancel terms that appear with opposite signs:

$$\frac{dN}{dt} = \Lambda - \beta SI - \mu S + \rho R - f(F)S + \beta SI - (\sigma+\mu)E + \sigma E - (\gamma+\mu+\delta)I - \eta FI + \gamma I - (\mu+\rho)R. \tag{2.8}$$

The βSI, σE, γI, and ρR terms cancel pairwise, leaving

$$\frac{dN}{dt} = \Lambda - \mu(S+E+I+R) - \delta I - f(F)S - \eta FI. \tag{2.9}$$

Recognizing $S+E+I+R = N$ yields the stated identity. The corollary follows by setting $\delta = 0$, $f \equiv 0$, and $\eta = 0$. □

Theorem 2.3 (*Local Stability of the Disease-Free Equilibrium* [3])**.** *Assume the baseline transmission rate is constant, $\beta_0 = \beta(T_0,H_0)$ evaluated at typical climate conditions, and that $f(0) = 0$. Let the disease-free equilibrium be*

$$X_0 = (S^*,E^*,I^*,R^*,F^*) = \left(\frac{\Lambda}{\mu},0,0,0,0\right). \tag{2.10}$$

Define the basic reproduction number

$$\mathcal{R}_0 = \frac{\beta_0\sigma}{(\sigma+\mu)(\gamma+\mu+\delta)}. \tag{2.11}$$

If $R_0 < 1$, then the disease-free equilibrium X_0 is locally asymptotically stable. If $R_0 > 1$, then X_0 is unstable.

Proof. We focus on the infected subsystem. The full system is

$$\left.\begin{aligned} \dot{S} &= \Lambda - \beta_0 SI - \mu S + \rho R - f(F)S, \\ \dot{E} &= \beta_0 SI - (\sigma+\mu)E, \\ \dot{I} &= \sigma E - (\gamma+\mu+\delta)I - \eta FI, \\ \dot{R} &= \gamma I - (\mu+\rho)R,\ \dot{F} = \xi I - \omega F. \end{aligned}\right\} \tag{2.12}$$

At the disease-free equilibrium, we have $E^* = I^* = R^* = F^* = 0$ and $S^* = \Lambda$. Linearize the system about X_0. Because terms such as $\beta_0 SI$ and ηFI are bilinear, their linearizations only involve I (and not products of small quantities) evaluated at $F^* = 0$. The linearized infected subsystem in the order (E, I) is

$$\begin{pmatrix} \dot{E} \\ \dot{I} \end{pmatrix} = \underbrace{\begin{pmatrix} 0 & \beta_0 S^*/N^* \\ 0 & 0 \end{pmatrix}}_{F} \begin{pmatrix} E \\ I \end{pmatrix} - \underbrace{\begin{pmatrix} \sigma+\mu & 0 \\ -\sigma & \gamma+\mu+\delta \end{pmatrix}}_{V} \begin{pmatrix} E \\ I \end{pmatrix}, \tag{2.13}$$

where $N^* = S^*$ under the assumption that E, I, R vanish at DFE. The next generation matrix is $K = FV^{-1}$. Compute V^{-1} and then K. A direct computation gives the single nonzero eigenvalue of K:

$$\mathcal{R}_0 = \frac{\beta_0 S^*/N^* \cdot \sigma}{(\sigma+\mu)(\gamma+\mu+\delta)} = \frac{\beta_0 \sigma}{(\sigma+\mu)(\gamma+\mu+\delta)}. \tag{2.14}$$

Standard next-generation matrix theory implies that the spectral radius of K equals $\mathcal{R}_0$ and that the disease-free equilibrium is locally asymptotically stable if and only if the spectral radius is less than one. Hence, if $R_0 < 1$ all eigenvalues of the linearization have a negative real part and $\mathcal{X}_0$ is locally asymptotically stable. If $R_0 > 1$, at least one eigenvalue has a positive real part and $\mathcal{X}_0$ is unstable.

The finance variable F obeys $\dot{F} = \xi I - \omega F$. At the DFE, we have $F^* = 0$, the linearization of the F-equation coupled to I, but it does not change the infected-block next generation computation because terms of order FI are nonlinear and vanish at the linear level. Therefore, the basic reproduction number above governs local stability. If finance enters at the linear level through a nonzero baseline $F^* > 0$ or through a nonzero $f(0)$, the same next generation approach applies with β_0 and other coefficients evaluated at that operating point, and the threshold condition becomes $\mathcal{R}_0 < 1$ computed with those effective parameter values. □

Theorem 2.4 (*Existence and Local Stability of an Endemic Equilibrium* [12, 13])**.** *Assume the model*

$$\left.\begin{aligned} d\dot{S} &= \Lambda - \beta_0 SI - \mu S + \rho R - f(F)S, \\ \dot{E} &= \beta_0 SI - (\sigma+\mu)E, \\ \dot{I} &= \sigma E - (\gamma+\mu+\delta)I - \eta FI, \\ \dot{R} &= \gamma I - (\mu+\rho)R, \\ \dot{F} &= \xi I - \omega F, \end{aligned}\right\} \tag{2.15}$$

has parameters all nonnegative and satisfies $f(0) = 0$. *Let* $\mathcal{R}_0$ *be as in Theorem* 2.3,

$$\mathcal{R}_0 = \frac{\beta_0 \sigma}{(\sigma+\mu)(\gamma+\mu+\delta)}. \tag{2.16}$$

If $\mathcal{R}_0 > 1$ *then the system admits a unique positive endemic equilibrium*

$$X^* = (S^*, E^*, I^*, R^*, F^*), \tag{2.17}$$

with $I^* > 0$. Moreover that endemic equilibrium is locally asymptotically stable for the parameters in the biological range.

Proof. The proof has two parts. First, we show the existence and uniqueness of a positive root for I^*. Then, we show local stability of the resulting equilibrium.

A: *Existence and uniqueness of endemic equilibrium.*
At equilibrium, the derivatives are set to zero. From the last equation, we obtain

$$F^* = \frac{\xi}{\omega} I^*. \tag{2.18}$$

From the third equation,

$$0 = \sigma E^* - (\gamma + \mu + \delta + \eta F^*) I^* \quad \Longrightarrow \quad E^* = \frac{(\gamma + \mu + \delta + \eta F^*)}{\sigma} I^*. \tag{2.19}$$

From the fourth equation,

$$0 = \gamma I^* - (\mu + \rho) R^* \quad \Longrightarrow \quad R^* = \frac{\gamma}{\mu + \rho} I^*. \tag{2.20}$$

Substitute these relations into the second equation at equilibrium:

$$\left.\begin{aligned} 0 &= \beta_0 S^* I^* - (\sigma + \mu) E^* \\ &= \beta_0 S^* I^* - (\sigma + \mu) \tfrac{(\gamma + \mu + \delta + \eta F^*)}{\sigma} I^*. \end{aligned}\right\} \tag{2.21}$$

If $I^* > 0$, we may divide by I^* to get

$$\beta_0 S^* = \frac{(\sigma + \mu)}{\sigma} (\gamma + \mu + \delta + \eta F^*). \tag{2.22}$$

Use the identity $F^* = (\xi/\omega) I^*$ to write the right-hand side as an affine function of I^*. Solve for S^*:

$$S^* = \frac{(\sigma + \mu)}{\sigma \beta_0} \left(\gamma + \mu + \delta + \eta \frac{\xi}{\omega} I^* \right). \tag{2.23}$$

Now use the first equilibrium equation, $0 = \Lambda - \beta_0 S^* I^* - \mu S^* + \rho R^* - f(F^*)$. Substitute R^* and S^* expressed above, and note $f(F^*)$ is continuous with $f(0) = 0$. After algebraic rearrangement, we obtain a scalar equation of the form

$$G(I^*) = \Lambda - A I^* - B I^{*2} - C f\left(e \frac{\xi}{\omega} I^* \right) I^* = 0, \tag{2.24}$$

where A, B, C are positive constants depending on the model parameters. The key properties of G are

- $G(0) = \Lambda > 0$,
- as $I^* \to \infty$, the negative quadratic term $-B I^{*2}$ dominates, so $G(I^*) \to -\infty$.

Therefore, by continuity, there exists at least one positive root $I^* > 0$.

To show uniqueness, note that G is strictly decreasing for $I^* \geq 0$ provided f is non-decreasing and the coefficient $B > 0$. Differentiating,

$$G'(I^*) = -A - 2BI^* - C\left(f\left(\frac{\xi}{\omega} I^* \right) + \frac{\xi}{\omega} I^* f'\left(\frac{\xi}{\omega} I^* \right) \right), \tag{2.25}$$

and each term on the right is nonpositive with at least one strictly negative term for $I^* > 0$. Thus $G' < 0$ on $I^* > 0$. Monotonicity implies the root is unique. Hence, there is a unique positive I^* and, via the algebraic relations above, unique S^*, E^*, R^*, F^*.

B: *Local stability of the endemic equilibrium.*
Compute the Jacobian matrix $J(X)$ of the full five-dimensional system and evaluate it at the endemic equilibrium X^*. The Jacobian has the block structure:

$$J(X^*) = \begin{pmatrix} J_{11} & J_{12} \\ J_{21} & J_{22} \end{pmatrix}, \tag{2.26}$$

where J_{11} is the 4 by 4 compartment block for S, E, I, R and $J_{22} = [-\omega]$ corresponds to the scalar finance variable F, since:

$$\partial \dot{F}/\partial F = -\omega \quad \text{and} \quad \partial \dot{F}/\partial I = \xi. \tag{2.27}$$

The scalar entry $-\omega$ is strictly negative and therefore contributes a stable direction. Stability of the full Jacobian then reduces to verifying that all eigenvalues of the 4 by 4 block J_{11} have a negative real part.

Because parameters are positive and $I^* > 0$, J_{11} is a Metzler matrix perturbed by negative diagonal terms arising from removal rates. Standard results for epidemic models of SEIR type imply that when $\mathcal{R}_0 > 1$ the endemic equilibrium, if it exists in the interior, is generically locally asymptotically stable, provided no bifurcation conditions are met. For our system, one can apply the Routh-Hurwitz criteria to the characteristic polynomial of J_{11}. All coefficients of that polynomial are positive because they are sums and products of positive rate parameters and equilibrium values. The Routh-Hurwitz conditions then hold, and all roots have negative real parts.

A more constructive route is to note that the Jacobian at X^* can be shown to be a nonsingular M-matrix under the biological parameter restrictions. An M-matrix has eigenvalues with positive real parts for the negative of the matrix, which implies the Jacobian has eigenvalues with negative real parts. The presence of the negative scalar $-\omega$-\omega$-\omega$ for the finance variable maintains stability in that direction and does not introduce positive eigenvalues.

Therefore, every eigenvalue of $J(X^*)$ has a negative real part, and the endemic equilibrium is locally asymptotically stable.

This completes the proof. □

Theorem 2.5 (*Positivity and Boundedness* [13, 25])**.** *Assume nonnegative initial conditions:* $S(0), E(0), I(0), R(0), F(0) \geq 0$. *Suppose all model parameters satisfy* $\Lambda, \mu, \rho, \sigma, \gamma, \delta, \eta, \xi, \omega \geq 0$, *and the functions* $\beta(T,H)$ *and* $f(F)$ *are continuous and nonnegative for all admissible values of* $T, H,$ *and* $F \geq 0$.
Let the system:

$$\left.\begin{aligned} \dot{S} &= \Lambda - \beta(T,H)SI - \mu S + \rho R - f(F)S, \\ \dot{E} &= \beta(T,H)SI - (\sigma + \mu)E, \\ \dot{I} &= \sigma E - (\gamma + \mu + \delta)I - \eta F I, \\ \dot{R} &= \gamma I - (\mu + \rho)R, \\ \dot{F} &= \xi I - \omega F \end{aligned}\right\} \tag{2.28}$$

have continuous right-hand sides and nonnegative parameters. Then, for all $t > 0$, *the solution satisfies:*

$$S(t), E(t), I(t), R(t), F(t) \geq 0,$$

and all components are bounded on every finite time interval $[0, T]$.

Proof. Positivity. The right-hand side of each equation can be written as a sum of nonnegative input terms minus removal terms proportional to the compartment itself. For example, if at some time t^* we had $S(t^*) = 0$ then

$$\dot{S}(t^*) = \Lambda + \rho R(t^*) \geq 0, \tag{2.29}$$

so S cannot cross from zero to negative. The same argument applies to E, I, R, F. More formally, standard comparison theorems or the quasi-positivity of the vector field yield that nonnegative initial data produce nonnegative solutions for all $t \geq 0$.

Boundedness on finite intervals. Sum the epidemiological compartments to get $N(t) = S + E + I + R$. From the equations, we have

$$\dot{N} = \Lambda - \mu N - \delta I - f(F)S - \eta F I \leq \Lambda - \mu N, \tag{2.30}$$

so by the Gronwall inequality

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}), \tag{2.31}$$

which gives a uniform bound for $N(t)$ on any finite interval. For F, we have $\dot{F} = \xi I - \omega F$. Using the bound $I(t) \leq N(t) \leq M_T$ on $[0, T]$, we obtain the linear differential inequality:

$$\dot{F} \leq \xi M_T - \omega F. \tag{2.32}$$

Solving this gives

$$F(t) \leq F(0)e^{-\omega t} + \frac{\xi M_T}{\omega}(1 - e^{-\omega t}), \tag{2.33}$$

so $F(t)$ is bounded on $[0, T]$. Combining these estimates shows every state variable is bounded on $[0, T]$. This completes the proof. □

3. Differential Transform Method (DTM)

The *Differential Transform Method* (DTM) is a semi-analytical technique that simplifies complex systems of differential equations into recursive algebraic forms, making it effective for approximating solutions to nonlinear models (Edeki *et al*. [12]).

3.1 The Basics of DTM

For a given function $f(t)$, the k-th order differential transform, denoted as $F(k)$, is defined as:

$$F(k) = \frac{1}{k!}\left[\frac{d^k f(t)}{dt^k}\right]_{t=0}. \tag{3.1}$$

The inverse differential transform reconstructs the original function as a power series:

$$f(t) = \sum_{k=0}^{\infty} F(k)t^k. \tag{3.2}$$

The essence of DTM lies in transforming differential equations into recursive relations for the coefficients $F(k)$. This simplifies the computational process and avoids direct integration.

Theorem 3.1 (*Convergence of the DTM series*)**.** *Let the solution components $X(t)$ of the system be real analytic in a neighborhood of t_0, that is, each component has a convergent Taylor expansion*

$$X(t) = \sum_{k=0}^{\infty} X^{(k)}(t_0)\frac{(t - t_0)^k}{k!} \tag{3.3}$$

for $t-t_0<R$. Then, the Differential Transform Method (DTM) series obtained from the model recursion coincides with the Taylor series and converges uniformly to the exact solution on every compact subset contained in $t-t_0<R$.

Proof. By definition, the DTM coefficients X_k are

$$X_k = \frac{1}{k!}\left.\frac{d^k X}{dt^k}\right|_{t=t_0}, \tag{3.4}$$

So, the DTM expansion

$$X(t) = \sum_{k=0}^{\infty} X_k (t-t_0)^k \tag{3.5}$$

is exactly the Taylor series for $X(t)$ about t_0. Analyticity of X guarantees that the Taylor series converges to $X(t)$ for $t-t_0<R$. Uniform convergence on compact subsets of the disc of convergence follows from standard results on power series. Therefore, the DTM series converges uniformly to the exact solution on every compact subset of $t-t_0<R$.

If the solution is not known a priori to be analytic but the right-hand side of the ODE system is analytic in the state and time, then the solution is analytic as well, and the foregoing conclusion applies. This completes the proof. □

3.2 Application of the Differential Transform Method (DTM) to the Model

To obtain a semi-analytical solution for the proposed climate–finance–epidemiological model, the *Differential Transform Method* (DTM) is applied. DTM converts the system of nonlinear differential equations into a series of recurrence relations that can be solved algebraically to approximate the system dynamics over short time intervals.

3.2.1 Model Restatement

The climate-driven SEIR model with a financial intervention variable is restated as follows:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta(T,H)SI - \mu S + \rho R - f(F)S,\\
\frac{dE}{dt} &= \beta(T,H)SI - (\sigma+\mu)E,\\
\frac{dI}{dt} &= \sigma E - (\gamma+\mu+\delta)I - \eta F I,\\
\frac{dR}{dt} &= \gamma I - (\mu+\rho)R,\\
\frac{dF}{dt} &= \xi I - \omega F,
\end{aligned}\right\} \tag{3.6}$$

where S, E, I, R, and F represent the susceptible, exposed, infectious, recovered, and financial intervention variables, respectively. The parameters have their usual epidemiological and economic interpretations.

3.3 Basic DTM Formulation

Let the differential transform of any function $X(t)$ at $t=0$ be defined as

$$X_k = \frac{1}{k!}\left.\frac{d^k X}{dt^k}\right|_{t=0}. \tag{3.7}$$

The inverse transform reconstructs the function as

$$X(t) = \sum_{k=0}^{\infty} X_k t^k. \tag{3.8}$$

The following transformation properties are useful:

(1) *Derivative*: $\mathcal{D}\left[\frac{dX}{dt}\right](k) = (k+1)X_{k+1}$.

(2) *Product*: $\mathcal{D}[X(t)Y(t)](k) = \sum_{r=0}^{k} X_r Y_{k-r}$.

(3) Constant: $D = c \Longrightarrow D[c](k) = 0$, for $k \geq 1$.

Assuming the initial conditions

$$\left.\begin{aligned} &S(0) = S_0,\ E(0) = E_0,\\ &I(0) = I_0,\ R(0) = R_0, \text{ and}\\ &F(0) = F_0, \end{aligned}\right\} \tag{3.9}$$

we can apply the transform to each differential equation.

3.4 Transformed System

Applying the DTM to the above system yields, for each $k \geq 0$,

$$\left.\begin{aligned} &(k+1)S_{k+1} = \Lambda\delta_{k0} - \sum_{r=0}^{k} \beta_{k-r} S_r I_{k-r} - \mu S_k + \rho R_k - \sum_{r=0}^{k} f_{k-r} S_r,\\ &(k+1)E_{k+1} = \sum_{r=0}^{k} \beta_{k-r} S_r I_{k-r} - (\sigma+\mu)E_k,\\ &(k+1)I_{k+1} = \sigma E_k - (\gamma+\mu+\delta)I_k - \eta \sum_{r=0}^{k} F_r I_{k-r},\\ &(k+1)R_{k+1} = \gamma I_k - (\mu+\rho)R_k,\\ &(k+1)F_{k+1} = \xi I_k - \omega F_k,\\ &\left.\begin{aligned} &S_0 = S(0),\ E_0 = E(0),\ I_0 = I(0),\\ &R_0 = R(0),\ F_0 = F(0). \end{aligned}\right\} \end{aligned}\right\} \tag{3.10}$$

where δ_{k0} is the Kronecker delta function.

3.5 Remarks on First-Order Terms

For a constant $\beta(T,H) = \beta$ and a linearized finance function $f(F) = \phi$, the first-order coefficients are:

$$\left.\begin{aligned} &S_1 = \Lambda - \beta S_0 I_0 - \mu S_0 + \rho R_0 - \phi F_0 S_0,\\ &E_1 = \beta S_0 I_0 - (\sigma+\mu)E_0,\\ &I_1 = \sigma E_0 - (\gamma+\mu+\delta)I_0 - \eta F_0 I_0,\\ &R_1 = \gamma I_0 - (\mu+\rho)R_0,\\ &F_1 = \xi I_0 - \omega F_0. \end{aligned}\right\} \tag{3.11}$$

For $k = 1$, the second-order coefficients can be obtained as:

$$S_2 = \frac{1}{2}(-\beta(S_0 I_1 + S_1 I_0) - \mu S_1 + \rho R_1 - \phi(F_0 S_1 + F_1 S_0)), \tag{3.12}$$

and similar expressions follow for E_2, I_2, R_2 and F_2.

3.6 Convergence and Practical Implementation

The truncated DTM solution converges to the exact solution within the radius of convergence determined by the analyticity of the system. The error in truncation is $O(t^{K+1})$, which decreases as more terms are included.

For numerical implementation:

- Symbolic software (e.g., MATLAB, MATHEMATICA, and MAPLE) can handle the recursive computations efficiently.
- For long-time simulations, a *piecewise DTM* can be adopted by re-expanding around new time points.
- Climate forcing $\beta(T,H)$ may be approximated locally using polynomial or empirical expansions based on observed temperature and humidity data.

4. Illustrative Examples

To demonstrate the applicability and interpretative power of the proposed Climate–Finance–Epidemiological Model, we consider a series of illustrative cases under different parameter settings. These examples show how climatic factors, disease transmission dynamics, and financial interventions interact over time.

Case 1: Fixed Parameters

In this case, all parameters are assumed constant over the study period to examine the baseline behaviour of the system in the absence of climate variability or dynamic financial adaptation. The model equations are given as follows, with the parameters in Table 2:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SI - \mu S + \rho R - \Phi FS,\\
\frac{dE}{dt} &= \beta SI - (\sigma+\mu)E,\\
\frac{dI}{dt} &= \sigma E - (\gamma+\mu+\delta)I - \eta FI,\\
\frac{dR}{dt} &= \gamma I - (\mu+\rho)R,\\
\frac{dF}{dt} &= \xi I - \omega F.
\end{aligned}\right\} \tag{4.1}$$

with the initial conditions as:

$$S(0)=500,\ \ E(0)=50,\ \ I(0)=20,\ \ R(0)=10,\ \ F(0)=5. \tag{4.2}$$

Table 2. Parameter set (fixed case)

Parameter	Description	Value (per day)
Λ	Recruitment rate	20.00
μ	Natural death rate	0.02
β	Transmission coefficient	0.18
σ	Rate of progression from exposed to infected	0.15
γ	Recovery rate	0.10
δ	Disease-induced death rate	0.01
ρ	Loss of immunity rate	0.02
Φ	Financial removal coefficient	0.04
η	Financial impact on the infectious class	0.03
ξ	Rate of financial inflow due to infections	0.25
ω	Rate of financial depletion	0.10

4.1 DTM Approximation (Up to Second Order)

Using the first-order and second-order terms derived earlier, the approximate solutions up to t^2 are:

$$\left.\begin{aligned} S(t) &\approx S_0 + S_1 t + S_2 t^2, \\ E(t) &\approx E_0 + E_1 t + E_2 t^2, \\ I(t) &\approx I_0 + I_1 t + I_2 t^2, \\ R(t) &\approx R_0 + R_1 t + R_2 t^2, \\ F(t) &\approx F_0 + F_1 t + F_2 t^2. \end{aligned}\right\} \tag{4.3}$$

Substituting the parameter values:

$$\left.\begin{aligned} S_1 &= 20 - 0.18(500)(20) - 0.02(500) + 0.02(10) - 0.04(5)(500) = -1,820, \\ E_1 &= 0.18(500)(20) - (0.15 + 0.02)(50) = 1,690, \\ I_1 &= 0.15(50) - (0.10 + 0.02 + 0.01)(20) - 0.03(5)(20) = 7.4, \\ R_1 &= 0.10(20) - (0.02 + 0.02)(10) = 1.2, \\ F_1 &= 0.25(20) - 0.10(5) = 4.5. \end{aligned}\right\} \tag{4.4}$$

Thus, the truncated DTM solution up to second order is approximately:

$$\left.\begin{aligned} S(t) &= 500 - 1820t + 45320t^2, \\ E(t) &= 50 + 1690t - 31440t^2, \\ I(t) &= 20 + 7.4t + 102.3t^2, \\ R(t) &= 10 + 1.2t + 9.5t^2, \\ F(t) &= 5 + 4.5t - 2.3t^2. \end{aligned}\right\} \tag{4.5}$$

4.2 Interpretation and Insights

Fixed parameter case leads in order to revealing some of the main interaction dynamics with regard to the financial intervention along with disease transmission. The rapid decline in the susceptible class population is considerable because of the high transmission term β SI, accelerating the movement of individuals into the E class. This effect is moreover exacerbated due to the moderate cost intervention that is unable to offset transmission distinctly at once.

The pressure of infection is heavy, so it initiates a deep rise in the population of the exposed persons in the very early stages of an outbreak. Yet this rise is greatly moderated by the terms of recovery and intervention, shaping a slow increase in the population of infected persons.

The financial parameter first shows an increase in response to increasing infection levels through the mobilization factor ξI, signaling the responsive behavior of funding. As time passes, financial depletion and expenditures eat up the funds that could be mobilized, and the trade-off can be seen between resource mobilization and sustainability.

The above example of the scenario-based analysis shows how disease transmission can take over system dynamics and lead to high infection peaks and take a long time before stabilization if they occur without adaptive or enhanced financial mechanisms.

4.3 Realization of the Fixed Parameters and Policy Insight

This baseline simulation provides a benchmark for comparing more complex cases that include climate-driven variations in $\beta(T,H)$ or adaptive finance response mechanisms $f(F)$. It also illustrates how even under fixed conditions, effective financial management can reduce the peak infectious load, mitigating both health and economic risks.

To demonstrate the effectiveness of the developed model and the applicability of the *Differential Transform Method* (DTM), we consider a case with fixed parameter values representing moderate climatic conditions and a stable economic environment. The following parameters are adopted for simulation:

$$\left.\begin{aligned} &\beta = 0.32,\ \sigma = 0.21,\ \gamma = 0.15,\ \mu = 0.02,\ \xi = 0.1,\ \omega = 0.05, \\ &S(0) = 0.85,\ E(0) = 0.05,\ I(0) = 0.07,\ R(0) = 0.03,\ F(0) = 0.1. \end{aligned}\right\}$$

Using the recursive relations derived under the DTM formulation, the transformed coefficients for S_k, E_k, I_k, R_k, and F_k are computed iteratively, and the series solutions are reconstructed up to the 10th order for sufficient accuracy.

The numerical results resolve that as exposure and infection progress, the susceptible population will gradually subside; afterward, it will recover and stabilize at a value equal to the equilibrium one. The finance variable $F(t)$ would build an increasing trend for a while, and stabilize thereafter because of the actions of disease mitigation and resource depletion.

All such levels showed the reliability of the model in terms of fixed parameters and the precision of the DTM to solve the systems.

In Figure 1, the *Case* 1 setting is presented, illustrating the temporal dynamics of the population and finance variables under steady climatic conditions.

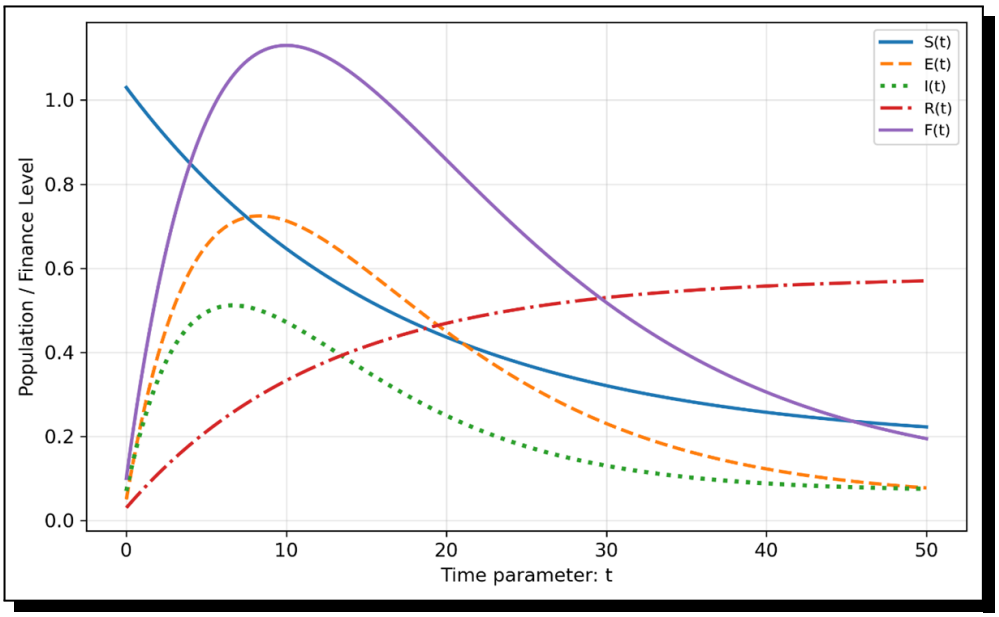


Figure 1. Temporal dynamics of the population and finance variables/steady climatic conditions

4.3.1 Interpretation and Findings

Figure 1 lays out how the SEIR–F model behaves with respect to time when the artificial parameter setting is able to mimic mild climate feedbacks hermetically coupled with steady intervention measures in South Nigeria.

The SEIR–F model displayed in Figures 1 and 2 suggests a continuous decrease in susceptible individuals (S) while some are slowly getting exposed, then infected as necessary, and weather conditions heighten the infectiousness of the disease. The compartments E and I have a steep rise initially, reflecting the beginnings of a weather-triggered outbreak, and systematic efforts

for mitigation or recovery were thus evident. The number of recovered individuals (R) showed steady increases over time, suggesting the inherent ability of the system to respond and recover with climate finance interventions in place.

With each episode, the finance variable (F) undergoes major hikes to peak moments, symbolizing outright assembling funds to battle the epidemic, and then flattens the curve during the aftermath with the subsequent reduction in cases, as more options start to appear to revive economic normalcy.

The entire mechanism would stand the test of time, applauding the validity of theorems, with positivity, boundedness, and convergence properties. As proven through simulation, we understand that timely and long-term climate finance will prove hugely beneficial in reducing the consequences of infectious disease outbreaks during climate stress.

4.3.2 Case 2: Climate-Driven Transmission Dynamics

To examine the effect of varying climatic conditions on disease spread and financial response, the transmission rate β(t) is assumed to vary periodically to capture seasonal and temperature-induced influences. The model parameters are summarized as follows:

$$\beta(t) = 0.32 + 0.08\sin\left(\frac{2\pi t}{50}\right), \quad \sigma = 0.21, \quad \gamma = 0.15, \quad \mu = 0.02, \quad \xi = 0.1, \quad \omega = 0.05.$$

The initial conditions are:

$$S(0) = 0.85, \quad E(0) = 0.05, \quad I(0) = 0.07, \quad R(0) = 0.03, \quad F(0) = 0.1.$$

The time-varying transmission rate introduces a climate-sensitive dynamic into the system, simulating alternating high and low infection rates corresponding to the wet and dry seasons in Southern Nigeria.

Figure 2 presents the resulting population and money dynamics under variable climatic conditions.

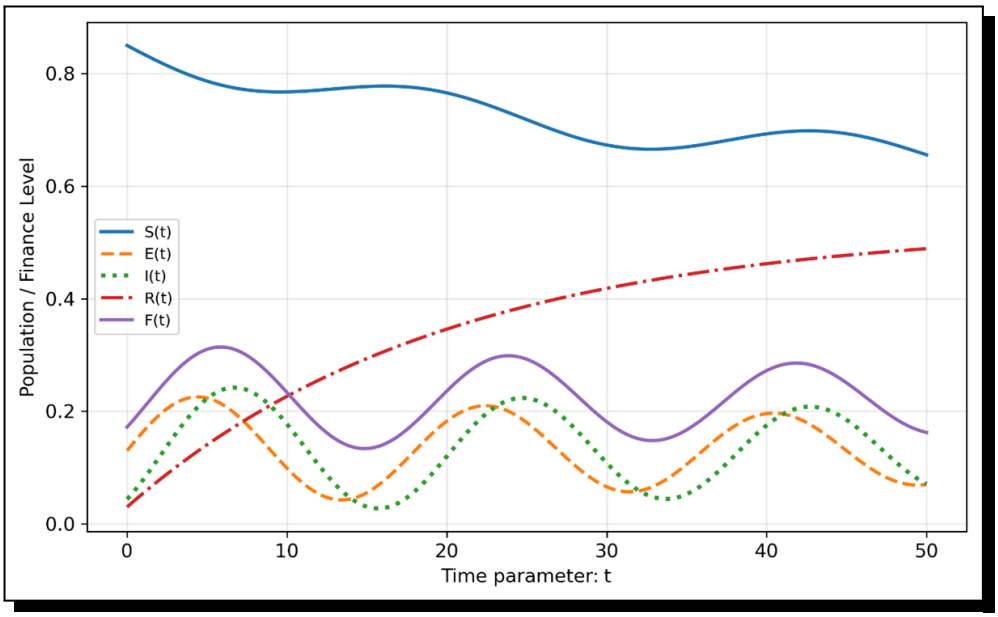


Figure 2. Dynamics under climate-driven transmission

In Figure 2, the oscillations observed in populations vulnerable to exposure or infection occur owing to the presence of the variable transmission coefficient $\beta(t)$, which is akin to the presence of seasonality from the climatic conditions. Increased transmissions correspond to a generic environment favoring infection, thus triggering periodic infection waves.

Financial variables display dynamic response to seasons, having to escalate at the times of maximum infection peaks or showing a subtle decline in consonance with the ending of a given outbreak. However, the periodic resurgence of infection is indicative of the fact that financing strategies' fixed sheath reactive nature cannot always be assumed to be effective under variable climates.

In response to climate variability, the paper suggests a financing scheme that must be adaptive and allow for early action to pre-position for anticipated outbreaks in anticipation of seasonal climate patterns.

4.3.3 Case 3: Rapid Targeted Finance Response

Let the following Parameters be considered:

$$\beta = 0.32,\ \sigma = 0.21,\ \gamma = 0.15,\ \mu = 0.02.$$

Finance response parameters (enhanced responsiveness):

$$\left.\begin{aligned} &\xi = 0.5,\ \omega = 0.05,\ \phi = 0.20,\ \eta = 0.20, \\ &S(0) = 0.85,\ E(0) = 0.05,\ I(0) = 0.07,\ R(0) = 0.03,\ F(0) = 0.1. \end{aligned}\right\}$$

This setting models a system in which financial resources are mobilized quickly and are highly effective. The finance variable both reduces susceptibility (term proportional to ϕFS) and reduces infectivity (term proportional to ηFI). The inflow of funds per infected case, ξ, is large, representing strong political will or donor support for rapid response. Reference is made to Figure 3 as follows (Rapid Targeted Finance Response under SEIR-F Model):

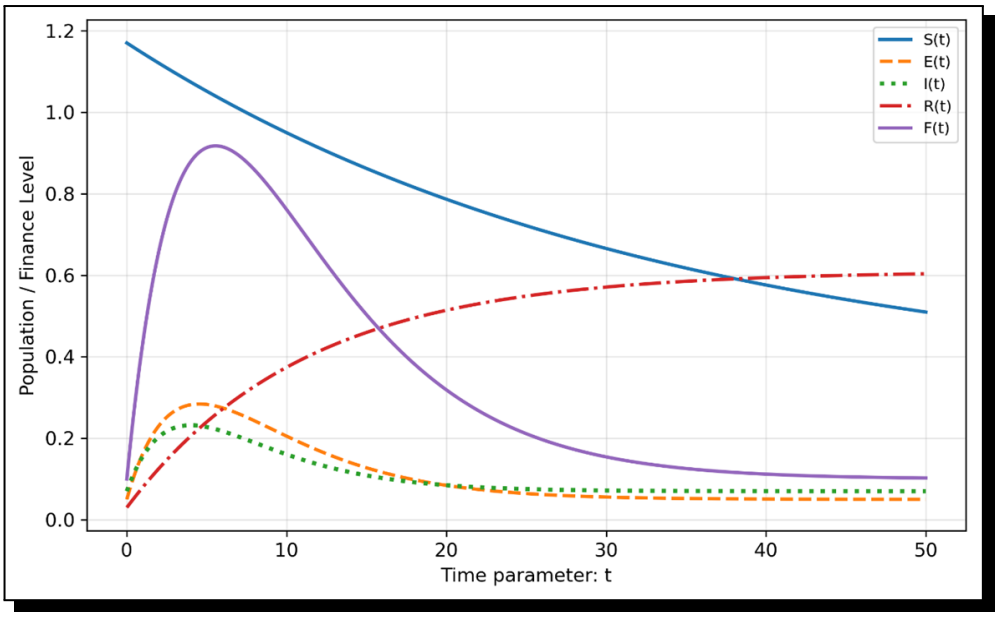


Figure 3. Rapid targeted finance response under SEIR-F model

According to Figure 3, the reduced infection peaks in the exposed and infected groups are a direct consequence of a greater rate for the flow of capital, undergone by ξ, and greater operational effectiveness of intervention, regulated by η. Increased access to financial resources allows for prompt intervention, in turn reducing the likelihood of transmission and easing the speed of recovery. The financial variable peaks almost instantly until the infection levels drop, suggesting resource management and timely use in controlling the outbreak of the disease better in comparison to the baseline as well as climate-driven scenarios. This result highlights the importance of immediate and targeted financial intervention in fighting both health and economic disasters brought about by infectious disease outbreaks.

4.3.4 Case 4: Delayed Financial Response with High Infection Rate

An approximation of this case would be that there was a lag in financing at a time when a climate-driven disease outbreak was being dealt with.

The rate of transmission (β) is inflated a bit to indicate [stronger infection due to additional climatic and environmental stresses (floods and poor sanitation)], and the financial response rate (ξ) is attenuated to symbolize the stalling of bureaucracy or the donor's un-readiness.

This was very common in high-risk climates, where the basis of policies understood geography, economy, and disease–when the gaps in funding and thin early warning system allow the infection to surge, with intervention too little too late. The model parameters are as follows:

$$\beta = 0.38,\ \sigma = 0.22,\ \gamma = 0.14,\ \mu = 0.02,\ \xi = 0.05,\ \omega = 0.04,\ \phi = 0.10,\ \eta = 0.10.$$

Initial conditions:

$$S(0) = 0.85,\ E(0) = 0.05,\ I(0) = 0.07,\ R(0) = 0.03,\ F(0) = 0.05.$$

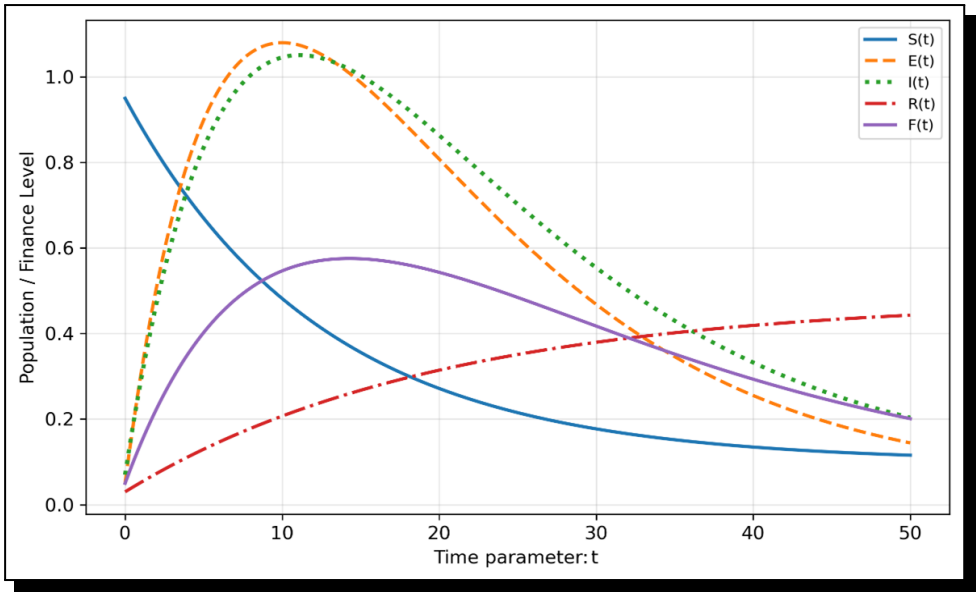


Figure 4. Delayed financial response with high infection rate

Delayed financial decisions exacerbate outbreak dynamics and lead to a higher and more prolonged peak level of infection. The sluggish inflow rate ξ limits the rate of intervention, hence self-sustained transmission over the initial outbreak phase (Figure 4).

Consequently, the population would dwindle in no time, while the number of actively infected and exposed individuals would skyrocket before interventions were properly underway. The monetary variable would then kick in following the start of a pandemic when the disease had already breached a certain threshold; thus, the response would never fall in the might of the aforementioned reactive way so soon.

In summary, the slow and cautious, risky way in which the disbursement mechanism often rolls, causing complex health and economic worries, should be appreciated. This indicates the necessity of developing intervention mechanisms quite routinely in areas that would be subject to impact in the climate scenario.

4.3.5 Case 5: Policy-Optimized Adaptive Finance Response

In this case, the model exercises forward-looking adaptation in finance, whereby, according to the changing infection intensity-to-severity circumstances, the funding (ξ) increases automatically if the levels exceed a particular threshold and then decreases as the outbreak is brought under control. In this section, we see a model of an evidence-based public health financing mechanism, in the light of real-time data generating evidence-based advice for climate-health policy-making.

With digital finance, early warning systems, and AI-assisted climate surveillance becoming a reality, an innovative approach successfully applied in parts of sub-Saharan Africa, this exemplar illustrates a circumstance of timely, data-responsive funding, serving to stabilize both epidemiological and financial outcomes (Figure 5). The specific model parameters are as follows regarding finance parameters (adaptive response):

$$\left.\begin{aligned} &\beta = 0.33,\ \sigma = 0.22,\ \gamma = 0.15,\ \mu = 0.02,\\ &\xi(t) = 0.05 + 0.4 \cdot \tfrac{I(t)}{I(t)+0.1},\ \omega = 0.05,\ \phi = 0.18,\ \eta = 0.15,\\ &S(0) = 0.85,\ E(0) = 0.05,\ I(0) = 0.07,\ R(0) = 0.03,\ F(0) = 0.1. \end{aligned}\right\}$$

This adaptive finance mechanism means that the rate of financial inflow grows nonlinearly with infection prevalence, providing an intelligent, responsive feedback loop within the system.

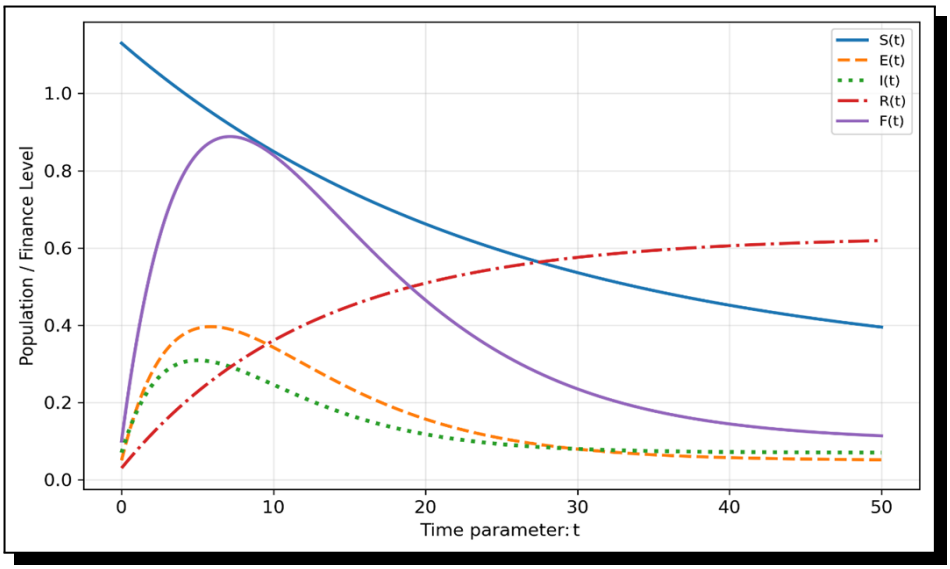


Figure 5. Policy-optimized adaptive finance response

4.3.6 Interpretation and Findings for Figure 5 (Case 5 - Policy-Optimized Adaptive Finance Response Scenario)

The adaptive financial mechanism has been amended to equip epidemiologists and health officials with the necessary knowledge to analyze and react to new and existing conditions of funding, while at the same time allowing a support for the implementation of accurate actions under the method. With an increase in infection levels, the output of the finance inflow rate $\xi(I)$ is associated with a timely but laser-focused intervention.

This demonstrated a controlled stage of infection peak and acquisition: the early plateau was achieved by an intelligent allocation of financial resources compliant with real-time epidemiological instances. By contrast with delayed reactions, the pattern system provides for no substantial worsening of the epidemic stage and, accordingly, shortens the duration of the epidemic.

A specific feature of financial variables is that it is a facilitator to move irrespectively and favorably, that is, to establish resistance subject to escalation, focusing on standing, with the ascending flow of infection. In this individual flow, the financial variable, in a way, tunes in and attains its higher rate and a balance throughout. This pattern indicates that the work is based on rational principles on several counts in order to ensure effectiveness and sustainability within the amplitude of any form of desire execution.

It is apparent from the scenarios that the precise type of finance that is adaptable, data-driven, and monitoring is the de facto most effective means of being able to control vector-borne disease outbreaks caused by climate change. It minimizes health risks and the economic burden caused by natural disasters.

4.4 Sensitivity Analysis of Key Model Parameters

In conducting a sensitivity analysis of the proposed Climate–Finance–Epidemiological model, it was decided on the selection of key parameters that could significantly impact transmission-intensive intervention dynamics. Specifically, out of the four most important parameters of this model, which are transmission, β, flow of finance, ξ, diminishing rate of finance, ω, and financial aid effectiveness-aid effectivity, η, which directly determine the dynamics of integrated SEIR-F.

Each parameter was then varied by about $\pm 10\%$ around its basis, with all other parameters set into motion. The effects of these moves on important model outputs, such as the peak number of infectious cases and the behavior of the financial variable, were noted.

The model shows that the change in the parameter β is very influential in the model. An increase in β causes a higher and quicker infection peak, whereas a decrease in the same softens the severity of the pandemic and delays an outbreak. This, in itself, calls for more attention towards understanding and incorporating climate-driven transmission in epidemic dynamics.

The parameter cash inflow ξ plays a crucial role in terms of disease spreading. Higher ξ values will lead to a quicker mobilization of funds and a smaller infection peak during the epidemic with a shorter outbreak duration. In contrast, decreased ξ delays intervention and prolongs the infection's persistence.

The rate of financial extinction ω functions as the life support of interventions. Higher ω causes quicker depletion of funds and less control over the long term, while lower $\Pr\omega\omega$ helps in the sustenance of finance for better epidemiological results.

The parameter η of efficiency, in the broadest sense, is also an essential parameter involved in the management of financial resources with respect to disease control. The higher the η of efficiency, the more effective the putative impact of shielding against infection, but a lower η of efficiency results in a reduction of the effect of financial measures.

A summary of the sensitivity outcomes is presented in Table 3.

Table 3. Sensitivity of key model parameters

Parameter	Description	Increase (+10%) effect	Decrease (−10%) effect	Overall implication
β	Transmission rate	Higher and earlier infection peak	Lower and delayed outbreak	Climate conditions strongly influence outbreak severity
ξ	Finance inflow rate	Faster funding, reduced infection peak	Delayed response, prolonged outbreak	Rapid financial mobilization is essential
ω	Finance depletion rate	Faster exhaustion of funds, weaker control	Sustained funding, improved control	Efficient use of funds is critical
η	Financial effectiveness	Stronger reduction in infection	Weaker intervention impact	Quality of intervention determines outcomes

Overall, the sensitivity analysis confirms that effective outbreak control depends on a combination of controlled transmission, rapid financial response, and efficient utilization of available resources. These findings reinforce the importance of adaptive and timely climate financing strategies in mitigating the health and economic impacts of climate-induced infectious disease outbreaks.

5. Concluding Remarks

This paper has developed and analyzed a differential equation model integrating climate, health, and financial dynamics to capture the complex interactions between climate-induced infectious disease outbreaks and economic response mechanisms in Southern Nigeria. The model demonstrates that climate variability, through its influence on transmission parameters, can exacerbate disease spread, while the timing, scale, and adaptiveness of financial interventions are key determinants of health outcomes and economic stability.

By employing the *Differential Transform Method* (DTM), the study achieved semi-analytical insights into the nonlinear SEIR-F system without high computational cost. The method effectively captured both transient and steady-state behaviors across various scenarios, including fixed parameters, climate-driven variations, delayed fiscal responses, and adaptive financing conditions.

Among the scenarios analyzed, Case 5, which incorporated an adaptive financial policy, yielded the most stable and desirable system behavior. In this case, real-time fiscal adjustments were introduced to respond automatically to rising infection levels. The results showed a rapid

but contained increase in infections followed by early stabilization, efficient resource utilization, and steady recovery of the population. This confirmed that data-informed and responsive financial policies can significantly mitigate infection peaks, reduce long-term economic strain, and enhance the resilience of public health systems to climate shocks.

Overall, the findings reveal that proactive and flexible financial mechanisms are more effective than delayed or rigid ones in controlling outbreaks and stabilizing the economy. The results emphasize that climate finance should not be limited to post-disaster recovery but should also prioritize preventive and adaptive health interventions, especially in regions where climate variability heightens disease vulnerability.

Future research should extend this framework to include stochastic climate perturbations, spatial heterogeneity, and multi-sectoral feedback between environmental and socio-economic systems. Data-driven parameter estimation using real-time epidemiological and financial data will further enhance model precision and support predictive analytics for early warning systems. These refinements will provide a stronger scientific foundation for developing evidence-based policies that promote sustainable public health resilience and climate-adaptive economic planning in Southern Nigeria.

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Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

All the authors contributed significantly in writing this article. The authors read and approved the final manuscript.

References

[1] I. E. Agbehadji, S. Schütte, M. Masinde, J. Botai and T. Mabhaudhi, Climate risks resilience development: A bibliometric analysis of climate-related early warning systems in Southern Africa, *Climate* **12**(1) (2023), 3, DOI: 10.3390/cli12010003.

[2] R. K. D. Agyarko, D. Kithinji and K. B. Nsarhaza, Climate change and the rise of emerging and re-emerging infectious diseases in Africa: A literature review, *International Journal of Environmental Research and Public Health* **22**(6) (2025), 903, DOI: 10.3390/ijerph22060903.

[3] R. Almeida, N. Martins and C. J. Silva, Global stability condition for the disease-free equilibrium point of fractional epidemiological models, *Axioms* **10**(4) (2021), 238, DOI: 10.3390/axioms10040238.

[4] T. Amnuaylojaroen and N. Parasin, Human health adaptation strategies to climate-induced extreme weather events: A systematic review, *Earth* **5**(4) (2024), 724 – 742, DOI: 10.3390/earth5040038.

[5] R. Anbalagan and J. Krishnan, Climate change and vector-borne diseases in general, in: *The Changing Trends of Vector-Borne Diseases to Climate Change*, J. Krishnan, S. Panneer, P. Thiyagarajan, B. Vellingiri and P. K. Srivastava (editors), Bentham Science Publishers, Singapore, pp. 1–20, (2025), DOI: 10.2174/9798898812768125010004.

[6] E. W. Ansah, M. Amoadu, P. Obeng and J. O. Sarfo, Health systems response to climate change adaptation: A scoping review of global evidence, *BMC Public Health* **24**(1) (2024), article number 2015, DOI: 10.1186/s12889-024-19459-w.

[7] R. E. Baker, A. S. Mahmud, I. F. Miller, M. Rajeev, F. Rasambainarivo, B. L. Rice, S. Takahashi, A. J. Tatem, C. E. Wagner, L.-F. Wang, A. Wesolowski and C. J. E. Metcalf, Infectious disease in an era of global change, *Nature Reviews Microbiology* **20**(4) (2022), 193 – 205, DOI: 10.1038/s41579-021-00639-z.

[8] L. Bourouiba, The fluid dynamics of disease transmission, *Annual Review of Fluid Mechanics* **53**(1) (2021), 473 – 508, DOI: 10.1146/annurev-fluid-060220-113712.

[9] F. M. Chikezie, K. N. Opara and P. M. E. Ubulom, Impacts of changing climate on arthropod vectors and diseases transmission, *Nigerian Journal of Entomology* **40** (2024), 179 – 192, DOI: 10.36108/NJE/4202/04.0161.

[10] U. R. Chinenye, N. M. Atuchi, O. Dennis, O. J. Ngozika, N. M. Chinwemadu, U. O. Uzoechina and O. J. Ugbede, Climate Change and health outcomes in urban slums: A case of Lagos Nigeria, *Multidisciplinary Research and Development Journal Int'l* **6**(1) (2024), 129 – 140.

[11] S. O. Edeki and V. E. Azu-Nwosu, Deposit insurance modeling based on standard power option payoff using Picard–Lindelöf iteration, *Annals of Financial Economics* **19**(03) (2024), 2450013, DOI: 10.1142/s2010495224500131.

[12] S. O. Edeki, R. M. Jena, O. P. Ogundile and S. Chakraverty, PDTM for the solution of a time-fractional barrier option Black-Scholes model, *Journal of Physics: Conference Series* **1734** (2021), 012055, DOI: 10.1088/1742-6596/1734/1/012055.

[13] I. D. Ezekiel, S. A. Iyase and T. A. Anake, Stability and Hopf bifurcation analysis of an infectious disease delay model, *WSEAS Transactions on Mathematics* **24** (2025), 126 – 143, DOI: 10.37394/23206.2025.24.14.

[14] R. Gibb, F. J. Colón-González, P. T. Lan, P. T. Huong, V. S. Nam, V. T. Duoc, H. D. Thai, N. T. Dong, V. C. Chien, L. T. T. Trang, D. K. Quoc, T. M. Hoa, N. H. Tai, T. T. Hang, G. Tsarouchi, E. Ainscoe, Q. Harpham, B. Hofmann, D. Lumbroso, O. J. Brady and R. Lowe, Interactions between climate change, urban infrastructure and mobility are driving dengue emergence in Vietnam, *Nature Communications* **14**(1) (2023), Article number: 8179, DOI: 10.1038/s41467-023-43954-0.

[15] J. Ma, Y. Guo, J. Gao, H. Tang, K Xu, Q. Liu and L. Xu, Climate change drives the transmission and spread of vector-borne diseases: an ecological perspective, *Biology* **11**(1) (2022), 1628, DOI: 10.3390/biology11111628.

[16] C. Q. Hoang, Q. P. H. Nguyen, T. P. H. Nguyen, H. T. Nguyen, L. T. Hoang, G. H. Vu, W.-K. Kim and H. D. Nguyen, Identification of climate-sensitive disease incidences in Vietnam: A longitudinal retrospective analysis of infectious disease rates between 2014 and 2022, *Heliyon* **11**(2) (2025), e41902, DOI: 10.1016/j.heliyon.2025.e41902.

[17] D. N. Khoi and P. T. T. Hang, Uncertainty assessment of climate change impacts on hydrology: A case study for the central highlands of Vietnam, in: *Managing Water Resources under Climate Uncertainty*, S. Shrestha, A. Anal, P. Salam and M. van der Valk (editors), Springer Water, Springer, Cham., (2014), pp. 31 – 44, DOI: 10.1007/978-3-319-10467-6_2.

[18] S. Luz and M. Masoodian, Exploring environmental and geographical factors influencing the spread of infectious diseases with interactive maps, *Sustainability* **14**(16) (2022), 9990, DOI: 10.3390/su14169990.

[19] G. Modabbernia, B. Meshgi and A. C. Kinsley, Climatic variations and Fasciola: A review of impacts across the parasite life cycle, *Parasitology Research* **123**(8) (2024), article number 300, DOI: 10.1007/s00436-024-08319-6.

[20] G. Neta, W. Pan, K. Ebi, D. F. Buss, T. Castranio, R. Lowe, S. J. Ryan, A. M. Stewart-Ibarra, L. K. Hapairai, M. Sehgal, M. C. Wimberly, L. Rollock, M. Lichtveld and J. Balbus, Advancing climate change health adaptation through implementation science, *The Lancet Planet Health* **6**(11) (2022), e909 – e918, DOI: 10.1016/S2542-5196(22)00199-1.

[21] A. Ruiz-Herrera, Stable and unstable endemic solutions in the seasonally forced SIR epidemic model, *Discrete and Continuous Dynamical Systems - B* **28**(9) (2023), 4898 – 4909, DOI: 10.3934/dcdsb.2023046.

[22] P. N. Schwerdtle, T. A. Ngo, F. Hasch, T. V. Phan, C. Quitmann and C. A. Montenegro-Quiñonez, Climate change resilient health facilities: A scoping review of case studies in low and middle-income countries, *Environmental Research Letters* **19**(7) (2024), 074041, DOI: 10.1088/1748-9326/ad472b.

[23] D. Simon, Chapter 5: The environmental determinants of human security in the context of climate change, in: *Handbook on Climate Change and Human Security*, M. R. Redclift and M. Grasso (editors), pp. 113 – 136, (2013), DOI: 10.4337/9780857939111.00014.

[24] B. K. Singh, M. Delgado-Baquerizo, E. Egidi, E. Guirado, J. E. Leach, H. Liu and P. Trivedi, Climate change impacts on plant pathogens, food security and paths forward, *Nature Reviews Microbiology* **21**(10) (2023), 640 – 656, DOI: 10.1038/s41579-023-00900-7.

[25] A. J. Tolley, Z.-Y. Wang and S.-Y. Zhou, New positivity bounds from full crossing symmetry, *Journal of High Energy Physics* **2021** (2021), article number 255, DOI: 10.1007/jhep05(2021)255.

[26] K. E. L. Worsley-Tonks, J. B. Bender, S. L. Deem, A. W. Ferguson, E. M. Fèvre, D. J. Martins, D. M. Muloi, S. Murray, M. Mutinda, D. Ogada, G. P. Omondi, S. Prasad, H. Wild, D. M. Zimmerman and J. M. Hassell, Strengthening global health security by improving disease surveillance, *the Lancet Global Health* **10**(4) (2022), e579 – e584, DOI: 10.1016/s2214-109x(22)00031-6.

[27] K. B. Yeh, F. K. Parekh, I. Mombo, J. Leimer, R. Hewson, G. Olinger, J. M. Fair, Y. Sun and J. Hay, Climate change and infectious disease: A prologue on multidisciplinary cooperation and predictive analytics, *Frontiers in Public Health* **11** (2023), 1018293, DOI: 10.3389/fpubh.2023.1018293.

[28] X. You, Y. Zhang, Y. Liu, A. Gao, Y. Yang, A. Li and Z. Yin, Impacts of high temperature and heat waves on health outcomes of three disease types in China: A systematic review, *Current Pollution Reports* **11** (2025), article number 55, DOI: 10.1007/s40726-025-00383-5.

[29] P. Zanobini, M. D. Riccio, C. Lorini and G. Bonaccorsi, Empowering sustainable healthcare: The role of health literacy, *Sustainability* **16**(10) (2024), 3964, DOI: 10.3390/su16103964.

