Modelling and Simulations Coursework
Zombie Outbreak Modelling

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Abstract

This report examines the dynamic of a fictional zombie outbreak using a SIR-based modelling approach. This study begins with a baseline SIR model and extends to incorporate a cure mechanism using Heaviside and logistic functions. analytical techniques such as non-dimensionalisation, steady-state analysis, and numerical simulations reveal critical factors influencing system behaviour. The final results highlights the importance of quick and effective intervention in controlling outbreaks.

1. Introduction

Imagine reading this report in your office at the Dyson Building on Imperial College's campus, only to be interrupted by groans outside the window. At first, you assume it's just the fourth years groaning about their master's projects, but soon you realise this is no ordinary affliction. The affected aren't simply unwell—they've become ravenous, reanimated corpses. This chilling scenario, often relegated to fiction, taps into humanity's deep-seated fear of losing control over life, death, and societal order.

Zombie outbreaks, though fictional, offer a fascinating parallel to real-world epidemics, where swift intervention and containment are critical. This report examines a hypothetical zombie outbreak using an SIR-inspired mathematical model to analyse key factors influencing epidemic dynamics. Starting with a base model of zombification, it extends to include a cure mechanism, simulating interventions like anti-viral medications. Analytical techniques, including non-dimensionalisation, steady-state analysis, and numerical simulations, highlight the roles of infection rates, cure effectiveness, and intervention timing. While fictional, the insights provide valuable perspectives on controlling real-world infectious diseases.

2. Base Model

To study the dynamics of a zombie outbreak, a baseline model must first be defined, along with its scope, parameters, and assumptions. The system is modelled using the SIR (Susceptible-Infectious-Recovered) framework, which allows for the incorporation of key assumptions and simplifications to focus on the essential aspects of the outbreak dynamics.

2.1 Scope of the Base Model

The model is limited to a fixed population in a closed environment where interactions between individuals drive the spread of the zombie infection. We will base this model of the paper 'WHEN ZOMBIES ATTACK!'.This assume homogeneous and instantaneous interaction among the population. The key variables are:

- $S(t)$: The number of susceptible individuals at time t
- $Z(t)$: The number of zombies at time t
- $R(t)$: The number of dead individuals at time t

The parameters include:

- α : Zombie Removal Rate
- β : Infection Rate
- ζ : Zombie Resurrection Rate
- δ : Susceptible Deathrate
- Π : Susceptible Birthrate

2.2 Assumptions

2.2.1 Small Time Scale

As the system plays out over a relatively short period of time, the assumption is that there will be no births, deaths, or migration in or out of the system to simplify the dynamics by focusing solely on disease spread. We will still include them as a variable but will assume ($\Pi = \delta = 0$) during numerical simulations.

2.2.2 Homogeneous Mixing

Every individual in the population has has an equal probability of interacting with each other. This ensures that the model can use averaged rates of zombification to reduce computational complexity.

2.2.3 Instantaneous Interaction

The rates of the interaction is proportional to the number of each population (e.g. $\beta SI)/.$ This is to ensure that we can use simple differential equation for the model without stochastic simulations.

2.2.4 Constant Parameters

All parameters are constant, zombies don't evolve to get better at infecting humans over time. This is to simplify calculations and focus on the inherent dynamics of the zombies.

2.2.5 Resurrection

Basing the zombies off from typical zombie media, dead bodies have a chance of reanimation (ζ).

2.3 System Behaviour

Therefore, this base model can be represented as:

$$\begin{aligned} S' &= \Pi - \beta SZ - \delta S \\ Z' &= \beta SZ + \zeta R - \alpha SZ \\ R' &= \delta S + \alpha SZ - \zeta R \end{aligned} \tag{1}$$

As this model is widely discussed in many literature, the key characteristics of this is that there are two outcomes: Zombies Domination ($S = 0,\ , Z \neq 0$) and Disease-Free State ($S \neq 0,\ , Z = 0$) - proving that human-zombie coexistence is impossible. This means that the disease-free equilibrium is always unstable and the doomsday equilibrium is asymptotically stable, meaning its more likely for the zombies to win unless humans act quickly. Therefore, in our iteration of this model, we will discuss on how the effectiveness and speediness of human response (introduction of cure to zombies) will impact the system.

3. Cure Model (Heaviside)

Throughout human history with infectious diseases, humans have consistently risen to the challenge by developing cures or vaccination to combat outbreaks. However, once the cure is manufactured, it takes time for it to be delivered to ground-zero, thus our new variable. Building on the base model, the cure model introduces the concept of a time-delayed cure delivery mechanism, where susceptible individuals can actively cure zombified individuals after a threshold time (τ) has passed, turning them back into humans. Therefore, this model assumes that the cure effectiveness depends on the interactions between susceptible individuals and zombies, requiring healthy humans to directly administer the cure to the zombies, where this behaviour is proportional to the populations of both groups, scaled

by the cure effectiveness rate (γ).

The new key parameters now include:

1. **The cure effectiveness (γ)**: This is under the assumption that the cure effectiveness will be constant and the cure have no shelf life.

2. **Time taken for the cure to be delivered (τ)**: This is under the assumption that past this threshold, the cure will be widely available, keeping γ constant.

For this iteration of the model, we will also assume instantaneous activation of the cure, represented by a Heaviside Step function. Where H is defined as,

$$H(t) = \begin{cases} 1, & \text{for } t \geq 1 \\ 0, & \text{for } t < 1 \end{cases} \tag{2}$$

The new model is therefore represented as,

$$\begin{aligned} S'(t) &= \Pi + \gamma S H(t-\tau) Z(t) - \beta S(t) Z(t) - \delta \\ Z'(t) &= \beta S(t) Z(t) + \zeta R(t) - \alpha S(t) Z(t) - \gamma S(t) Z(t) H(t-\tau) \\ R'(t) &= \delta + \alpha S(t) Z(t) - \zeta R(t) \end{aligned} \tag{3}$$

Right away, we can see that there would be 2 distinct behaviours between $t < \tau$ and $t \geq \tau$ due to the piecewise characteristic. As the system is discontinuous at $t = \tau$, we know that potential function (V) does not exist, meaning there will be no closed orbits (no need to do Poincaré map analysis).

3.1 Non-Dimensionalisation analysis

While we can generally see the characteristics of the model, we can derive the non-dimensionalised version of the system to better highlight the dominant interactions. Let us based the characteristic timescale on β (See Appendix **??**):

$$\begin{aligned} \frac{d\hat{S}}{d\hat{t}} &= \hat{\Pi} + \hat{\gamma}\hat{S} H(\hat{t} - \hat{\tau})\hat{Z} - \hat{S}\hat{Z} - \hat{\delta}, \\ \frac{d\hat{Z}}{d\hat{t}} &= \hat{S}\hat{Z} + \hat{\zeta}\hat{R} - \hat{\alpha}\hat{S}\hat{Z} - \hat{\gamma}\hat{S}\hat{Z} H(\hat{t} - \hat{\tau}), \\ \frac{\hat{R}}{\hat{t}} &= \hat{\delta} + \hat{\alpha}\hat{S}\hat{Z} - \hat{\zeta}\hat{R}. \end{aligned} \tag{4}$$

Now we can investigate the dominant terms in each equation. For the susceptible population, at early stages (when Z is small), the birth rate (Π) may dominate the system. However, generally, the $\hat{S}\hat{Z}$ is likely to be dominant. Once the cure is introduced, depending on γ, the cure may dominate the system. The opposite would be true for zombies. However the interaction between zombies and the removed population would be dominated by the resurrection rate.

3.2 Steady State Analysis

Knowing that γ and τ greatly impact the system, finding the transition in the system is critical. Most importantly is we want to know what would happen when the cure is fully active. As the model exhibit piecewise behaviour, we can investigate the 2 behaviours that this model encapsulates ($t < \tau$) and ($t > \tau$). Let logistic weight function equal to 1 with constant population ($\Pi = \delta = 0$).

3.2.1 $t < \tau$

$$\begin{aligned} -\beta SZ &= 0 \\ -\beta SZ + \zeta R - \alpha SZ &= 0 \\ \alpha SZ - \zeta R & \end{aligned} \tag{5}$$

Similar to the base equation, we have 2 trivial equilibria:

- Disease-Free Equilibrium $(S, Z, R) = (S, 0, 0)$, where there are only susceptible
- Doomsday Equilibrium $(S, Z, R = (S, 0, 0))$

Before the cure is delivered, co-existence between humans and zombies cannot exist. With β dominating the dynamics of the zero, by definition of an outbreak, the infection rate cannot be zero ($\beta \neq 0$). This means that the basic reproductive number (R_0) of this system is:

$$R_0 = \frac{\beta}{\alpha} \tag{6}$$

In this timeframe, the only way for humans to win is to kill more zombies than being turned into one.

3.2.2 $t \geq \tau$

However, once the cure has been delivered ($t \geq \tau$), the system of equations becomes (See Appendix A.2)

$$\begin{aligned} \gamma SZ - \beta SZ &= 0 \\ \beta SZ + \gamma R - \alpha SZ - \gamma SZ &= 0 \\ \alpha SZ - \zeta R &= 0 \end{aligned} \tag{7}$$

As expected, in this case we have 2 trivial equilibria. We know this because the linearity of the system remained constant and the difference between the new and the base model is the piecewise characteristic. This means that once the cures are delivered, the effective reproductive number changes to $R_{eff} = \frac{\beta+\alpha}{\alpha+\gamma}$. Therefore, if $R_eff < 1$, it would mean that the cure would successfully eradicate the zombie population over time. To analyse the stability to the equilibria of this system, the Jacobian can be defined as:

$$J = \begin{bmatrix} (\gamma-\beta)Z & (\gamma-\beta)S & 0 \\ (\beta-\alpha-\gamma)Z & (\beta-\alpha-\gamma)S & \zeta \\ \alpha Z & \alpha S & -\zeta \end{bmatrix} \tag{8}$$

Where the eigenvalue is:

$$det(J-\lambda I) = \begin{bmatrix} (\gamma-\beta)Z & (\gamma-\beta)S & 0 \\ (\beta-\alpha-\gamma)Z & (\beta-\alpha-\gamma)S-\lambda & \zeta \\ \alpha Z & \alpha S & -\zeta-\lambda \end{bmatrix} \tag{9}$$

$$det(J-\lambda I) = \Big[(\gamma-\beta)Z - \lambda\{[(\beta-\alpha-\gamma)S-\lambda](-\zeta-\lambda)-\zeta\alpha S)\}\Big] \tag{10}$$

we can investigate our model numerically on Wolfram Mathematica and Python.

3.3 Numerical Analysis

Immediately, we can investigate the impact of each variable numerically though phase portrait and time-series plot. For numerical analysis, let us assume the system is playing over a small period of time removing birth and death rate ($\Pi = \delta = 0$). For our initial evaluation these are the variables:

- Zombie Killing Rate (α) = 0.05
- Infection Rate (β)= 0.053
- Resurrection Rate (ζ) = 0.005
- Cure Effectiveness (γ) = 0.3
- Cure Delivery Time (τ) = 3
- Initial Susceptible Population (S_0) = 500
- Initial Zombie Population (Z_0) = 1
- Initial Removed Population (S_0) = 0

From this initial setup we produce phase plot and time-series of our system.

From Figure 1, it is evident that the system exhibits extreme sensitivity to its initial conditions. Additionally, Figure 2 highlights the piecewise linear characteristics of the system. This pronounced sensitivity strongly suggests that the system may be chaotic. To confirm this, we will calculate the system's Lyapunov exponent before proceeding further.

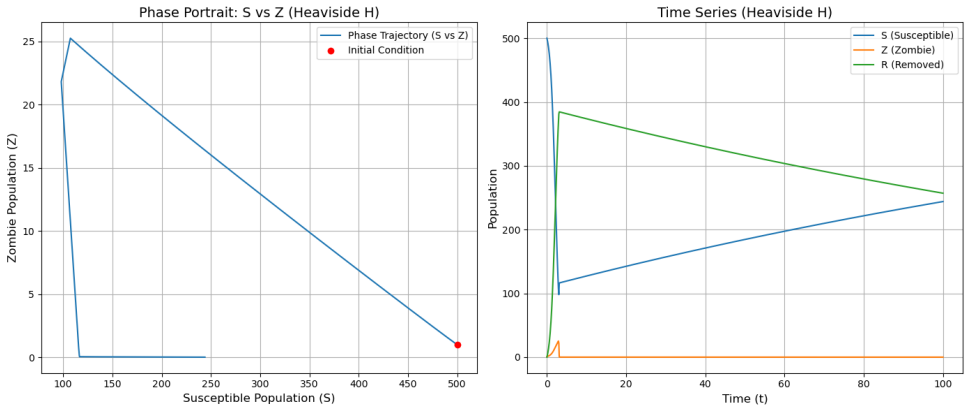


Figure 1: Initial Phase Plot and Time Series Plot

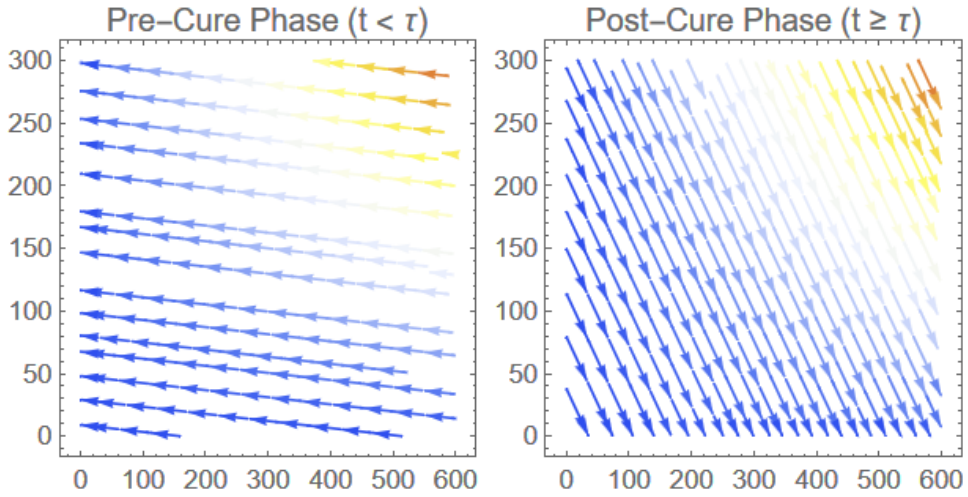


Figure 2: Initial Streamplot before and after τ

3.3.1 Lyapunov Exponent Analysis

Using a numerical solver, we determined that the Lyapunov exponent of this system is $\lambda_{\tau=3} = -0.0117$, when $\tau = 3$ but shifted to $\lambda_{\tau=5} = 0.3487$ when $\tau = 5$, signalling a transition to instability. This change suggests that the increased delay destabilises the system, leading to exponential divergence of trajectories, which is consistent with the onset of oscillatory or chaotic behaviour. This highlights the critical influence of the delay parameter τ in determining the stability and dynamical behaviour of the system, where we know that at $\tau = 3$, the humans would gain access to the cure before the population drops below 1. Further analysis of this variable will be discussed in later section.

Referring to Figure 3, when $\tau = 5$, we can explain the behaviour of each section. The small oscillations near $t = 0$ likely represent the transient dynamics as the system stabilises and the eradication of humans progresses. During this phase, the system behaves linearly, exhibiting weaker divergence. Once $t \geq \tau$, the activation of Heaviside function introduces non-linear effects, causing the trajectories to diverge exponentially, which reflects the system's sensitivity to initial conditions. The plateau at later times signifies the saturation of divergence, constrained by the bounded phase space, where the total population remains

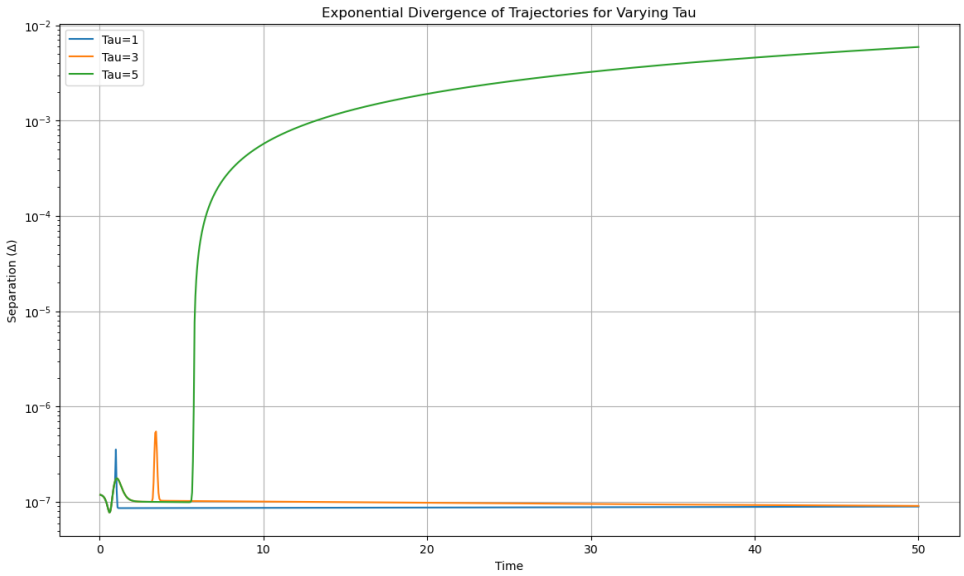
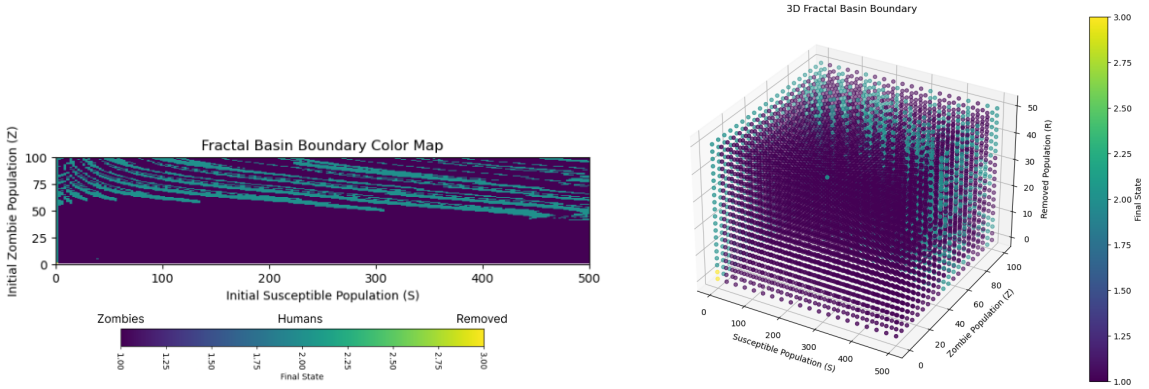


Figure 3: Lyapunov Plot

constant as $Total\ Population = S(t) + Z(t) + R(t)$. From the plot we can see that the separation seems to approach 10^{-2}, as the population number is much greater than this value, the perturbation is negligible.

3.3.2 Fractal Basin Boundary Analysis

Knowing that the system can be chaotic, Fractal analysis help us better understand self-similar patterns and sensitivity to initial conditions. Through this, we can identify region where the system exhibit stability, unpredictability, or transitions between different states in helping us better understand the critical thresholds for intervention.



(a) 2D Fractal Map

(b) 3D Fractal Map

Figure 4: Caption describing the comparison between 2D and 3D fractal maps.

From Figure 4a, the transition regions between dominant states exhibit intricate, self-similar patterns characteristic of fractals. These boundaries highlight the system's extreme sensitivity to initial conditions, where even small perturbations can result in drastically different outcomes. Furthermore, the large purple region reinforces the findings

from the base model, confirming that the zombie outbreak equilibrium is asymptotically stable over a significant portion of the phase space.

3.3.3 Fixed Point Stability Analysis of Initial Parameters

Using numerical representations of our variables, we calculated the eigenvalues for the fixed point:

$$(\lambda_S, \lambda_Z, \lambda_R) = (-0.054 + 1.37 \times 10^{-14}i, 90.0 + 2.02 \times 10^{-17}i, 0.048 - 1.79 \times 10^{-14}i) \quad (11)$$

. The presence of both a negative real eigenvalue (-0.054) and positive real eigenvalues (90.0 and 0.048) indicates that this fixed point exhibits characteristics of a saddle point. Even-though there is a complex term it is negligible, so its not a Hopf bifurcation. Specifically, the negative eigenvalue corresponds to a stable manifold, where trajectories contract towards the fixed point, while the positive eigenvalues represent unstable manifolds, where trajectories diverge away from it. The imaginary components are negligible and do not significantly affect the system's local stability. This mix of stability and instability confirms the saddle-point nature of this fixed point.

3.3.4 Variable Analysis

Knowing that the system is extremely sensitive to the starting conditions, we can analyse the system's sensitivity to parameters. As humans, we'll be focusing on the conditions for us to win. Referring back to the steady state, we can take partial derivatives of $\frac{dS}{dt}$ with respect to each parameter:

$$\frac{\partial(\frac{dS}{dt})}{\partial \Pi} = 1, \quad \frac{\partial(\frac{dS}{dt})}{\partial \gamma} = SH(t,\tau)Z, \quad \frac{\partial(\frac{dS}{dt})}{\partial \beta} = -SZ, \quad \frac{\partial(\frac{dS}{dt})}{\partial \delta} = -1 \qquad (12)$$

From 12, we can observe that the critical parameters are β and γ. However, as γ is delayed by τ, its counteracting behaviour is less influential on the system compared to β. In reality, the infection rate (β) is often uncontrollable, leaving the effectiveness of the cure (γ) as humanity's primary hope. The value of γ directly impacts the system's chaotic behaviour, with higher γ introducing stronger non-linear interactions between S and Z, helping to stabilize the dynamics. Conversely, when γ is small, these non-linear interactions are weaker, leading to unstable oscillations and potentially amplifying chaos.

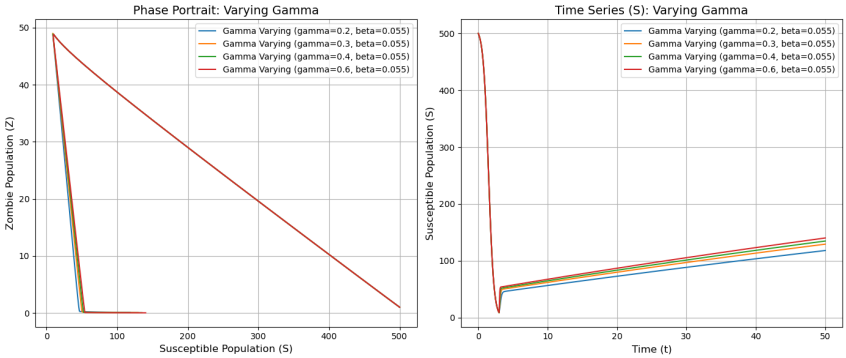


Figure 5: Phase plot & Time Series Plot with varying γ

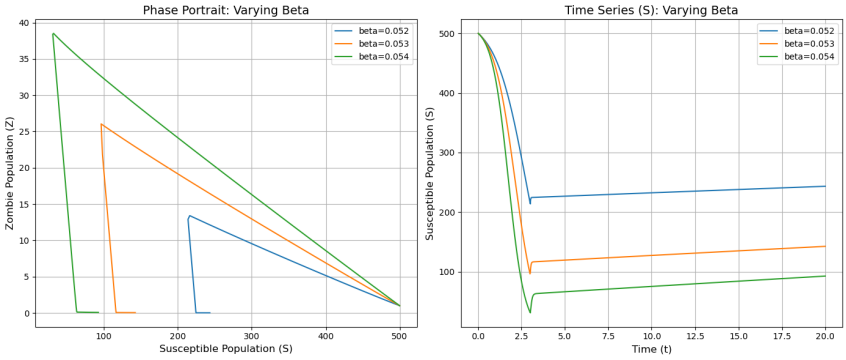


Figure 6: Phase plot & Time Series Plot with varying β

From Figure 5 and 6, we can see that β is a much more impactful on the system behaviour as expected. Now, recalling the importance of τ discussed in the Lyapunov analysis section:

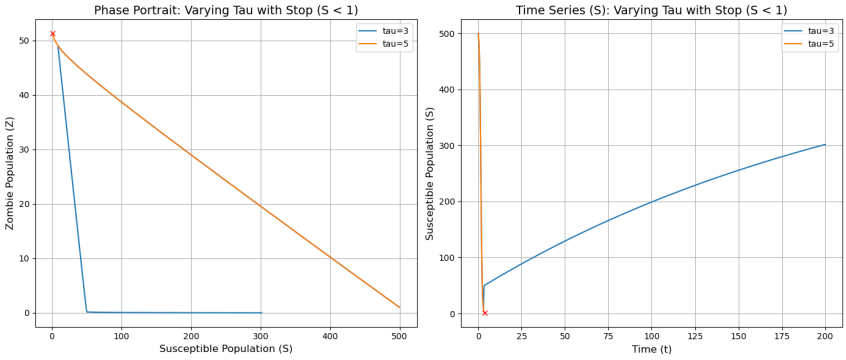


Figure 7: Phase plot & Time Series Plot with varying τ

Since the steady-state analysis disregards the time component, the effect of the delay parameter τ was not included in that part of the analysis. However, as shown in Figure 7, the delay in delivering the cure have a significant impact. When the delay ($\tau = 5$) is too long, the susceptible population reaches zero before the cure is available, highlighting the importance of timely intervention. As the current mathematical model does not account for this, we introduced a condition in the plot to mark the point where the susceptible population drops below 1. The next step is to perform a bifurcation analysis to explore

the critical parameter values and examine how the system's behaviour changes as these parameters vary. However, both python and Wolfram struggled to numerically solve due to the Heaviside function discontinuity, therefore the model must be adjusted.

4. Cure Model Approximation

As Heaviside function introduce undefined behaviour and creating abrupt changes that affect step-size sensitivity, we need to use logistic function to create a smooth transition instead. Assume:

$$H(t-\tau) \approx \frac{1}{1+e^{k(t-\tau)}} \tag{13}$$

Therefore, assuming $k = 100$ to imitate the quick changes in Heaviside function, the new model is

$$\begin{aligned} S'(t) &= \Pi + \gamma S \cdot \tfrac{Z(t)}{1+e^{\tau-t}} - \beta S(t)Z(t) - \delta \\ Z'(t) &= \beta S(t)Z(t) + \zeta R(t) - \alpha S(t)Z(t) - \gamma S(t) \cdot \tfrac{Z(t)}{1+e^{\tau-t}} \\ R'(t) &= \delta + \alpha S(t)Z(t) - \zeta R(t) \end{aligned} \tag{14}$$

4.1 Bifurcation Analysis

From this, we can investigate the bifurcation point for the change in the variables. Using the base assumption from Numerical Analysis section (Section 3.3), for the cure effectiveness (γ):

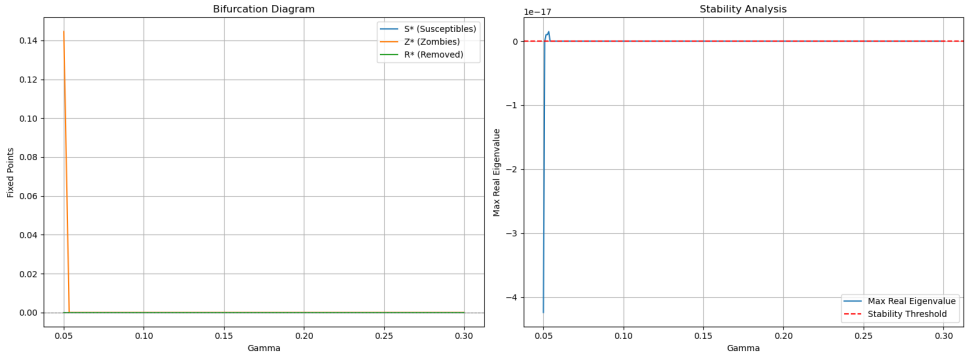


Figure 8: Gamma Bifurcation and Stability Plot

From Figure 8, we can observe that the critical gamma value is approximately 0.05. This means that when τ is small, it barely matters what the effectiveness of the cure is, as long as it is being deployed thats enough to eliminate the zombies.

For β, with the current parameters, the critical value is around 0.0311. If β is any larger, it would result in the zombies winning.

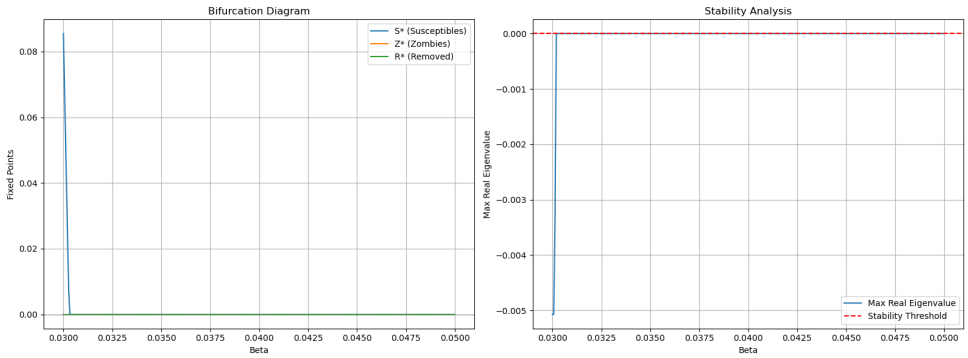


Figure 9: Beta Bifurcation and Stability Plot

4.2 Finding Critical τ Value

As τ is time dependent, we cannot do bifurcation analysis to find its critical value. Therefore, we instead analyse its timeseries plot for varying value of τ:

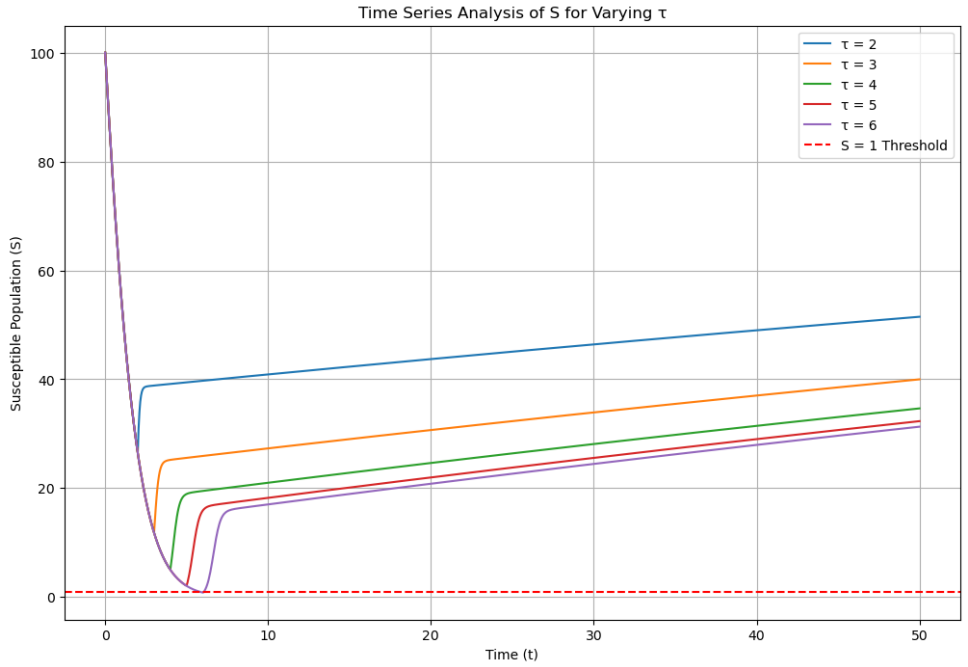


Figure 10: Tau Timeseries Plot

Through iterating through the values of τ, we found that the critical value is approximately 5.799. This means that for humans to survive under the standard assumption, the time it takes for the cure to be delivered into the area is less than 5.799 days.

5. Influence of Logistic Function's Steepness

When we use logistic function to represent heavside function, the assumption was that if the steepness, represented by k, is high enough; the logistic function would approximately have the same characterstic. However, what if k varies? In the story context, it may be that there is a set of time where the cure may be delivered to the affected area but needed

time to setup in order to administer the cure at its full efficiency. To investigate this we can plot susceptible population over time:

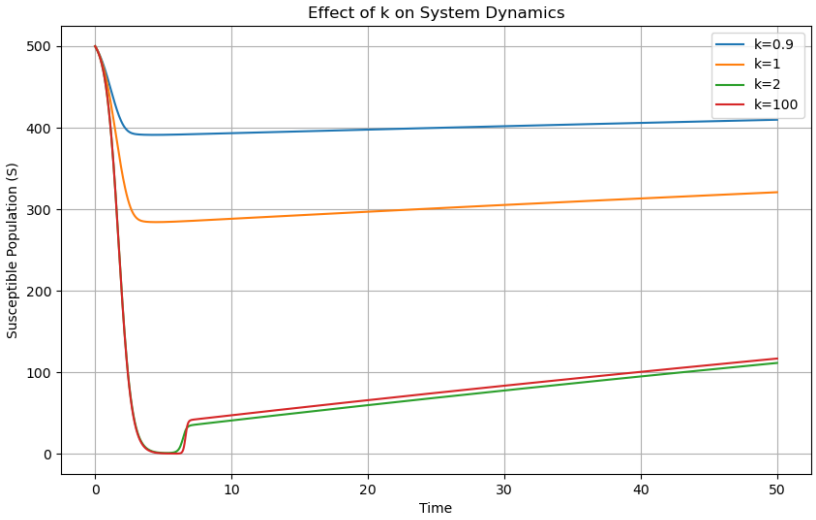


Figure 11: Effects of k on the system dynamics

From Figure 11, the impact of k on the system dynamics is minimal, except when k is small (e.g. $k \leq 1$ in this specific parameter set). In such cases, the smoother transition of the logistic function activates the cure term earlier, often preventing human extinction. However, for sufficiently high k, its exact value becomes negligible in influencing the system's behaviour, demonstrating that the logistic function is an effective approximation of the Heaviside step function for our application.

6. Conclusion

In conclusion, this report analyses demonstrates the critical dynamics of a zombie outbreak under various modelling scenarios. The **Base Model**, grounded in the SIR framework, establishes that without intervention, the system gravitates toward either a doomsday equilibrium or an unstable disease-free state.

The introduction of the **Cure Model**, incorporating Heaviside (then logistic functions), highlights the profound impact of cure effectiveness (γ) and delivery time (τ). Through numerical analysis, we found that while γ impact how fast the human population recovers, the most important variables are β and τ. For the default parameter set, human survival is only possible if β remains below 0.0311. However, as β is largely uncontrollable in a real outbreak, humanity's survival hinges on minimizing τ, with the critical threshold being 5.80 days.

Although the proposed model simplifies reality, future work could refine it by incorporating stochastic effects, heterogeneous populations, and spatial dynamics. Ultimately, this study emphasizes the importance of rapid and effective interventions in outbreak

scenarios, offering valuable insights—even if faced with a fictional crisis like zombie outbreaks.

Appendix

A. System of Equation Derivation

A.1 Heaviside Function ($t < \tau$) Equilibria Derivation

A.1.1 Full System of Equations

:

$$-\beta SZ = 0$$
$$\beta SZ + \zeta R - \alpha SZ = 0$$
$$\alpha SZ - \zeta SZ = 0$$

Using the first equation ($-\beta SZ = 0$):

$$-\beta SZ = 0 \Rightarrow \beta SZ = 0$$

Since $\beta > 0$ (infection rate cannot be zero), this implies:

$$S = 0 \quad or \quad Z = 0$$

A.1.2 Case 1: $Z = 0$

If $Z = 0$, substitute into second and third equations giving:

$$\beta S(0) + \zeta R - \alpha S(0) = 0 \Rightarrow \zeta R = 0 \Rightarrow R = 0.$$

Therefore, the only solution is (S,Z,R) = (S,0,0) where S can be any non-negative value, representing the disease-free equilibrium.

A.1.3 Case 2: $S = 0$

If $S = 0$, substitute into second and third equations giving:

$$\beta(0)Z + \zeta R - \alpha(0)Z = 0 \Rightarrow \zeta R = 0 \Rightarrow R = 0.$$

Therefore the only solution is $(S, Z, R) = (0, Z, 0)$, where Z can be any non-negative value. This represents the doomsday equilibrium, where the zombies take over the world.

A.1.4 Case 3: $S \neq 0, Z \neq 0$

From the the third equation,

$$\alpha SZ - \zeta R = 0 \Rightarrow R = \frac{\alpha SZ}{\zeta}$$

Substitute R into the second equation,

$$\beta SZ + \zeta(\frac{\alpha SZ}{\zeta}) - \alpha SZ = 0$$

This simplifies to $\beta SZ = 0$. As infection rate (β) cannot be zero, there is no co-existence equilibrium

A.2 Heaviside Function ($t > \tau$) Equilibria Derivation

Full System of Equations:

$$\gamma SZ - \beta SZ = 0$$
$$\beta SZ + \zeta R - \alpha SZ = 0$$
$$\alpha SZ - \zeta R = 0$$

As $S \geq 0,\ Z \geq 0$, we can solve the first equation as it implies that,

$$\gamma - \beta = 0 \ \textit{or} \ S = 0 \ \textit{or} \ Z = 0.$$

Assume that S and Z are not 0, we can substitute $\gamma = \beta$ in the second and third equations. Equation (2):

$$\beta SZ + \zeta R - \alpha SZ - \beta SZ = 0$$

Simplify:

$$\zeta R - \alpha SZ = 0$$

Equation (3):

$$\alpha SZ - \zeta R = 0$$

As the Equation (2) and Equation (3) are the same, we conclude that,

$$R = \frac{\alpha SZ}{\zeta}$$

When substituted R back into the system, we get:

$$\gamma SZ - \beta SZ = 0$$
$$\beta SZ - \gamma SZ = 0$$

At the equilibrium there are no additional constraints in the reduced system, meaning

the steady-state values would depend on initial conditions and parameter values.

A.3 Full Workout for Cure Model Jacobian & Eigenvalue

A.3.1 Define the system

$$F(S,Z,R) = \begin{bmatrix} \gamma SZ - \beta SZ \\ \beta SZ + \zeta R - \alpha SZ - \gamma SZ \\ \alpha SZ - \zeta R \end{bmatrix}$$

A.3.2 Find the Jacobian

$$J = \begin{bmatrix} \frac{\partial F_1}{\partial S} & \frac{\partial F_1}{\partial Z} & \frac{\partial F_1}{\partial R} \\ \frac{\partial F_2}{\partial S} & \frac{\partial F_2}{\partial Z} & \frac{\partial F_2}{\partial R} \\ \frac{\partial F_3}{\partial S} & \frac{\partial F_3}{\partial Z} & \frac{\partial F_3}{\partial R} \end{bmatrix}$$

$$J = \begin{bmatrix} (\gamma - \beta)Z & (\gamma - \beta)S & 0 \\ (\beta - \alpha - \gamma)Z & (\beta - \alpha - \gamma)S & \zeta \\ \alpha Z & \alpha S & -\zeta \end{bmatrix}$$

A.3.3 Find the eigenvalue

$$det(J - \lambda I) = 0$$

$$det(J - \lambda I) = \begin{vmatrix} (\gamma - \beta)Z - \lambda & (\gamma - \beta)S & 0 \\ (\beta - \alpha - \gamma)Z & (\beta - \alpha - \gamma)S - \lambda & \zeta \\ \alpha Z & \alpha S & -\zeta - \lambda \end{vmatrix}$$