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, beginning 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 were then utilised to 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 31 To study the dynamics of a zombie outbreak, a baseline model must first be defined, along with its scope, parameters, and assumptions. This system is modelled using the SIR (Susceptible-Infectious-Recovered) framework, which allows for the incorporation of 34 key assumptions and simplifications to focus on the essential aspects of the outbreak 35 dynamics. 36 2.1Scope of the Base Model 37 This model will be based on the model discussed in WHEN ZOMBIES ATTACK! paper (Smith et al., 2009). The main assumption here would be that the system is played out in a short period of time with fixed total population in a closed environment. The key 40 variables are: 41 • S(t): The number of susceptible individuals at time t 42 • Z(t): The number of zombies at time t 43 • R(t): The number of dead individuals at time t The parameters include: 45 • α : Zombie Removal Rate 46 • β : Infection Rate 47 • ζ : Zombie Resurrection Rate 48 • δ : Susceptible Deathrate 49 • Π : Susceptible Birthrate 50 2.2Assumptions 51 2.2.1 Small Time Scale 52 As the system plays out over a relatively short period of time, the assumption is that there 53 will be no births, deaths, or migration in or out of the system to simplify the dynamics by 54 focusing solely on disease spread. We will still include them as a variable but will assume 55 $(\Pi = \delta = 0)$ during numerical simulations. 56 2.2.2 Homogeneous Mixing 57 Every individual in the population has an equal probability of interacting with each other. This ensures that the model can use averaged rates of zombification to reduce 59

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computational complexity.

2.2.3 Instantaneous Interaction

The rates of the interaction is proportional to the number of each population (e.g. βSI)/. 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.

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2.2.5 Resurrection 68

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:

$$S' = \Pi - \beta SZ - \delta S$$

$$Z' = \beta SZ + \zeta R - \alpha SZ$$

$$R' = \delta S + \alpha SZ - \zeta R$$
(1)

As this model is widely discussed in the WHEN ZOMBIES ATTACK! paper (Smith et al., 2009), we will quickly go through its system behaviour. The key characteristics of this model is that there are two outcomes: Zombies Domination $(S=0,\ ,Z\neq 0)$ and Disease-Free State $(S\neq 0,\ ,Z=0)$ - meaning that human-zombie coexistence is impossible. Additionally, 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 mathematical model. 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 zombie. 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 97 that past this threshold, the cure will be widely available, keeping γ constant. 98

For this iteration of the model, we will also assume instantaneous activation of the 99 cure, represented by a Heaviside Step function. In the context of the story line, we 100 assume that the moment the cure is available in an area, the population will be able to 101 immediately administer it at maximum efficiency. Where H is defined as,

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

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The new model is therefore represented as,

$$S'(t) = \Pi + \gamma SH(t - \tau)Z(t) - \beta S(t)Z(t) - \delta$$

$$Z'(t) = \beta S(t)Z(t) + \zeta R(t) - \alpha S(t)Z(t) - \gamma S(t)Z(t)H(t - \tau)$$

$$R'(t) = \delta + \alpha S(t)Z(t) - \zeta R(t)$$
(3)

Right away, we can see that there would be 2 distinct behaviours between $t < \tau$ and 104 $t \ge \tau$ due to the piecewise characteristic due to the Heaviside function. Additionally, 105 another immediate insight is that the system is discontinuous at $t = \tau$, meaning there is 106 no potential function (V), thus no closed orbits (no need to do Poincaré map analysis). 107

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 ??):

$$\frac{d\hat{S}}{d\hat{t}} = \hat{\Pi} + \hat{\gamma}\hat{S}H(\hat{t} - \hat{\tau})\hat{Z} - \hat{S}\hat{Z} - \hat{\delta},$$

$$\frac{d\hat{Z}}{d\hat{t}} = \hat{S}\hat{Z} + \hat{\zeta}\hat{R} - \hat{\alpha}\hat{S}\hat{Z} - \hat{\gamma}\hat{S}\hat{Z}H(\hat{t} - \hat{\tau}),$$

$$\frac{\hat{R}}{\hat{t}} = \hat{\delta} + \hat{\alpha}\hat{S}\hat{Z} - \hat{\zeta}\hat{R}.$$
(4)

Now we can investigate the dominant terms in each equation. For the relationship 112 between the susceptible population and the zombie population, at early stages (when Z is 113 small), the human's birth rate (Π) may dominate the system over an extended period of 114 time. However, as the main assumption of the model is short-time scale of the scenario, 115 $\Pi=0$ and this term can be eliminated. This means that, generally, the $\hat{S}\hat{Z}$ term is 116

going to be the most dominant (especially considering humanity survival in mind). Once 117 the cure is introduced, depending on γ , the cure term may dominate the system, saving 118 humanity. In the context of the interaction between zombies and the removed population 119 (the non-walking dead), this term is dominated by the resurrection rate (ζ). 120

3.2 Steady State Analysis

As γ and τ is our variable of interest introduced in this model, finding the transition in the system is critical. Most importantly, we want to know what would happen when the transition in the system is critical. Most importantly, we want to know what would happen when the transition in the system is critical. Most importantly, we want to know what would happen when the transition in the system is critical. Most importantly, we want to know what would happen when the transition in the system is critical. Most importantly, we want to know what would happen when the transition in the system is critical. Most importantly, we want to know what would happen when the transition in the system is critical. As the model exhibit piecewise behaviour, we can investigate the 2 the behaviours that this model encapsulates: $(t < \tau)$ and $(t > \tau)$.

3.2.1
$$t < \tau$$

$$-\beta SZ = 0$$

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

$$\alpha SZ - \zeta R$$
(5)

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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))

The steady-state analysis further proves that co-existence is impossible, as this would only be true if $\beta=0$. By the definition of an outbreak, this is not possible, resulting in the system's basic reproductive number (R_0) to be:

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

In this timeframe (before cures arrive), the only way for humans to win is to kill more 133 zombies than being turned into one.

3.2.2
$$t \geq au$$

However, once the cure has been delivered $(t \ge \tau)$, the system of equations becomes (See 136 Appendix A.2)

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

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

$$\alpha SZ - \zeta R = 0$$
(7)

As expected, in this case we have 2 trivial equilibria. We know this because the 138 linearity of the system remained constant and the difference between the new and the 139 base model is the piecewise characteristic. This means that once the cures are delivered, 140 the effective reproductive number changes to $R_{eff} = \frac{\beta + \alpha}{\alpha + \gamma}$. Talking from a perspective of 141

a human, our chance of survival would require $R_{eff} < 1$, meaning the effectiveness of the 142 cure is a critical parameter in our survival. To analyse the stability to the equilibria of 143 this system, the Jacobian can be defined as:

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

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Where the eigenvalue is:

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

$$det(J - \lambda I) = \left[(\gamma - \beta)Z - \lambda \{ [(\beta - \alpha - \gamma)S - \lambda](-\zeta - \lambda) - \zeta \alpha S) \} \right]$$
 (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 148 portrait and time-series plot. For numerical analysis, let us assume the system is playing 149 over a small period of time removing birth and death rate ($\Pi = \delta = 0$). From trial and 150 error parameter tuning, these are the 'default' parameter values: 151

- Zombie Killing Rate (α) = 0.05
- Infection Rate (β) = 0.053
- Resurrection Rate $(\zeta) = 0.005$
- Cure Effectiveness $(\gamma) = 0.3$
- Cure Delivery Time $(\tau)=3$
- Initial Susceptible Population $(S_0) = 500$
- Initial Zombie Population $(Z_0) = 1$
- Initial Removed Population $(S_0) = 0$

From this initial setup we produce the initial phase plot and time-series plot of our $_{160}$ system.

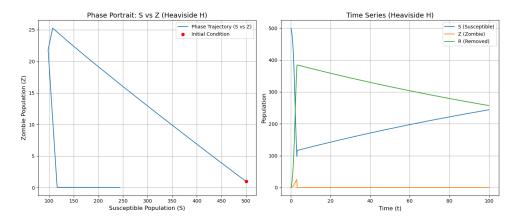


Figure 1: Initial Phase Plot and Time Series Plot

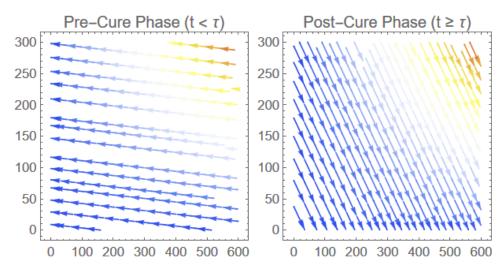


Figure 2: Initial Streamplot before and after τ

From Figure 1, it is evident that the system exhibits extreme sensitivity to its initial 162 conditions while Figure 2 highlights the piecewise linear characteristics of the system. 163 While the piecewise linear characteristic follows our model design intension, the pronounced initial condition sensitivity strongly suggests that the system may be chaotic. 165 To confirm this, we investigate the system's Lyapunov exponent before proceeding further. 166

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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 170 a transition to instability. This change suggests that the increased delay destabilises 171 the system, leading to exponential divergence of trajectories, which is consistent with the 172 onset of oscillatory or chaotic behaviour. This highlights the critical influence of the delay 173 parameter τ in determining the stability and dynamical behaviour of the system, where 174 we know that at $\tau=3$, the humans would gain access to the cure before the population 175 drops below 1. This clearly shows the significance of τ in our system, a characteristic that 176 cannot be discovered through Steady State Analysis alone.

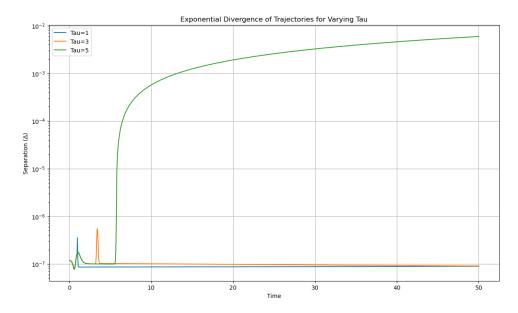


Figure 3: Lyapunov Plot

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 that 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 is sensitive to initial conditions and occasionally become chaotic, 189 fractal analysis help us better understand self-similar patterns and sensitivity. Through 190 this, we can identify region where the system exhibit stability, unpredictability, or trans191 itions between different states in helping us better understand the critical thresholds for 192 intervention.

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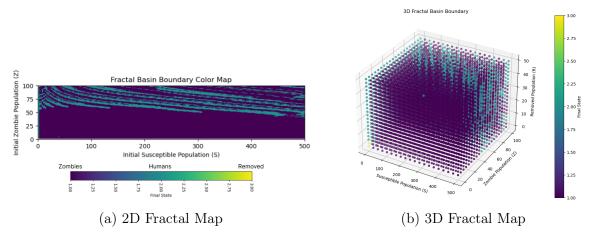


Figure 4: Caption describing the comparison between 2D and 3D fractal maps.

From Figure 4a, the transition regions between dominant states exhibit intricate, 194 self-similar patterns characteristic of fractals. These boundaries highlight the system's 195 extreme sensitivity to initial conditions, where even small perturbations can result in 196 drastically different outcomes. Furthermore, the large purple region reinforces the findings 197 from the base model, confirming that the zombie outbreak equilibrium is asymptotically 198 stable over a significant portion of the phase space.

Fixed Point Stability Analysis of Initial Parameters 3.3.3

Using numerical representations of our variables, we calculated the eigenvalues for the 201 fixed point: 202

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

The presence of both a negative real eigenvalue (-0.054) and positive real eigenvalues (90.0 203 and 0.048) indicates that this fixed point exhibits characteristics of a saddle point. There 204 is a small complex term, but it is negligible, so its not a Hopf bifurcation. Specifically, 205 the negative eigenvalue corresponds to a stable manifold, where trajectories contract towards the fixed point, while the positive eigenvalues represent unstable manifolds, where 207 trajectories diverge away from it. The imaginary components are negligible and do not 208 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, the next step 212 is to analyse the system's sensitivity to parameters. As humans, we'll be focusing on the 213 conditions for us to win (disease-free state). Referring back to the steady state, we can 214 take partial derivatives of $\frac{dS}{dt}$ with respect to each parameter: 215

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

From 12, we can observe that the critical parameters are β and γ . However, as γ is 216 delayed by τ , its counteracting behaviour is less influential on the system compared to 217 β . In reality, the infection rate (β) is often uncontrollable, leaving the effectiveness of 218 the cure (γ) as humanity's primary hope. The value of γ directly impacts the system's 219 chaotic behaviour, with higher γ introducing stronger non-linear interactions between S 220 and Z, helping to stabilize the dynamics. Conversely, when γ is small, these non-linear 221 interactions are weaker, leading to unstable oscillations and potentially amplifying chaos. 222

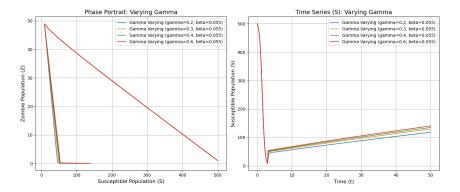


Figure 5: Phase plot & Time Series Plot with varying γ

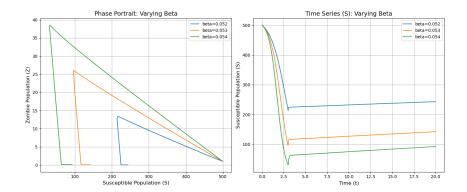


Figure 6: Phase plot & Time Series Plot with varying β

From Figure 5 and 6, we can see that β is a much more impactful on the system 223 behaviour as expected. Now, recalling the importance of τ discussed in the Lyapunov 224 analysis section:

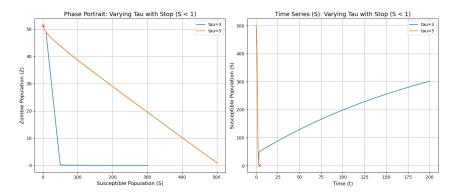


Figure 7: Phase plot & Time Series Plot with varying τ

Since the steady-state analysis disregards the time component, the effect of the delay 226 parameter τ was not included in that part of the analysis. However, as shown in Figure 7, 227 the delay in delivering the cure have a significant impact. When the delay ($\tau = 5$) is too 228 long, the susceptible population reaches zero before the cure is available, highlighting the 229 importance of timely intervention. As the current mathematical model does not account 230 for this, we introduced a condition in the plot to mark the point where the susceptible 231 population drops below 1. The next step is to perform a bifurcation analysis to explore 232 the critical parameter values and examine how the system's behaviour changes as these 233 parameters vary. However, both python and Wolfram struggled to numerically solve due 234 to the Heaviside function discontinuity, therefore the model must be adjusted. 235

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:

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

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Therefore, assuming k=100 to imitate the quick changes in Heaviside function, the 240 new model is represented as:

$$S'(t) = \Pi + \gamma S \cdot \frac{Z(t)}{1 + e^{\tau - t}} - \beta S(t) Z(t) - \delta$$

$$Z'(t) = \beta S(t) Z(t) + \zeta R(t) - \alpha S(t) Z(t) - \gamma S(t) \cdot \frac{Z(t)}{1 + e^{\tau - t}}$$

$$R'(t) = \delta + \alpha S(t) Z(t) - \zeta R(t)$$
(14)

4.1 Bifurcation Analysis

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

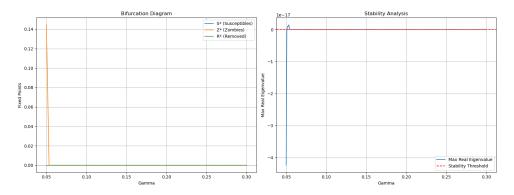


Figure 8: Gamma Bifurcation and Stability Plot

From Figure 8, we can observe that the critical cure effectiveness (γ) value is approximately 0.05. This means that it barely matters what the effectiveness of the cure is, as 247 long as it is being deployed fast enough (small τ) to eliminate the zombies before they 248 eliminate all of the susceptible population.

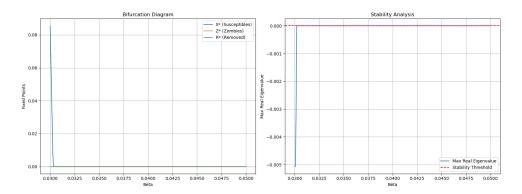


Figure 9: Beta Bifurcation and Stability Plot

For β , with the 'default' parameters, the critical value is around 0.0311. If the infection 250 rate (β) is any larger, it would result in the zombies winning. Realistically, we wouldn't 251 be able to control the infection rate, therefore the only chance of humanity surivial would 252 be the understanding of τ 's critical value. 253

4.2Finding Critical τ Value

As τ is time dependent, we cannot do bifurcation analysis to find its critical value. Therefore, we instead analyse its time-series plot for varying value of τ : 256

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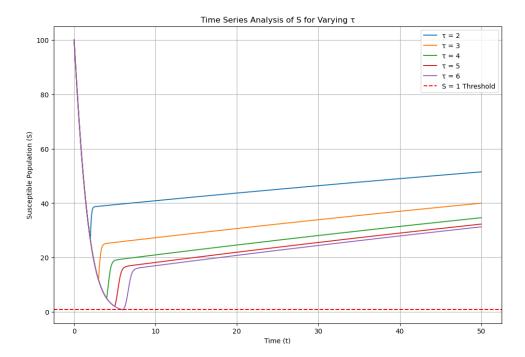


Figure 10: Tau Timeseries Plot

Through iterating through the values of τ , we found that the critical value is approx-257 imately 5.799. This means that for humans to survive under the 'default assumption, the 258 cure must be delivered to the affected area in less than 5.799 days. 259

5. Influence of Logistic Function's Steepness

When we use logistic function to represent heavside function, the assumption was that if 261 the steepness, represented by k, is high enough; the logistic function would approximately 262 have the same characteristic. However, what if k varies? In the story context, it may be 263 that there is a set of time where the cure may be delivered to the affected area but needed 264 time to setup in order to administer the cure at its full efficiency. To investigate this we 265 can plot susceptible population over time: 266

From Figure 11, the impact of k on the system dynamics is minimal, except when k is 267 small (e.g. $k \leq 1$ in this specific parameter set). In such cases, the smoother transition of 268 the logistic function activates the cure term earlier, often preventing human extinction. 269 However, for sufficiently high k, its exact value becomes negligible in influencing the 270 system's behaviour, demonstrating that the logistic function is an effective approximation 271 of the Heaviside step function for our application.

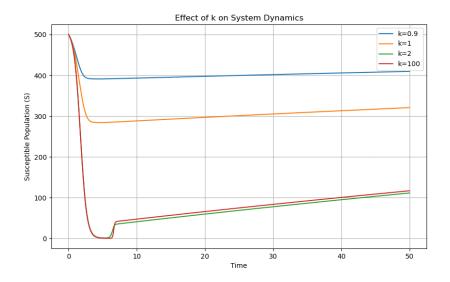


Figure 11: Effects of k on the system dynamics

Conclusion 6.

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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 func- 278 tions), highlights the profound impact of cure effectiveness (γ) and delivery time (τ). 279 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 281 set, human survival is only possible if β remains below 0.0311. However, as β is largely 282 uncontrollable in a real outbreak, humanity's survival hinges on minimizing τ , with the 283 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, 286 this study emphasizes the importance of rapid and effective interventions in outbreak 287 scenarios, offering valuable insights—even if faced with a fictional crisis like zombie outbreaks.

References 290

Smith, R., Alemi, A., Dickison, M., Eyre, S., Hawkins, S., & Huynh, D. (2009). When 291 zombies attack!: Mathematical modelling of an outbreak of zombie infection. http://doi.org/10.1007/j.com/10.1007/j //math.uchicago.edu/~shmuel/Modeling/WHEN%20ZOMBIES%20ATTACK!- 293 %20MATHEMATICAL%20MODELLING%20OF%20AN%20OUTBREAK%20OF%94 20ZOMBIE%20INFECTION.pdf 295

Appendix	296					
A. System of Equation Derivation	297					
A.1 Heaviside Function $(t < \tau)$ Equilibria Derivation	298					
A.1.1 Full System of Equations	299					
: $-\beta SZ = 0$ $\beta SZ + \zeta R - \alpha SZ = 0$ $\alpha SZ - \zeta SZ = 0$	300					
Using the first equation $(-\beta SZ = 0)$:	301					
$-\beta SZ = 0 \Rightarrow \beta SZ = 0$						
Since $\beta > 0$ (infection rate cannot be zero), this implies:	302					
S = 0 or $Z = 0$						
A.1.2 Case 1: $Z = 0$	303					
If $Z = 0$, substitute into second and third equations giving:	304					
$\beta S(0) + \zeta R - \alpha S(0) = 0 \Rightarrow \zeta R = 0 \Rightarrow R = 0.$						
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: $S = 0$	307					
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: $S \neq 0, Z \neq 0$	311					
From the third equation,	312					

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

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 315

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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,

$$\gamma - \beta = 0$$
 or $S = 0$ or $Z = 0$.

Assume that S and Z are not 0, we can substitute $\gamma = \beta$ in the second and third 319 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 325 the steady-state values would depend on initial conditions and parameter values. 326

A.3	Full Wor	kout for	Cure	Model	Jacobian	&	Eigenvalu	ıe
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A.3.1 Define the system 328

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

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

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

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