Global Dynamics of an SIR Model with Post-Infection Mortality and Partial Immunity Brendan Shrader, Zhisheng Shuai



MOTIVATION

- There is growing interest in studying epidemiological models that incorporate post-infection mortality and partial immunity. This is because there are diseases, such as COVID, that have both of these properties.
- In this project, we investigate how the choice of transmission term influences disease dyanmics in a model that includes post-infection mortality and partial immunity.
- We are particularly interested in characterizing parameter regions where periodic solutions exist.

OVERVIEW

• We incorporate post-infection mortality into the recovered class as an additional death rate, α_R . We incorporate partial immunity a constant ϵ in front of the incidence rate from the recovered to infected class. Our model uses the following compartment diagram:



Figure 1. Compartment Diagram

• From this compartment diagram, we have the following system of differential equations.

$$\dot{S} = r(N) - f(S, I, N) - dS$$

$$\dot{I} = f(S, I, N) + \epsilon f(R, I, N) - \gamma I - dI - \alpha_I I$$

$$\dot{R} = \gamma I - \epsilon f(R, I, N) - dR - \alpha_R R$$

$$N = S + I + R$$

• Our new model uses different recruitment and incidence functions compared to the original model [3].

Model	Recruitment $r(N)$	Incidence $f(S, I, N)$	
Original	Λ	λSI	"Mass-action Incident
New	bN	$\beta SI/N$	"Standard Incidence

• The incidence rate that a model uses depends on assumptions about how individuals interact and how the infectiousness of the disease depends on population size.

Incidence Rate	Mass Action	Standard
Response to Population Size	$\lambda SI \propto N^2$	$\beta SI/N \propto N$



Figure 2. Standard Incidence: Spread-out population; assumes that every susceptible individual interacts with a proportion of the infected population.



Figure 3. Mass-action Incidence: Dense population; assumes that every susceptible individual interacts with every infectious individual.

Original Model

4000

6000

750

Time (Weeks)

1000

Time (Weeks)

Susceptible

University of Central Florida

NUMERICAL RESULTS

Parameters

$$\Lambda = b = d = 0.0004$$
$$\beta = 2.5$$
$$\gamma = 1$$
$$\epsilon = 1$$
$$\alpha_I = 0$$
$$\alpha_R = 0.0008$$



ANALYTICAL RESULTS

The new model evolves according to the system of differential equations:

$$\dot{S} = bN - \frac{\beta SI}{N} - dS$$
$$\dot{I} = \frac{\beta SI}{N} + \frac{\epsilon \beta RI}{N} - \gamma I - dL$$
$$\dot{R} = \gamma I - \frac{\epsilon \beta RI}{N} - dR - \alpha_R$$

Because of our choice of recruitment and incidence functions, we can make the change of variables s = S/N, i = I/N, and r = R/N to reduce this model to 2 dimensions. $\dot{s} = b - bs - \beta si + \alpha_I si + \alpha_R s(1 - s - i)$

$$i = \beta si + \epsilon \beta i (1 - s - i) - (b + \alpha_I + \gamma)i +$$

s, i, and r are population proportions, so we restrict our attention to the feasible set Γ .



Figure 4. $\Gamma = \{(s, i) : s + i \le 1, s \ge 0, i \ge 0\}$

THEOREMS

Following the next-generation matrix method [1], define the Basic Reproductive Number (\mathcal{R}_0) as $\mathcal{R}_0 = \frac{\rho}{b + \gamma + \alpha_I}.$

1. If $\mathcal{R}_0 \leq 1$, the Disease Free Equilibrium is globally stable in Γ . Proof uses a novel Lyapunov Function and modification of Lasalle's Invariance Principle [2].

2. If $\mathcal{R}_0 > 1$, there is a unique Endemic Equilibrium and the Disease Free Equilibrium is unstable.

3. If $\mathcal{R}_0 > 1$ and $\alpha_I \leq \alpha_R + \epsilon \beta$, the Endemic Equilibrium is globally stable in Γ .

Proof uses Dulac Criterion and Poincaré-Bendixson Theorem.

ice"





$$I - \alpha_I I$$

 $- \alpha_I i^2 + \alpha_R i (1 - s - i)$

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PARAMETERS				
Parameter	Biological Meaning	Range		
Λ , b	Birth rate	$\Lambda = b > 0$		
eta,λ	Transmission Coefficient	$\beta = \lambda > 0$		
ϵ	Immunity	$0 \le \epsilon \le 1$		
d	Death rate	d > 0		
γ	Recovery rate	$\gamma > 0$		
$lpha_I$	Disease-induced mortality	$\alpha_I \ge 0$		
$lpha_R$	Post-infectious mortality	$\alpha_R \ge 0$		

- From our numerical analysis, we found that the new model does not produce cycles in cases when the original model does; for example, when $\alpha_I = 0$.
- We have analytically shown that the Disease Free Equilibrium is globally stable if $\mathcal{R}_0 \leq 1$, and that cycles cannot occur when $\alpha_I < \alpha_R + \epsilon \beta$.
- Thus, we have shown that the choice of recruitment and incidence functions can have an impact on global dynamics, particularly on the existence of cycles.

- We are interested in applying our Lyapunov function to other models. In fact, it can be used to prove the global stability of the disease-free equilibrium in the original model [3].
- We would like to further examine the impact of post-infection mortality on the endemicity. We do not have a mathematical explanation for why the endemicity of each compartment responds to \mathcal{R}_0 in the following manner:



- [1] P. van den Driessche and James Watmough, *Reproduction numbers and sub-threshold* endemic equilibria for compartmental models of disease transmission, Mathematical Biosciences. 180 (2002), pp. 29-48, DOI 10.1016/S0025-5564(02)00108-6.
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- [3] Chadi M. Saad-Roy, Simon A. Levin, Bryan T. Grenfell, and Mike Books, Epidemiological Impacts of Post-Infection Mortality, Royal Society Proceeding B. 290 (2023), 2002, DOI 10.1098/rspb.2023.0343.

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CONCLUSIONS

FUTURE RESEARCH

Figure 5. Final proportion of susceptible, infectious, and recovered classes with respect to \mathcal{R}_0

REFERENCES

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