

MODELING A VIRAL EPIDEMIC WITH A CONCURRENT “MISINFODEMIC”

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OVERVIEW

1 BACKGROUND

2 METHODOLOGY

3 OUR MODELS

4 APPENDIX

5 FINAL REMARKS

THE KERMACK-MCKENDRICK THEORY

DEFINITION (*SIR* MODEL)

Let S denote the number of **susceptible** individuals in a population, I the number of **infectious** individuals, and R the number of **recovered** individuals. Then, the *SIR* model of infection is the system

$$\left\{ \begin{array}{l} \frac{d}{dt} S = - \underbrace{\beta SI}_{\text{Infection}} \\ \frac{d}{dt} I = \underbrace{\beta SI}_{\text{Infection}} - \underbrace{\gamma I}_{\text{Recovery}} \\ \frac{d}{dt} R = \underbrace{\gamma I}_{\text{Recovery}} \end{array} \right. \quad (1)$$

THE KERMACK-MCKENDRICK THEORY

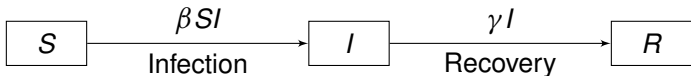


FIGURE: Basic *SIR* transfer diagram

THE KERMACK-MCKENDRICK THEORY

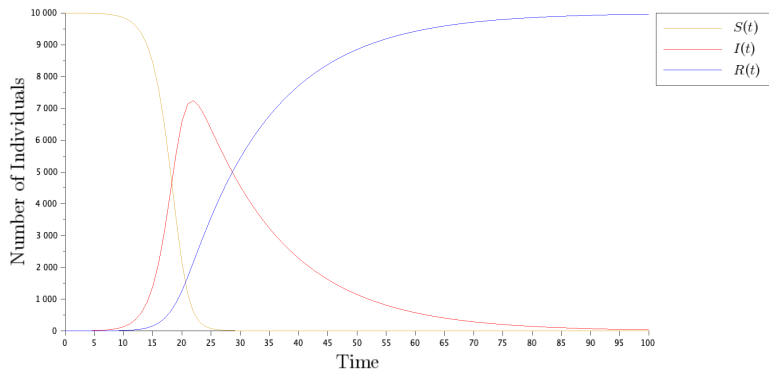


FIGURE: Basic *SIR* plot over time t with $\beta = \frac{7}{10}$, $\gamma = \frac{1}{15}$

COMPARTMENTAL ODE MODELS

DEFINITION (COMPARTMENTAL MODEL)

Together, we take a (i) system of time-dependent equations, (ii) its associated transfer diagram, (iii) and the resultant plot over time t to be a compartmental model of infectious disease.

In our case, a **compartment** refers to one of the population stocks. For instance, in the basic *SIR* model, the susceptible, infected, and recovered populations are the compartments, and the equations model an individuals **transfer** between compartments.

REMARK

In a Kermack-McKendrick population, we assume that the rate of births and rate of deaths are equal, so they are not considered in the transfers.

BUILDING COMPLEXITY

Our approach to modeling certain traits of an infectious disease is to gradually build complexity, beginning from the *SIR* model:

- (I) Identify the characteristic to be added to the altered model.
- (II) Construct a logical transfer diagram with transfer ratios.
- (III) Use the transfer ratios to write the equations explicitly.
- (IV) Plot the equations numerically.

REMARK

Hereafter, we will write the time derivative $\frac{d}{dt}F$ as \dot{F} .

NUMERICAL SOLUTIONS

DEFINITION (EULER'S METHOD)

Given t_0 , $y(t_0)$, and \dot{y} , set $y_0 = y(t_0)$. Then, choose a step size Δt such that each interval of time is given by $t_{n+1} = t_n + \Delta t$. We can take

$$y_{n+1} = y_n + \dot{y}\Delta t,$$

so $y_n \approx y(t_n)$.

We use Euler's method, alongside higher-order versions such as RK4 to approximate and plot S , I , R from \dot{S} , \dot{I} , \dot{R} .

INFORMATION DISPERSAL

We begin by adding an information spread, where a **misinformed** population M “learns” via contact with an **knowledgeable** population K , stratifying the susceptible population into 2 groups:

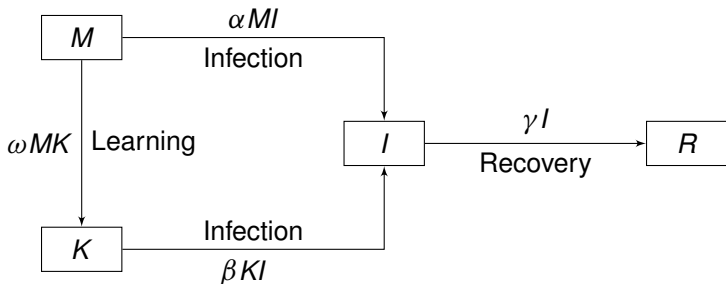


FIGURE: *MKIR* transfer diagram with information dispersal

INFORMATION DISPERSAL

Converting this into ODEs yields the system

$$\left\{ \begin{array}{l} \dot{M} = - \underbrace{\omega MK}_{\text{Learning}} - \underbrace{\alpha MI}_{\text{Infection}} \\ \dot{K} = \underbrace{\omega MK}_{\text{Learning}} - \underbrace{\beta KI}_{\text{Infection}} \\ \dot{i} = \underbrace{(\alpha M + \beta K)I}_{\text{Infection}} - \underbrace{\gamma I}_{\text{Recovery}} \\ \dot{R} = \underbrace{\gamma I}_{\text{Recovery}} \end{array} \right. \quad (2)$$

INFORMATION DISPERSAL

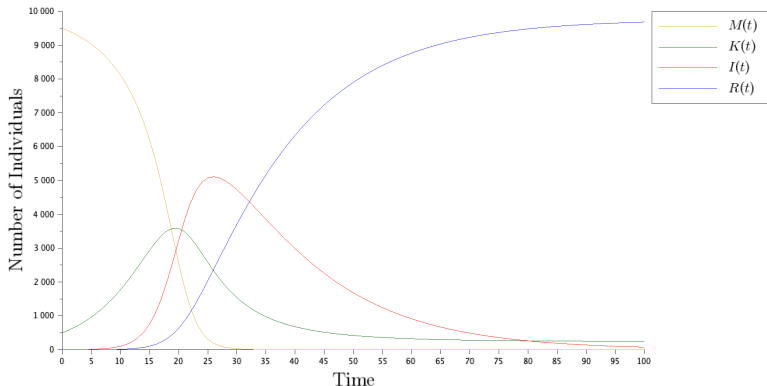


FIGURE: *MKIR* with $\omega = \frac{15}{100}$, $\alpha = \frac{7}{10}$, $\beta = \frac{2}{10}$, $\gamma = \frac{1}{15}$

EXPOSURE LATENCY

Now, we add an “exposure latency,” where there is a non-contagious **exposed** period E in between when an individual is susceptible and infected:

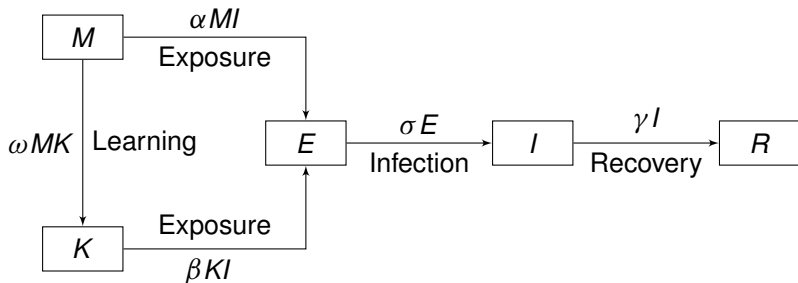


FIGURE: MKEIR transfer diagram with exposure latency

EXPOSURE LATENCY

$$\left\{ \begin{array}{l}
 \dot{M} = - \underbrace{\omega MK}_{\text{Learning}} - \underbrace{\alpha MI}_{\text{Exposure}} \\
 \dot{K} = \underbrace{\omega MK}_{\text{Learning}} - \underbrace{\beta KI}_{\text{Exposure}} \\
 \dot{E} = \underbrace{(\alpha M + \beta K)I}_{\text{Exposure}} - \underbrace{\sigma E}_{\text{Infection}} \\
 \dot{i} = \underbrace{\sigma E}_{\text{Infection}} - \underbrace{\gamma I}_{\text{Recovery}} \\
 \dot{R} = \underbrace{\gamma I}_{\text{Recovery}}
 \end{array} \right. \quad (3)$$

EXPOSURE LATENCY

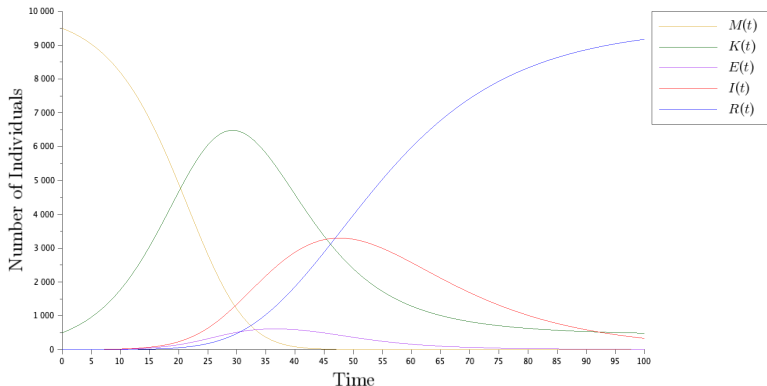


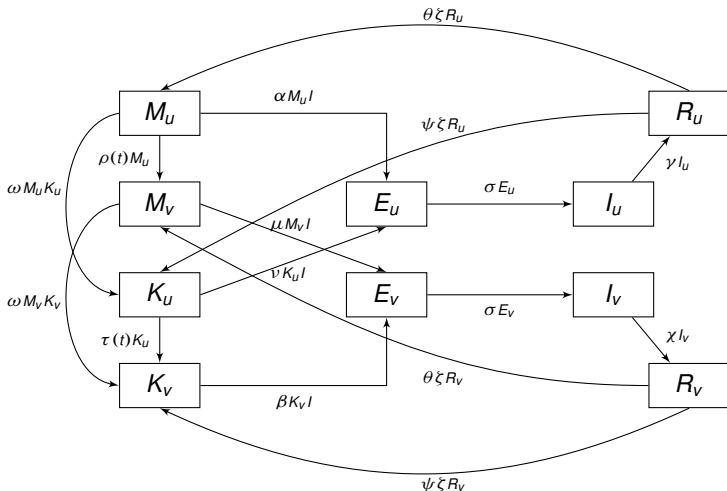
FIGURE: *MKEIR* with $\omega = \frac{15}{100}$, $\alpha = \frac{7}{10}$, $\beta = \frac{2}{10}$, $\sigma = \frac{1}{2}$, $\gamma = \frac{1}{15}$

VACCINATION AND TEMPORARY IMMUNITY

Q: What if we were to add a **vaccination** mechanism and make post-infection **immunity temporary**?

A: The model becomes *very messy*!

VACCINATION AND TEMPORARY IMMUNITY



VACCINATION AND TEMPORARY IMMUNITY

$$\left\{ \begin{array}{l}
 \dot{M}_U = - \underbrace{\omega M_U K_U}_{\text{Learning}} - \underbrace{\rho(t) M_U}_{\text{Vaccination}} - \underbrace{\alpha M_U I}_{\text{Exposure}} + \underbrace{\theta \zeta R_U}_{\text{Loss of Immunity}} \\
 \dot{M}_V = - \underbrace{\omega M_V K_V}_{\text{Learning}} + \underbrace{\rho(t) M_U}_{\text{Vaccination}} - \underbrace{\mu M_V I}_{\text{Exposure}} + \underbrace{\theta \zeta R_V}_{\text{Loss of Immunity}} \\
 \dot{K}_U = \underbrace{\omega M_U K_U}_{\text{Learning}} - \underbrace{\tau(t) K_U}_{\text{Vaccination}} - \underbrace{\nu K_U I}_{\text{Exposure}} + \underbrace{\psi \zeta R_U}_{\text{Loss of Immunity}} \\
 \dot{K}_V = \underbrace{\omega M_V K_V}_{\text{Learning}} + \underbrace{\tau(t) K_U}_{\text{Vaccination}} - \underbrace{\beta K_V I}_{\text{Exposure}} + \underbrace{\psi \zeta R_V}_{\text{Loss of Immunity}} \\
 \dot{E}_U = \underbrace{(\alpha M_U + \nu K_U) I}_{\text{Exposure}} - \underbrace{\sigma E_U}_{\text{Infection}}
 \end{array} \right. \quad (4)$$

$$\left\{ \begin{array}{l}
 \dot{E}_v = \underbrace{(\mu M_v + \beta K_v)I}_{\text{Exposure}} - \underbrace{\sigma E_v}_{\text{Infection}} \\
 \dot{I}_u = \underbrace{\sigma E_u}_{\text{Infection}} - \underbrace{\gamma I_u}_{\text{Recovery}} \\
 \dot{I}_v = \underbrace{\sigma E_v}_{\text{Infection}} - \underbrace{\chi I_v}_{\text{Recovery}} \\
 \dot{R}_u = \underbrace{\gamma I_u}_{\text{Recovery}} - \underbrace{\zeta R_u}_{\text{Loss of Immunity}} \\
 \dot{R}_v = \underbrace{\chi I_v}_{\text{Recovery}} - \underbrace{\zeta R_v}_{\text{Loss of Immunity}} \\
 I = \underbrace{I_u + I_v}_{\text{Total Infected}}
 \end{array} \right. \quad (5)$$

$\rho(t)$ and $\tau(t)$ are linear vaccination rates “turned on” at time $t = t_v$.

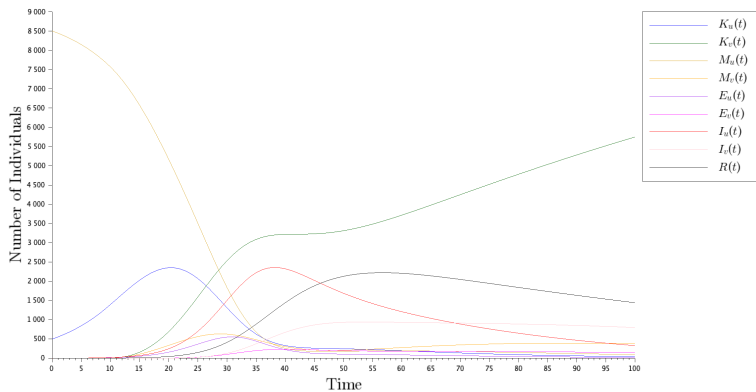


FIGURE: $MKEIVR$ plotted over time t , where $R(t) = R_u(t) + R_v(t)$

AN AGENT-BASED MODEL

DEFINITION (ABM)

An **agent-based model** (ABM) is a stochastic model that codes agents (i.e. people, animals, etc.) which interact with each other probabilistically via pre-set parameters.

Q: What if we ran an ABM of our *MKEIVR* model?

A: It turns out, the plots agree *extremely* well!

AN AGENT-BASED MODEL

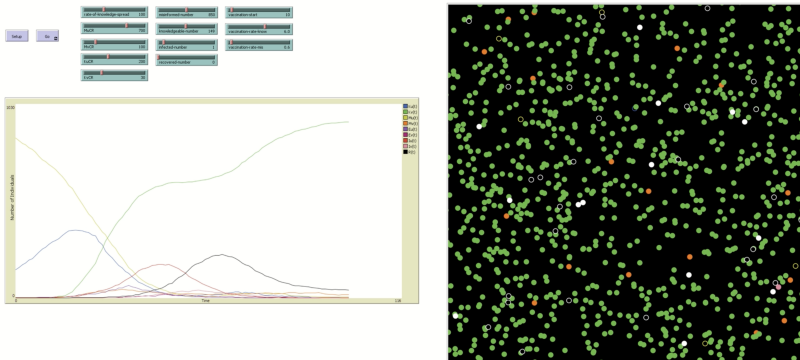


FIGURE: Still of *MKEIVR* spatial simulation and plot

APPENDIX: A PROSPECTIVE 2-DOSE MODEL

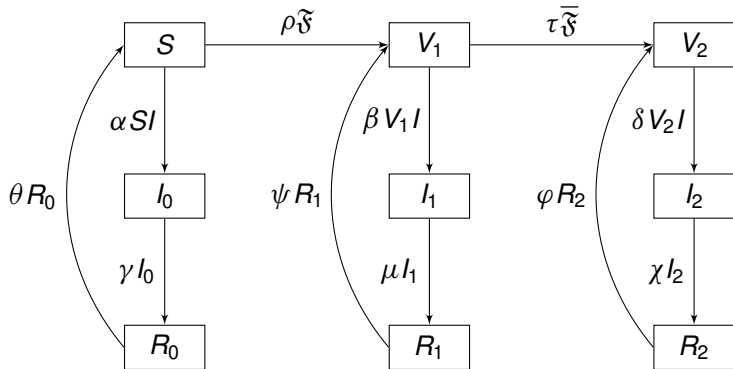


FIGURE: SV_2IR transfer diagram including two doses of vaccination, where $\mathfrak{F} = \min\{S, \lambda F\}$ and $\bar{\mathfrak{F}} = \min\{S, (1 - \lambda)F\}$, given a stock F of vaccines

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Any and all inquiries regarding our modeling techniques, results, and prospective models are welcome at dheeran2@illinois.edu.