Simulation of Epidemic Dynamics Using a Modified SEIRS Model

Árpád Elekes
0000-0002-5878-6566
Budapest, Hungary
elekes.a@freemail.hu

***Abstract*—A modified SEIRS epidemic model is introduced and then used to simulate some scenarios, such as possible recurrence of the disease and the impact of restrictive control measures on the dynamics. Also spatial spreading investigated among neighboring regions.**

***Keywords*—epidemic modeling, epidemic simulation, compartment models, SEIRS model**

I. Introduction

This work was motivated obviously by the recent Covid-19 pandemic (and an accidental encounter with an introduction [1],[2]). Initially the main questions of interest were the conditions for a possible recurrence and the spatial spreading of the disease among neighboring interconnected regions. It turned out however, that the model parameters presently cannot reliably be assessed to address these issues realistically enough. This is partly the reason why the treatise here has become more general. So, what follows is a straightforwardly extended SEIRS (still rather simplified) model of a contagious disease and the investigation of some interesting scenarios (so not a full extent examination) based on the simulation of this model.

The most common quantitative method of describing epidemic dynamics is the use of compartment models, represented by a system of differential equations. Here the SEIRS model from this family was choosen as an initial model to extend, and solved numerically for different initial conditions and parameter settings.

II. THE SEIRS BASE MODEL

A. *Features of the SEIRS model without vital dynamics*

- Compartments of the model: susceptible *S*, exposed *E*, infectious *I*, recovered *R*.
- The parameters: transmission rate β, virulence rate σ, recovery rate *γ*, resusceptibility rate *ρ*.
- The progression in the model: a susceptible individual by contacting the disease can be infected, first not contagious yet (in the exposed group), then after the incubation period can be infectious, and even later can recover. If the immunity only temporary, re-susceptibility is possible (Fig. 1.).

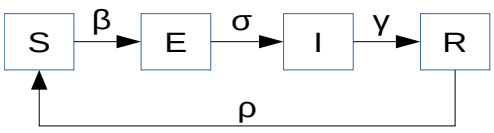

Fig.1.The SEIRS model.

B. *The governing equations of the system*

$$\dot{S} = -\beta SI + \rho R \quad (1)$$

$$\dot{E} = \beta SI - \sigma E \quad (2)$$

$$\dot{I} = \sigma E - \gamma I \quad (3)$$

$$\dot{R} = \gamma I - \rho R \quad (4)$$

III. THE PROPOSED EXTENDED SEIRS MODEL

A. *The additional features and differences of the new model over the base SEIRS model are:*

- In order to be able to take into account the mortality from the disease a new *D* compartment is introduced for the deceased part of the population (Fig. 2.).

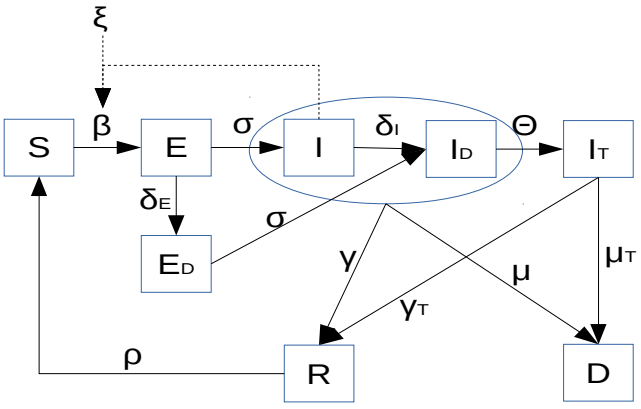

Fig. 2. The SEIRSDx model.

- Counter measures (i.e. quarantine, hygiene, social distancing...) conditional on some signal (i.e. the number of fatalities or new cases rises above a limit) have a decreasing effect on the transmission rate: $0 \le \xi \le 1$ in (5).
- Here the original SEIRS infectious group separated for three distinct compartments: the non-diagnosed *I*, the diagnosed and non-treated I_D , and the diagnosed and medically treated I_T . The treatment is supposed to be beneficial on the recovery, that is why the *γ* recovery and *μ* mortality rates of the treated and not treated class can be different.
- A susceptible individual by contacting the disease and becoming infected, first not yet infectious (in the

exposed group), then after the incubation period (can be zero) can be virulent. If diagnostication possible after the undiagnosability period then according to the diagnostic capacity rate the infected can be diagnosed and in this case, automatically separated from the rest of the population. The diagnosed then, depending on the actual health service capacity, can be medically treated, and depending on the recovery rate and mortality rate later can recover or decease. If the immunity only temporary, a recovered person can become susceptib again depending on the re-susceptibility rate.

- The diagnostic capacity rate δ (number of diagnosis/ time) is assumed to be constant (11).
- The actual medical treatment capacity $\theta(t)$ is the number of new cases could be provided with medical care at time t, which is the available capacity remaining from an initial (θ_{MAX} constant) value diminished by the already treated group $I_T(t)$ (12).
- The infectious groups and the recovered group R stratified by delay (from a reference) time (while the S, D groups are not). This allows treating the transitions between these groups (delay) time dependent, not just constants: recovery rate γ_{NT}, mortality rate μ_{NT}, treated recovery rate γ_T, treated mortality rate μ_T, resusceptibility rate ρ are all can be functions of time (starting from the joining time to the given compartment).
- In the simplest (constant) case (which is used here in the later case studies) it is possible to set the starting point in time of the:
  - virulence (latency, incubation period),
  - diagnosability,
  - recovery,
  - mortality,
  - resusceptibility.

B. The major equations

$$\dot{S} = -\beta\xi S I + \rho R \tag{5}$$

$$\dot{E} = \beta\xi S I - \sigma E - \delta_E \tag{6}$$

$$\dot{E}_D = \delta_E - \sigma E_{ND} \tag{7}$$

$$\dot{I} = \sigma E - \delta_I - (\gamma+\mu) I \tag{8}$$

$$\dot{I}_D = \sigma E_{ND} + \delta_I - (\gamma+\mu) I_D - \theta \tag{9}$$

$$\dot{I}_T = \theta - (\gamma_T+\mu_T) I_T \tag{10}$$

$$\delta = \text{const} = \delta_E + \delta_I \tag{11}$$

$$\theta = \theta_{MAX} - I_T \tag{12}$$

$$\dot{R} = \gamma(I+I_D) + \gamma_T I_T - \rho R \tag{13}$$

$$\dot{D} = \mu(I+I_D) + \mu_T I_T \tag{14}$$

$$\delta_E = \delta \frac{E}{(S+E+I)} \tag{15}$$

$$\delta_I = \delta \frac{I}{(S+E+I)} \tag{16}$$

C. What is still missing

What is not considered here, but for some disease could be an important factor too:

- vaccination
- vital dynamics (demography)
- age differences
- gender differences
- no distinction is made between the E—>E_D and I—>I_D transitions (14),(15), but obviously should. (symptomatic vs. asymptomatic case) $\delta = \delta_{Testing}$ (=const) + no. of symptomatic cases (for diseases which have fully diagnostic symptomes)

IV. SOME RESULTS

A. Epidemic recurrence

One way of how cyclic disease wave can develop is when the immunity is not final, so after some time the members of the recovered population can become susceptible again.

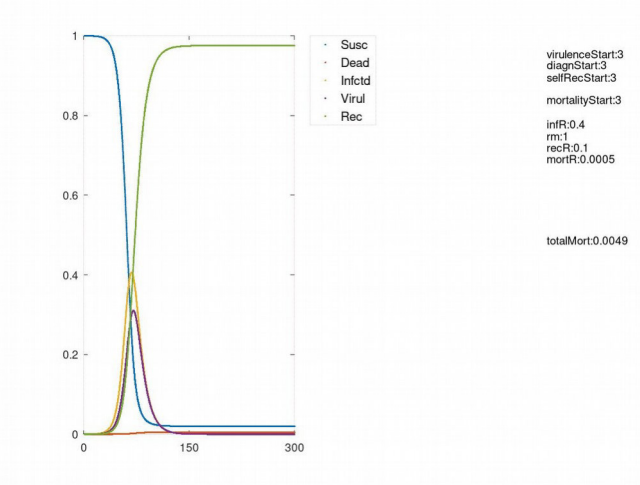

Fig.3. A possible scenario without recurrence

An epidemic second and third wave can develop also when the restriction measures introduced and applied alternately, even without re-susceptibility Fig.8. .

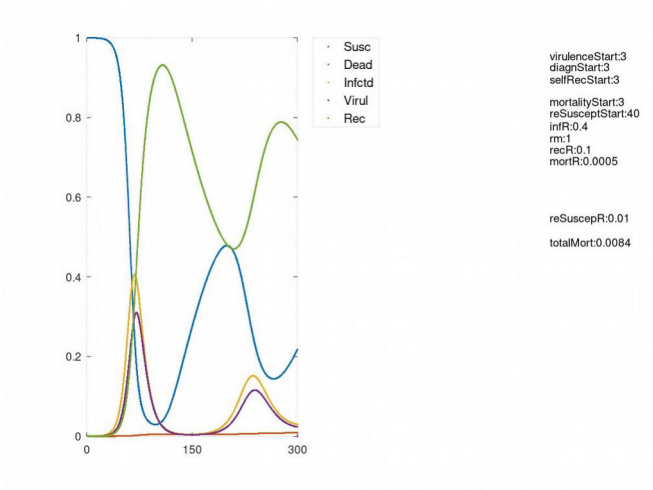

Fig.4. Recurrence scenario with the same settings except now recurrence starts after 40 days with re-susceptibility rate 1%.

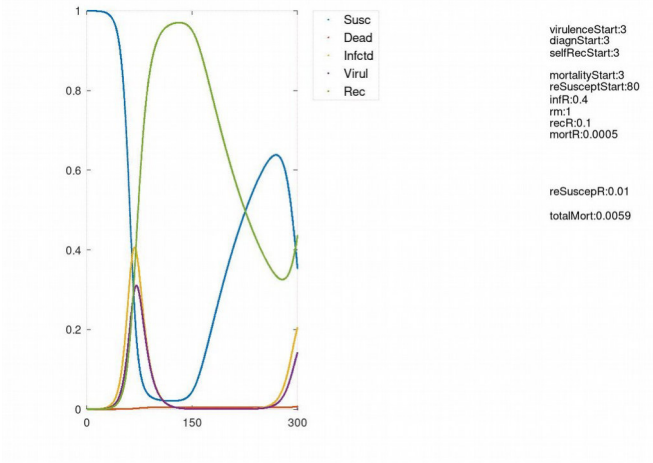

Fig.5. Recurrence scenario with the same settings except now recurrence starts after 80 days.

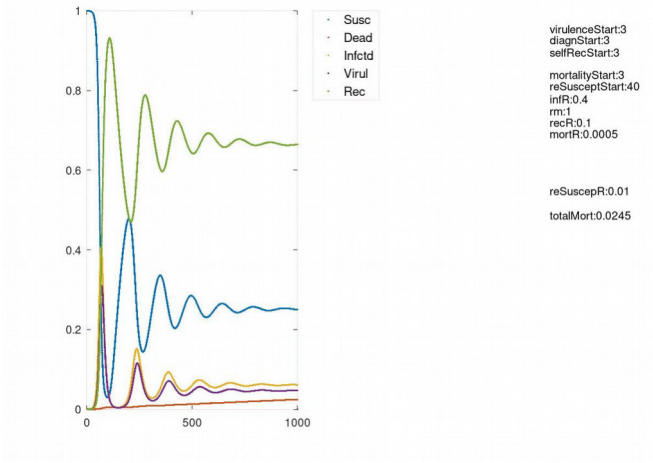

Fig.6. The longer run of Fig.4. scenario.

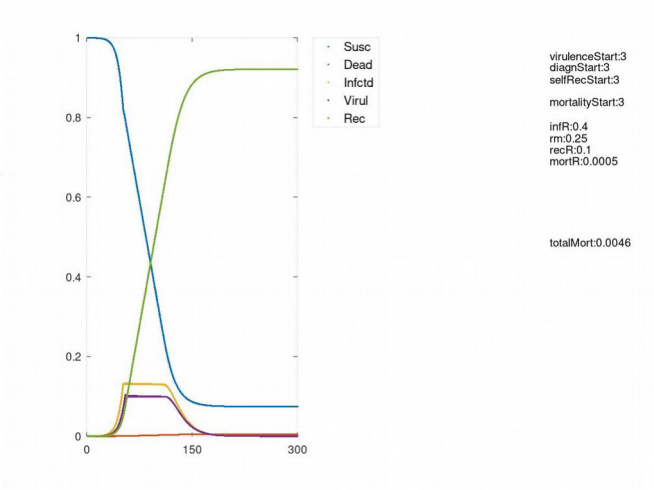

Fig.7. The scenario of Fig.3. with control measure effect $\xi = 0.25$. The restriction is applied is when the number of infected cases exceeded the 15% of the non-infected population.

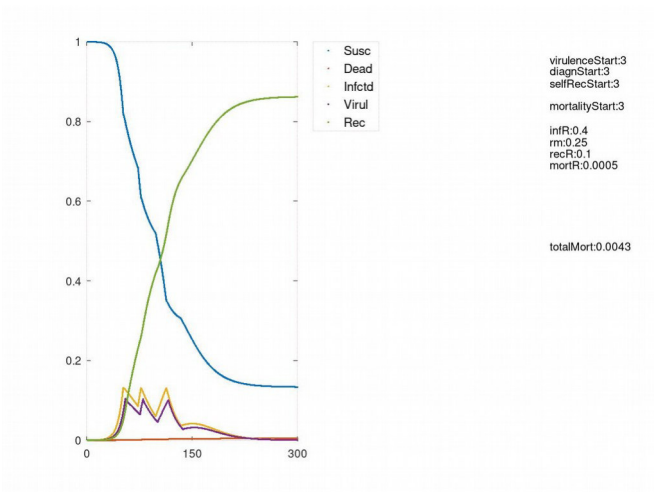

Fig.8. Recurrence scenario of Fig.4. settings (still without re-susceptibility) but here the restriction last for a prefixed 3 weeks (the restriction starting signal is the same).

B. Spatial spreading of an epidemic

One of the basic assumptions of compartment models is the homogeneous mixing of the population, which is not realistic over the entire course of an epidemic and on the larger territories considered. That is why it can be interesting to divide a major territory to subareas with possibly different population densities and even maybe some parameters Fig.9. Here some population exchange (only the non-diagnosed, so non-quarantined) is allowed between connected areas.

Also the parameters of real life epidemics have a rather random nature, so it can be interesting to include this feature in the compartment models too (Fig.10.).

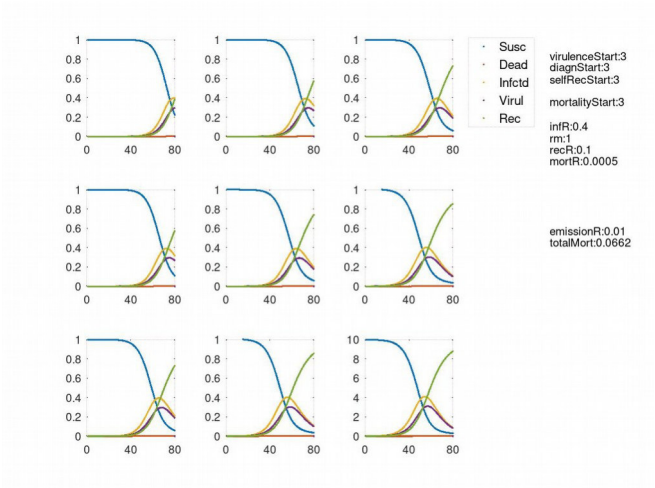

Fig.9. Epidemic spreading on a grid of 3x3 areas, starting from the bottom right.

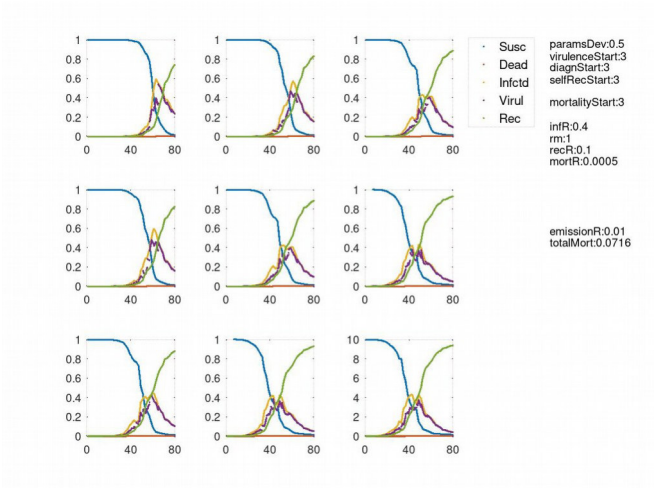

Fig.10. A more realistic looking Fig.9. case with parameters randomly changing (normal distribution, with standard deviation 0.5).

CONCLUSION

The difficulty of prediction of course lies not in the math but in the biology, namely: how to interpret the highly random real life statistics to obtain or measure the relevant parameters of the models.

REFERENCES

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