Assignment 2

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April 16, 2020

Note: Matlabs ode45 with the tolerances given in the assignment (section 2) was used for all simulations except the discrete model. Implementations are included in section 6.

1 Initial conditions

Suppose we treat the variables S, E, I, R as number of individuals, so that $S + E + I + R = N$ is the population size. Also let F denote the force of infection, $F = N^{-1}\beta I$. Then introducing a scale factor $Z \in \mathbb{R}^+$, so that $N_z = ZN$ and $F_z = N^{-1}\beta I_z = N^{-1}\beta ZI$, we get

$$\begin{aligned} S_z' &= -F_z S_z = -Z^2 FS = Z^2 S' \\ E_z' &= F_z S_z - \kappa E_z = Z^2 FS - Z\kappa E \\ I_z' &= -\gamma I_z + \kappa E_z = Z(-\gamma I + \kappa E) = ZI' \\ R_z' &= \gamma I_z = ZR' \end{aligned}$$

Notice that the solutions S_z, I_z and R_z can be written as (for example)

$$\int S_z' dt = Z^2 \int S' dt \ ,$$

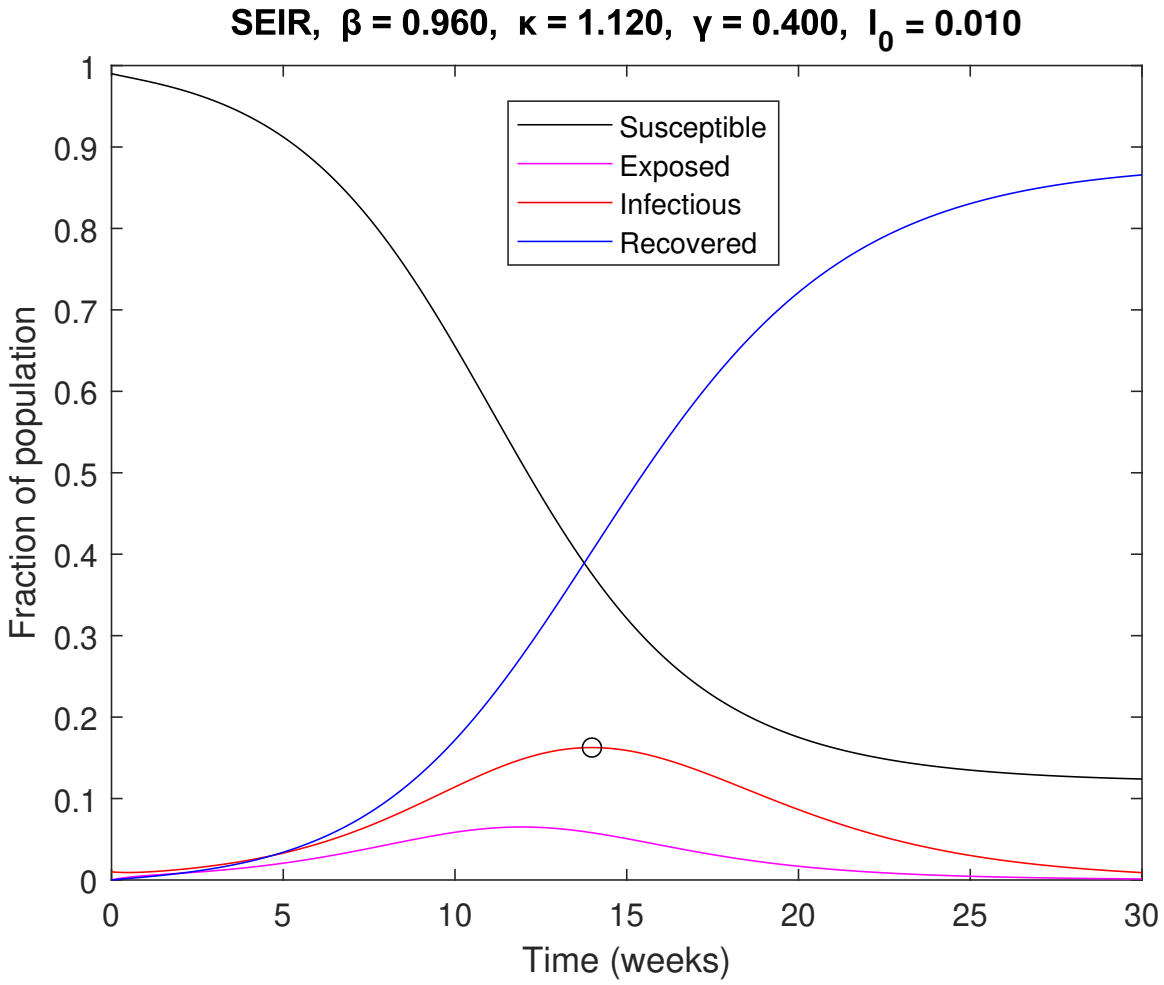
and similarly for I_z and R_z. However, we cannot do the same for E_z. This is no good if we want to scale a solution for various population sizes. So instead, we make the system independent of population size by treating the variables as fractions of the population rather than number of individuals. Let initial conditions $S_0, E_0, I_0, R_0 \in [0, 1]$ such that $S_0 + E_0 + I_0 + R_0 = 1$ and $F = \beta I$. We then get

$$\begin{aligned} S' &= -FS = -\beta IS \\ E' &= FS - \kappa E = \beta IS - \kappa E \\ I' &= -\gamma I + \kappa E \\ R' &= \gamma I \ , \end{aligned}$$

and solutions can simply be scaled by N to obtain data in terms of number of individuals. If we wanted to model a population of 14 000 with 7 infectious individuals at $t = 0$, we would take $I_0 = \dfrac{7}{14000} = 0.0005$, i.e. 0.05% of the population are infectious. Then $S_0 = 1 - 0.0005$.

2 First simulation

At $t = 10$, we have $(S, E, I, R) = (0.6563, 0.0585, 0.1139, 0.1713)$. So approximately 65.6% of the population are susceptible, 5.9% exposed, 11.4% infectious and 17.1% recovered.
We should expect to see the infectious fraction reach its peak when people are recovering at the same rate as they are becoming infectious. That is, when $R' = \kappa E \iff \gamma I = \kappa E \iff I' = 0$.



We see that I goes to zero, and S approaches some fixed point S_*. Since $S' \leq 0$ and the population is fixed in this model, (no birth- or death rate), and we also assume that the recovered fraction has permanent immunity, there is nothing to affect S other than exposure. Thus, S must eventually decrease to a point where exposure levels out, i.e. $\beta IS = \kappa E$. Then I must also level out as E begins to decrease, which then slows the decrease of S (S' increases as I decreases). Eventually the system converges on an equilibrium where $S = S_*$ and $R = 1 - S_*$. This equilibrium should be stable for all initial conditions (assuming $\gamma, \beta > 0$), since otherwise

$$\lim_{t\to\infty} I(t) > 0 \implies \lim_{t\to\infty} R'(t) = \gamma \lim_{t\to\infty} I(t) > 0 \,,$$

in which case R would grow without bound, which would be contradictory.

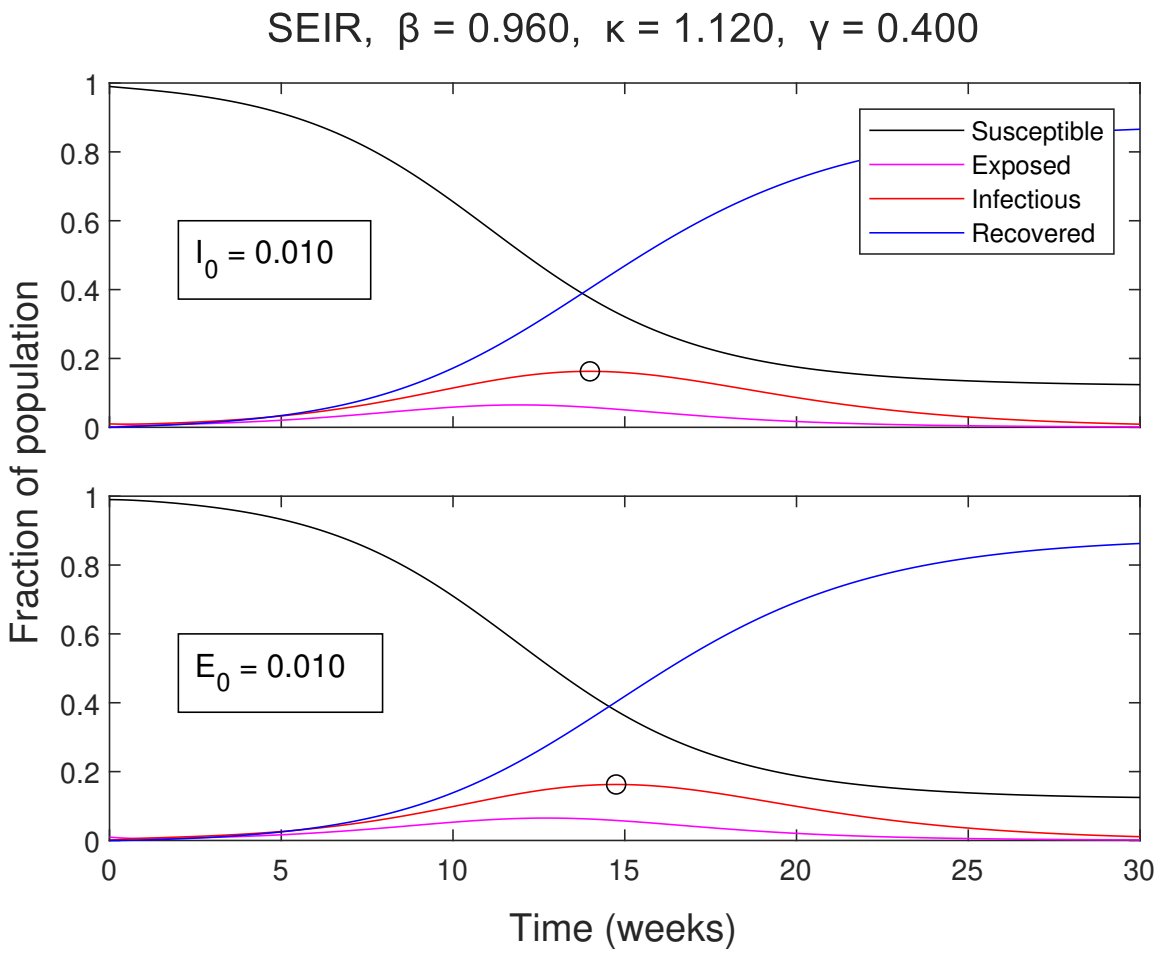


Figure 1: Starting with infectious vs starting with exposed

Staring with exposed individuals rather than infectious does not significantly affect the evolution with these parameters. We see that starting with exposed individuals shifts the infectious peak forward by about a five days, from $t = 13.99$ to $t = 14.75$. This is along the lines of what one might expect, since I' depends linearly on κE. With a smaller value of κ, i.e. a longer incubation time, the time of peak infection is shifted farther and vice versa.

With the discrete model, we have $(S, E, I, R) = (0.7869, 0.0443, 0.0749, 0.094)$ at $t = 10$. If we use our initial simulation as ground truth, that is a mean local relative error of about 31%. However, the model is stable with $h = 1$ and exhibits the same behaviour as its continuous counterpart. Thus the previous conclusions do not change, even though the propagated error is probably quite large. Using the discrete model in a real scenario would not be a good idea though.

3 Impact

For a first simulation, I set $\alpha = 0.04, \mu_h = 0.0175, \mu_n = 0.5$ based on the given data. The other parameters are set to the midpoints of the suggested intervals. Taking $I_h = 1$ produces the following results, starting with 3 infectious individuals out of 10 million:

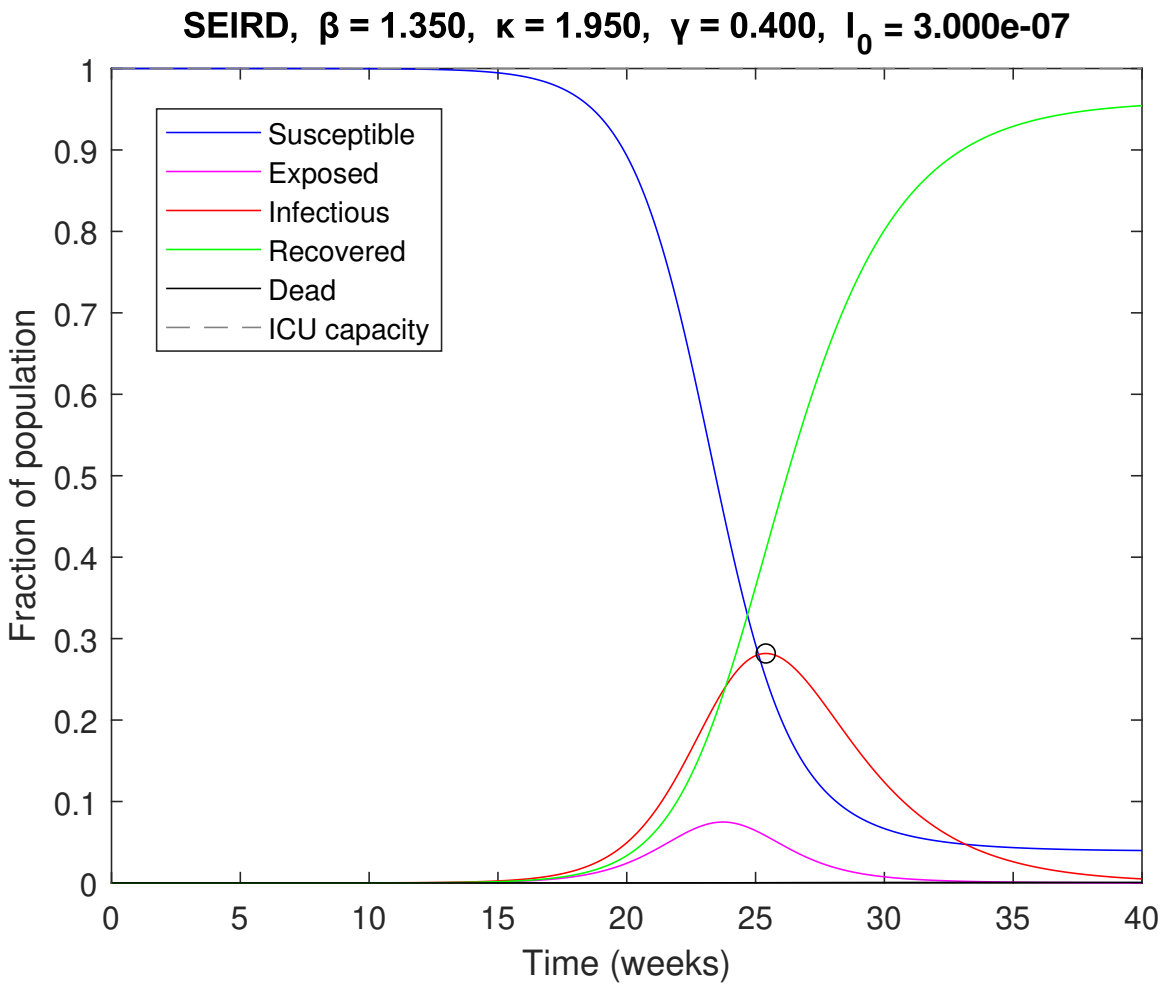


Figure 2: $\alpha = 0.04,\ \mu_h = 0.0175,\ \mu_n = 0.5,\ I_h = 1$

At $t = 40$ we have $D = 0.0007$, i.e. the death toll is 0.07% of the population, which comes out to 7000 people. Setting I_h to a more realistic value of 10^{-4} brings the death toll up by orders of magnitude, to 1.86% or 186 000 people.

As for the initial infectious population, it can increase by many orders of magnitude without affecting the evolution of the system other than shifting it in time. That is, the onset of the epidemic is quicker, but it has the same behaviour and equilibrium. The death toll with ICU capacity of 1000 and initial infectious population of 100 000 comes out to 1.87%, shown in figure 3.

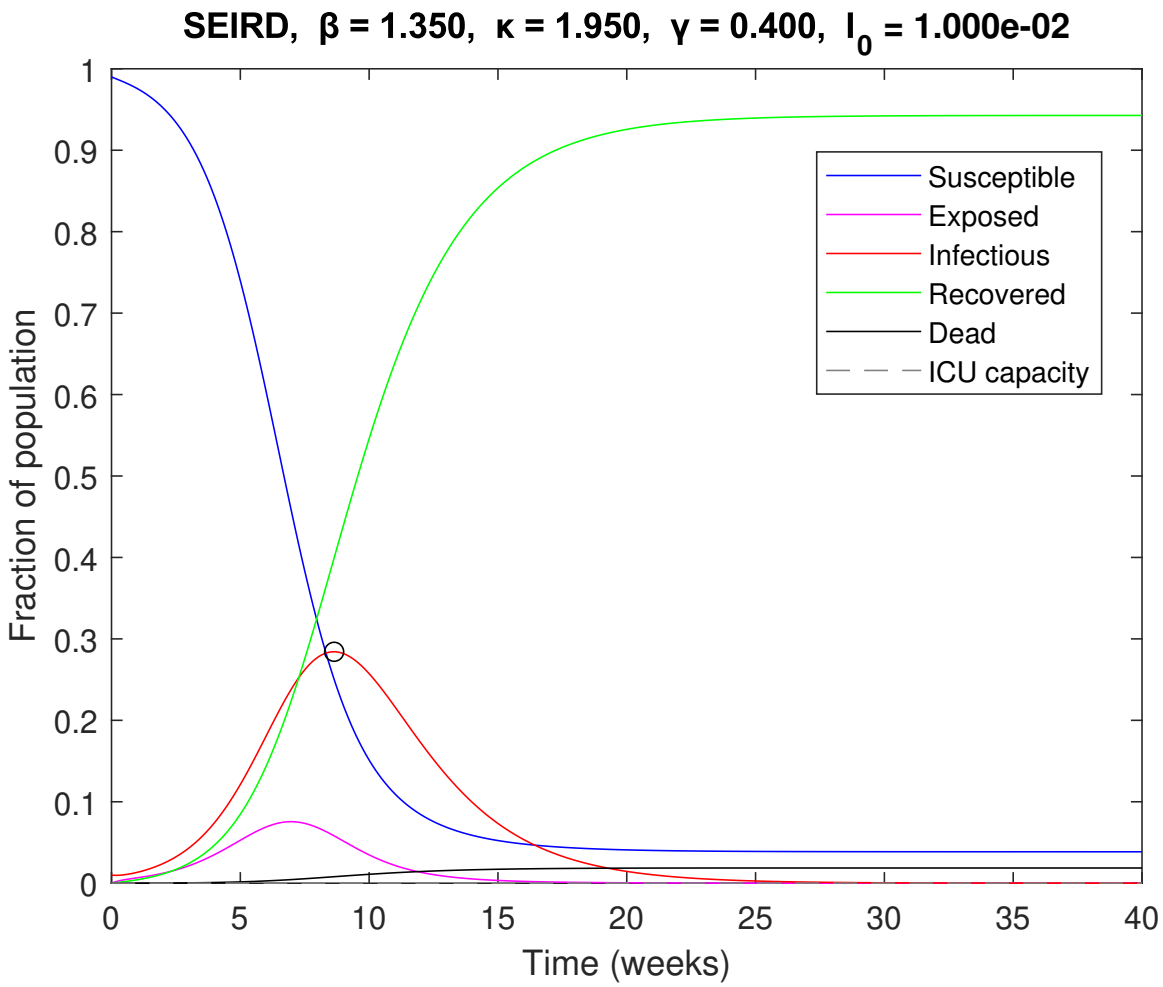


Figure 3: $\alpha = 0.04,\ \mu_h = 0.0175,\ \mu_n = 0.5,\ I_h = 10^{-4}$

4 Countermeasures

I will make the following assumptions:

All acute cases are symptomatic

50% of the *total* cases are asymptomatic

Asymptomatic cases are never acute

This implies that out of the total infectious population, 50% minus those that are hospitalized can be subject to quarantine. Thus at least 50% of I will always be contributing to exposure. Then the calculation would look like

$$I - 0.6(0.5I - H) - 0.95H = (1 - 0.3)I + (0.6 - 0.95)H = 0.7I - 0.35H\ ,$$

where $H = \min\{\alpha I,\ I_h\}$. That is, $c_1 = 0.7$ and $c_2 = -0.35$. Note that this imposes a restriction on α; since asymptomatic cases cannot be acute, we must have $\alpha \in [0,\ 0.5]$.

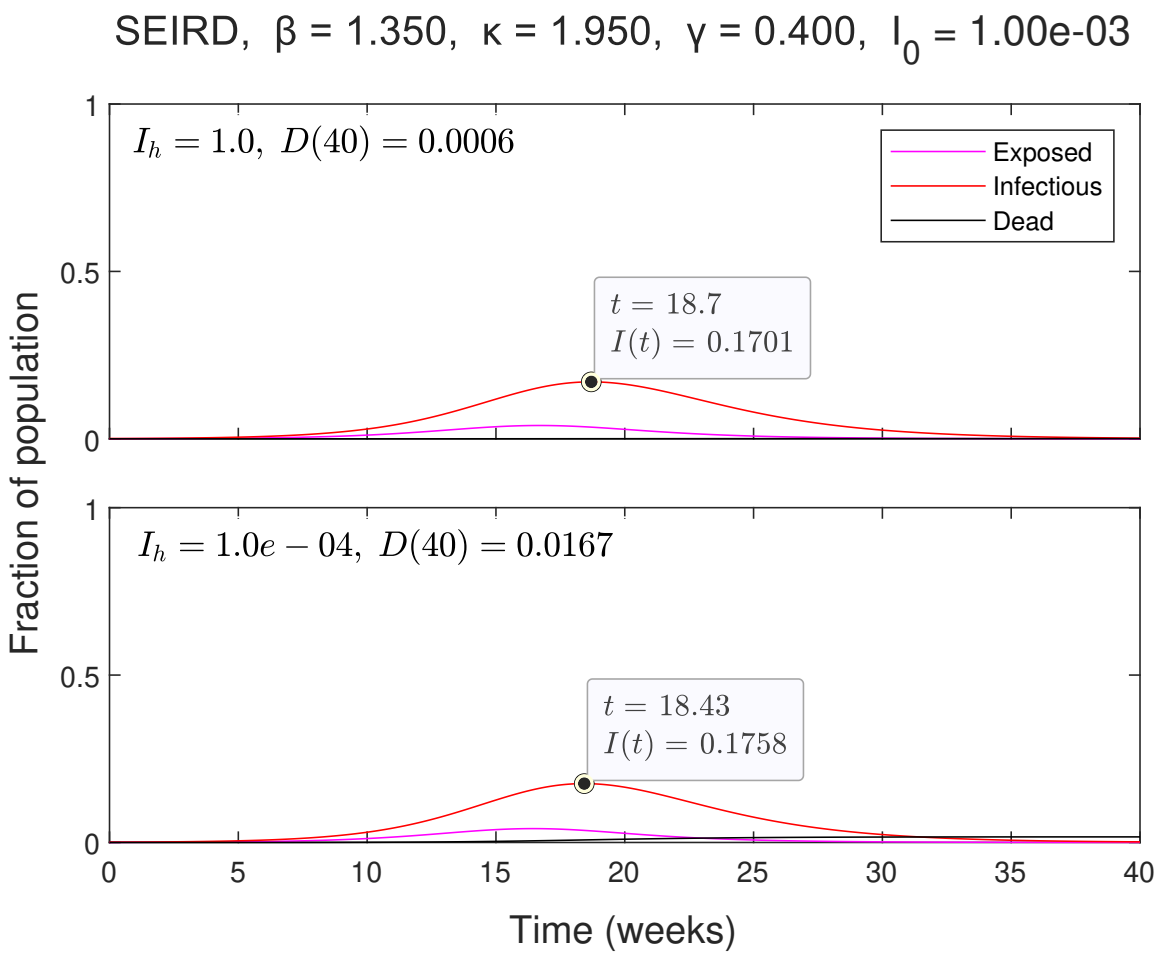


Figure 4: Model with asymptomatic cases, quarantine and reduced exposure in hospitals.
$\alpha = 0.04,\ \mu_h = 0.0175,\ \mu_n = 0.5$

As with the previous simulations, we see a huge impact on the death toll when reducing the ICU capacity. The difference is on the same order of magnitude as before, but in both cases the death toll is lower; 0.06% vs 0.07% and 1.67% vs 1.86%. The impact of quarantine can be seen in the significantly flatter infection curve, peaking at 17.58% vs 28.42%, which should be the main contributing factor in reducing the death toll since the mortality is very high in untreated acute cases.

If we ditch the quarantine and instead want to reduce the rate of contact for the susceptible and non-hospitalized individuals in such a way that the force of infection F remains the same, then we would like to determine β_2 such that

$$F_2 = \beta_2(I - H) + \beta_1 0.05H = F_1 = \beta_1(0.7I - 0.35H)\ .$$

This equation is not consistent, however equating the coefficients of I yields $\beta_2 = 0.7\beta_1 = 0.945$. Setting $\beta = 0.945$ produces very similar results to the quarantine simulation, with a death toll of 16.6% and an infection peak of 17.56%. So a reduction in contact rate of 30% is sufficient.

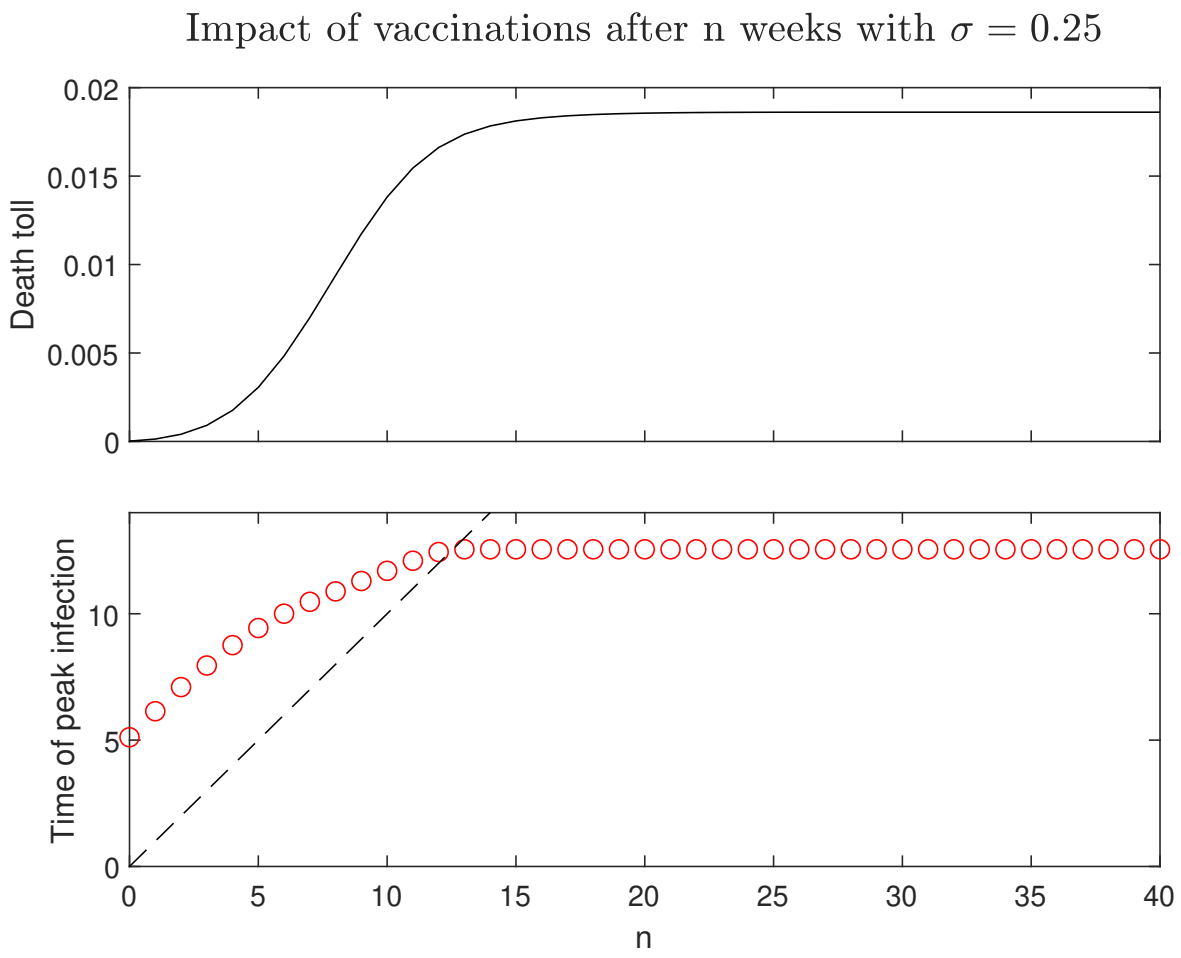


Figure 5: No countermeasures except for vaccinations.
$\alpha = 0.04,\ \mu_h = 0.0175,\ \mu_n = 0.5,\ I_h = 10^{-4}$

Looking at the death toll and peak of infection as functions of n, we see that starting vaccinations after the peak has very little effect.

5 Conclusions

There is an obvious limitation to our models, which is touched upon in section 2. That is the fact that we are not including vital dynamics, and we are also equating recovery with permanent immunity. This means that the models are not useful for modelling long term dynamics of a virus that remains endemic in the population. They are however useful for modelling an isolated epidemic. Out of our initial parameters, the relation between β and γ has the most impact on the outcome, which is not surprising since β scales the nonlinear term IS, which provides the exponential growth of E and subsequently I. If we have a basic reproduction number $R_0 = \beta\gamma^{-1} < 1$, then I will not grow at all.

Introducing zombies, we will start the last revision of the model, where

$$\begin{aligned}
H &= \min(\alpha I, I_h) \\
A_u &= \max(0, \alpha I - I_h) \\
F &= \beta\big((1-qs)I + (q-\rho_h)H\big)
\end{aligned}$$

$$\begin{aligned}
S' &= -FS - \delta_n(t)\sigma S \\
E' &= FS - \kappa E \\
I' &= \kappa E - \gamma I \\
R' &= \gamma\big((1-\mu_h)H + (1-\mu)A_u + (1-\alpha)I\big) \\
D' &= \gamma(\mu_h H + \mu A_u) \\
V' &= \delta_n(t)\sigma S
\end{aligned}$$

The parameters q and s are quarantine success rate and percentage with symptoms, respectively. We will model the zombie state by introducing an additional compartment Z. The transition from I to D will now instead be from I to Z, since there is typically not much of an incubation time for reanimation (it's a matter of hours or even minutes. Source: Hollywood). It is also widely known that sufficient brain trauma will kill a zombie. Therefore, we will introduce a parameter ζ which will determine the rate of transition from Z to D. That is, ζ is the average capability of the healthy population to fight back. Finally, while recovery or vaccination grants immunity to the virus, it does not make people invulnerable. Thus we will introduce yet another parameter μ_z to account for the probability of death resulting from the physical trauma of a zombie attack. So we will have transitions $S \to E$ and $S \to Z$ as well as $R \to D$ and $V \to D$. It would be reasonable to assume that people try to avoid contact with zombies, but on the other hand, zombies will actively seek out the living. Therefore, to simplify things a bit, we will use the same rate of contact β for these transitions. The new model will look like this:

$$\begin{aligned}
F &= \beta\big((1-qs)I + (q-\rho_h)H\big) \\
F_z &= \beta(1-\mu_z)Z
\end{aligned}$$

$$\begin{aligned}
S' &= -(F+F_z)S - \delta_n(t)\sigma S - \beta\mu_z ZS \\
E' &= (F+F_z)S - \kappa E \\
I' &= \kappa E - \gamma I \\
R' &= \gamma\big((1-\mu_h)H + (1-\mu)A_u + (1-\alpha)I\big) - \beta\mu_z RZ \\
D' &= \beta\big(\zeta(S+R+V) + \mu_z(R+V)\big)Z \\
V' &= \delta_n(t)\sigma S - \beta\mu_z VZ \\
Z' &= \gamma(\mu_h H + \mu A_u) + \beta\big(\mu_z S - \zeta(S+R+V)\big)Z
\end{aligned}$$

Setting $\mu_z = 0.6$, $\zeta = 0.1$ and running the simulation with our previous countermeasure parameters of 60% quarantine success rate and 50% asymptomatic cases, we get the following dire scenario:

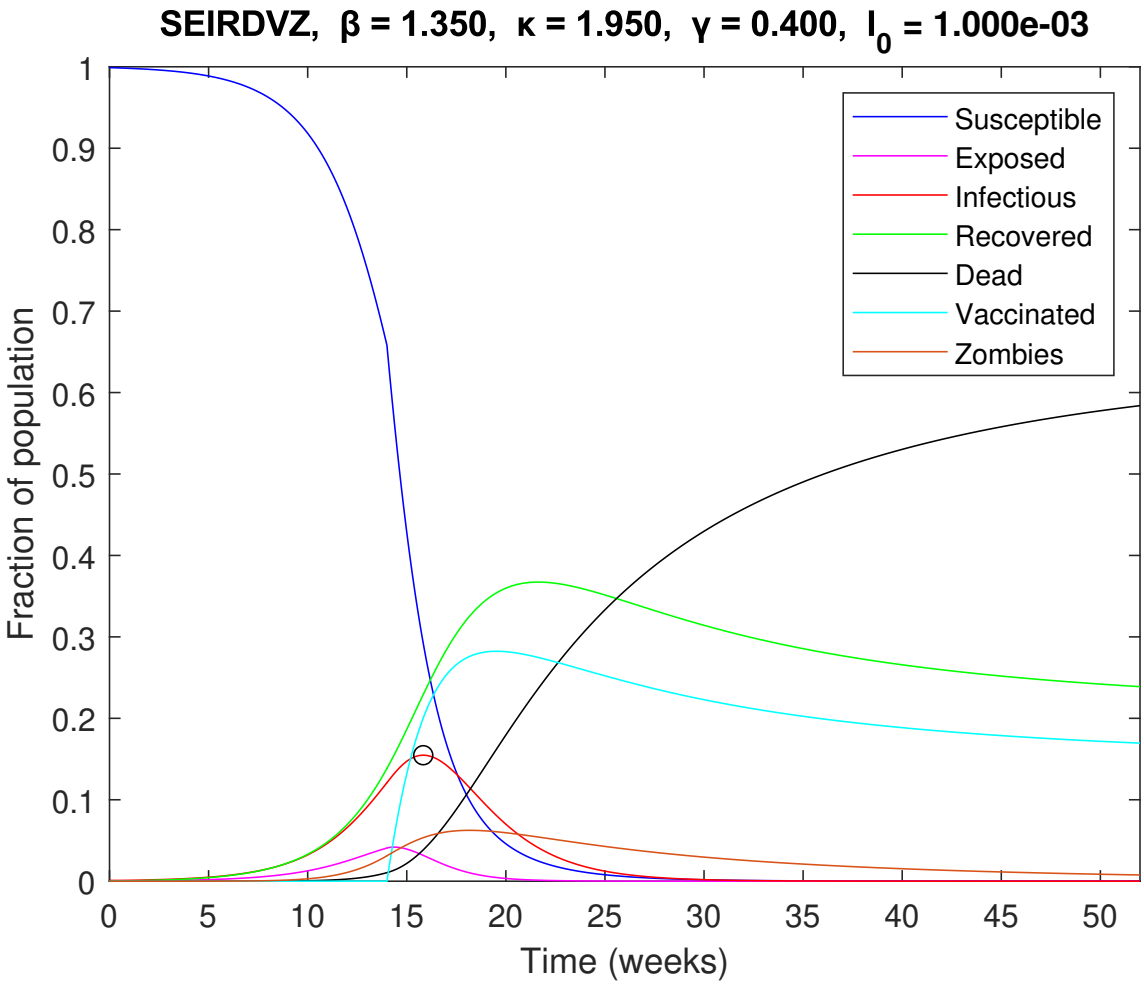


Figure 6: Model with zombies, using our initial countermeasure parameters.
$\alpha = 0.04,\ \mu_h = 0.0175,\ \mu_n = 0.5,\ \mu_z = 0.6,\ \zeta = 0.1,\ I_h = 10^{-4}$

Here, a whopping 58.4% of the population perish. The main contributing factors to this massive death toll is the rather high mortality rate of coming into contact with a zombie and the rather low rate at which zombies are killed off. The feedback loop of zombies generating more zombies becomes very fast. Also, people keep dying long after the infection trails off.

6 Code

My implementations of the models are included here. All of the code including scripts can also be checked out at `https://github.com/Fredrik-M/BERV-MCS/tree/master/MCS/lab_2`

6.1 First implementation

```
% p :: struct
% p.beta  -- average contact rate
% p.kappa -- average incubation rate
% p.gamma -- average recovery rate

function ddt = SEIR_ODE(t, seir, p)
    ddt = zeros(4, 1);
    [S,E,I,R] = feval(@(c)c{:}, num2cell(seir));
    F = p.beta * seir(3);

    ddt(1) = -F * S;
    ddt(2) = F * S - p.kappa * E;
    ddt(3) = p.kappa * E - p.gamma * I;
    ddt(4) = p.gamma * I;
end
```

6.2 Euler

```
function [t, y] = euler_fwd(f, tspan, y0, h)
    n      = 1 + round((tspan(2) - tspan(1)) / h);
    t      = linspace(tspan(1), tspan(2), n)';
    h_     = t(2) - t(1);
    y      = zeros(length(t), length(y0));
    y(1,:) = y0;

    for i = 2:length(t)
        y(i,:) = y(i - 1,:)' + f(t(i), y(i - 1,:)) * h_;
    end
end
```

6.3 Including deceased

```
% p.beta  -- average contact rate
% p.kappa -- average incubation rate
% p.gamma -- average recovery rate
% p.alpha -- percentage of infections with acute symptoms
% p.mu    -- baseline mortality among acute cases
% p.mu_h  -- mortality (acute cases) with hostpital treatment
% p.h     -- hospital ICU capacity (percent of population)

function ddt = SEIRD_ODE(t, seird, p)
    ddt = zeros(5, 1);
    [S,E,I,R,D] = feval(@(c)c{:}, num2cell(seird));
```

```
    acute_untreated = max(0, p.alpha * seird(3) - p.h);
    acute_treated   = min(p.alpha * seird(3), p.h);
    F = p.beta * I;

    ddt(1) = -F * S;
    ddt(2) = F * S - p.kappa * E;
    ddt(3) = p.kappa * E - p.gamma * I;
    ddt(4) = p.gamma * (1 - p.mu_h) * acute_treated ...
             + p.gamma * (1 - p.mu) * acute_untreated ...
             + p.gamma * (1 - p.alpha) * I;
    ddt(5) = p.gamma * p.mu_h * acute_treated ...
             + p.gamma * p.mu * acute_untreated;
end
```

6.4 Including vaccination and countermeasures

```
% p.beta  -- average contact rate
% p.kappa -- average incubation rate
% p.gamma -- average recovery rate
% p.s     -- percentage of infections with symptoms :: [0, 1]
% p.alpha -- percentage of infections with acute symptoms :: [0, 1 - p.s]
% p.mu    -- baseline mortality among acute cases
% p.mu_h  -- mortality (acute cases) with hostpital treatment
% p.h     -- hospital ICU capacity (percent of population)
% p.q     -- quarantine effectivness (percent of symptomatic cases)
% p.rho_h -- hospital exposure reduction
% p.sigma -- average vaccination rate
% p.n     -- time to develop vaccine

function ddt = SEIRDV_ODE_QSS(t, seirdv, p)
    ddt = zeros(5, 1);
    [S,E,I,R,D,V] = feval(@(c)c{:}, num2cell(seirdv));

    acute_untreated = max(0, p.alpha * I - p.h);
    acute_treated   = min(p.alpha * I, p.h);
    F = p.beta * (I * (1 - p.q * p.s) + acute_treated * (p.q - p.rho_h));

    ddt(1) = -F * S - (t >= p.n) * p.sigma * S;
    ddt(2) = F * S - p.kappa * E;
    ddt(3) = p.kappa * E - p.gamma * I;
    ddt(4) = p.gamma * (1 - p.mu_h) * acute_treated ...
             + p.gamma * (1 - p.mu) * acute_untreated ...
             + p.gamma * (1 - p.alpha) * I;
```

```
    ddt(5) = p.gamma * p.mu_h * acute_treated ...
             + p.gamma * p.mu * acute_untreated;
    ddt(6) = (t >= p.n) * p.sigma * S;
end
```

6.5 Zombies

```
% p.beta  -- average contact rate
% p.kappa -- average incubation rate
% p.gamma -- average recovery rate
% p.s     -- percentage of infections with symptoms :: [0, 1]
% p.alpha -- percentage of infections with acute symptoms :: [0, 1 - p.s]
% p.mu    -- baseline mortality among acute cases
% p.mu_h  -- mortality (acute cases) with hostpital treatment
% p.h     -- hospital ICU capacity (percent of population)
% p.q     -- quarantine effectivness (percent of symptomatic cases)
% p.rho_h -- hospital exposure reduction
% p.sigma -- average vaccination rate
% p.n     -- time to develop vaccine
% p.zeta  -- average zombie-fighting capacity
% p.mu_z  -- average mortality of zombie attack

function ddt = SEIRDVZ_ODE_QSS(t, seirdvz, p)
    ddt = zeros(5, 1);
    [S,E,I,R,D,V,Z] = feval(@(c)c{:}, num2cell(seirdvz));

    acute_untreated = max(0, p.alpha * I - p.h);
    acute_treated   = min(p.alpha * I, p.h);
    F   = p.beta * (I * (1 - p.q * p.s) + acute_treated * (p.q - p.rho_h));
    F_z = p.beta * (1 - p.mu_z) * Z;

    ddt(1) = -(F + F_z) * S - (t >= p.n) * p.sigma * S - p.beta * p.mu_z * Z * S;
    ddt(2) = (F + F_z) * S - p.kappa * E;
    ddt(3) = p.kappa * E - p.gamma * I;
    ddt(4) = p.gamma * (1 - p.mu_h) * acute_treated ...
             + p.gamma * (1 - p.mu) * acute_untreated ...
             + p.gamma * (1 - p.alpha) * I ...
             - p.beta * p.mu_z * R * Z;
    ddt(5) = p.beta * (p.zeta * (S + R + V) + p.mu_z * (R + V)) * Z;
    ddt(6) = (t >= p.n) * p.sigma * S - p.beta * p.mu_z * V * Z;
    ddt(7) = p.gamma * p.mu_h * acute_treated ...
             + p.gamma * p.mu * acute_untreated ...
             + p.beta * (p.mu_z * S - p.zeta * (S + R + V)) * Z;
end
```