

FIT3139 Assignment 1 Q 3

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Part A:

Flow Diagram:

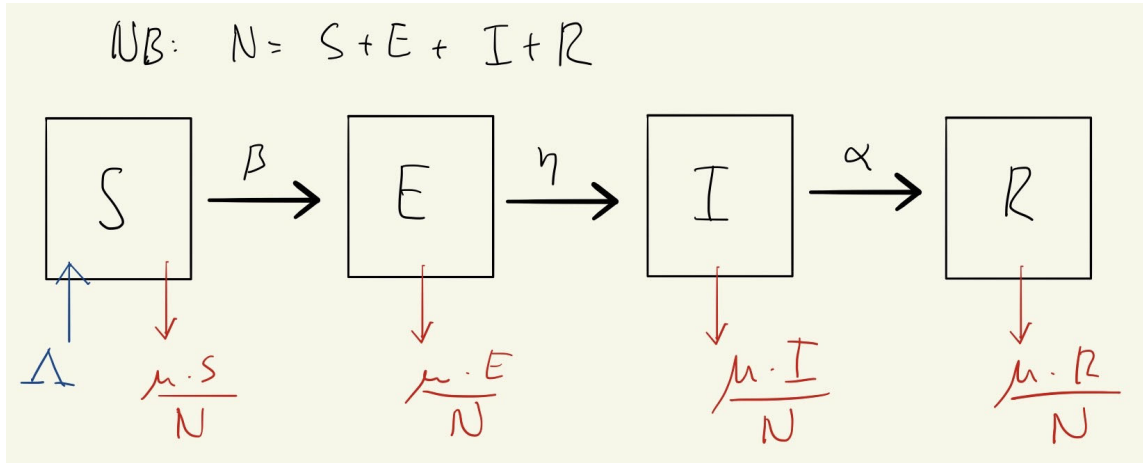


Fig 3.a.1: Flow diagram for a demographic SEIR model

Set of Equations:

The set of equations for this model may be given as:

$$\begin{aligned}\dot{S} &= -\beta SI + \Lambda - \mu \cdot \frac{S}{N} \cdot N = -\beta SI + \Lambda N - \mu S \\ \dot{E} &= \beta SI - \eta E - \mu E \\ \dot{I} &= \eta E - \alpha I - \mu I \\ \dot{R} &= \alpha I - \mu R\end{aligned}$$

Expression of N and long-run population size:

Logically we know that:

$$\dot{L} = \dot{S} + \dot{E} + \dot{I} + \dot{R}$$

Therefore we can expand and solve this as:

$$\begin{aligned}\dot{N} &= \frac{dN}{dt} = \dot{S} + \dot{E} + \dot{I} + \dot{R} \\ &= \beta SI - \beta SI + \eta E - \eta E + \alpha I - \alpha I + \Lambda N - \mu(S + E + I + R) \\ \frac{dN}{dt} &= \Lambda - N\mu\end{aligned}$$

This is a first-order separable non-homogeneous ODE (i.e.: $y' + p(x)y = q(x)$), which has a general solution in the form:

$$y(x) = \gamma(x) \cdot \int \frac{q(x)}{\gamma(x)} \cdot dx + C \cdot \gamma(x)$$

where $\gamma(x) = e^{-\int p(x) \cdot dx}$. Applying this to N we get:

$$\begin{aligned}\gamma(t) &= e^{-\int \mu \cdot dt} = e^{-\mu t} \\ \therefore N(t) &= e^{-\mu t} \cdot \int \frac{\Lambda}{e^{-\mu t}} \cdot dt + C \cdot e^{-\mu t}, \quad C \in \mathbb{R}^+ \\ &= e^{-\mu t} \cdot \frac{\Lambda}{\mu} \cdot e^{\mu t} + C \cdot e^{-\mu t} \\ N(t) &= \frac{\Lambda}{\mu} + C \cdot e^{-\mu t}\end{aligned}$$

Applying an initial condition of $N(0) = N_0$, we get a final equation of:

$$N(t) = \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right) e^{-\mu t}$$

This equation gives a very clear long-run stabilisation for $N(t)$ given that e^{-t} converges to 0 as t goes to infinity. We can see this as:

$$\begin{aligned}\lim_{t \rightarrow \infty} (N(t)) &= \lim_{t \rightarrow \infty} \left(\frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right) e^{-\mu t} \right) \\ &= \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right) \cdot 0 \\ &= \frac{\Lambda}{\mu}\end{aligned}$$

Thus, in the long run, the total population size converges to $\frac{\Lambda}{\mu}$.

Long-term behaviour:

Below are simulations of this SEIR model under the scenario. As required by the question, $N_0 = 1000$ and $E_0 = 1$. In all three, the values of β , η and α were set the same, at values of $\beta = 0.002$ which is roughly analogous to 0.2% of all possible interactions between infected and susceptible people resulting in expose, $\eta = 0.05$ resulting in a roughly 20 day latent period, and $\alpha = 0.1$ resulting in a roughly 10 day recovery period.

In the simulations below, both of the population shrinkage simulations use values of μ and Λ of 0.0007 and 0.4 respectively. This is done to give rough 10% annual death rate over 365 days. The ‘birth’ rate is equivalent to a person arriving every 2.5 days, reasonable for a community this size. These values are also large enough that the effects of demographics are clearly obvious. In the expansion simulation, the value for Λ is increased to 1, or one ‘birth’ a day.

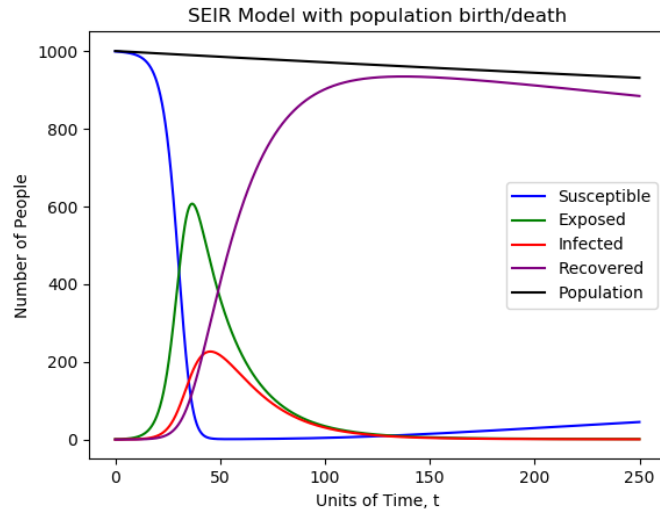


Fig 3.a.2: Short-run simulation with long-run population below the initial value



Fig 3.a.3: Long-run plot with pop. expansion

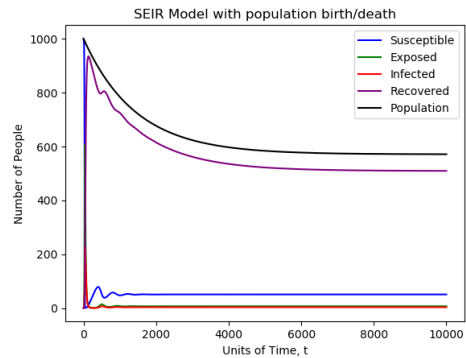


Fig 3.a.4: Long-run plot with pop. shrinkage

These simulations can be compared with a basic SEIR model as such:

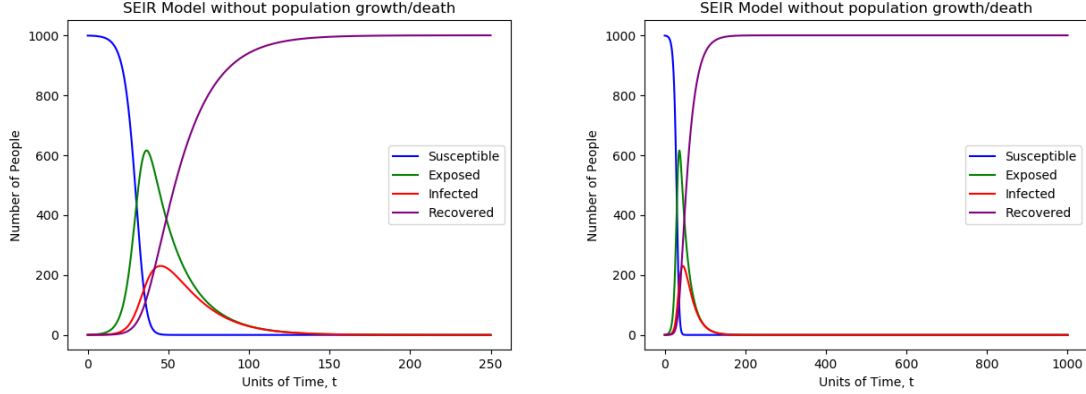


Fig 3.a.5: Short-run (left) and long-run (right) simulations of a basic SEIR model

From this we can see three main differences in the behaviour of these two models: a change in the total population, repeated waves of the disease, and convergence to a steady-state where the disease doesn't die out.

Firstly, the change in the population. As we derived above, unless someone chooses μ and Λ such that $N_0 = \Lambda/\mu$, the population is going to converge to some value other than the initial population value. This is clearly visible in figures 3.a.2 to 3.a.4 above via the black line.

Secondly, we may observe repeated diminishing spikes of disease in the demographic model as the disease resurges in the new susceptible population. This behaviour isn't observed in the initial model, where the disease spikes once. As a result of our births there are always susceptible people available for the disease to infect, and after initially burning through the susceptible population, the disease re-emerges in the form of aftershocks as the susceptible population builds up. These shocks gradually diminish as the system converges towards a steady-state.

This leads me on to my final point: that the disease does not die out like in the basic model. As a result of the new births and the non-discrete nature of the data, some portion of the population remains infected, resulting in a steady-state forming where some portion of the population remains infected.

Part B:

Flow Diagram:

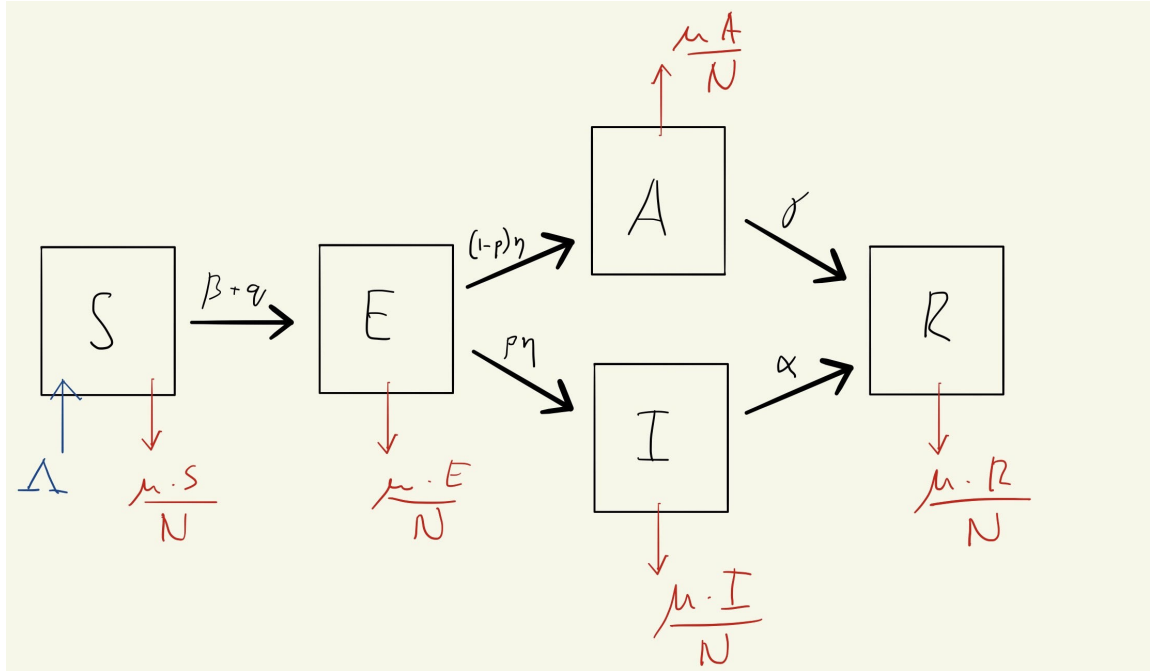


Fig 3.b.1: Flow diagram for a demographic SEIAR model

Set of Equations:

The set of equations for this model may be given as:

$$\dot{S} = -\beta SI - qSA + \Lambda - \mu S$$

$$\dot{E} = \beta SI + qSA - \eta E - \mu E$$

$$\dot{I} = p\eta E - \alpha I - \mu I$$

$$\dot{A} = (1-p)\eta E - \gamma A - \mu A$$

$$\dot{R} = \alpha I + \gamma A - \mu R$$

Long-term behaviour:

In the simulations below, for all values carried over from part A, I have retained the values given in that section (including Λ and μ as given for the shrinkage and growth plots).

For the new constants, q was set at 0.001, or roughly half β . The value of p was set at 0.1 to accentuate the impacts of the asymptomatic group. The value for γ was set at 0.2.

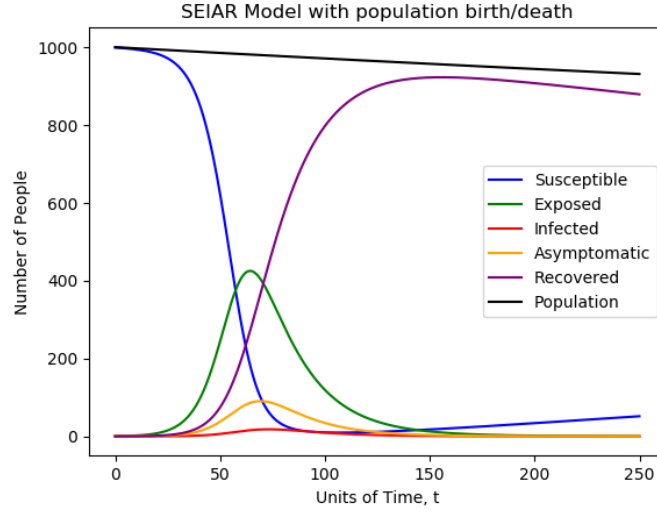


Fig 3.b.2: Short-run simulation with long-run population below the initial value

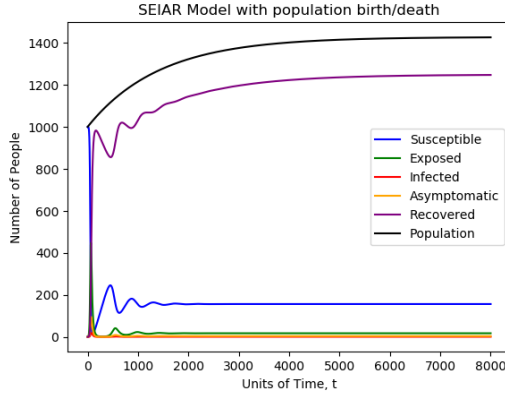


Fig 3.b.3: Long-run plot with pop. expansion

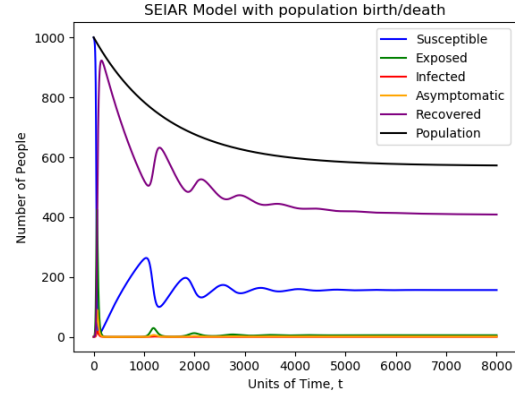


Fig 3.a.4: Long-run plot with pop. shrinkage

From these graphs we can see the overall effects of the asymptomatic group - a lower initial peak, reduced infected values, and wider oscillations as the populations converges to a higher steady-state susceptible population. This is because, *ceteris paribus*, the division of those with the disease into a less infection asymptomatic group as well as the standard infectious group slows the 'speed' of the disease down and reduces its infectiousness due to the reduced infectiousness and quicker recovery

of the asymptomatic group. This results in a higher steady-state susceptible population as the disease is less effective at moving through the population.

To understand this more thoroughly we must examine each new parameter (p , q and γ) independently:

Parameter p :

We can examine the long-term effects as:

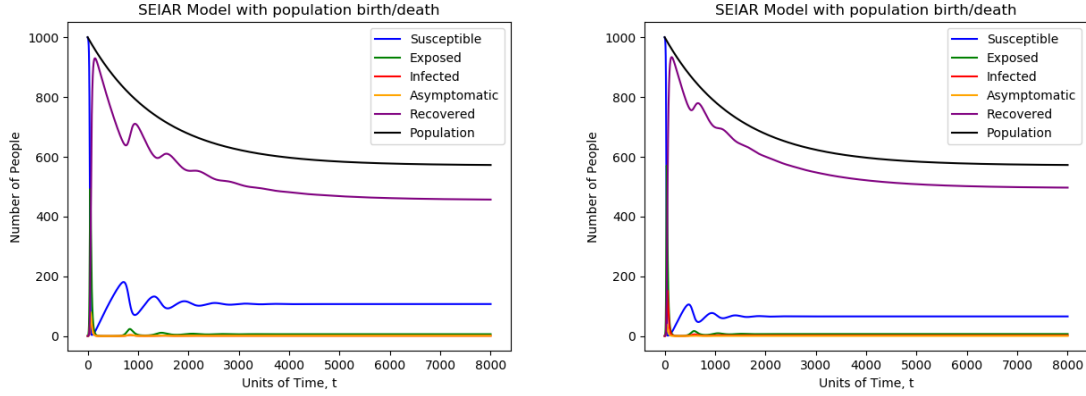


Fig 3.b.4: A low value of $p = 0.3$ (left) and a high value $p = 0.7$ (right) in the long term

The value of p essentially dictates how close the SEIAR model is to a SEIR model - in fact, a very high value for p is near-indistinguishable from a SEIR model. As a result of this, a high p value produces a more 'effective' disease that results in a smaller steady-state susceptible population in the long term.

Parameter q :

We can examine the long-term effects as:

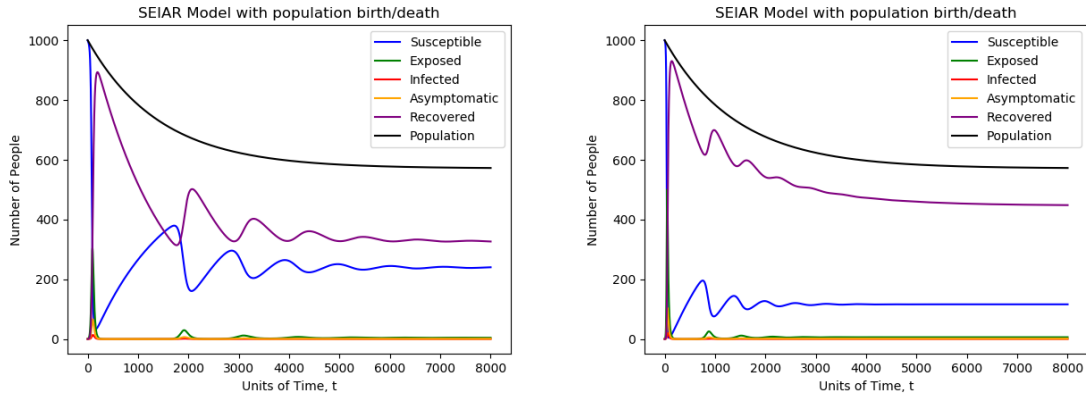


Fig 3.b.4: A low value of $q = 0.0005$ (left) and a high value $q = 0.0015$ (right) in the long term

The value of q dictates how close to the infectious group the asymptomatic group gets from an infectiveness point of view, so from theory a higher q value would result in a smaller proportion of the population remaining susceptible in the long run. This is clearly reflected in the data, along with more sizeable oscillations as the data converges due, again, to the lower infectiveness enabling susceptible cases to build up (and as such create oscillations in the data).

Parameter γ :

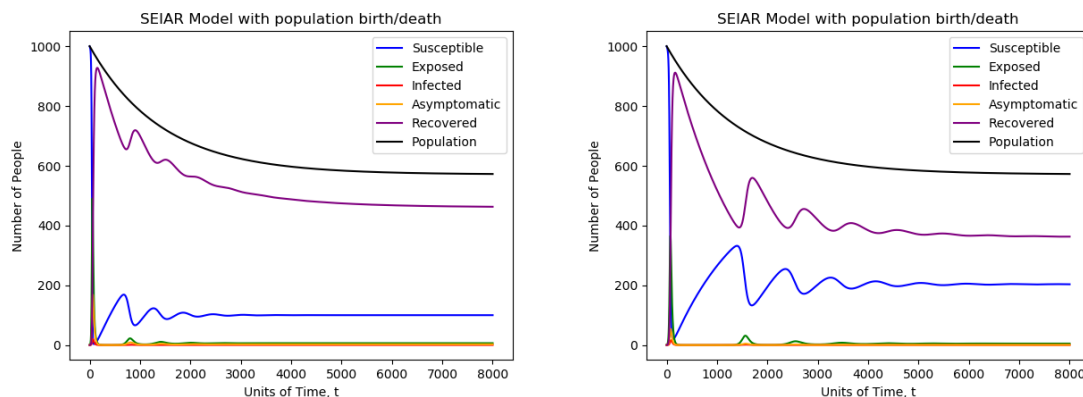


Fig 3.b.4: A low value of $\gamma = 0.11$ (left) and a high value $\gamma = 0.3$ (right) in the long term

From theory, we know that γ dictates how long asymptomatic cases remain infectious before recovery, so lower γ values should result in a higher steady-state susceptible population from the reduced infective time of asymptomatic cases reducing the overall effectiveness of the disease. We may observe this in the data and, like with q above, we also observe more sizeable oscillations as the data converges again due to the lower infectiveness reduced asymptomatic recovery times produce (*ceteris paribus*).

Overall, where p , q and γ mirror their SEIR counterparts, the long term state of the systems are very similar. However, when these values diverge, the disease as a whole becomes significantly weaker, resulting in a long term situation where a larger proportion of the total population as still susceptible.