# Structured compartment models of infection in Python

*Epidemiological models*: We consider a population aggregated by age into $M$ groups labelled by $i = 1, 2, \ldots M$. The population within age group $i$ is partitioned into susceptibles $S_i$, asymptomatic infectives $I_i^a$, symptomatic infectives $I_i^s$ and removed individuals $R_i$. The sum of these is the size of the population in age group $i$, $N_i = S_i + I_i^a + I_i^s + R_i$ [1–4]. We ignore vital dynamics and the change in age structure on the time scale of the epidemic. Therefore each $N_i$ and, consequently, the total population size

$$N = \sum_{i=1}^{M} N_i$$

remain constant in time. We assume that the rate of infection of a susceptible individual in age group $i$ is

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \left( C_{ij}^a \frac{I_j^a}{N_j} + C_{ij}^s \frac{I_j^s}{N_j} \right), \quad i, j = 1, \ldots M \quad (1)$$

where $\beta$ is the probability of infection on contact (assumed intrinsic to the pathogen) and $C_{ij}^a$ and $C_{ij}^s$ are, respectively, the number of contacts between asymptomatic and symptomatic infectives in age-group $j$ with susceptibles in age-group $i$ (reflecting the structure of social contacts). We take the age-independent recovery rate $\gamma$ to be identical for both asymptomatic and symptomatic individuals whose fractions are, respectively, $\alpha$ and $\bar{\alpha} = 1 - \alpha$.

## I. SIR

With these assumptions the progress of the epidemic is governed by the age-structured SIR model

$$\begin{aligned} \dot{S}_i &= -\lambda_i(t) S_i, \\ \dot{I}_i^a &= \alpha \lambda_i(t) S_i - \gamma_{I^a} I_i^a, \\ \dot{I}_i^s &= \bar{\alpha} \lambda_i(t) S_i - \gamma_{I^s} I_i^s, \\ \dot{R}_i &= \gamma_{I^a} I_i^a + \gamma_{I^s} I_i^s. \end{aligned} \quad (2)$$

The age structure of the population is specified the proportions $N_i/N$ and the contact structure by the matrices $C_{ij}^a$ and $C_{ij}^s$. We assume that symptomatic infectives reduce their contacts compared to asymptomatic infectives and set $C_{ij}^s = f C_{ij}^a \equiv f C_{ij}$, where $0 \leq f \leq 1$ is the proportion by which this self-isolation takes place. Here

- $\gamma_{I^a}$ is the recovery rate for asymptomatic infectives
- $\gamma_{I^s}$ is the recovery rate for symptomatic infectives
- $\beta$ is the probability of infection on contact
- $\alpha$ is the fraction of asymptomatic infectives
- $f$ is the fraction for reduction in contacts of the symptomatic infectives

## II. SEIR

We can add an exposed class, that has caught the infection but is not infectious, to the SIR model to obtain an SEIR model. The rate of infection remains unchanged as before, but the equations now change to

$$\begin{aligned} \dot{S}_i &= -\lambda_i(t) S_i, \\ \dot{E}_i &= \lambda_i(t) S_i - \gamma_E E_i \quad (3) \\ \dot{I}_i^a &= \alpha \gamma_E E_i - \gamma_{I^a} I_i^a, \quad (4) \\ \dot{I}_i^s &= \bar{\alpha} \gamma_E E_i - \gamma_{I^a} I_i^s, \\ \dot{R}_i &= \gamma_{I^a} I_i^a + \gamma_{I^s} I_i^s. \end{aligned}$$

Assuming an exponentially distributed incubation time distribution, $1/\gamma_E$ can be interpreted as the average incubation period.

## III. SEAIR

This model is an extension of the SEIR model, introducing the additional class A, which is both asymptomatic and infectious. In other words, this models shows what ensues if *everyone* who gets infected, undergoes a latency period where they are both asymptomatic and infectious. This class is potentially quite important, as there is some evidence that people are infectious before they start showing symptoms [**?** ].

$$\begin{aligned} \dot{S}_i &= -\lambda_i(t) S_i \\ \dot{E}_i &= \lambda_i(t) S_i - \gamma_E E_i \\ \dot{A}_i &= \gamma_E E_i - (\alpha \gamma_{A \to I^s} + \bar{\alpha} \gamma_{A \to I^a}) A_i \\ \dot{I}_i^a &= \alpha \gamma_{A \to I^a} A_i - \gamma_{I^a} I_i^a \\ \dot{I}_i^s &= \bar{\alpha} \gamma_{A \to I^s} A_i - \gamma_{I^s} I_i^s \\ \dot{R}_i &= \gamma_{I^a} I_i^a + \gamma_{I^s} I_i^s \end{aligned}$$

The $A$ and $I^a$ classes should behave virtually the same (so their contact matrices should be equal). The two are kept distinct to keep track of the fact that some people remain asymptomatic even in the $I$ stage.

Since it's difficult to find data on the ratio of $I^s$ to $I^a$, it is possible to disregard the distinction and simply use $I$ instead.

## IV. SEAIRQ

We introduce the $Q$-class, which represents people who have been tested and put into quarantine (and can therefore not infect anyone else).

$$
\begin{aligned}
\dot{S}_i &= -\lambda_i(t)S_i - \tau_S S_i \\
\dot{E}_i &= \lambda_i(t)S_i - (\gamma_E + \tau_E)E_i \\
\dot{A}_i &= \gamma_E E_i - (\alpha\gamma_{A\to I^s} + \bar{\alpha}\gamma_{A\to I^a} + \tau_A)A_i \\
\dot{I}_i^a &= \alpha\gamma_{A\to I^a}A_i - (\gamma_{I^a} + \tau_{I^a})I_i^a \\
\dot{I}_i^s &= \bar{\alpha}\gamma_{A\to I^s}A_i - (\gamma_{I^s} + \tau_{I^s})I_i^s \\
\dot{R}_i &= \gamma_{I^a}I_i^a + \gamma_{I^s}I_i^s \\
\dot{Q}_i &= \tau_S S_i + \tau_E E + \tau_A A + \tau_{I^s}I_i^s + \tau_{I^a}I_i^a
\end{aligned}
$$

Here $\tau_{E,A,I^s,I^a}$ is the testing rate in the population, these are in general different for different classes. I've presumed that people in the incubation stage $E$ can also be tested, which may or may not be the case.

The $\tau_S$ terms model the effects of false-positives, resulting in susceptibles being put into quarantine.

Note that this model does not keep track of what happens to people once they're put into $Q$ (which is especially important to do if $\tau_S > 0$). Since $Q$ is a closed system, this can all be done after the initial SEAIR simulation has been completed.

## V. SIKR

We now use method of stages to write an age-structured $k-$staged SIkR model

$$
\begin{aligned}
\dot{S}_i &= -\lambda_i(t)S_i, \\
\dot{I}_i^1 &= \lambda_i(t)S_i - k\gamma_I I_i^1, && (5)\\
\dot{I}_i^2 &= k\gamma_I I_i^1 - k\gamma_I I_i^2, && (6)\\
&\vdots && (7)\\
\dot{I}_i^k &= k\gamma_I I_i^{n-1} - k\gamma_I I_i^k, && (8)\\
\dot{R}_i &= k\gamma_I I_i^k.
\end{aligned}
$$


[1] R. M. Anderson, B. Anderson, and R. M. May, *Infectious diseases of humans: dynamics and control* (Oxford university press, 1992).

[2] M. J. Keeling and P. Rohani, *Modeling infectious diseases in humans and animals* (Princeton University Press, 2011).

[3] S. Towers and Z. Feng, "Social contact patterns and control strategies for influenza in the elderly," Math. Biosci. **240**, 241–249 (2012).

[4] N. M. Ferguson *et al.*, "Strategies for mitigating an influenza pandemic," Nature **442**, 448–452 (2006).