Structured compartment models of infection in Python

I. EPIDEMIOLOGICAL MODELS

We consider a population aggregated by age into M groups labelled by $i = 1, 2, \dots M$. In what follows, we provide several mathematical models of infection which have been implemented in PyRoss.

A. SIR

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 in this model. Therefore each N_i and, consequently, the total population size

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

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

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \left(C_{ij}^a(t) \frac{I_j^a}{N_j} + C_{ij}^s(t) \frac{I_j^s}{N_j} \right), \quad i, j = 1, \dots M \tag{2}$$

where β 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 γ to be identical for both asymptomatic and symptomatic individuals whose fractions are, respectively, α_i and $\bar{\alpha}_i = 1 - \alpha_i$. The social contact matrix C_{ij} denotes the average number of contacts made per day by an individual in class i with an individual in class j. Clearly, the total number of contacts between group i to group j must equal the total number of contacts from group j to group i, and thus, $N_i C_{ij} = N_j C_{ji}$. In what follows, the contact matrix is both dependent on the time and state.

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

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

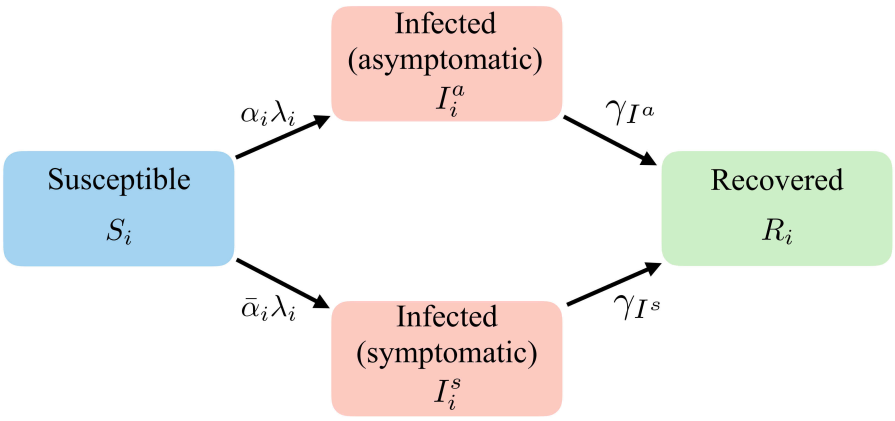

Figure 1: **Schematics of the SIR model.**

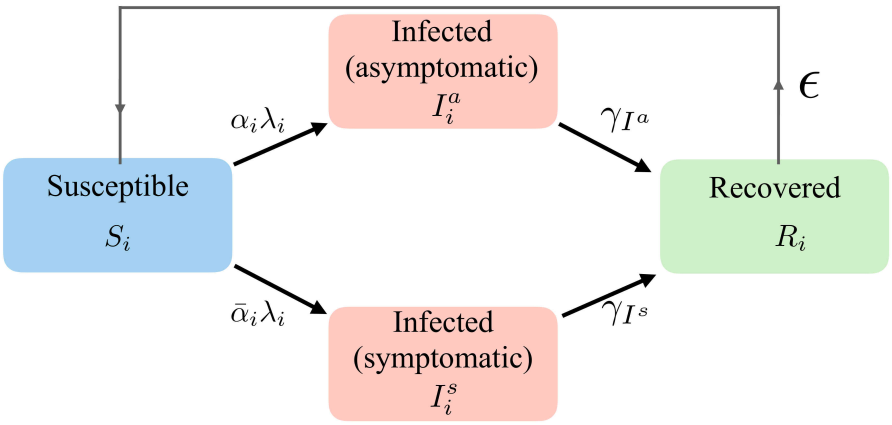

Figure 2: **Schematics of the SIRS model.**

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 $C_{ij}^s = f^s C_{ij}^a \equiv f^s C_{ij}$, where $0 \leq f^s \leq 1$ is the proportion by which this self-isolation takes place. Here

- γ_{I^a} is the recovery rate for asymptomatic infectives
- γ_{I^s} is the recovery rate for symptomatic infectives
- β is the probability of infection on contact
- α_i is the fraction of asymptomatic infectives
- f^s is the fraction for reduction in contacts of the symptomatic infectives

The SIR model allows for lowest bounds on number of infectives on fitting to a given data. The SIR model does not account for incubations. In what follows, we add more features to SIR model and derive more detailed models.

B. SIRS

We now extend the age-structured SIR model to allow for recovered persons to be susceptible and change in population of each age group. The dynamics of the resulting SIRS model is:

$$\begin{aligned}
\dot{S}_i &= -\lambda_i(t)S_i + \sigma_i + \epsilon(\gamma_{I^a} I_i^a + \gamma_{I^s} I_i^s) \\
\dot{I}_i^a &= \alpha_i \lambda_i(t) S_i - \gamma_{I^a} I_i^a + l_i \\
\dot{I}_i^s &= \bar{\alpha}_i \lambda_i(t) S_i - \gamma_{I^a} I_i^s \\
\dot{R}_i &= \gamma_{I^a} I_i^a + \gamma_{I^s} I_i^s. \\
\dot{N}_i &= \sigma_i + l_i
\end{aligned} \tag{4}$$

Here ϵ is fraction of recovered who is susceptible. σ_i denotes of the arrival of new susceptibles, while l_i are new asymptomatic infectives. This means that N_i is now dynamical.

The rate of infection of a susceptible individual in age group i is

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \left(C_{ij}^a \frac{I_j^a}{N_j} + C_{ij}^s \frac{I_j^s}{N_j} \right), \tag{5}$$

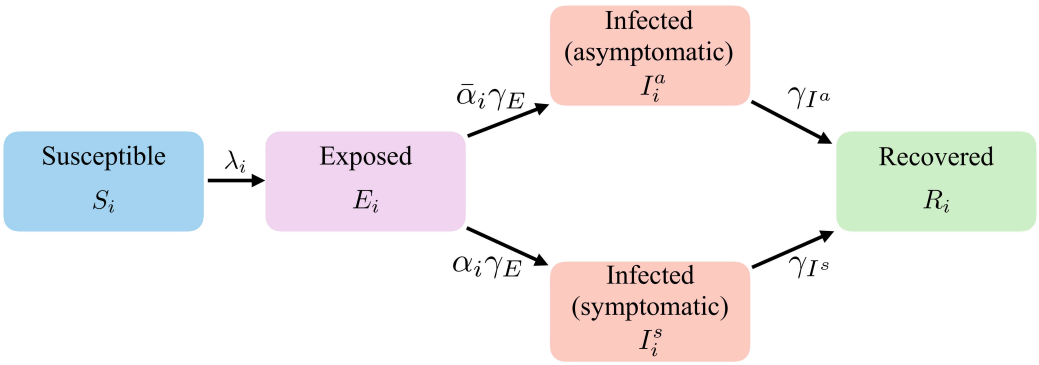

Figure 3: **Schematics of the SEIR model.**

C. 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

$$\begin{aligned}
\dot{S}_i &= -\lambda_i(t)S_i, \\
\dot{E}_i &= \lambda_i(t)S_i - \gamma_E E_i \\
\dot{I}_i^a &= \alpha_i\gamma_E E_i - \gamma_{I^a} I_i^a, \\
\dot{I}_i^s &= \bar{\alpha}_i\gamma_E E_i - \gamma_{I^a} I_i^s, \\
\dot{R}_i &= \gamma_{I^a} I_i^a + \gamma_{I^s} I_i^s.
\end{aligned} \tag{6}$$

The rate of infection of a susceptible individual in age group i is

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \left(C_{ij}^a \frac{I_j^a}{N_j} + C_{ij}^s \frac{I_j^s}{N_j} \right), \tag{7}$$

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

D. SEI5R

We now extend SEIR model to have fives types of infectives (I_i^h: infectives who are hospitalized, I_i^c: infectives who are in ICU, and I_i^m: mortality) to obtain:

$$\begin{aligned}
\dot{S}_i &= -\lambda_i(t)S_i + \sigma_i, \\
\dot{E}_i &= \lambda_i(t)S_i - \gamma_E E_i \\
\dot{I}_i^a &= \alpha_i\gamma_E E_i - \gamma_{I^a} I_i^a, \\
\dot{I}_i^s &= \bar{\alpha}_i\gamma_E E_i - \gamma_{I^s} I_i^s, \\
\dot{I}_i^h &= h_i\gamma_{I^s} I_i^s - \gamma_{I^h} I_i^h, \\
\dot{I}_i^c &= c_i\gamma_{I^h} I_i^h - \gamma_{I^c} I_i^c, \\
\dot{I}_i^m &= m_i\gamma_{I^c} I_i^c, \\
\dot{R}_i &= \gamma_{I^a} I_i^a + \bar{h}_i\gamma_{I^s} I_i^s + \bar{c}_i\gamma_{I^h} I_i^h + \bar{m}_i\gamma_{I^c} I_i^c. \\
\dot{N}_i &= \sigma_i - m_i\gamma_{I^c} I_i^c
\end{aligned} \tag{8}$$

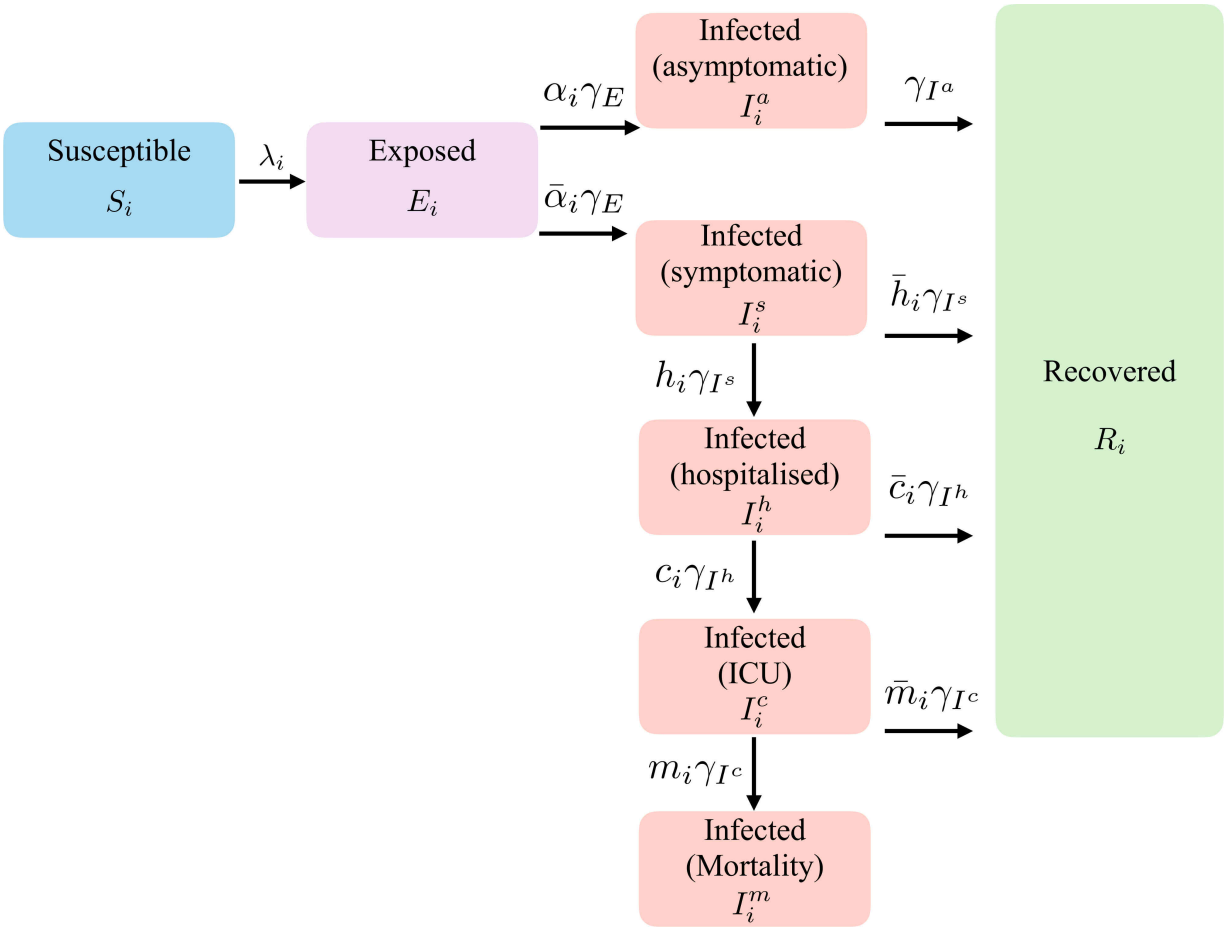

Figure 4: **Schematics of the SEI5R model.**

The rate of infection of a susceptible individual in age group i is

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \left(C_{ij}^a \frac{I_j^a}{N_j} + C_{ij}^s \frac{I_j^s}{N_j} + C_{ij}^h \frac{I_j^h}{N_j} \right), \tag{9}$$

Here $\bar{h}_i = 1 - h_i$, $\bar{m}_i = 1 - m_i$, $C_{ij}^s = f^s C_{ij}^a \equiv f^s C_{ij}$ and $C_{ij}^s = f^h C_{ij}^a \equiv f^h C_{ij}$. I^c is ICU cases and I^m is the mortality due to the infection.

E. SIkR

We now use method of stages to write an age-structured $k-$staged SIkR \newcommand{\bx}{\boldsymbol{x}}model $\dot{I}_k(t) = \gamma_I N_I (I_{k-1} - I_k)$

$$\begin{aligned}
\dot{S}_i &= -\lambda_i(t) S_i, \\
\dot{I}_i^1 &= \lambda_i(t) S_i - k\gamma_I I_i^1, \\
\dot{I}_i^2 &= k\gamma_I I_i^1 - k\gamma_I I_i^2, \\
&\vdots \\
\dot{I}_i^k &= k\gamma_I I_i^{n-1} - k\gamma_I I_i^k, \\
\dot{R}_i &= k\gamma_I I_i^k.
\end{aligned} \tag{10}$$

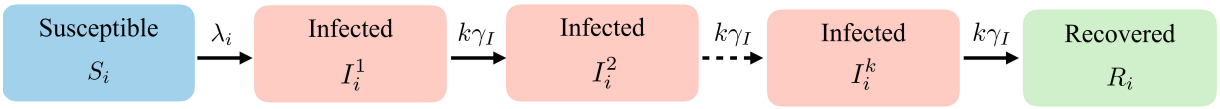

Figure 5: **Schematics of the SIkR model.**

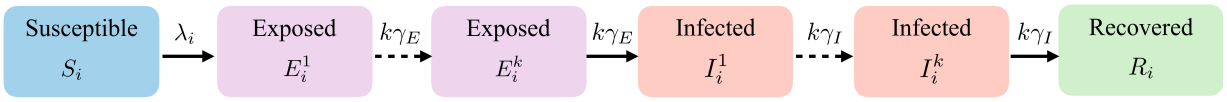

Figure 6: **Schematics of the SEkIkR model.**

The rate of infection of a susceptible individual in age group i is

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \sum_{n=1}^{k} C_{ij} \frac{I_j^n}{N_j}, \tag{11}$$

F. SEkIkR

The above method of stages for SIR can be extended to SEIR model to obtain an age-structured $k-$staged SEkIkR model to obtain

$$\begin{aligned}
\dot{S}_i &= -\lambda_i(t) S_i, \\
\dot{E}_i^1 &= \lambda_i(t) S_i - k\gamma_E E_i^1 \\
\dot{E}_i^2 &= k\gamma_E E_i^1 - k\gamma_E E_i^2 \\
&\vdots \\
\dot{E}_i^k &= k\gamma_E E_i^{k-1} - k\gamma_E E_i^k \\
\dot{I}_i^1 &= k\gamma_E E_i^k - k\gamma_I I_i^1, \\
\dot{I}_i^2 &= k\gamma_I I_i^1 - k\gamma_I I_i^2, \\
&\vdots \\
\dot{I}_i^k &= k\gamma_I I_i^{n-1} - k\gamma_I I_i^k, \\
\dot{R}_i &= k\gamma_I I_i^k.
\end{aligned} \tag{12}$$

The rate of infection of a susceptible individual in age group i is

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \sum_{n=1}^{k} C_{ij} \frac{I_j^n}{N_j}, \tag{13}$$

G. 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

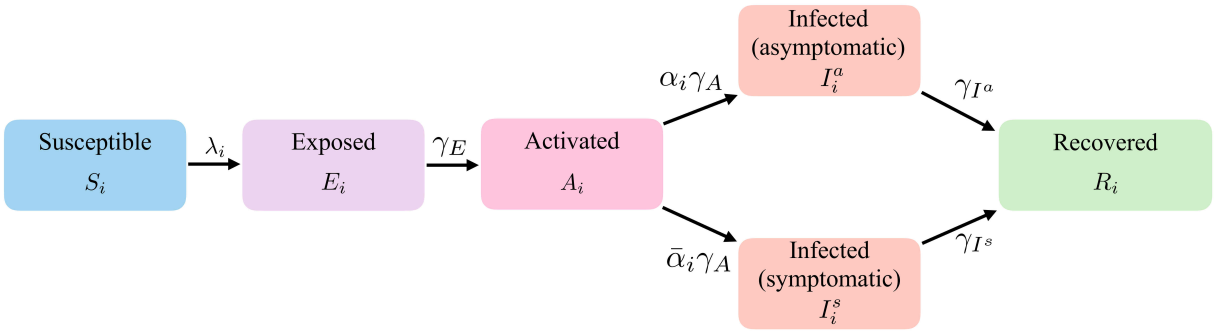

Figure 7: **Schematics of the SEAIR model.**

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

$$\begin{aligned}
\dot{S}_i &= -\lambda_i(t) S_i \\
\dot{E}_i &= \lambda_i(t) S_i - \gamma_E E_i \\
\dot{A}_i &= \gamma_E E_i - \gamma_A A_i \\
\dot{I}_i^a &= \alpha_i \gamma_A A_i - \gamma_{I^a} I_i^a \\
\dot{I}_i^s &= \bar{\alpha}_i \gamma_A A_i - \gamma_{I^s} I_i^s \\
\dot{R}_i &= \gamma_{I^a} I_i^a + \gamma_{I^s} I_i^s
\end{aligned} \tag{14}$$

The rate of infection of a susceptible individual in age group i is

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \left(C_{ij}^a \frac{I_j^a}{N_j} + C_{ij}^a \frac{A_j}{N_j} + C_{ij}^s \frac{I_j^s}{N_j} \right), \tag{15}$$

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.

H. SEAIRQ

We now introduce the Q-class, which represents people who have been tested and put into quarantine (and can therefore not infect anyone else). This point of Q-class is to model proper contact tracing. The dynamics of the SEAIRQ model is given as:

$$\begin{aligned}
\dot{S}_i &= -\lambda_i(t) S_i \\
\dot{E}_i &= \lambda_i(t) S_i - (\gamma_E + \tau_E) E_i \\
\dot{A}_i &= \gamma_E E_i - (\gamma_A + \tau_A) A_i \\
\dot{I}_i^a &= \alpha_i \gamma_A A_i - (\gamma_{I^a} + \tau_{I^a}) I_i^a \\
\dot{I}_i^s &= \bar{\alpha}_i \gamma_A A_i - (\gamma_{I^s} + \tau_{I^s}) I_i^s \\
\dot{R}_i &= \gamma_{I^a} I_i^a + \gamma_{I^s} I_i^s \\
\dot{Q}_i &= \tau_S S_i + \tau_E E + \tau_A A + \tau_{I^s} I_i^s + \tau_{I^a} I_i^a
\end{aligned} \tag{16}$$

The rate of infection of a susceptible individual in age group i is

$$\lambda_i(t) = \beta \sum_{j=1}^{M} \left(C_{ij}^a \frac{I_j^a}{N_j} + C_{ij}^a \frac{A_j}{N_j} + C_{ij}^s \frac{I_j^s}{N_j} \right), \tag{17}$$

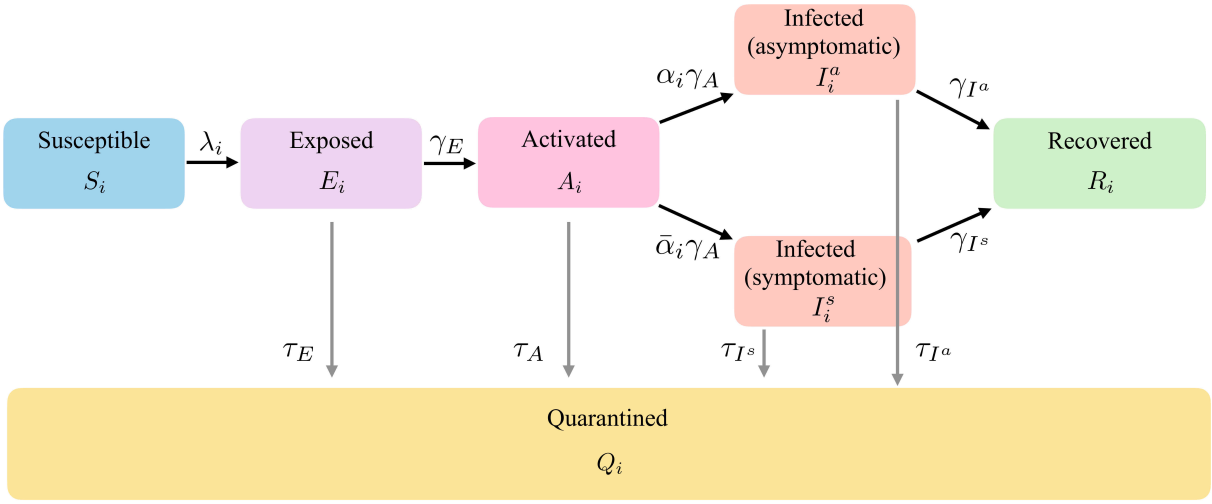

Figure 8: **Schematics of the SEAIRQ model.**

Here τ_{E,A,I^s,I^a} is the testing rate in the population, these are in general different for different classes. We have presumed that people in the incubation stage E can also be tested. The τ_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.

I. SEAI5R

We now extend SEIR model to have fives types of infectives (I_i^h: infectives who are hospitalized, I_i^c: infectives who are in ICU, and I_i^m: mortality) to obtain:

$$
\begin{aligned}
\dot{S}_i &= -\lambda_i(t)S_i + \sigma_i, \\
\dot{E}_i &= \lambda_i(t)S_i - \gamma_E E_i \\
\dot{A}_i &= \gamma_E E_i - \gamma_A A_i \qquad (18)\\
\dot{I}_i^a &= \alpha_i\gamma_A A_i - \gamma_{I^a} I_i^a, \qquad (19)\\
\dot{I}_i^s &= \bar{\alpha}_i\gamma_A A_i - \gamma_{I^s} I_i^s, \\
\dot{I}_i^h &= h_i\gamma_{I^s} I_i^s - \gamma_{I^h} I_i^h, \\
\dot{I}_i^c &= c_i\gamma_{I^h} I_i^h - \gamma_{I^c} I_i^c, \\
\dot{I}_i^m &= m_i\gamma_{I^c} I_i^c, \\
\dot{R}_i &= \gamma_{I^a} I_i^a + \bar{h}_i\gamma_{I^s} I_i^s + \bar{c}_i\gamma_{I^h} I_i^h + \bar{m}_i\gamma_{I^c} I_i^c. \\
\dot{N}_i &= \sigma_i - m_i\gamma_{I^c} I_i^m
\end{aligned}
$$

The rate of infection of a susceptible individual in age group i is

$$
\lambda_i(t) = \beta \sum_{j=1}^{M} \left(C_{ij}^a \frac{I_j^a}{N_j} + C_{ij}^a \frac{A_j}{N_j} + C_{ij}^s \frac{I_j^s}{N_j} + C_{ij}^h \frac{I_j^h}{N_j} \right), \qquad (20)
$$

Here $\bar{h}_i = 1 - h_i$, $\bar{m}_i = 1 - m_i$, $C_{ij}^s = f^s C_{ij}^a \equiv f^s C_{ij}$ and $C_{ij}^s = f^h C_{ij}^a \equiv f^h C_{ij}$. I^c is ICU cases and I^m is the mortality.

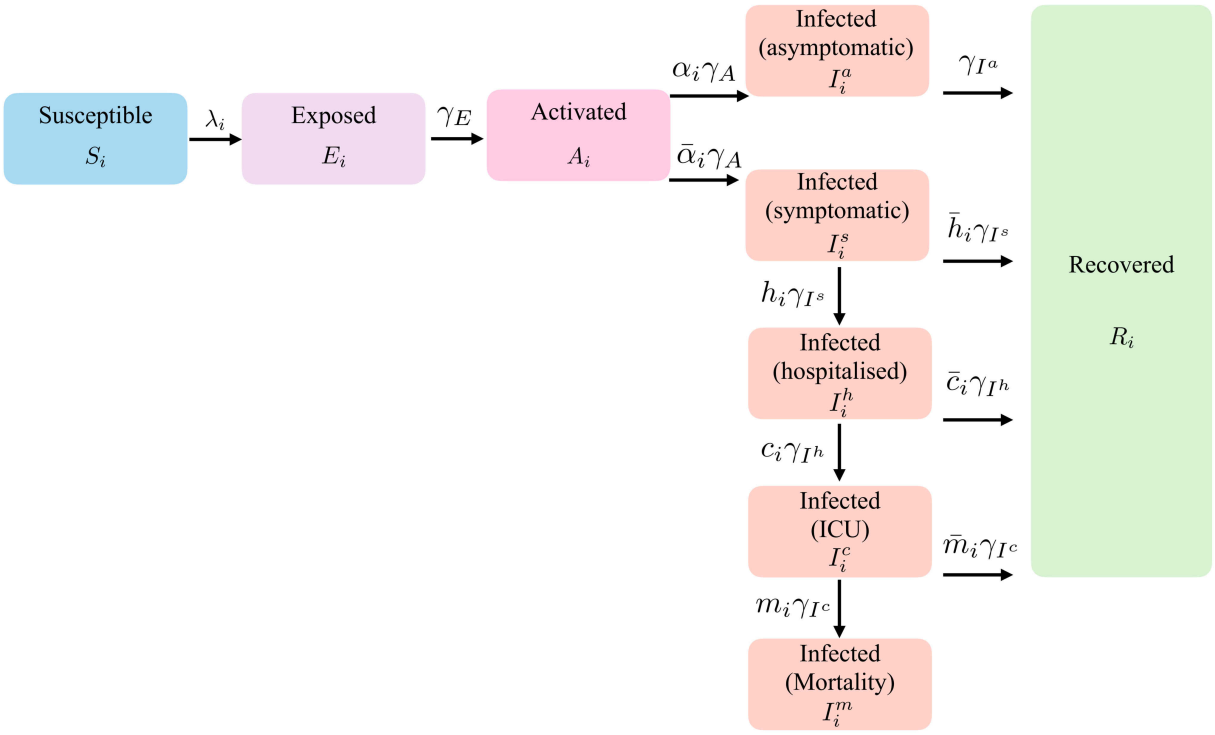

Figure 9: **Schematics of the SEAI5R model.**

[1] R. M. Anderson, B. Anderson, and R. M. May, *Infectious diseases of humans: dynamics and control* (Oxford university press, 1992).
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[3] S. Towers and Z. Feng, Math. Biosci. **240**, 241 (2012).
[4] N. M. Ferguson et al., Nature **442**, 448 (2006).