

REEM - Model Description

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This is the description of the REEM epidemic model. REEM stands for Renewal Equation based Epidemic Model. Unlike most implementations of epidemic models that are either based on differential equations or agent-based models, here the model is implemented with a renewal equation, with a discrete daily time step and is stochastic.

1 Pathogen transmission

We consider an infectious pathogen spreading in a closed population of size N . The number of individuals susceptible to infection on day t is noted $S(t)$. Let $I(t)$ be the incidence on day t , \mathcal{R}_0 the basic reproduction number. The intrinsic generation interval distribution of the pathogen is noted g .

The infection process is described by the following discrete-time equations:

$$j(t) = \mathcal{R}_0 \left(\frac{S(t)}{N} \right)^{\exp(\alpha)} B(t) \sum_{k=1}^{t-1} g(k) j(t-k) \quad (1)$$

$$I(t) \sim \text{Pois}(j(t)) \quad (2)$$

$$S(t) = \max(0, S(t-1) - I(t)) \quad (3)$$

The time-dependent variable $j(t)$ represents the *mean* incidence driven by a *deterministic* pathogen transmission process. The actual incidence at time t , $I(t)$, is simply drawn from a Poisson distribution with mean $j(t)$. The parameter α is a real number affecting the tail-end of the epidemic curve and has been associated with a simple way to model transmission heterogeneity. When $\alpha = 0$, contacts are modelled as homogeneous (like in the canonical ODE-based SIR model). The deterministic function B is introduced to model exogenous changes in the transmission rate (for example, to represent public health interventions). When $B(t) = 1$ for all times t , there is no change in the intrinsic transmission rate and the epidemic follows its “natural” course.

1.1 Interpretation of the parameter α

The fraction $S(t)/N$ in [Equation 1](#) represents the proportion of the population that is *available* to be infected at time t . If contacts among individuals of the population are completely random and homogeneous, then the fraction of the population that is potentially *exposed* to infection is indeed $S(t)/N$. This is because any infectious individual has a probability $S(t)/N$ to contact a susceptible individual.

However, in reality, contacts are not truly homogeneous because of the intrinsic structures of human populations (eg, households, workplaces, social events, etc.). Hence, the actual average proportion of susceptible individuals exposed to an infectious person may either be larger (if infectious individuals “seek” susceptibles) or lower (if infectious individuals tend to cluster together more than with susceptibles).

30 The relationship between the susceptible fraction that is exposed and the one effectively exposed can
 31 be modelled with a power law. A negative power does not really make sense in this context, so it is
 32 parameterised with an exponential, $\exp(\alpha)$, to ensure its positivity. Examples of a power-law relationship
 33 is illustrated in Figure 1.

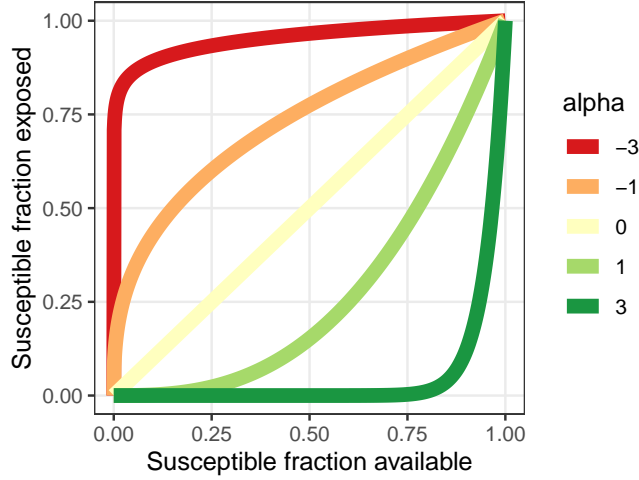


Figure 1: Relationship between the fraction of susceptible individuals that are effectively exposed to infection, as a function of the fraction of susceptible individuals that are available in the population.

34 2 Fecal/urinary shedding in wastewater

35 We assume that fecal and/or urinary shedding of pathogen RNA/DNA occurs during the course of infec-
 36 tion of an individual. In communities where a sewer system exists, a large amount of pathogen RNA/DNA
 37 is deposited in wastewater. Sampling wastewater at strategic locations (e.g., a wastewater treatment plant)
 38 and measuring the pathogen RNA/DNA concentration (using molecular techniques like qPCR or ddPCR)
 39 can indicate the prevalence of infections in the community. Although shedding can occur through both
 40 fecal and urinary routes, here we simply focus on the fecal route for simplicity.

41 Fecal shedding depends on pathogen. Here, we focus on a description inspired by SARS-CoV-2, as this
 42 is the most studied fecal shedding to date. Fecal shedding occurs shortly after infection (although we
 43 do not know exactly how long after), probably peaks around the time of symptoms onset and carries on
 44 several days after the infection has been “cleared” (i.e., the individual has effectively recovered, and is
 45 not diseased any more).

46 The fecal shedding distribution, noted f , gives the *average* amount of pathogen RNA/DNA shed with
 47 time by one infected individual. More precisely, $f(\tau)$ is the average amount of RNA/DNA shed τ
 48 days after infection.

49 There were $I(t - k)$ individuals infected at time $t - k$ and all have been infected for k days at time t ,
 50 hence they contribute $f(k) \times I(t - k)$ RNA/DNA copies in the wastewater on day t . Then, we sum over
 51 all past cohorts infected k days ago. Hence, the amount of RNA/DNA deposited on day t in wastewater
 52 by the population is:

$$W_d(t) = \sum_{k=1}^{t-1} f(k) I(t - k) \quad (4)$$

53 Wastewater can travel long distances (several kilometers) before reaching the sampling location determined
 54 for epidemiological surveillance. The sewer system can generate complex fluid dynamics where RNA/DNA

55 can settle and resuspend. Hence, there can be a significant delay between the time RNA/DNA is deposited
 56 in the wastewater and the time it reaches the sampling location. Moreover, the wastewater is a harsh
 57 environment which can degrade RNA/DNA during its journey between the shedding and sampling points.
 58 We model the concentration present at the sampling site on day t as:

$$W_p(t) = \sum_{k=1}^{t-1} \psi(k) W_d(t-k) e^{-\kappa k} \quad (5)$$

59 The parameter κ represents the average RNA/DNA decay in wastewater and the function ψ the average
 60 time lag for the RNA/DNA copies to reach the sampling point from the shedding point.

61 Finally, the RNA/DNA concentration in wastewater reported depends on multiple steps performed in a
 62 laboratory. Each step generates measurement error that is simply modelled as a Gaussian process. The
 63 reported concentration for a sample collected at time t is defined as

$$W_r(t) = \mathcal{N}(W_p(t), \sigma_w) \quad (6)$$

64 3 Hospital admissions

65 Like fecal shedding in wastewater, the number of hospital admissions is modelled as simply resulting from
 66 incidence, with a time lag. More precisely, we define h as the average fraction of infected individuals that
 67 will be hospitalized because of the severity of their infection. The function h is the distribution of the
 68 time lags between infection and hospital admission. In other words, $h(\tau)$ is the proportion of individuals
 69 infected τ days ago that will be hospitalized today. Hence, the number of hospital admissions on day t is
 70 given by:

$$H(t) = \sum_{k=1}^{t-1} h(k) I(t-k) \quad (7)$$

71 For implementation convenience, the function h is reparameterized such that the time repartition and the
 72 total quantity of hospital admissions are separated:

$$h(k) = \frac{\eta(k)}{\sum_{i>0} \eta(i)} h^* \quad (8)$$

73 Hence, a cohort of infected individuals infected at the same time in the past will contribute the fraction
 74 $\sum_{k>0} h(k) = h^*$ of its size to hospital admissions. The repartition of the hospital admission lags is defined
 75 with the unitless function η .