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Research article

An SIS epidemic model with individual variation

Philip K. Pollett*

School of Mathematics and Physics, The University of Queensland, Qld 4072, Australia

* **Correspondence:** Email: pkp@maths.uq.edu.au.

Abstract: We study an extension of the stochastic SIS (Susceptible-Infectious-Susceptible) model in continuous time that accounts for variation amongst individuals. By examining its limiting behaviour as the population size grows we are able to exhibit conditions for the infection to become endemic.

Keywords: Epidemics; Markov processes; SIS Model; limit theorems; quasi stationarity

1. Introduction

The *SIS (Susceptible-Infectious-Susceptible) Model* was introduced by Weiss and Dishon [1] to study infections in a closed population of n individuals, infections that do not confer any long lasting immunity (gonorrhea, or the common cold, for example). If $Y(t)$ is the number of infectives at time t, then $(Y(t),\, t \geq 0)$ is a continuous-time Markov chain taking values in $\{0, 1, \ldots, n\}$ with transitions

$$\begin{aligned} Y \to Y+1 \quad &\text{at rate} \quad \frac{\lambda}{n} Y(n-Y) \qquad &&\text{(infection)} \\ Y \to Y-1 \quad &\text{at rate} \quad \mu Y \qquad &&\text{(recovery).} \end{aligned}$$

The large-n behaviour of this model is well understood, because the proportion of infectives $Y(t)/n$ obeys the following *law of large numbers*, which is an application of Theorem 3.1 of [2]; to the best of my knowledge, this result has not been stated explicitly elsewhere.

Theorem 1. *If $Y(0)/n \to y_0$ as $n \to \infty$, then $(Y(t)/n)$ converges in probability, uniformly over finite time intervals, to the solution of the ODE*

$$\frac{\mathrm{d}y}{\mathrm{d}t} = \lambda y(1-\rho-y), \tag{1.1}$$

where $\rho = \mu/\lambda$, namely

$$y(t) = \frac{(1-\rho)y_0}{y_0 + (1-\rho-y_0)\mathrm{e}^{-\lambda(1-\rho)t}}, \qquad y(0) = y_0.$$

We see immediately that if $\lambda > \mu$ then (1.1) has a globally stable equilibrium at $y^* = 1 - \rho$, while if $\lambda < \mu$ the disease-free state 0 is globally stable. The two behaviours are illustrated in Figure 1: quasi stationarity (the infection becomes endemic) versus evanescence (the infection dies out). Notice how the sample paths of the process $(Y(t)/n)$ (blue) "track" the limiting deterministic path $y(t)$ (green).

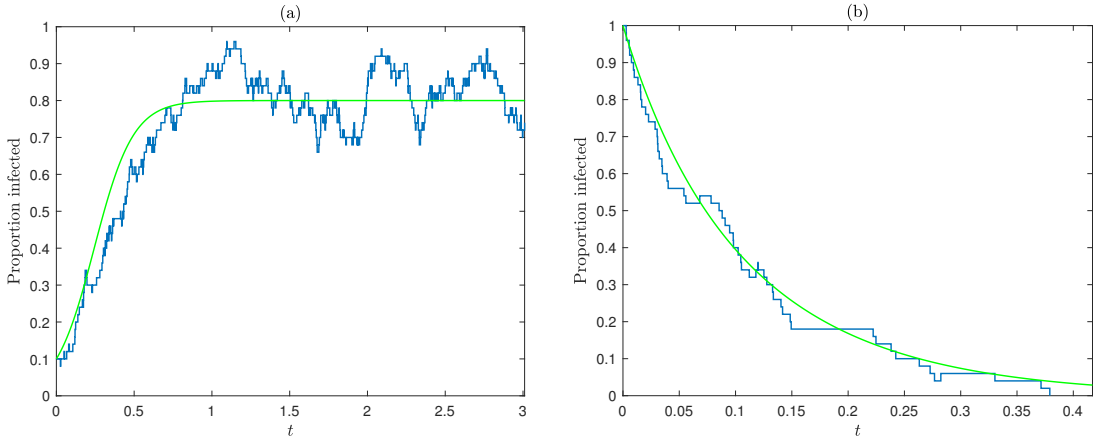

Figure 1. Simulation of the SIS model with $n = 50$ individuals, and with infection and recovery rates (a) $\lambda = 10$, $\mu = 2$ ($\rho < 1$, $y^* = 0.8$), and (b) $\lambda = 2$, $\mu = 10$ ($\rho > 1$).

Several elaborations of the basic SIS model have been proposed where the assumption of homogeneity among individuals is relaxed. Perhaps the most is extensive is the study of epidemics where transmission occurs within households [3–8]. Another [9] assumes that individuals encounter one another in a communal meeting place. We incorporate heterogeneity by assuming that individuals have their own, possibly different, infection and recovery rates. In particular, we assume that individual i ($i = 1, \dots, n$) has an exponentially distributed recovery period with mean μ_i^{-1} and a resistance level λ_i^{-1} (both finite). Let $X_i^{(n)}$ be 1 or 0 according to whether individual i is infected or not, and let $X^{(n)} = (X_1^{(n)}, \dots, X_n^{(n)})$ be the state of the population. Suppose that $(X^{(n)}(t),\ t \geq 0)$ is a continuous-time Markov chain taking values in $\{0, 1\}^n$ with transitions

$$\begin{array}{lll} (\dots, 0, \dots) \to (\dots, 1, \dots) & \text{at rate} & \lambda_i \bar{X}^{(n)} \\ (\dots, 1, \dots) \to (\dots, 0, \dots) & \text{at rate} & \mu_i, \\ \quad\uparrow \qquad\qquad\uparrow & & \\ \text{Position } i\ (i = 1, \dots, n) & & \end{array} \tag{1.2}$$

where $\bar{X}^{(n)} = \frac{1}{n}\sum_{j=1}^{n} X_j^{(n)}$ is the *proportion* of the population that is infected. So we assume here that each infected individual makes the same contribution to the infection potential of the group of infectives. The model in described in [9] has $\mu_i = 0$ and λ_i to be interpreted as the proportion of time individual i spends in the communal meeting place. Daley et al. [13] also consider the case where $\mu_i = 0$ for all i and study the mean duration of the epidemic.

Notice that the disease free state $\mathbf{0} = (0, 0, \dots, 0)$ is the sole absorbing state and, since (λ_i) and (μ_i) are strictly positive, the remaining states form a communicating class from which $\mathbf{0}$ is accessible, and indeed reached with probability 1. None-the-less, the process may exhibit quasi stationarity. By investigating the large-n behaviour of the model, we can determine conditions for such a quasi equilibrium

to be achieved. We will see that accounting for variation amongst individuals can lead to different conclusions about equilibrium behaviour to those reached for the standard SIS model. Several examples are given to show a range of behaviour. For example, the model presented here may predict quasi stationarity in cases where the standard model predicts evanescence.

2. Large-n behaviour

Because the population is now heterogeneous, a quite different approach is needed. We will follow McVinish and Pollett [10], who considered a setup that also encapsulates metapopulation models, and other population models. Think of the individual characteristics $\theta_i := (\lambda_i, \mu_i)$ as (random) points in a subset S of $\mathbb{R}^2_+$, and define sequences of random measures $(\sigma^{(n)})$ and random-measure-valued processes $(m_t^{(n)},\ t \geq 0)$ by

$$\sigma^{(n)}(B) = \#\{\theta_i \in B\}/n \qquad \text{and} \qquad m_t^{(n)}(B) = \#\{\theta_i \in B : X_{i,t}^{(n)} = 1\}/n,$$

where B is any Borel subset of S. We will suppose that $\sigma^{(n)} \xrightarrow{d} \sigma$ (weak convergence) for some non-random (probability) measure σ, and then hope to establish that $m_t^{(n)} \xrightarrow{d} m^{(n)}$, so that properties of $X_t^{(n)}$ can be approximated for large n. In particular, if the individual characteristics were chosen from some non-negative bivariate distribution, then $\sigma^{(n)} \xrightarrow{d} \sigma$ would follow as a *consequence* of the law of large numbers.

The sequences $(\sigma^{(n)})$ and $(m_t^{(n)})$ may be defined, equivalently, by

$$\int h(\theta)\sigma^{(n)}(\mathrm{d}\theta) = \frac{1}{n}\sum_{i=1}^{n} h(\theta_i) \qquad \text{and} \qquad \int h(\theta)m_t^{(n)}(\mathrm{d}\theta) = \frac{1}{n}\sum_{i=1}^{n} X_{i,t}^{(n)}\, h(\theta_i), \tag{2.1}$$

for h in $C_b(S)$, the class of bounded continuous functions that map S to $\mathbb{R}$. For example if $(h \equiv 1)$ then $m_t^{(n)}(S) = \int m_t^{(n)}(\mathrm{d}\theta) = \frac{1}{n}\sum_{i=1}^{n} X_{i,t}^{(n)}$, the proportion of population that is infected at time t.

Our main result is a consequence of Theorem 1 of [10].

Theorem 2. *Suppose that $\sigma^{(n)} \xrightarrow{d} \sigma$ and $m_0^{(n)} \xrightarrow{d} m_0$ for some non-random measures σ and m_0. Then, the sequence of measure-valued processes $(m_t^{(n)},\ t \geq 0)$ converges weakly to the unique solution $(m_t,\ t \geq 0)$ of*

$$(h, m_t) = (h, m_0) + \int_0^t L(h, m_s)\,\mathrm{d}s, \qquad h \in C_b(S), \tag{2.2}$$

where (notation) $(h, m) = \int h(\theta)m(\mathrm{d}\theta)$, and

$$L(h, m_t) := m_t(S)\left(\int \lambda h(\theta)\sigma(\mathrm{d}\theta) - \int \lambda h(\theta)m_t(\mathrm{d}\theta)\right) - \int \mu h(\theta)m_t(\mathrm{d}\theta).$$

Lemma 5 of [10] implies that $m_t(B) \leq \sigma(B)$, for all Borel sets B, and in particular that m_t is absolutely continuous with respect to σ. It follows that m_t has a (uniquely determined σ-a.e.) Radon-Nikodym derivative ϕ_t (≥ 0) with respect to σ: $m_t(B) = \int_B \phi_t(\theta)\sigma(\mathrm{d}\theta)$. And further it implies that $\phi_t \leq 1$.

Now we may differentiate both sides of (2.2) with respect to σ to show that $\phi_t(\lambda, \mu)$ must satisfy

$$\phi_t = \phi_0 + \int_0^t \left(\lambda(1 - \phi_s)\int \phi_s(\theta)\sigma(\mathrm{d}\theta) - \mu\phi_s\right)\mathrm{d}s,$$

or equivalently,

$$\frac{\mathrm{d}\phi}{\mathrm{d}t} = \lambda(1-\phi_t)\int \phi_t(\theta)\sigma(\mathrm{d}\theta) - \mu\phi_t. \tag{2.3}$$

Note that $\phi_t(\theta)$ can be interpreted as the limiting probability that an individual with characteristics $\theta = (\lambda, \mu)$ is infected at time t.

3. Long-term behaviour

Equation (2.3) can be used to study the long-term ($t \to \infty$) behaviour of our model. For example, any equilibrium ϕ_{eq} of (2.3) must satisfy $0 = \lambda(1-\phi_{\mathrm{eq}})\int \phi_{\mathrm{eq}}(\theta)\sigma(\mathrm{d}\theta) - \mu\phi_{\mathrm{eq}}$. On setting $\psi = \int \phi_{\mathrm{eq}}(\theta)\sigma(\mathrm{d}\theta)$, we see that

$$\phi_{\mathrm{eq}}(\lambda,\mu)\ (=\phi_{\mathrm{eq}}(\theta)\ ) = \frac{\lambda\psi}{\lambda\psi+\mu},$$

and so, on integrating this over $(\lambda, \mu) \in S$, we find that ψ must solve the equation

$$\psi = R(\psi) := \int \frac{\lambda\psi}{\lambda\psi+\mu}\,\sigma(\mathrm{d}\lambda, \mathrm{d}\mu). \tag{3.1}$$

The following result characterizes the large-n equilibrium behaviour of our model in terms of $r_0 := R'(0+)$ ($\leq \infty$). It follows from Theorems 2, 3 and 4 of [10], noting that their Condition D′ holds in the present setting. Note also that since the integrand in (3.1) is less that 1, and σ is a probability measure, we can differentiate under the integral to obtain

$$R'(\psi) = \int \frac{\lambda\mu}{(\lambda\psi+\mu)^2}\,\sigma(\mathrm{d}\lambda, \mathrm{d}\mu),$$

which implies that $r_0 = \int(\lambda/\mu)\,\sigma(\mathrm{d}\lambda, \mathrm{d}\mu)$.

Theorem 3. *(a) If $r_0 \leq 1$, then $\psi = 0$ is the only fixed point of R, and $\phi_{\mathrm{eq}} = 0$ is globally stable, that is, for all ϕ_0, $\phi_t \to 0$ on S. The latter entails $m_t(B) \to 0$, for all B, and hence the disease free state is globally stable.*

(b) If $r_0 > 1$, then R has two fixed points, 0 and a positive fixed point ψ_*, and if $(m_0(S\,) =)\ (\phi_0, \sigma) > 0$, then

$$\phi_t \to \phi_* := \frac{\lambda\psi_*}{\lambda\psi_*+\mu}.$$

The latter entails $m_t(B) \to m_*(B)$, for all B, where

$$m_*(B) = \int_B \phi_*(\theta)\sigma(\mathrm{d}\theta) = \int_B \frac{\lambda\psi_*}{\lambda\psi_*+\mu}\sigma(\mathrm{d}\lambda, \mathrm{d}\mu),$$

in particular $m_*(S\,) = \psi_*$, thus implying quasi stationarity.

Example 1. Suppose that the individual characteristics are chosen independently from $\Gamma(a_\lambda, r_\lambda)$ and $\Gamma(a_\mu, r_\mu)$ distributions, respectively. Notation: r_λ and r_μ are *rate* parameters. We have that

$$r_0 = \frac{a_\lambda}{r_\lambda}\cdot\frac{r_\mu}{a_\mu - 1}.$$

Figure 2 illustrates (a) quasi-stationary behaviour, and (b) evanescence. In (a), the green line is at $\psi_* = 0.8035$, very close to 0.8 (dashed red), which is the equilibrium of the homogeneous model using $\lambda = 10$ and $\mu = 2$. Figure 3 also illustrates quasi-stationary behaviour, now with $\psi_* = 0.2435$, but the corresponding homogeneous model ($\lambda = 1.4$ and $\mu = 1.5$) predicts evanescence. This disparity between the two models happens when

$$\frac{a_\mu - 1}{r_\mu} < \frac{a_\lambda}{r_\lambda} < \frac{a_\mu}{r_\mu}.$$

Note that ψ_* was evaluated by way of fixed point iteration of (3.1) using Matlab's `integral2`, which evaluates double integrals numerically.

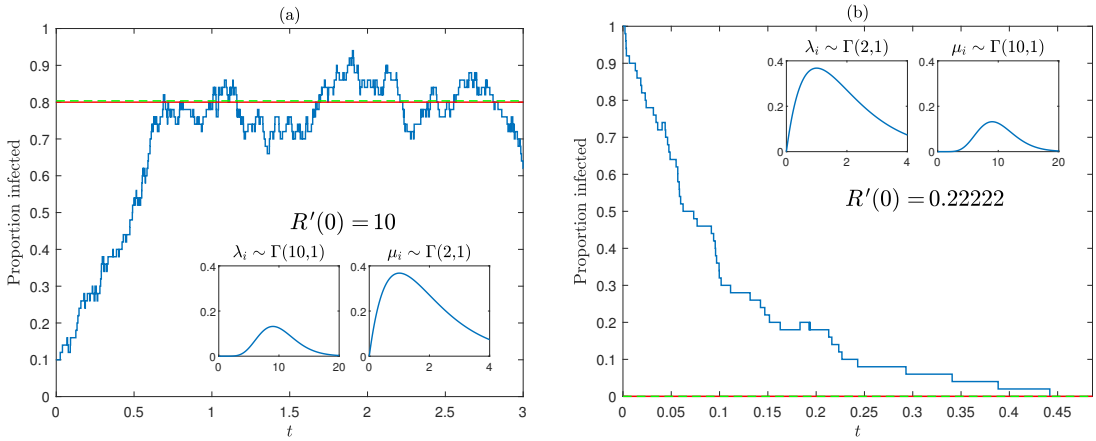

Figure 2. Simulation of the SIS model with $n = 50$ individuals, and with independent Gamma distributed infection and recovery rates.

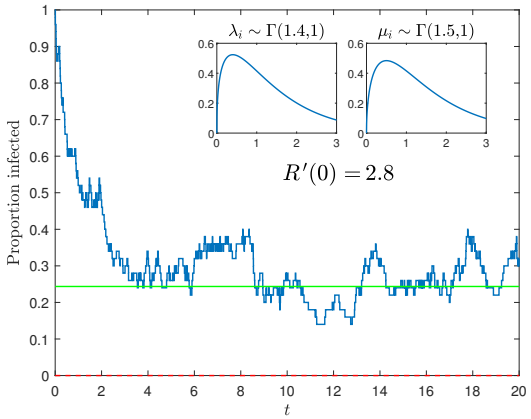

Figure 3. Simulation of the SIS model with $n = 50$ individuals, and with independent Gamma distributed infection and recovery rates.

Example 2. Suppose that the individual characteristics are chosen from a bivariate Gamma distribution of the form given in Theorem 2 of [11]. The marginals are Gamma distributions with a common

rate parameter, which we denote by r, and shape parameters a_λ and a_μ, respectively, and correlation $\rho = \sqrt{a_\lambda a_\mu}/c$, where $c > 0$. Using the representation Eq.3 on Page 768 of [11], we find that

$$r_0 = \frac{a_\lambda}{r} \cdot \frac{r}{a_\mu - 1} = \frac{a_\lambda}{a_\mu - 1}.$$

The simplest case has $r = 1$, and $b_\lambda = b_\mu = 1$ (which necessitates $a_\lambda = a_\mu = c - 1$, and $c > 1$), and $\sigma(\mathrm{d}\lambda, \mathrm{d}\mu) = f(\lambda, \mu)\, d\lambda\, \mathrm{d}\mu$, with

$$f(x, y) = \begin{cases} C(xy)^{c-2}\Gamma(2 - c, x) & \text{if } y \le x \\ C(xy)^{c-2}\Gamma(2 - c, y) & \text{if } y > x, \end{cases}$$

where $C^{-1} = \Gamma(c)/(c - 1)^2$, and $\Gamma(a, x) = \int_x^\infty t^{a-1}\mathrm{e}^{-t}\,\mathrm{d}t$ is the complementary incomplete gamma function. Figure 4 illustrates quasi-stationary behaviour with $\psi_* = 0.1941$, noting the corresponding homogeneous model ($\lambda = \mu = 1.5$) predicts evanescence.

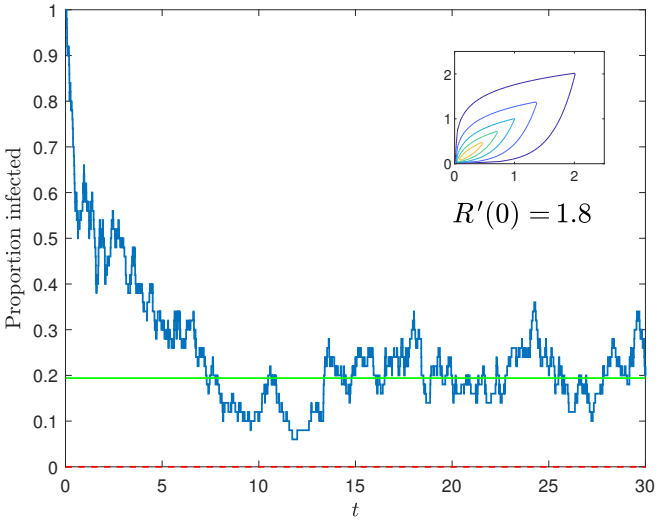

Figure 4. Simulation of the SIS model with $n = 50$ individuals, and with positively correlated ($\rho = 0.6$) Gamma distributed infection and recovery rates.

4. Moments

We can get a handle on various statistics associated with the process by way of the moment generating function (MGF) of m_t:

$$M_t(a, b) = \int \mathrm{e}^{a\lambda + b\mu} m_t(\mathrm{d}\lambda, \mathrm{d}\mu),$$

which will be defined for (a, b) in a region containing the origin. On setting $h(\theta)$ $(= h(\lambda, \mu)) = \exp(a\lambda + b\mu)$ in (2.2) we obtain an integral equation for M_t:

$$M_t(a, b) = M_0(a, b) + \int_0^t \left(m_s(S) \left(\frac{\partial}{\partial a}\Sigma(a, b) - \frac{\partial}{\partial a} M_s(a, b) \right) - \frac{\partial}{\partial b} M_s(a, b) \right) \mathrm{d}s,$$

where $\Sigma(a,b) = \int \mathrm{e}^{a\lambda+b\mu}\sigma(d\lambda, d\mu)$ is the MGF of σ. This can be differentiated to obtain the PDE

$$\frac{\partial}{\partial t}M_t(a,b) + M_t(0,0)\frac{\partial}{\partial a}M_t(a,b) + \frac{\partial}{\partial b}M_t(a,b) = M_t(0,0)\Phi(a,b), \tag{4.1}$$

where

$$\Phi(a,b) = \frac{\partial}{\partial a}\Sigma(a,b) = \int \lambda\, \mathrm{e}^{a\lambda+b\mu}\sigma(\mathrm{d}\lambda, \mathrm{d}\mu).$$

On setting $(\partial/\partial t)M_t(a,b) = 0$, we see that any equilibrium M_{eq} of (4.1) must satisfy

$$\psi\frac{\partial}{\partial a}M_{\mathrm{eq}}(a,b) + \frac{\partial}{\partial b}M_{\mathrm{eq}}(a,b) = \psi\Phi(a,b), \tag{4.2}$$

where $\psi = M_{\mathrm{eq}}(0,0)$. This PDE is easily solved using the method of characteristics. We find that

$$M_{\mathrm{eq}}(a,b) = M_{\mathrm{eq}}(0,0) + \int \frac{\psi\lambda}{\mu+\psi\lambda}(e^{a\lambda+b\mu} - 1)\,\sigma(\mathrm{d}\lambda, \mathrm{d}\mu).$$

Under the conditions of Theorem 3 (b), namely that $r_0 > 1$ and $(m_0(S)=)\ (\phi_0, \sigma) > 0$, we will have that $M_t(a,b) \to M_{\mathrm{eq}}(a,b)$, M_{eq} being the MGF of m_*:

$$M_{\mathrm{eq}}(a,b) = \int e^{a\lambda+b\mu}m_*(\mathrm{d}\lambda, \mathrm{d}\mu).$$

We will necessarily have $M_{\mathrm{eq}}(0,0) = \psi_*$, and, since $R(\psi_*) = \psi_*$,

$$M_{\mathrm{eq}}(a,b) = \int \frac{\psi_*\lambda}{\mu+\psi_*\lambda}e^{a\lambda+b\mu}\sigma(\mathrm{d}\lambda, \mathrm{d}\mu).$$

Example 3. Suppose that the individual characteristics are chosen from independent exponential distributions with rate parameters l and m: $\sigma(\mathrm{d}\lambda, \mathrm{d}\mu) = l\mathrm{e}^{-l\lambda}\, m\mathrm{e}^{-m\mu}\,\mathrm{d}\lambda\,\mathrm{d}\mu$. With this choice we can perform some explicit calculations. For example,

$$R(\psi) = \begin{cases} \frac{\rho\psi}{(\rho-\psi)^2}\log\left(\frac{\rho}{\psi}\right) - \frac{\psi}{\rho-\psi} & \text{if } 0 < \psi \neq \rho \\ \frac{1}{2} & \text{if } 0 < \psi = \rho \\ 0 & \text{if } \psi = 0, \end{cases}$$

where $\rho = l/m$. Therefore,

$$R'(\psi) = \begin{cases} \frac{\rho(\rho+\psi)}{(\rho-\psi)^3}\log\left(\frac{\rho}{\psi}\right) - \frac{2\rho}{(\rho-\psi)^2} & \text{if } 0 < \psi \neq \rho \\ \frac{1}{6\rho} & \text{if } 0 < \psi = \rho. \end{cases}$$

So, we have $R'(0+) = \infty$. This implies a stable equilibrium. In particular, R has two fixed points, 0 and a positive fixed point ψ_*. Again, a simple calculation yields

$$M_{\mathrm{eq}}(a,b) = \frac{lm\psi_*}{(l-a-\psi_*(m-b))^2}\log\left(\frac{l-a}{\psi_*(m-b)}\right) - \frac{lm\psi_*}{(l-a)(l-a-\psi_*(m-b))}, \quad a < l,\ b < m.$$

Notice that $M_{\text{eq}}(0,0) = R(\psi_*) = \psi_*$. For example,

$$\int \mu m_*(\mathrm{d}\lambda, \mathrm{d}\mu) = \frac{\partial M_{\text{eq}}}{\partial b}(0,0) = \frac{\psi_*(l + m\psi_*)}{(l - m\psi_*)^2} - \frac{2lm\psi_*^2}{(l - m\psi_*)^3} \log\left(\frac{l}{m\psi_*}\right),$$

a quantity which approximates the large-n long-term behaviour of (refer to (2.1))

$$\int \mu m_t^{(n)}(\mathrm{d}\lambda, \mathrm{d}\mu) = \frac{1}{n} \sum_{i=1}^{n} \mu_i X_{i,t}^{(n)}, \tag{4.3}$$

being the average recovery rate of infected individuals. This is illustrated in Figure 5, where the average recovery rate of infected individuals (cyan) is plotted along with the value of (4.3) (yellow).

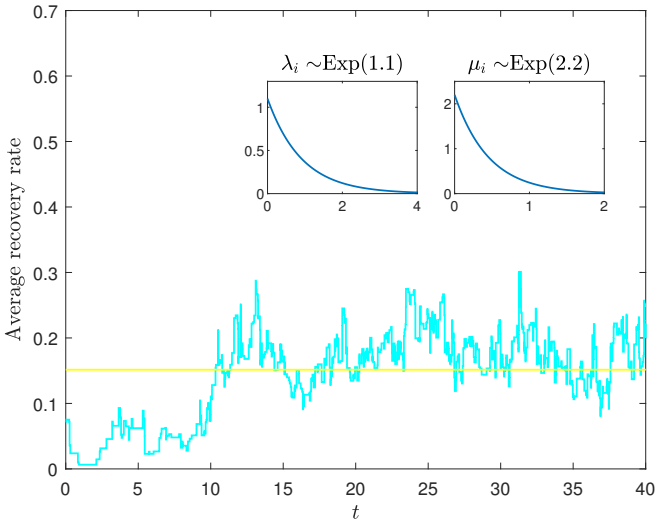

Figure 5. Simulation of the SIS model with $n = 50$ individuals, and with independent exponentially distributed infection and recovery rates.

5. Conclusion

We have presented a model for an SIS infection that incorporates variation of individual characteristics. By imagining that these characteristics follow a point process on a subset of $\mathbb{R}_+^2$, we are able to exploit existing results on random measures, and random measure-valued processes, to obtain large-population limits, and then predict long-term behaviour. These results provide a means of studying various statistics associated with the process, including the proportion of individuals infected. Several examples demonstrate a range of behaviour that cannot be gleaned from the standard stochastic SIS model based on mean values of the characteristics. Explicit results on the moment generating function of the (large-n) limiting process allow us to estimate additional quantities such as the average infection rates and average recovery rates.

The methods described here do not provide explicit results on the *quality* of the approximations presented, and in particular how large n would need to be for our approximations to be faithful. It might be possible to adapt the results in Chapter 4 of [12], based on their discrete-time counterparts [13], to

provide explicit rates of convergence by attempting to bound weighted sums of differences:

$$\frac{1}{n}\sum_{i=1}^{n} h(\theta_i)|X_{i,t}^{(n)} - p_{i,t}|,$$

where $p_t = (p_{i,t},\ i = 1 \dots n)$ is some suitable deterministic law of motion; this is the subject of ongoing research. A simple extension to the model presented would be to replace the upward transition rate in (1.2) by $a + \lambda_i \bar{X}^{(n)}$, where $a > 0$, thus incorporating an external source of infection as in [14]. Now the disease-free state **0** would no longer be an absorbing state, but the methods employed here could be brought to bear, thus providing a means of estimating equilibrium behaviour.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Conflict of interest

The authors declare there is no conflict of interest.

References

1. G.H. Weiss, M. Dishon, On the asymptotic behaviour of the stochastic and deterministic models of an epidemic, *Math. Biosci.*, **11** (1971), 261–265. https://doi.org/10.1016/0025-5564(71)90087-3
2. T. G. Kurtz, Solutions of ordinary differential equations as limits of pure jump Markov processes, *J. Appl. Probab.*, **7** (1970), 49–58. https://doi.org/10.2307/3212147
3. F. Ball, Stochastic and deterministic models for SIS epidemics among a population partitioned into households, *Math. Biosci.*, **158** (1999), 41–67. https://doi.org/10.1016/S0025-5564(98)10060-3
4. F. G. Ball, O. D. Lyne, Optimal vaccination policies for stochastic epidemics among a population of households, *Math. Biosci.*, **177–178** (2002), 333–354. https://doi.org/10.1016/S0025-5564(01)00095-5
5. T. Britton, P. Neal, The time to extinction for an SIS-household-epidemic model, *J. Math. Biol.*, **61** (2010), 763–779. https://doi.org/10.1007/s00285-009-0320-5
6. D. Clancy, P. O'Neill, Exact bayesian inference and model selection for stochastic models of epidemics among a community of households, *Scand. J. Statist.*, **34** (2007), 259–274. https://doi.org/10.1111/j.1467-9469.2006.00522.x
7. D. Hiebler, Moment equations and dynamics of a household SIS epidemiological model, *J. Math. Biol.*, **68** (2006), 1315–1333. https://doi.org/10.1007/s11538-006-9080-1
8. P. Neal, Efficient likelihood-free bayesian computation for household epidemics, *Stat. Comput.*, **22** (2012), 1239–1256. https://doi.org/10.1007/s11222-010-9216-x
9. V. Cane, R. McNamee, The spread of infection in a heterogeneous population, *J. Appl. Probab.*, **19A** (1982), 173–184. https://doi.org/10.1017/S0021900200034549

10. R. McVinish, P. K. Pollett, The deterministic limit of a stochastic logistic model with individual variation, *Math. Biosci.*, **241** (2013), 109–114. https://doi.org/10.1016/j.mbs.2012.10.001

11. S. Nadarajaha, A. K. Gupta, Some bivariate gamma distributions. *Appl. Math. Letters*, **19** (2006), 767–774. https://doi.org/10.1016/j.aml.2005.10.007

12. L. Hodgkinson, *Approximations for finite spin systems and occupancy processes*. PhD thesis, School of Mathematics and Physics, The University of Queensland, 2019.

13. L. Hodgkinson, R. McVinish, P. K. Pollett. Normal approximations for discrete-time occupancy processes, *Stochastic Process. Appl.*, **130** (2020), 6414–6444. https://doi.org/10.1016/j.spa.2020.05.016

14. P. Stone, H. Wilkinson-Herbots, V. Isham, A stochastic model for head lice infections, *J. Math. Biol.*, **56** (2008), 743–763. https://doi.org/10.1007/s00285-007-0136-0

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