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# Stochastic and deterministic models for SIS epidemics among a population partitioned into households

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### Abstract

Models for the spread of an SIS epidemic among a population consisting of m households, each containing n individuals, are considered and their behaviour is analysed under the practically relevant situation when m is large and n small. A threshold parameter  $R_*$  is determined. For the stochastic model it is shown that the epidemic has a non-zero probability of taking off if and only if  $R_* > 1$ , and the extension to unequal household sizes is also considered. For the deterministic model, with households of size 2, it is shown that if  $R_* \leq 1$  then the epidemic dies out, whilst if  $R_* > 1$  the epidemic settles down to an endemic equilibrium. The usual basic reproductive ratio  $R_0$  does not provide a good indicator for the behaviour of these household epidemic models unless the household size n is large. © 1999 Elsevier Science Inc. All rights reserved.

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## 1. Introduction

There has been considerable interest recently in models for the spread of an epidemic among a community of households, see e.g. Refs. [4,6–13]. All of

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these previous studies have been concerned with SIR (susceptible  $\rightarrow$  infective  $\rightarrow$  removed) epidemics, and hence with models that cannot display endemic behaviour. The aim of this paper is to examine, within a population of households setting, the simplest class of epidemic models that can show endemic behaviour, namely SIS (susceptible  $\rightarrow$  infected  $\rightarrow$  susceptible) epidemics.

The standard SIS model, which was introduced by Ross (see e.g. [25]), is a model for the spread of an epidemic among a homogeneously mixing population of n individuals, in which an infective individual becomes susceptible again as soon as its infectious period terminates. For  $t \ge 0$ , let  $Y_n(t)$  denote the number of infectives at time t. Then in the stochastic version of the standard SIS model,  $\{Y_n(t); t \ge 0\}$  is a continuous time Markov chain, with state space  $\{0, 1, \ldots, n\}$  and transition probabilities

$$P(Y_n(t + \Delta t) = j \mid Y_n(t) = i) = \begin{cases} \frac{\lambda}{n} i(n - i)\Delta t + o(\Delta t) & \text{if } j = i + 1, \\ \gamma i \Delta t + o(\Delta t) & \text{if } j = i - 1, \\ o(\Delta t) & \text{otherwise.} \end{cases}$$
(1)

Thus a typical infective makes infectious contacts at the points of a Poisson process with rate  $\lambda$  during an infectious period which follows a negative exponential distribution with mean  $\gamma^{-1}$ , and the individuals contacted at successive contacts are chosen independently and uniformly from the n individuals in the population.

Suppose that  $Y_n(0) = ny_n(0)$ , where  $y_n(0) \to y(0) \in (0, 1]$  as  $n \to \infty$ . Then as  $n \to \infty$ , the process describing the density of infectives,  $\{n^{-1}Y_n(t); t \ge 0\}$ , converges, in a sense that is made clear in Ref. [19] to a deterministic limit described by the differential equation

$$\frac{\mathrm{d}y}{\mathrm{d}t} = \lambda y(1-y) - \gamma y. \tag{2}$$

The *reproductive ratio* (see e.g. Ref. [15]) of the epidemic is  $R_0 = \lambda/\gamma$ . It is shown in Ref. [16] that if  $R_0 \le 1$  then the origin is the only equilibrium point of Eq. (2) and  $y(t) \to 0$  as  $t \to \infty$ , whilst if  $R_0 > 1$  there is a second equilibrium point  $y^* = 1 - R_0^{-1}$  and, provided y(0) > 0,  $y(t) \to y^*$  as  $t \to \infty$ . Thus the epidemic invades the population, in the sense that an initial trace of infection gives rise to a true epidemic, if and only if  $R_0 > 1$  and the epidemic will persist, i.e. remain endemic, if and only if  $R_0 > 1$ . Hence  $R_0 = 1$  provides both an invasion threshold and a persistence threshold for the deterministic SIS epidemic. Similar results hold for a very wide class of deterministic epidemic models.

Consider now the stochastic SIS model and suppose that  $Y_n(0) = a$ . During the early stages of the epidemic, all infectious contacts are likely to be with susceptibles, so the process of infectives can be approximated by a birth-and-death process having birth rate  $\lambda$  and death rate  $\gamma$ . The extinction probability for this birth-and-death process is  $[\min(R_0^{-1}, 1)]^a$ , see e.g. Ref. [14], p. 253. Thus in a large population the epidemic will have a non-zero probability of 'taking

off only if  $R_0 > 1$ . This threshold behaviour can be made precise, as  $n \to \infty$ , either by using the coupling argument of Ball and Donnelly [5] or by adapting the sandwiching method of Whittle [27]. Note that although the deterministic and the stochastic SIS models have the same invasion threshold  $R_0 = 1$ , the interpretation of the threshold behaviour in the two models is quite different.

Nåsell (see e.g. Ref. [23]) defines invasion and persistence thresholds for the stochastic SIS epidemic in terms of the expected time to extinction from a single initial infective and from an infective population that is distributed according to the quasi-stationary distribution of  $\{Y_n(t); t \ge 0\}$ , respectively. However, his methods do not extend easily to more complicated models.

Suppose now that the population consists of m households, labelled  $1, 2, \ldots, m$ , each containing n individuals, and that a typical infective makes within- and between-household contacts at rates  $\lambda_{\rm W}$  and  $\lambda_{\rm B}$ , respectively. The standard deterministic formulation of this model is

$$\frac{dy_i}{dt} = \lambda_W y_i (1 - y_i) + \frac{\lambda_B}{m - 1} (1 - y_i) \sum_{j \neq i} y_j - \gamma y_i \quad (i = 1, 2, \dots, m),$$
 (3)

where  $y_i(t)$  denotes the density of susceptibles in household i at time t. Let  $\mathbf{y}(t) = (y_1(t), y_2(t), \dots, y_m(t))^{\top}$ , where  $\top$  denotes transpose, and  $R_0 = (\lambda_{\mathbf{W}} + \lambda_{\mathbf{B}})/\gamma$ . If  $R_0 \le 1$  then the origin  $\mathbf{0}$  is the only equilibrium point of Eq. (3) and moreover that equilibrium point is globally asymptotic stable, in the sense that for any  $\mathbf{y}(0) \in [0, 1]^m$ ,  $\mathbf{y}(t) \to \mathbf{0}$  as  $t \to \infty$ . If  $R_0 > 1$  there is a second (endemic) equilibrium point at  $\mathbf{y}^* = (y^*, y^*, \dots, y^*)$ , where  $y^* = 1 - R_0^{-1}$ , which is globally asymptotic stable in  $[0, 1]^m \setminus \{\mathbf{0}\}$ . See Ref. [21] for details.

The results of Kurtz [19] show that the deterministic model (3) provides a good approximation to its more realistic stochastic counterpart if the initial density of infectives is non-zero, the number of households m is fixed and the household size n is large. However, this is *not* the situation of practical interest, which is a population consisting of a large number of small households. Thus, in this paper the asymptotic situation when  $m \to \infty$  with n fixed is considered. In Section 2, the invasion threshold is analysed from a stochastic perspective for a very general household SIS epidemic model, whilst in Section 3, the persistence threshold is studied from a deterministic perspective, using an appropriate model. A few concluding comments are given in Section 4.

## 2. Invasion threshold

## 2.1. Household SIS epidemic model

Consider the following model for the spread of an SIS epidemic among a population of m households, each containing n individuals. Attach to each individual in the population independent and identically distributed *infectious* 

careers  $\mathcal{H} = \{\mathcal{H}^{(k)}; k=1,2,\ldots\}$ , where for  $k=1,2,\ldots,\mathcal{H}^{(k)} = (T_1^{(k)},\eta_W^{(k)},\eta_B^{(k)}),T_1^{(k)}$  is the time elapsing between an individual's kth infection and it becoming susceptible again, and  $\eta_W^{(k)}$  and  $\eta_B^{(k)}$  are point processes of times, relative to an individual's kth infection, at which within-household and between-household contacts are made, respectively. Each within-household contact of a specific infective is with an individual chosen independently and uniformly from the n individuals in the same household. Each between-household contact of a specific infective is with an individual chosen independently and uniformly from the m individuals in the population. If the individual contacted by the infective is susceptible, then it becomes infected and adopts the appropriate  $\mathcal{H}^{(k)}$  of its infectious career, otherwise nothing happens. Initially there are m - 1 susceptibles and one infective, who has just been infected for the first time. The epidemic ceases as soon as there are no infectives present in the population.

Denote the above epidemic model by  $E_{m,n}$ . Note that the possibilities of within-household contacts with oneself and between-household contacts with one's own household involve no real loss of generality since, for any realistic model, the point processes  $\eta_W^{(k)}$  and  $\eta_B^{(k)}$  ( $k=1,2,\ldots$ ) can be modified accordingly. Note also that the components  $\mathscr{H}^{(k)}$  ( $k=1,2,\ldots$ ) of  $\mathscr{H}$  need not be identically distributed, or even independent. Also, for  $k=1,2,\ldots$ , the components  $T_1^{(k)}$ ,  $\eta_W^{(k)}$  and  $\eta_B^{(k)}$  need not be independent and the model also implicitly includes the possibility of a latent period and a recovery period after infection before an individual becomes susceptible again. Specifically, for  $k=1,2,\ldots$ , suppose that after its kth infection, an infected individual has a latent period of length  $T_L^{(k)}$  before it is able to infect others. This is followed by an infectious period of length  $\tilde{T}_1^{(k)}$ , which is in turn followed by a recovery period of length  $T_R^{(k)}$  before it becomes susceptible again. Then such a model is included in the above framework by setting  $T_1^{(k)} = T_L^{(k)} + \tilde{T}_1^{(k)} + T_R^{(k)}$  and choosing the probabilistic laws of  $\mathscr{H}^{(k)}$  so that, with probability one,  $\eta_W^{(k)}$  and  $\eta_B^{(k)}$  only have points in the (possibly random) interval  $[T_L^{(k)}, T_L^{(k)} + \tilde{T}_1^{(k)}]$ . However, such detail is not required for our general results and thus it is not included explicitly in our model.

## 2.2. Construction of epidemic and approximating branching process

In order to examine the asymptotic behaviour of  $E_{m,n}$  as the number of households  $m \to \infty$ , it is fruitful to construct realisations of  $E_{m,n}$ , for  $m = 1, 2, \ldots$ , on the same probability space as follows. Let  $(\Omega, \mathcal{F}, P)$  be a probability space on which are defined the following independent sets of random quantities:

(i)  $\mathcal{H}_{ij}$  (i = 1, 2, ...; j = 1, 2, ..., n), independent and identically distributed according to  $\mathcal{H}$ ;

- (ii)  $\mathcal{C}_{ij}$  (i = 1, 2, ...; j = 1, 2, ...), independent and uniformly distributed on  $\{1, 2, ..., n\}$ ;
- (iii)  $U_1, U_2, \ldots$ , independent and uniformly distributed on (0, 1).

For m = 1, 2, ..., the epidemic  $E_{m,n}$  is constructed as follows. Label the households  $1, 2, \dots, m$  and for  $i = 1, 2, \dots, m$  label the individuals in household  $i(i,1),(i,2),\ldots,(i,n)$ . Also, give the individuals population-based labels  $1, 2, \dots, N$ , where N = mn and the population-based label of individual (i,j) is (i-1)n+j. Suppose that the initial infective is individual (1,1). For  $i=1,2,\ldots,m$ , let  $r_i^{(m)}$  denote the *i*th household to be infected by the epidemic  $E_{m,n}$ , so  $r_1^{(m)} = 1$ . For i = 1, 2, ..., m and j = 1, 2, ..., n, the jth individual to be infected for the first time in household  $r_i^{(m)}$  adopts the infectious career  $\mathcal{H}_{ij}$ , which can be written as  $\mathcal{H}_{ij} = \{\mathcal{H}_{ij}^{(k)}; k = 1, 2, ...\}$ , where, for  $k = 1, 2, ..., \mathcal{H}_{ij}^{(k)} = (T_{1ij}^{(k)}, \eta_{W_{ij}}^{(k)}, \eta_{B_{ij}}^{(k)})$ . Thus the initial infective adopts the infectious career  $\mathscr{H}_{11}$  and, during its first infection, makes contacts governed by the point processes  $\eta_{W_{11}}^{(1)}$  and  $\eta_{B_{11}}^{(1)}$ . For  $i=1,2,\ldots,m$  and  $j=1,2,\ldots$ , the individual contacted at the jth within-household contact in household  $r_i^{(m)}$ , is given by  $(r_i^{(m)}, \mathcal{C}_{ij})$ . For  $k = 1, 2, \ldots$ , the individual contacted at the kth betweenhousehold contact occurring in  $E_{m,n}$  has population-based label given by  $\chi_k^{(m)} = [NU_k] + 1$ , where, for  $x \in \mathbb{R}$ , [x] denotes the greatest integer  $\leq x$ . If a contacted individual is infective then nothing happens. If it is susceptible then, for some i, j and k, it will be the kth infection of the jth individual to be infected for first time in household  $r_i^{(m)}$ , so it starts making contacts according to the point processes  $\eta_{W_{ij}}^{(k)}$  and  $\eta_{B_{ij}}^{(k)}$ . If j=k=1, so the contacted individual is the first to be infected in household  $r_i^{(m)}$ , then the contacted individual swaps his labels with those of  $(r_i^{(m)}, 1)$ . The epidemic ceases as soon as there are no infectives in the population.

The sets of infectious careers and associated contact numbers can be used to define a general (Crump-Mode-Jagers) branching process on  $(\Omega, \mathcal{F}, P)$ , which will approximate the early stages of the epidemic  $E_{m,n}$  when the number of households m is large. For i = 1, 2, ..., the infectious careers  $\mathcal{H}_{i1}, \mathcal{H}_{i2}, ..., \mathcal{H}_{in}$ and the contact numbers  $\mathscr{C}_{i1}, \mathscr{C}_{i2}, \dots, \mathscr{C}_{in}$  can be used to define a single household SIS epidemic, with initially one infective (the individual labelled 1) and n-1 susceptibles, by ignoring the point processes  $\eta_{B_{ij}}^{(k)}(j=1,2,\ldots,n;k=1,2,\ldots,n)$ 1, 2...) governing between-household contacts. Let  $D_i$  denote the duration of this single household SIS epidemic and let  $\eta_i^G$  denote the point process, obtained using  $\eta_{B_{ij}}^{(k)}(j=1,2,\ldots,n;k=1,2\ldots)$ , of times relative to the first infection of the initial infective at which infectives in this single household epidemic would make between-household contacts if they were allowed to. Then  $(D_i, \eta_i^G)$  (i = 1, 2, ...) are independent and identically distributed. Thus they can be used to define a general branching process, initiated by a single ancestor, in which a typical individual lives until age D and reproduces at ages according to  $\eta^{G}$ . See e.g. Ref. [17], Ch. 6, for a discussion of general branching processes.

## 2.3. Main convergence theorem and threshold behaviour

For  $t \ge 0$ , let  $Z_m(t)$  be the number of households in  $E_{m,n}$  that are infectious at time  $t(m=1,2,\ldots)$  and let Z(t) be the number of individuals that are alive in the branching process at time t. Note that when determining  $Z_m(t)$ , an individual is deemed to be infectious if it is latent or recovering from infection. For t>0, let T(t) be the total number of individuals born in (0, t] in the branching process. Let  $T(\infty) = \lim_{t \to \infty} T(t)$ . It is assumed that the branching process is non-explosive, i.e. that P(C) = 1, where  $C = \{\omega \in \Omega : T(t, \omega) < \infty \ \forall t > 0\}$ .

**Theorem 2.1.** Let  $A = \{\omega \in \Omega : T(\infty, \omega) < \infty\}$  denote the set on which the branching process becomes extinct. Then:

- (a)  $\lim_{m\to\infty} \sup_{0\leq t\leq\infty} |Z_m(t,\omega)-Z(t,\omega)| = 0$  for P-almost all  $\omega\in A$ ;
- (b) for any  $t_0 > 0$ ,

$$\lim_{m \to \infty} \sup_{0 \le t \le t_0} |Z_m(t,\omega) - Z(t,\omega)| = 0 \quad \textit{for P-almost all } \omega \in A^c.$$

**Proof.** The key observation in proving the theorem is that  $Z_m(t,\omega) = Z(t,\omega)$   $(0 \le t < \tau_m(\omega))$ , where  $\tau_m(\omega)$  is the time of the first between-household contact in  $E_{m,n}$  with a previously infected household. Let  $B = \{\omega \in \Omega : U_i \times (\omega) \ne U_i(\omega) \ \forall i \ne j\}$ . Then P(B) = 1, see e.g. Ref. [2], Lemma 2.

Fix  $\omega \in A \cap B$ . Then  $T(\infty, \omega) < \infty$  and, since  $\omega \in B$ ,  $\varepsilon(\omega) > 0$ , where  $\varepsilon(\omega) = \min\{|U_i(\omega) - U_j(\omega)| : i, j = 1, 2, \dots, T(\infty, \omega), i \neq j\}$ . Then for  $m > \max\{\varepsilon(\omega)^{-1}, (n-1)/(n\min\{U_i(\omega) : i = 1, 2, \dots, T(\infty, \omega)\})\}$ , the individuals  $\chi_k^{(m)}(\omega) = [mnU_k(\omega)] + 1$  ( $k = 1, 2, \dots, T(\infty, \omega)$ ) will belong to distinct households, none of which will be household 1. (If  $\chi_k^{(m)}(\omega)$  and  $\chi_l^{(m)}(\omega)$  belong to the same household then  $|mnU_k(\omega) - mnU_l(\omega)| < n$ , which implies that  $m < 1/|U_k(\omega) - U_l(\omega)|$  and hence that  $m < \varepsilon(\omega)^{-1}$ . Alternatively, if  $\chi_k^{(m)}(\omega)$  belongs to household 1 then  $[mnU_k(\omega)] + 1 \leq n$ , which implies that  $m \leq (n-1)/(nU_k(\omega))$  and hence that  $m \leq (n-1)/(n\min\{U_i(\omega) : i = 1, 2, \dots, T(\infty, \omega)\})$ . Thus, for  $m > \max\{\varepsilon(\omega)^{-1}, (n-1)/(n\min\{U_i(\omega) : i = 1, 2, \dots, T(\infty, \omega)\})\}$ , every between-household contact in  $E_{m,n}$  is with a previously uninfected household and hence every birth in the branching process will correspond to a new infected household in  $E_{m,n}$ . This proves part (a), since  $P(A \cap B) = P(A)$  as P(B) = 1.

Now fix  $\omega \in A^c \cap B \cap C$ . Then, since  $\omega \in C$ , given  $t_0 > 0$ ,  $T(t_0, \omega) < \infty$  and the above argument can be repeated but with  $T(\infty, \omega)$  replaced by  $T(t_0, \omega)$ . This proves part (b), since  $P(A^c \cap B \cap C) = P(A^c)$  as P(B) = P(C) = 1.

For m = 1, 2, ..., let  $T_m(\infty)$  be the total number of households that receive infection during the epidemic  $E_{m,n}$ . Note that  $T_m(\infty)$  includes the initially

infected household, whereas  $T(\infty)$  does not include the initial ancestor in the branching process.

**Theorem 2.2.**  $\lim_{m\to\infty} T_m(\infty) = 1 + T(\infty)$  almost surely.

**Proof.** If  $\omega \in A \cap B$  then  $T(\infty, \omega) < \infty$  and  $\lim_{m \to \infty} T_m(\infty, \omega) = 1 + T(\infty, \omega)$  since, as in the proof of Theorem 2.1,  $Z_m(t, \omega) = Z(t, \omega)$  ( $0 \le t < \infty$ ) for all sufficiently large m. If  $\omega \in A^c \cap B \cap C$  then  $T(\infty, \omega) = \infty$ , so for  $k = 1, 2, \ldots$ , let  $\tilde{\tau}_k(\omega) = \inf\{t: T(t, \omega) \ge k\}$ . Then  $\tilde{\tau}_k(\omega) < \infty$  so, as in the proof of Theorem 2.1, for fixed k,  $Z_m(t, \omega) = Z(t, \omega)$  ( $0 \le t \le \tilde{\tau}_k(\omega)$ ) for all sufficiently large m. Thus  $\lim_{m \to \infty} T_m(\infty, \omega) \ge k$  ( $k = 1, 2, \ldots$ ) and hence  $\lim_{m \to \infty} T_m(\infty, \omega) = \infty = T(\infty, \omega)$ . Theorem 2.2 follows since  $P((A \cap B) \cup (A^c \cap B \cap C)) = 1$ .

We are now in a position to prove an invasion threshold theorem for our stochastic household SIS epidemic model. A *global epidemic* is said to occur if in the limit as the number of households  $m \to \infty$  the epidemic affects infinitely many households.

**Corollary 2.3.** Let  $R = \eta^G((0, \infty))$  be the total number of between-household contacts emanating from a typical single-household SIS epidemic. Let  $R_* = E[R]$  and  $f(s) = E[s^R]$  be the probability generating function of R. Then, as the number of households  $m \to \infty$ ,

- (a) a global epidemic occurs with non-zero probability if and only if  $R_* > 1$ ,
- (b) the probability of a global epidemic is 1 p, where p is the smallest root of f(s) = s in [0,1],
- (c) the probability generating function of the limiting total number of households that receive infection  $T(\infty) + 1$ , h(s) say, satisfies h(s) = sf(h(s)). Further.

$$P(T(\infty) + 1 = k) = k^{-1}P(R_1 + R_2 + \dots + R_k = k)$$
  $(k = 1, 2, \dots),$  where  $R_1, R_2, \dots$  are independent and identically distributed copies of  $R$ .

**Proof.** The distribution of  $T(\infty)$  is the same as that of the embedded Galton-Watson process with offspring distribution the distribution of R. Corollary 2.3 follows immediately from Theorem 2.2 and standard results (see e.g. Ref. [17], Ch. 2) for Galton-Watson processes.

The previous discussion has assumed that the epidemic is started by a single initial case. It can easily be extended to the situation where there are  $a < \infty$  initial infectives. In particular, the threshold behaviour described by Corollary 2.3(a) still holds. If the initial infectives are in distinct households then the probability of a global epidemic is  $1 - p^a$ , where p is as in Corollary 2.3(b), and

Corollary 2.3(c) can also be modified appropriately. The situation is more complicated if some households have more than one initial infectives, since then in the approximating branching process the initial ancestors no longer all have the same offspring distribution as R. In such cases, the probability of a global epidemic is most easily determined by conditioning on the size of the first generation in the embedded Galton–Watson process, as in Ref. [6] for household SIR epidemics.

## 2.4. Unequal household sizes

In practice, it is most unlikely that all the households in a population will be of equal size, so we now consider an extension of the preceding theory to the case of unequal household sizes. Thus suppose that the population consists of  $m_n$  households of size n (n = 1, 2, ...), where the total number of households  $m = \sum_{n=1}^{\infty} m_n$  and the total number of individuals  $N = \sum_{n=1}^{\infty} n m_n$  are both finite. As before, the epidemic is defined in terms of infectious careers, with the infectious careers of different individuals being independent and the infectious careers of all individuals in households of a given size being identically distributed. For n = 1, 2, ..., let  $\mathcal{H}^{(n)} = \{\mathcal{H}^{(n,k)}; k = 1, 2, ...\}$  denote the infectious career of a typical individual residing in a household of size n, where  $\mathcal{H}^{(n,k)} = (T_{\mathrm{I}}^{(n,k)}, \eta_{\mathrm{W}}^{(n,k)}, \eta_{\mathrm{B}}^{(n,k)})$   $(k = 1, 2, \ldots)$ . As before, each within-household (between-household) contact of a given infective is with an individual chosen independently and uniformly from the individuals within the same household (population). For ease of exposition, it is assumed that initially there is one infective, who has just been infected for the first time and who is chosen uniformly from the N individuals in the population, though this assumption can be relaxed.

A realisation of the above epidemic model can be constructed on a probability space  $(\Omega, \mathcal{F}, P)$  as follows. Define on  $(\Omega, \mathcal{F}, P)$  the following independent sets of random quantities:

- (i) for  $n = 1, 2, ..., \mathcal{H}_{ij}^{(n)} (i = 1, 2, ...; j = 1, 2, ..., n)$  independent and identically distributed according to  $\mathcal{H}^{(n)}$ ;
- (ii) for  $n = 1, 2, ..., C_{ij}^{(n)}$  (i = 1, 2, ...; j = 1, 2, ...), independent and uniformly distributed on  $\{1, 2, ..., n\}$ ;
- (iii)  $U_0, U_1, \ldots$  independent and uniformly distributed on (0, 1). Label the households  $1, 2, \ldots, m$  in increasing order of size. For  $i = 1, 2, \ldots, m$ , let  $n_i$  denote the size of household i and label the individuals in that household  $(i, 1), (i, 2), \ldots, (i, n_i)$ . Also, give the individuals population-based labels  $1, 2, \ldots, N$ , with individual (i, j) having population-based label  $\sum_{k=1}^{i-1} n_k + j$ , the sum being vacuous if i = 1.

The epidemic is constructed similarly to before, except now the initial infective has population-based label  $[NU_0]+1$  and for  $n=1,2,\ldots$  and  $i=1,2\ldots$ ,  $\mathscr{H}_{i1}^{(n)},\mathscr{H}_{i2}^{(n)},\ldots,\mathscr{H}_{in}^{(n)}$  are the infectious careers of the individuals in the

*i*th household of size n to be infected by the epidemic, again allocated in order of initial infection, and  $C_{i1}^{(n)}, C_{i2}^{(n)}, \ldots$  give the individuals contacted at successive within-household contacts in that household. For  $k=1,2,\ldots$ , the individual contacted at the kth between-household contact occurring in the epidemic has population-based label given by  $\chi_k = [NU_k] + 1$ . As before, the first person to be infected in a household swaps his labels with the individual labelled 1 in that household.

In order to prove a threshold theorem for the epidemic with unequal household sizes, we consider a sequence of such epidemics,  $E_v$  ( $v=1,2,\ldots$ ), all defined on  $(\Omega,\mathcal{F},P)$ , with the epidemic  $E_v$  being among a population consisting of  $m_n^{(v)}$  households of size n ( $n=1,2,\ldots$ ) and thus comprising  $m^{(v)}=\sum_{n=1}^{\infty}m_n^{(n)}$  households and  $N^{(v)}=\sum_{n=1}^{\infty}nm_n^{(v)}$  individuals, where  $m^{(v)}$  and  $N^{(v)}$  are both finite. We assume that:

- (i)  $m^{(v)} \to \infty$  as  $v \to \infty$ ;
- (ii)  $\frac{m_n^{(v)}}{m^{(v)}} \to \theta_n$  as  $v \to \infty$  (n = 1, 2, ...);
- (iii)  $\sum_{n=1}^{\infty} \theta_n = 1$  and  $\sum_{n=1}^{\infty} n\theta_n < \infty$ .

Thus as  $v \to \infty$  the number of households tends to  $\infty$  and the proportions of households of different sizes tends to a proper probability distribution having finite mean. These conditions, which are very mild, ensure that the epidemic processes converge to a branching process as  $v \to \infty$ . For  $n = 1, 2, \ldots$ , let  $\alpha_n^{(v)} = n m_n^{(v)} / N^{(v)}$ ,  $\alpha_n = n \theta_n / \sum_{k=1}^{\infty} k \theta_k$ ,  $\beta_n^{(v)} = \sum_{k=1}^n \alpha_k^{(v)}$  and  $\beta_n = \sum_{k=1}^n \alpha_k$ . Then, for  $n = 1, 2, \ldots, \alpha_n^{(v)} \to \alpha_n$  and  $\beta_n^{(v)} \to \beta_n$  as  $v \to \infty$ . Note that, for  $n = 1, 2, \ldots, \alpha_n^{(v)}$  is the probability that an individual chosen uniformly at random from the population among which  $E_v$  is spreading resides in a household of size n and  $\alpha_n$  is the corresponding limiting probability.

The approximating branching process can now be constructed as follows. For  $n=1,2,\ldots$  and  $i=1,2,\ldots$ , the infectious careers  $\mathcal{H}_{i1}^{(n)},\mathcal{H}_{i2}^{(n)},\ldots,\mathcal{H}_{in}^{(n)}$  and contact numbers  $C_{i1}^{(n)},C_{i2}^{(n)},\ldots$  can be used to construct an SIS epidemic among a single household of size n, with one initial infective (the individual labelled 1). Use that epidemic to define  $D_i^{(n)}$  and  $\eta_{Gi}^{(n)}$  similarly to  $D_i$  and  $\eta_i^G$  of Section 2.2. Then  $(D_i^{(n)}, \eta_{Gi}^{(n)})$   $(n = 1, 2, \dots; i = 1, 2, \dots)$  are independent and, for  $n = 1, 2, ..., (D_i^{(n)}, \eta_{Gi}^{(n)})$  (i = 1, 2, ...) are identically distributed, say according to  $(D^{(n)}, \eta_G^{(n)})$ . Thus  $(D_i^{(n)}, \eta_{Gi}^{(n)})$   $(n = 1, 2, \dots; i = 1, 2, \dots)$  can be used to define a multitype branching process, with type corresponding to household size n, in which, for  $k = 1, 2, \dots$ , the kth individual born into the process is of type *n* if and only if  $U_k \in (\beta_{n-1}, \beta_n]$ , where  $\beta_0 = 0$ . The process has one initial ancestor, whose type is determined by using  $U_0$  in the same fashion. For n = $1, 2, \dots$  and  $i = 1, 2, \dots$ , the *i*th individual of type *n* ever alive in the branching process adopts the life-history given by  $(D_i^{(n)}, \eta_{Gi}^{(n)})$ . Note that, since  $U_0, U_1, \ldots$ are independent and identically distributed, the branching process can be represented as a single-type branching process, in which a typical individual lives until age D and reproduces at ages according to  $\eta^{G}$ , where  $(D, \eta^{G})$  is a

mixture of  $(D^{(1)}, \eta^{(1)}), (D^{(2)}, \eta^{(2)}), \ldots$  with respective mixing probabilities  $\alpha_1, \alpha_2, \ldots$ 

For  $t\geqslant 0$  and  $n=1,2,\ldots$ , let  $Z_{v}^{(n)}(t)$  be the number of households of size n in the epidemic  $E_{v}$  that are infectious at time  $t(v=1,2,\ldots)$  and let  $Z^{(n)}(t)$  be the number of type-n individuals that are alive in the branching process at time t. For  $t\geqslant 0$ , let  $T^{(n)}(t)$  be the total number of type-n individuals born in (0,t] in the branching process  $(n=1,2,\ldots)$  and let  $T(t)=\sum_{n=1}^{\infty}T^{(n)}(t)$ . Also, let  $T^{(n)}(\infty)=\lim_{t\to\infty}T^{(n)}(t)$   $(n=1,2,\ldots)$  and  $T(\infty)=\lim_{t\to\infty}T(t)$ . Again it is assumed that the branching process is non-explosive, i.e. that P(C)=1, where  $C=\{\omega\in\Omega: T(t,\omega)<\infty\quad \forall t<0\}$ .

**Theorem 2.4.** Let  $A = \{\omega \in \Omega : T(\infty, \omega) < \infty\}$  denote the set on which the branching process becomes extinct. Then:

- (a)  $\lim_{\substack{\nu \to \infty \\ \nu = 0}} \sup_{n=1,2,\dots} \sup_{0 \leqslant t < \infty} |Z_{\nu}^{(n)}(t,\omega) Z^{(n)}(t,\omega)| = 0 \text{ for $P$-almost all } \omega \in A;$
- (b) for any  $t_0 > 0$ ,

$$\lim_{{\scriptscriptstyle \nu}\to\infty}\sup_{{\scriptscriptstyle n}=1,2,\dots}\sup_{{\scriptscriptstyle 0}\,\leqslant\,t\,\leqslant\,t_0}|Z^{(n)}_{\scriptscriptstyle \nu}(t,\omega)-Z^{(n)}(t,\omega)|=0\quad \textit{for $P$-almost all $\omega\in A^c$}.$$

**Proof.** Let  $B = \{\omega \in \Omega: U_i(\omega) \neq U_j(\omega) \forall i \neq j\}$ , so P(B) = 1, and let  $D = \bigcap_{i=0}^{\infty} \bigcap_{n=1}^{\infty} \{\omega: U_i(\omega) \neq \beta_n\}$ , so P(D) = 1 since  $D^c$  is a countable union of sets each having probability zero. Fix  $\omega \in A \cap B \cap D$ . Then  $T(\infty, \omega) < \infty$  and, since  $\beta_n^{(v)} \to \beta_n$  (n = 1, 2, ...) as  $v \to \infty$  and  $U_i(\omega) \notin \bigcup_{n=1}^{\infty} \{\beta_n\}$  (i = 0, 1, ..., T  $(\infty, \omega))$ , the types of the  $T(\infty, \omega) + 1$  individuals ever alive in the branching process are the same as those of the corresponding between-household contacts in  $E_v$ , for all sufficiently large v, say for  $v \geq v_0(\omega)$ . Also, let  $n_0(\omega) = \max_{k=0,1,...,T(\infty,\omega)}\chi_k^*(\omega)$ , where for  $k = 0, 1, ..., \chi_k^*$  denotes the type of the kth individual born in the branching process. Let  $\mathscr{E}(\omega) = \min\{|U_i(\omega) \cdots U_j \times (\omega)|: i, j = 0, 1, ..., T(\infty, \omega), i \neq j\}$ , then since  $N^{(v)} \to \infty$  as  $v \to \infty$ , there exists  $v_1(\omega)$  such that  $N^{(v)} \geq n_0(\omega)\mathscr{E}(\omega)^{-1}$  for all  $v \geq v_1(\omega)$ . Thus, as in the proof of Theorem 2.1, for  $v \geq \max(v_0(\omega), v_1(\omega))$ , all the between-household contacts occurring in  $E_v$  (including the initial infective) are with individuals in distinct households and hence necessarily give rise to a newly infected household. This proves Theorem 2.4(a), since  $P(A \cap B \cap D) = P(A)$ . Part (b) is proved similarly.

For v = 1, 2, ..., let  $T_v^{(n)}(\infty)$  be the total number of households of size n that receive infection during  $E_v$  (n = 1, 2, ...) and let  $T_v(\infty) = \sum_{n=1}^{\infty} T_v^{(n)}(\infty)$ . The following theorem is proved analogously to Theorem 2.2, and hence its proof is omitted.

**Theorem 2.5.** For n = 1, 2, ..., let  $\delta_n = 1$  if the initial ancestor in the branching process is of type n and let  $\delta_n = 0$  otherwise. Then,

- (a)  $\lim_{v\to\infty} T_v^{(n)}(\infty) = T^{(n)}(\infty) + \delta_n \ (n=1,2,\ldots)$  almost surely; (b)  $\lim_{v\to\infty} T_v(\infty) = T(\infty) + 1$  almost surely.

For  $n = 1, 2, ..., \text{ let } R^{(n)} = \eta_G^{(n)}((0, \infty))$  denote the total number of betweenhousehold contacts emanating from a typical single-household SIS epidemic, with initially one infective and n-1 susceptibles. Let  $R = \eta_G((0,\infty))$  denote the corresponding quantity for a typical household whose size has distribution given by  $\alpha_n$   $(n=1,2,\ldots)$ . Let  $R_*^{(n)}=E[R^{(n)}]$  and  $f^{(n)}(s)=E[s^{R^{(n)}}]$   $(n=1,2,\ldots)$ . Also let  $R_*=E[R]$  and  $f(s)=E[s^R]$ . Then  $R_*=\sum_{n=1}^{\infty}\alpha_n\,R_*^{(n)}$  and  $f(s)=\sum_{n=1}^{\infty}\alpha_n\,f^{(n)}(s)$ . With this notation, Corollary 2.3 now holds for the epidemic with unequal household sizes. In particular, global epidemics can only occur if  $R_* > 1$ .

# 2.5. Specialisation to model with Poisson contacts and exponential infectious periods

Return to the equal household size setting of Section 2.1 and suppose that the components  $\mathcal{H}^{(k)} = (T_{\rm I}^{(k)}, \eta_{\rm W}^{(k)}, \eta_{\rm B}^{(k)}) \ (k=1,2,\ldots)$  of the infectious career  $\mathcal{H}$ are independent and identically distributed, according to  $(T_{\rm I}, \eta_{\rm W}, \eta_{\rm R})$  say. Suppose also that  $T_1$ ,  $\eta_W$  and  $\eta_B$  are independent, with  $T_I$  following a negative exponential distribution with mean  $\gamma^{-1}$  and  $\eta_{\rm W}$  and  $\eta_{\rm B}$  following homogeneous Poisson processes with rates  $\lambda_W$  and  $\lambda_B$ , respectively. Then, if between-household contacts are ignored, the spread of the epidemic within a single household follows the Markov single population SIS epidemic model, described by Eq. (1) with  $\lambda = \lambda_{\rm W}$ .

Now consider that single population model with population size n, suppress the explicit dependence of  $Y_n(t)$  on n, and suppose that Y(0) = 1. Let  $N^*$  denote the total number of recoveries that occur in  $\{Y(t); t \ge 0\}$ , where a recovery is said to occur each time an infective individual becomes susceptible again, and let  $T_A$  denote the sum of the corresponding  $N^*$  infectious periods. Note that, provided  $\gamma > 0$ , the epidemic eventually becomes extinct almost surely and hence  $N^*$  and  $T_A$  are both finite almost surely. In the household epidemic  $E_{m,n}$ , each infective is making between-household contacts at rate  $\lambda_B$ , so the total number of between-household contacts emanating from a single-household SIS epidemic,  $R = \eta^{G}((0, \infty))$ , follows a Poisson distribution with random mean  $\lambda_B T_A$ . Thus the invasion threshold parameter is given by  $R_* = E[R] = \lambda_B E[T_A]$ . The following theorem gives an explicit expression for  $E[T_A]$ , and hence for  $R_*$ , but first some more notation is introduced.

For k = 0, 1, ..., n, let  $\psi_k(s, \theta) = E[s^{N^*} \exp(-\theta T_A) | Y(0) = k]$   $(0 \le s \le 1;$  $\theta \geqslant 0$ ),  $\mu_k = E[N^*|Y(0) = k]$  and  $\tilde{\mu}_k = E[T_A|Y(0) = k]$ . Note that  $\psi_k(s,\theta)$  is the joint probability generating function and moment generating function of the pair of random variables  $(N^*, T_A)$ . Let  $\psi(s, \theta) = (\psi_1(s, \theta), \psi_2(s, \theta), \dots, \psi_n(s, \theta))^\top$ ,  $\mathbf{\mu} = (\mu_1, \mu_2, \dots, \mu_n)^\top$  and  $\tilde{\mathbf{\mu}} = (\tilde{\mu}_1, \tilde{\mu}_2, \dots, \tilde{\mu}_n)^\top$ . Let  $\beta = \lambda n^{-1}$  and  $\rho = (\tilde{\mu}_1, \tilde{\mu}_2, \dots, \tilde{\mu}_n)^\top$ .

 $\gamma/\beta$ ;  $\rho$  is often referred to as the relative removal-rate. Let A(s) be the  $n \times n$ matrix function given by

$$A(s) = \begin{bmatrix} 0 & \frac{n-1}{n-1+\rho} & 0 & \cdots & \cdots & 0\\ \frac{\rho s}{n-2+\rho} & 0 & \frac{n-2}{n-2+\rho} & 0 & \cdots & 0\\ 0 & \frac{\rho s}{n-3+\rho} & 0 & \frac{n-3}{n-3+\rho} & 0 & 0\\ \vdots & \ddots & \ddots & \ddots & \ddots\\ \vdots & & & \frac{\rho s}{1+\rho} & 0 & \frac{1}{1+\rho}\\ 0 & \cdots & \cdots & 0 & s & 0 \end{bmatrix}.$$

Let  $\mathbf{b}(s) = (\frac{\rho s}{n-1+\rho}, 0, 0, \dots, 0)^{\top}$ , where  $\mathbf{b}(s)$  has dimension  $n \times 1$ ,  $\mathbf{c} = (\frac{\rho}{n-1+\rho}, \frac{\rho}{n-2+\rho}, \dots, \frac{\rho}{1+\rho}, 1)^{\top}$  and  $D(\theta)$  be the  $n \times n$  diagonal matrix function, whose kth diagonal element is  $(n-k+\rho)/(n-k+\rho+\beta^{-1}\theta)$ .

## Theorem 2.6.

(a) 
$$\psi(s, \theta) = (I - D(\theta)A(s))^{-1}D(\theta)\mathbf{b}(s) \quad (0 \le s \le 1; \theta \ge 0);$$
  
(b)  $\mathbf{\mu} = (I - A(1))^{-1}\mathbf{c};$ 

(b) 
$$\mu = (I - A(1))^{-1} \mathbf{c}$$
;

(c) 
$$\tilde{\boldsymbol{\mu}} = \gamma^{-1} \boldsymbol{\mu};$$
  
(d)  $\mu_1 = \frac{(n-1)!}{\rho^{n-1}} \left( 1 + \rho + \frac{\rho^2}{2!} + \dots + \frac{\rho^{n-1}}{(n-1)!} \right).$ 

**Proof.** Suppose that Y(0) = k, where  $1 \le k \le n$ , and let  $T = \inf\{t > 0 : Y(t) \ne k\}$ be the time of the first jump of  $\{Y(t); t \ge 0\}$ . Conditioning on (T, Y(T)), noting that this initial sojourn contributes kT to  $T_A$  and the first jump increments  $N^*$  by 1 if Y(T) = k-1, and using the Markov property of  $\{Y(t); t \ge 0\}$  yields

$$\begin{split} \psi_k(s,\theta) &= \int\limits_0^\infty (\beta k(n-k) + \gamma k) \, \exp\{-(\beta k(n-k) + \gamma k)u\} \, \exp\{-ku\theta\} \\ &\times \left\{ \frac{\beta k(n-k)}{\beta k(n-k) + \gamma k} \psi_{k+1}(s,\theta) + \frac{\gamma ks}{\beta k(n-k) + \gamma k} \psi_{k-1}(s,\theta) \right\} \, \mathrm{d}u \\ &= \frac{n-k+\rho}{n-k+\rho+\beta^{-1}\theta} \left\{ \frac{n-k}{n-k+\rho} \psi_{k+1}(s,\theta) + \frac{\rho s}{n-k+\rho} \psi_{k-1}(s,\theta) \right\} \end{split} \tag{4}$$

with the convention that  $\psi_{n+1}(s,\theta) = 0$ . Clearly,  $\psi_0(s,\theta) = 1$ , so Eq. (4) can be expressed in matrix-vector form as

$$\psi(s,\theta) = D(\theta)(A(s)\psi(s,\theta) + \mathbf{b}(s)), \tag{5}$$

with solution

$$\psi(s,\theta) = (I - D(\theta)A(s))^{-1}D(\theta)\mathbf{b}(s) \quad (0 \leqslant s \leqslant 1; \theta > 0),$$

proving part (a). (Note that the non-singularity of  $I - D(\theta)A(s)$  follows using Lemma B1 of Seneta [26], since it is easily shown that  $\{D(\theta)A(s)\}^k \to 0$  elementwise as  $k \to \infty$ .) To prove (b), let  $\partial_1 \psi(s, \theta)$  denote the elementwise partial derivative of  $\psi$  with respect to s, evaluated at  $(s, \theta)$ . Differentiating (5) partially with respect to s yields

$$(I - D(\theta)A(s))\partial_1 \psi(s,\theta) - D(\theta)A'(s)\psi(s,\theta) = D(\theta)\mathbf{b}'(s),$$

where A'(s) denotes the first derivative of A(s) etc. Hence,

$$\mu = \partial_1 \psi(1,0) = (I - A(1))^{-1} (A'(1)\mathbf{1} + \mathbf{b}(1)),$$

where **1** denotes the  $n \times 1$  column vector of ones, since D(0) = I,  $\mathbf{b}'(1) = \mathbf{b}(1)$  and  $\psi(1,0) = \mathbf{1}$ . It is easily verified that  $A'(1)\mathbf{1} + \mathbf{b}(1) = \mathbf{c}$ , so part (b) follows.

Now let  $\partial_2 \psi(s, \theta)$  denote the elementwise partial derivative of  $\psi$  with respect to  $\theta$ , evaluated at  $(s, \theta)$ . Then differentiating (5) partially with respect to  $\theta$  yields

$$(I - D(\theta)A(s))\partial_2 \psi(s,\theta) - D'(\theta)A(s)\psi(s,\theta) = D'(\theta)\mathbf{b}(s).$$

Hence,

$$\tilde{\mathbf{\mu}} = -\partial_2 \psi(1,0) = -(I - A(1))^{-1} D'(0) \ (A(1)\mathbf{1} + \mathbf{b}(1))$$
$$= -(I - A(1))^{-1} D'(0)\mathbf{1}.$$

A simple calculation shows that  $D'(0)\mathbf{1} = -\gamma^{-1}\mathbf{c}$  and hence part (c) follows. Note that part (c) is a Wald's identity for the SIS epidemic, cf. Corollary 2.2 of Ref. [3] which proves a similar result for a stochastic SIR (susceptible  $\rightarrow$  infected  $\rightarrow$  removed) epidemic model.

To prove part (d) note first that conditioning on Y(T), similarly as in the derivation of Eq. (4), yields

$$\mu_k = \frac{n-k}{n-k+\rho}\mu_{k+1} + \frac{\rho}{n-k+\rho}(1+\mu_{k-1}) \quad (k=1,2,\ldots,n).$$

with  $\mu_{k-1} = 0$ .

Thus

$$(n-k+\rho)\mu_k = (n-k)\mu_{k+1} + \rho\mu_{k-1} + \rho \quad (k=1,2,\ldots,n)$$
(6)

and clearly  $\mu_0=0$ . Let  $\varphi_k=\mu_k-\mu_{k-1}$   $(k=1,2,\ldots,n+1)$ . Then it follows from Eq. (6) that

$$\varphi_k = 1 + \frac{n-k}{\rho} \varphi_{k+1} \quad (k = 1, 2, \dots, n),$$
(7)

so, in particular,  $\varphi_n = 1$ . Now let  $\zeta_k = \varphi_{n-k}$  (k = 0, 1, ..., n - 1), so  $\zeta_0 = 1$  and, from Eq. (7),

$$\zeta_k = 1 + \frac{k}{\rho} \zeta_{k-1} \quad (k = 1, 2, \dots, n-1).$$

Using induction on k then shows that

$$\zeta_k = \frac{k!}{\rho^k} \left( 1 + \rho + \frac{\rho^2}{2!} + \dots + \frac{\rho^k}{k!} \right) \quad (k = 0, 1, \dots, n - 1).$$

Hence, since  $\mu_0 = 0$ , for  $k = 1, 2, \dots, n$ ,

$$\mu_k = \sum_{i=1}^k (\mu_i - \mu_{i-1}) = \sum_{i=1}^k \varphi_i = \sum_{i=1}^k \zeta_{n-i} = \sum_{i=n-k}^{n-1} \zeta_i,$$

and, in particular,  $\mu_1 = \zeta_{n-1}$ , proving part (d).

We return now to the setting at the start of this section, i.e. to a household SIS epidemic model, with constant household size n, in which, at each infection, an infectious individual makes within-household (between-household) contacts at the points of a Poisson process with rate  $\lambda_{\rm W}(\lambda_{\rm B})$  throughout an infectious period which follows a negative exponential distribution with mean  $\gamma^{-1}$ . Thus, in the notation of Theorem 2.6,  $\rho = n\gamma/\lambda_{\rm W}$ . By linearly rescaling the time axis, assume without loss of generality that  $\gamma = 1$ . It then follows from Theorem 2.6 and the discussion preceding it that

$$R_* = \lambda_{\rm B} \frac{(n-1)!}{\rho^{n-1}} \sum_{k=0}^{n-1} \rho^k / k! \quad \text{with } \rho = n/\lambda_{\rm W}.$$
 (8)

Fig. 1. shows graphs of critical values of  $(\lambda_W, \lambda_B)$  so that  $R_* = 1$  for various household sizes n, together with the corresponding graph for  $R_0 = 1$  for the equivalent standard deterministic model (cf. Eq. (3)), for which  $R_0 = (\lambda_W + \lambda_B)/\gamma$ . (Equivalent graphs for the corresponding SIR model are also shown and these will be discussed later.) Note that, unless n is large,  $R_0$  is not a good indicator as to whether the household SIS epidemic can invade a susceptible population, for the reasons outlined in Section 1. The graph for the case n = 1 is constant since, if the households are all of size 1 there can be no spread of infection within a household, so the value of  $\lambda_W$  is irrelevant. Note also that if  $\lambda_W \geqslant 1$  then, provided  $\lambda_B > 0$ , global epidemics can always occur provided that the household size n is sufficiently large, whilst if  $\lambda_W < 1$  they can only occur for sufficiently large n if  $\lambda_B > 1 - \lambda_W$ . Thus there is a phase transition as  $\lambda_W$  passes through its single household threshold of one.

As a practical matter, the formula (8) for  $R_*$  would be difficult to compute for large household size n, so an asymptotic approximation to it is useful. The following such approximation follows directly from Nåsell [24] p. 909, on noting that  $R_*$  is related to the quantity  $S^{(1)}$  of Ref. [24] by  $R_* = \lambda_{\rm B} S^{(1)}$ , with  $T = \lambda_{\rm W}$  and N = n. As  $n \to \infty$ ,

$$R_* \sim \frac{\lambda_{\rm B}}{\lambda_{\rm W}} \sqrt{n} \frac{\Phi(r\sqrt{n})}{\phi(r\sqrt{n})},$$
 (9)

where  $\phi(x) = \exp(-x^2/2)/\sqrt{2\pi}$  and  $\Phi(x) = \int_{-\infty}^{x} \phi(t) dt$  denote the standard normal density and distribution functions, respectively, and

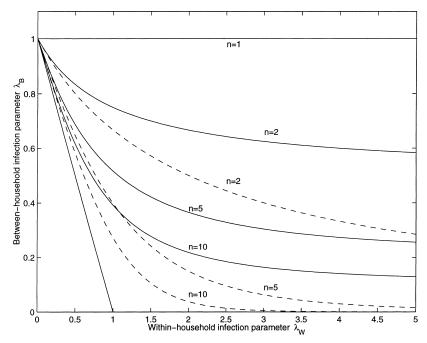


Fig. 1. Critical values of  $(\lambda_W, \lambda_B)$  so that  $R_* = 1$  for SIS (solid line) and SIR (dashed line) epidemics.

$$r = \begin{cases} r_1 = \operatorname{sign}(\lambda_{\mathrm{W}} - 1) \sqrt{2(\log \lambda_{\mathrm{W}} - 1 + \lambda_{\mathrm{W}}^{-1})} & \text{if } \lambda_{\mathrm{W}} \geqslant 1, \\ r_2 = 1 - \lambda_{\mathrm{W}}^{-1} & \text{if } \lambda_{\mathrm{W}} \leqslant 1. \end{cases}$$

Moreover, the asymptotic approximation (9) is uniform in  $\lambda_{\rm W}$ .

Eq. (9) can be used to give a formal description of the above-mentioned phase transition, which also reflects the threshold behaviour of the single population SIS epidemic since  $R_* = \lambda_{\rm B} E[T_{\rm A}] = \lambda_{\rm B} \gamma^{-1} E[N^*]$ . If  $\lambda_{\rm W} < 1$  then Eq. (9), together with  $\Phi(x)/\phi(x) \sim -x^{-1}$  as  $x \to \infty$ , yields that  $R_* \sim \lambda_{\rm B} (1-\lambda_{\rm W})^{-1}$  as  $n \to \infty$ , whereas if  $\lambda_{\rm W} > 1$  then it follows from Eq. (9) using Stirling's formula that  $R_* \sim \lambda_{\rm B} \lambda_{\rm W}^{n-1} \exp(n \lambda_{\rm W}^{-1}) n!/n^n$ . The passage through the critical value  $\lambda_{\rm W} = 1$  can be described by setting  $\lambda_{\rm W} = 1 + x/\sqrt{n}$  and then Eq. (9) shows that  $R_* \sim \lambda_{\rm B} \sqrt{n} \Phi(x)/\phi(x)$  as  $n \to \infty$ , where  $x = {\rm O}(1)$  as  $n \to \infty$ .

It is instructive to compare the threshold  $R_*=1$  of the household SIS epidemic model with that of the corresponding SIR epidemic model, obtained by setting  $\eta_W^{(k)}=\eta_B^{(k)}=\emptyset$  ( $k=2,3,\ldots$ ), which is discussed in e.g. Ref. [6]. Thus an individual can only be infected once and after its infectious period it plays no further role in the epidemic. Suppose that the households are all of size n and that the components  $(T_I^{(1)},\eta_W^{(1)},\eta_B^{(1)})$  of  $\mathscr{H}^{(1)}$  are independent, with  $\eta_W^{(1)}$  and  $\eta_B^{(1)}$  following homogeneous Poisson processes with rates  $\lambda_W$  and  $\lambda_B$ , respectively,

and  $T_{\rm I}^{(1)}$  following an arbitrary but specified distribution having moment generating function  $\phi_{T_{\rm I}}(\theta)=E[\exp(-\theta T_{\rm I})]$   $(\theta\geqslant 0)$ . Then  $R_*=\lambda_{\rm B}E[T_{\rm A}]=\lambda_{\rm B}E[T_{\rm I}]E[N^*]$ , as before, but note that now  $N^*$  is the total number of removals in a single population SIR epidemic and thus takes values in  $0,1,\ldots,n$ . See Ref. [4] for details, where it is shown that

$$E[N^*] = n - 1 - \sum_{k=0}^{n-1} {n-1 \choose k} \alpha_k \phi_{T_1}(k\lambda_{\mathbf{W}}/n),$$

where  $\alpha_0, \alpha_1, \dots, \alpha_{n-1}$  are determined recursively by

$$\sum_{i=0}^k \binom{k}{i} \alpha_i \phi_{T_1}(i\lambda_{\mathbf{W}}/n) = k \quad (k=0,1,\ldots,n-1).$$

Fig. 1 also shows graphs of critical values of  $(\lambda_W, \lambda_B)$  so that  $R_* = 1$  for various household sizes n, for SIR epidemics, when  $T_{\rm I}$  follows a negative exponential distribution with mean 1 (so  $\phi_{T_1}(\theta) = 1/(1+\theta)$  ( $\theta \ge 0$ )). When n=1, the graphs of  $R_*=1$  for the SIS and SIR epidemics are the same. This is because when the households are of size 1, there is no internal spread of infection within a household, so the approximating branching process is the same for both models. When n > 1, the graph of  $R_* = 1$  for the SIS epidemic lies below that of the SIR epidemic. This is because individuals can be infected more than once in the SIS epidemic, and consequently  $T_A$  will be stochastically larger in the SIS epidemic than in the SIR epidemic. As the household size  $n \to \infty$ , the graphs of  $R_* = 1$  for the SIS and SIR epidemics converge to the same (deterministic) limit. This is because for large n, the process of infected individuals in the early stages of either the SIS or SIR epidemic may be approximated by a multitype branching process, with type corresponding to household. The approximating branching process assumes that every infectious contact is with a susceptible individual, so it will be the same for both the SIS and SIR epidemics, although the branching process will provide a closer approximation to the SIS epidemic than to the SIR one. Indeed, it is readily seen that the SIS epidemic is sandwiched between the SIR epidemic and the branching process, since, unlike in the SIR epidemic, if the same individual is contacted more than once in the SIS epidemic there is always the possibility that it has become susceptible again.

It is also interesting to compare the asymptotic behaviour of the graphs as  $\lambda_{\rm W} \to \infty$ . Let  $\lambda_{\rm B}^{\rm CRIT}(\lambda_{\rm W})$  denote the critical value of  $\lambda_{\rm B}$  as a function of the  $\lambda_{\rm W}$ . In the SIR model, letting  $\lambda_{\rm W} \to \infty$  yields the model for highly infectious diseases studied by Becker and Dietz [9], in which if one individual in a household is infected then the whole household becomes infected. Thus  $E[N^*] = n$ , so  $R_* = \lambda_{\rm G} n E[T_{\rm I}]$  and hence  $\lambda_{\rm B}^{\rm CRIT}(\lambda_{\rm W}) \sim 1/(n E[T_{\rm I}])$  as  $\lambda_{\rm W} \to \infty$ . In the SIS model, as  $\lambda_{\rm W} \to \infty$ , as soon as an individual is removed in the single population epidemic it becomes infected again. Thus  $E[N^*] \to \infty$  as  $\lambda_{\rm W} \to \infty$  so  $\lambda_{\rm B}^{\rm CRIT}(\lambda_{\rm W}) \to 0$  as

 $\lambda_W \to \infty$ . Moreover, it follows easily from Eq. (8) that  $\log \lambda_B^{CRIT}(\lambda_W) \sim -\log E[T_I] - (n-1)\log n - \log((n-1)!) - (n-1)\log \lambda_W$  as  $\lambda_W \to \infty$ . The behaviour of  $\lambda_B^{CRIT}(\lambda_W)$  for the two models is illustrated in Fig. 2, which is Fig. 1 for  $\lambda_W > 1$ , drawn on a log-log-scale. Note that when  $\lambda_W = 10$ , which is not atypical for diseases like measles and influenza etc, for households of size 5  $\lambda_B^{CRIT}$  is several orders of magnitude smaller for the SIS model than for the SIR model.

#### 3. Persistence threshold

## 3.1. Stochastic model and its deterministic approximation

The stochastic household SIS epidemic described in Section 2.1 ultimately goes extinct with probability 1. However, for certain values of its parameters the extinction time can be extremely large. In such circumstances the persistence behaviour of the model when the number of households is large can be studied by using a deterministic approximation. In order for the deterministic model to provide a good approximation to the more realistic stochastic model it is necessary that the sizes of all the quantities described by the deterministic model be large, so that probabilistic effects in some sense average out. The classical approach to a deterministic model for the household setting would be to derive a system of differential equations governing the number of infectives in each household, but, as indicated in the introduction, such an approach is inappropriate as the households are typically small. Suppose instead we were to derive a system of differential equations governing the number of households with k infectives  $(k = 0, 1, \dots, n)$  then, if the number of households in the population is large and the epidemic has taken off, all of these quantities will be large and hence the deterministic model is likely to provide a good approximation. Note that this view of the model is not as detailed as before, since it no longer describes the evolution of the epidemic at the individual household level, although it is clearly sufficient to analyse the endemic behaviour of the model. The approximation of the stochastic model by a deterministic one can be made precise by considering a sequence of stochastic models indexed by the number of households in the population. That approach is used here to formulate the deterministic model, although it can be written down directly by considering the effects of various within- and between- household infections and removals on the number of households containing a given number of infectives.

Consider the Markov household SIS model described at the start of Section 2.5 and suppose that the population comprises m households each of size n. For  $t \ge 0$  and  $k = 0, 1, \ldots, n$ , let  $X_k^{(m)}(t)$  denote the number of households that contain exactly k infectives at time t, and let  $\mathbf{X}^{(m)}(t) = (X_0^{(m)}(t), X_1^{(m)}(t), \ldots, X_n^{(m)}(t))^{\top}$ . Then  $\{\mathbf{X}^{(m)}(t); t \ge 0\}$  is a continuous time Markov chain,

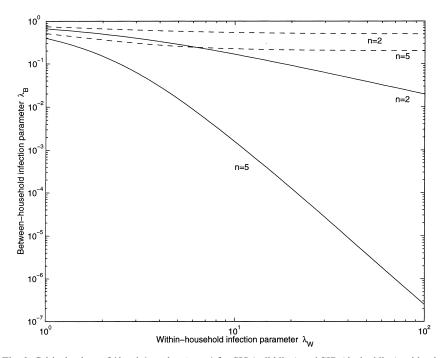


Fig. 2. Critical values of  $(\lambda_W, \lambda_B)$  so that  $R_* = 1$  for SIS (solid line) and SIR (dashed line) epidemics.

with state space  $\Delta_n^{(m)} = \{\mathbf{i} = (i_0, i_1, \dots, i_n)^\top \in \mathbb{Z}^{n+1} : i_k \ge 0 \ (k = 0, 1, \dots, n), \sum_{k=0}^n i_k = m\}$ . Define the following operators on the elements of  $\Delta_n^{(m)}$ : for  $k = 0, 1, \dots, n-1$ , the infection operator  $I_k$  by

$$I_k(\mathbf{i}) = (i_0, i_1, \dots, i_{k-1}, i_k - 1, i_{k+1} + 1, i_{k+2}, \dots, i_n)^{\mathsf{T}}$$

and for k = 1, 2, ..., n, the removal operator  $R_k$  by

$$R_k(\mathbf{i}) = (i_0, i_1, \dots, i_{k-1} + 1, i_k - 1, i_{k+1}, \dots, i_n)^{\top}.$$

Thus  $I_k$  and  $R_k$  correspond respectively to an infection and a removal in a household that previously contained k infectives. The transition probabilities of  $\{\mathbf{X}^{(m)}(t); t \geq 0\}$  are given by

$$P(\mathbf{X}^{(m)}(t + \Delta t) = \mathbf{j} | \mathbf{X}^{(m)}(t) = \mathbf{i})$$

$$= \begin{cases} \left(\frac{\lambda_{\mathbf{W}}}{n} k(n - k) i_k + \frac{\lambda_{\mathbf{B}}}{nm} \sum_{l=1}^{n} (n - k) l i_l i_k\right) \Delta t + o(\Delta t) & \text{if } \mathbf{j} = I_k(\mathbf{i}), \\ \gamma k i_k \Delta t + o(\Delta t) & \text{if } \mathbf{j} = R_k(\mathbf{i}), \\ o(\Delta t) & \text{otherwise.} \end{cases}$$

Suppose that  $\mathbf{X}^{(m)}(0) = m\mathbf{x}_m(0)$ , where  $\mathbf{x}_m(0) \to \mathbf{x}(0) \in \Delta_n \setminus \{\mathbf{0}\}$  as  $m \to \infty$  and  $\Delta_n$  is the simplex  $\{(x_0, x_1, \dots, x_n)^\top \in \mathbb{R}^{n+1} : x_k \ge 0 \ (k = 0, 1, \dots, n), \sum_{k=0}^n x_k = 1\}$ . Then the results of Kurtz [19] show that, as number of households  $m \to \infty$ ,  $\{m^{-1}\mathbf{X}^{(m)}(t); t \ge 0\}$  converges to the deterministic limit  $\mathbf{x}(t)$   $(0 \le t < \infty)$ , described by the system of differential equations

$$\frac{\mathrm{d}x_k}{\mathrm{d}t} = f_k(x_0, x_1, \dots, x_n) \quad (k = 0, 1, 2, \dots, n), \tag{10}$$

where

$$f_0(x_0, x_1, \dots, x_n) = -\frac{\lambda_B}{n} x_0 \sum_{l=1}^n l x_1 + \gamma x_1,$$

$$f_k(x_0, x_1, \dots, x_n) = -\frac{\lambda_W}{n} k(n-k) x_k - \frac{\lambda_B}{n} (n-k) x_k \sum_{l=1}^n l x_l - \gamma k x_k$$

$$+ \frac{\lambda_W}{n} (k-1)(n-k+1) x_{k-1}$$

$$+ \frac{\lambda_B}{n} (n-k+1) x_{k-1} \sum_{l=1}^n l x_l$$

$$+ \gamma (k+1) x_{k+1} \quad (k=1, 2, \dots, n-1),$$

$$f_n(x_0, x_1, \dots, x_n) = \frac{\lambda_W}{n} (n-1) x_{n-1} + \frac{\lambda_B}{n} x_{n-1} \sum_{l=1}^n l x_l - n \gamma x_n.$$

As made clear above, the deterministic model (10) should be viewed as an approximation to its stochastic cousin. Note from the associated limit theorem that the approximation requires the initial proportion of infectives to be strictly positive, so it cannot be used to describe the spread of infection from a few initial cases. In that situation the branching process approximation of Section 2 can be used for the early stages of the epidemic and, if the epidemic takes-off, the deterministic approximation will take over after a random time which can be related to the asymptotic behaviour of the branching process, cf. [22,1], although the piecing together of these two epidemic approximations is still awaiting a fully rigorous mathematical description.

## 3.2. Households of size 2

Suppose that n = 2. Let  $\beta_W = \lambda_W/2$ ,  $\beta_B = \lambda_B/2$  and assume, without loss of generality, that  $\gamma = 1$ . Note that  $\sum_{k=0}^{2} x_k = 1$ . Thus, the system of differential equations (10) is now completely specified by

$$\frac{dx_1}{dt} = f(x_1, x_2), \quad \frac{dx_2}{dt} = g(x_1, x_2),$$
 (11)

where

$$\begin{split} f(x_1, x_2) &= -\beta_{\mathbf{W}} x_1 - \beta_{\mathbf{B}} x_1 (x_1 + 2x_2) - x_1 + 2\beta_{\mathbf{B}} (1 - x_1 - x_2) (x_1 + 2x_2) \\ &+ 2x_2, g(x_1, x_2) \\ &= \beta_{\mathbf{W}} x_1 + \beta_{\mathbf{B}} x_1 (x_1 + 2x_2) - 2x_2. \end{split}$$

Let  $\Delta = \{(x_1, x_2)^{\top} \in \mathbb{R}^2 : x_1 \ge 0, x_2 \ge 0, x_1 + x_2 \le 1\}$ . It follows from Eq. (8) that, for the present model,  $R_* = 2\beta_B(1 + \beta_W)$ .

**Theorem 3.1.** If  $R_* \leq 1$  then the origin  $\mathbf{0}$  is the only equilibrium point of the system of differential equation (11) in  $\Delta$ , and  $\Delta$  is an ASR (asymptotic stability region) for this equilibrium point. If  $R_* > 1$  then there is a second equilibrium point  $\mathbf{x}^* = (x_1^*, x_2^*)^\top$ , where  $x_1^*$  is the smallest root of the quadratic

$$\beta_{\rm B}^2 \beta_{\rm W} x^2 - \beta_{\rm B} (2\beta_{\rm B} + 3\beta_{\rm W} + 2\beta_{\rm B} \beta_{\rm W} + \beta_{\rm W}^2) x + 2\beta_{\rm B} (1 + \beta_{\rm W}) - 1 = 0 \tag{12}$$

and

$$x_2^* = \frac{x_1^*(\beta_W + \beta_B x_1^*)}{2(1 - \beta_B x_1^*)}. (13)$$

Moreover,  $\Delta \setminus \{0\}$  is an ASR for this equilibrium point.

**Proof.** We determine first the existence of the equilibrium points in  $\Delta$ . Now  $\mathbf{x} = (x_1, x_2)^{\top}$  is an equilibrium point of Eq. (11) if and only if  $f(\mathbf{x}) = g(\mathbf{x}) = 0$ . Clearly  $f(\mathbf{0}) = g(\mathbf{0}) = 0$ , so  $\mathbf{0}$  is an equilibrium point. Let  $\mathbf{x} \in \Delta \setminus \{\mathbf{0}\}$  be an equilibrium point. Then  $g(\mathbf{x}) = 0$  implies that

$$x_2 = \frac{x_1(\beta_W + \beta_B x_1)}{2(1 - \beta_B x_1)}. (14)$$

Thus  $x_1 = 0$  implies that  $x_2 = 0$ , so we may assume that  $x_1 > 0$ . Clearly, for  $x_1 > 0, x_2 > 0$  if and only if  $x_1 < \beta_B^{-1}$ . We now show that  $x_1 < \beta_B^{-1}$  ensures that  $\mathbf{x} \in \Delta$ . Suppose that  $x_1 < \beta_B^{-1}$ . Then using Eq. (14),  $x_1 + x_2 \le 1$  if and only if  $(x_1 - \alpha)^2 + 2 - \alpha^2 \ge 0$ , where  $\alpha = 1 + \beta_B^{-1}(1 + \frac{1}{2}\beta_W)$ . Clearly,  $(x_1 - \alpha)^2 + 2 - \alpha^2 \ge 0$  if  $\alpha \le \sqrt{2}$ , whilst if  $\alpha > \sqrt{2}$  we require  $x_1 \ge \alpha + \sqrt{2 - \alpha^2}$  or  $x_1 \le \alpha - \sqrt{2 - \alpha^2}$ . A little algebra shows that  $\alpha - \sqrt{2 - \alpha^2} > \beta_B^{-1}$ . Thus  $0 < x_1 < \beta_B^{-1}$  implies that  $x_2 > 0$  and  $x_1 + x_2 \le 1$  and hence is a necessary and sufficient condition for  $\mathbf{x} \in \Delta \setminus \{0\}$ .

Substituting Eq. (14) into  $f(x_1, x_2) - g(x_1, x_2) = 0$  shows that  $x_1$  satisfies the quadratic equation (12), which may be written as  $h(x) = ax^2 + bx + c = 0$ . Suppose that c = 0. Then, since  $x_1 > 0, x_1 = -b/a = (2\beta_B + 3\beta_W + 2\beta_B\beta_W + \beta_W^2)/(\beta_B\beta_W) > 3\beta_B^{-1} > \beta_B^{-1}$ , so  $\mathbf{x} \notin \Delta \setminus \{\mathbf{0}\}$ . Suppose that  $c \neq 0$ . Then it is easily shown that h(x) = 0 has two real roots. Now h(x) has a minimum at  $x = -b/2a = (2\beta_B + 3\beta_W + 2\beta_B\beta_W + \beta_W^2)/(2\beta_B\beta_W) > 1$ . hence only the small-

est root of h(x) = 0 can lead to  $\mathbf{x} \in \Delta \setminus \{0\}$ . Let  $x_1^*$  be the smallest root of h(x) = 0. Then  $x_1^* > 0$  if and only if c > 0 and a little algebra shows that if c > 0 then  $x_1^* < \beta_B^{-1}$ . Thus the system (11) has two equilibrium points if and only if c > 0 (i.e. if and only if  $R_* > 1$ ) and if  $R_* > 1$  the non-zero equilibrium point is located as stated in Theorem 3.1.

We next examine the local asymptotic stability of the equilibrium point at the origin. Linearising the system (11) about the origin leads to

$$\frac{dx_1}{dt} = ax_1 + bx_2, \quad \frac{dx_2}{dt} = cx_1 + dx_2, \tag{15}$$

where  $a=2\beta_{\rm B}-1-\beta_{\rm W},\ b=2(1+2\beta_{\rm B}),\ c=\beta_{\rm W}$  and d=-2. Let  $p=a+b,\ q=ad-bc$  and  $\delta=p^2-4q$ . Then  $q=2(1-2\beta_{\rm B}(1+\beta_{\rm W}))$  and  $p=2\beta_{\rm B}-3-\beta_{\rm W}$ . Further,  $\delta>0$  and if q>0 then p<0. It follows, see e.g. Ref. [18] p. 173, that the equilibrium point at the origin is a saddle point if  $R_*>1$  and a stable node if  $R_*<1$ .

We turn now to the local asymptotic stability of the endemic equilibrium point  $\mathbf{x}^*$ , when it exists. Writing  $\mathbf{y} = \mathbf{x} - \mathbf{x}^*$  and linearising leads to the system (15), with  $\mathbf{x}$  replaced by  $\mathbf{y}$  and  $a = 2\beta_{\rm B} - 1 - \beta_{\rm W} - 2\beta_{\rm B}(3x_1^* + 4x_2^*), b = 2(1 + 2\beta_{\rm B} - 4(x_1^* + x_2^*)), c = \beta_{\rm W} + 2\beta_{\rm B}(x_1^* + x_2^*)$  and  $d = -2(1 - \beta_{\rm B}x_1^*)$ . Lengthy algebra then shows that, in the above notation,  $p = -2\beta_{\rm B} - 2\sqrt{(\beta_{\rm W} + 2\beta_{\rm B})^2 + 4\beta_{\rm W}}$  and  $\theta = (1 + \beta_{\rm W} - 2\beta_{\rm G})^2 + 4\beta_{\rm W} - (\beta_{\rm W} + 2\beta_{\rm B})^2 + 4\beta_{\rm W}$ . Thus  $\delta > 0$  and if  $R_* > 1$  then q > 0 and p < 0, so  $\mathbf{x}^*$  is a stable node if  $R_* > 1$ .

Next, we consider global asymptotic stability of the equilibrium points. First note that no solution paths of Eq. (11) leave  $\Delta$ . If  $R_* < 1$  then the origin is the only equilibrium point of Eq. (11) in  $\Delta$  and hence the system (11) has no closed path solution in the interior of  $\Delta$ . Further, the origin is an attractor and hence  $\Delta$  is an ASR for the origin. If  $R_* > 1$  then the origin is a saddle point and it is easily shown that the repulsive direction is into  $\Delta$  and the attractive direction is not in  $\Delta$ . Also,  $\mathbf{x}^*$  is an attractor. Thus, provided the system (11) has no closed path solution in the interior of  $\Delta$ ,  $\Delta \setminus \{\mathbf{0}\}$  is an ASR for the equilibrium point  $\mathbf{x}^*$ . To rule out the possibility of closed path solutions, let  $b(x_1, x_2) = x_1^{-1}$ . Then

$$\frac{\partial}{\partial x_1}(fb) + \frac{\partial}{\partial x_2}(gb) = -(4\beta_B x_2(1-x_2) + 2x_2 + 2x_1 + \beta_G x_1^2)/x_1^2,$$

which is strictly negative throughout the interior of  $\Delta$ . Hence, by Theorem 3.1 of Ref. [16], Eq. (11) has no closed path solution in the interior  $\Delta$ , as required.

Finally, we consider the case  $R_* = 1$ , when linearisation does not resolve the stability of the equilibrium point at the origin. This case can be resolved by considering the trajectories of  $(x,y)^{\top}$ , where  $x = x_1 + x_2$  and  $y = x_2$ , in the phase space  $\Delta^* = \{(x,y)^{\top} \in [0,1]^2 : x \ge y\}$ . The set of points for which dx/dt = 0 and dy/dt = 0 can be described by increasing functions  $y = h_1(x)$  and

 $y = h_2(x)$ , respectively, satisfying  $h_1(0) = h_2(0) = 0$  and  $h_1(x) > h_2(x)$  for  $0 < x \le 1$ . Let  $S_1 = \{(x,y) \in \Delta^* : 0 \le y < h_2(x)\}$ ,  $S_2 = \{(x,y) \in \Delta^* : h_2(x) < y < h_1(x)\}$  and  $S_3 = \{(x,y) \in \Delta^* : y > h_1(x)\}$ . Then dx/dt < 0 and dy/dt > 0 on  $S_1$ , dx/dt < 0 and dy/dt < 0 on  $S_2$ , and dx/dt > 0 and dy/dt < 0 on  $S_3$ , from which it follows that  $\Delta$  is an ASR for the equilibrium point at the origin.

Note that if  $\beta_{\rm W}=0$  the model reduces to the standard homogeneously mixing single population deterministic SIS epidemic model (2), with  $\lambda=2\beta_{\rm B}$  and  $\gamma=1$ . Thus  $R_*=2\beta_{\rm B}=R_0$  and from Section 1, if  $R_*>1$  a proportion  $1-R_0^{-1}=(2\beta_{\rm B}-1)/2\beta_{\rm B}$  of individuals are infected at the endemic equilibrium. It follows that the endemic proportions of households with 0, 1 and 2 infectives are  $1/4\beta_{\rm B}^2$ ,  $(2\beta_{\rm B}-1)/4\beta_{\rm B}^2$  and  $(2\beta_{\rm B}-1)^2/4\beta_{\rm B}^2$ , respectively, in agreement with Eqs. (12) and (13). (Note that Eq. (12) is now reduced to a linear equation.)

Fig. 3 shows for various values of  $R_* > 1$ , graphs of how the endemic equilibrium  $\mathbf{x}^*$  varies as  $\beta_W$  ranges over  $[0,\infty)$ , with  $\mathbf{x}^*$  for  $\beta_W = 0$  being marked by an asterisk. Note that for fixed  $R_*$ , neither  $x_1^*$  or  $x_2^*$  is necessarily monotonic in  $\beta_W$ . Note also that  $x_1^* \to 0$  as  $\beta_W \to \infty$ , as expected. Fig. 4 shows for various values of  $R_* > 1$ , graphs of how the proportion  $1/2(x_1^* + 2x_2^*)$  of

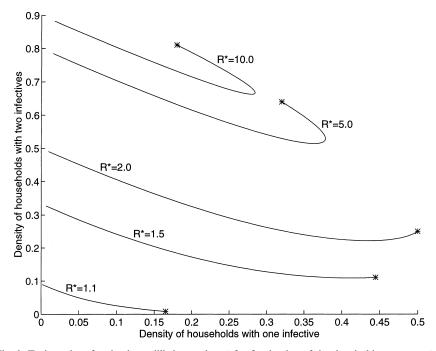


Fig. 3. Trajectories of endemic equilibrium point  $\mathbf{x}^*$  for fixed value of the threshold parameter  $R_*$ , as the within group infection rate  $\beta_W$  ranges over  $[0, \infty)$ .

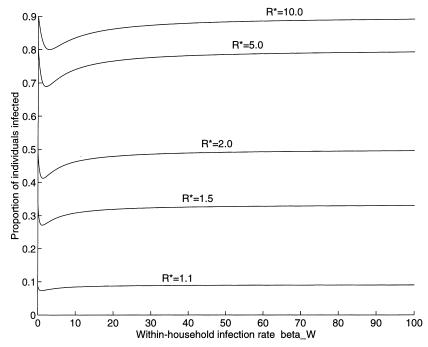


Fig. 4. Graphs for fixed values of  $R_*$  of how the endemic proportion of individuals infected varies with  $\beta_W$ .

individuals infected at the endemic equilibrium varies with  $\beta_W$ . Again, this is not monotonic in  $\beta_W$ . Figs. 5 and 6 show typical phase curves, obtained using MATLAB, for the system (11) when  $R_* < 1$  and  $R_* > 1$ , respectively.

## 3.3. Households of size $\geqslant 3$

It is not straightforward to extend the results of the previous subsection to the case  $n \ge 3$ . Let  $\mathbf{x} = (x_1, x_2, \dots, x_n)^{\top}$ . Then it is easily seen that the origin is an equilibrium point for the system of differential equations which govern  $\mathbf{x}(t) (0 \le t < \infty)$ . Moreover, linearising that system about the origin leads to the deterministic version of the approximating branching process for the Markov SIS household epidemic model of Section 2.5. The evolution of that branching process can be described in terms of the numbers of households containing k infectives  $(k = 1, 2, \dots, n)$ . It is then a linear Markov process, so its deterministic version describes the mean number of households containing k infectives  $(k = 1, 2, \dots, n)$ . It then follows from branching process theory that these numbers either grow or decay exponentially, according to whether  $R_* > 1$  or  $R_* < 1$ . Thus, in the deterministic household SIS epidemic model, the

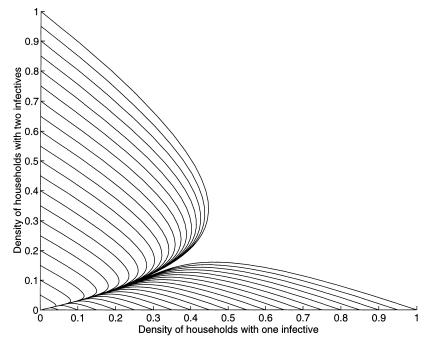


Fig. 5. Typical phase curves for the system (11) when  $\beta_W = 0.5$  and  $\beta_B = 0.25$ .

equilibrium point at the origin is stable if  $R_* < 1$  and unstable if  $R_* > 1$ . This argument can be extended to the case of unequal household sizes.

It does not seem straightforward to show that the origin is the only equilibrium point if  $R_* \leq 1$  and that there is precisely one other equilibrium point if  $R_* > 1$ . Also, the methods used to prove global asymptotic stability when n=2 no longer apply for n>2 and an approach based on Liapunov functions is likely to be required, although this may prove difficult in practice. However, it is tempting to conjecture that the analogue of Theorem 3.1 will hold when n>2, and also for the case of unequal household sizes. Numerical investigations can be used to support or refute these conjectures.

## 4. Concluding comments

A threshold parameter  $R_*$  has been determined for models for the spread of an SIS epidemic among a population partitioned into a large number of households. In the stochastic models, the probability that an epidemic takes off is non-zero if and only if  $R_* > 1$ . In the deterministic model, with households of size 2, the epidemic dies out if  $R_* \le 1$  and it settles down to an endemic equi-

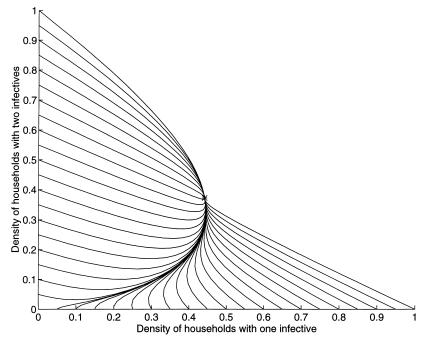


Fig. 6. Typical phase curves for the system (11) when  $\beta_W = 0.5$  and  $\beta_B = 1.0$ . (The endemic equilibrium point is marked by an asterisk.)

librium if  $R_* > 1$ . Moreover, it seems likely that this will also be true for models with other distributions of household size. Although the above results are qualitatively the same as those for more standard epidemic models, which correspond to large households, the threshold parameter is quite different. It is the threshold parameter that is of paramount importance in determining vaccination strategies to prevent epidemics. Thus appropriate account should be taken of household structure when formulating and analysing models to inform public health policy.

The models and methods of this paper can in principle be extended to more general SIS epidemic models, e.g. including different types of individuals, and to other epidemic models, such as SIR epidemics with vital dynamics. The branching process approximation of Section 2 provides a flexible framework for determining the threshold parameter  $R_*$  for such models, and the coupling argument underlying the proof of Theorem 2.1 provides a general method for proving a stochastic invasion threshold theorem. The appropriate method of constructing a deterministic model for the spread of an epidemic among a community of households is that of Section 3.1, viz. by classifying each household according to its numbers of infectives, susceptibles etc. at a given

time t and then deriving a set of differential equations governing the evolution of the densities of households of different classifications.

The results of Sections 2 and 3 are based on approximations to a stochastic model that become exact in the limit as the number of households  $m \to \infty$ . In practice, it is important to know how accurate the approximations are for a given finite m, or equivalently how large m must be for the approximations to be useful. Ball and Donnelly [5] provide methods for obtaining bounds on the total variation distance between an epidemic and its approximating branching process and a central limit theorem for fluctuations of the epidemic about its deterministic limit can, at least in principle, be proved using results in Ref. [20]. However, the bounds on the total variation distance are often coarse and the central limit theorem requires  $m \to \infty$ . Thus, in the absence of analytic results, carefully planned simulation studies are required to determine the accuracy of the approximation. Such computer intensive methods can be applied to models which are more realistic than those that are currently susceptible to mathematical analysis. They may also indicate features of models worthy of further mathematical investigation.

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