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THE FINAL OUTCOME OF AN EPIDEMIC MODEL WITH SEVERAL DIFFERENT TYPES OF INFECTIVE IN A LARGE POPULATION

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Abstract

We consider a stochastic model for the spread of an epidemic amongst a closed homogeneously mixing population, in which there are several different types of infective, each newly infected individual choosing its type at random from those available. The model is based on the carrier-borne model of Downton (1968), as extended by Picard and Lefèvre (1990). The asymptotic distributions of final size and area under the trajectory of infectives are derived as the initial population becomes large, using arguments based on those of Scalia-Tomba (1985), (1990). We then use our limiting results to compare the asymptotic final size distribution of our model with that of a related multi-group model, in which the type of each infective is assigned deterministically.

CARRIER-BORNE EPIDEMICS; EPIDEMICS IN HETEROGENEOUS POPULATIONS; SIZE OF EPIDEMIC; AREA UNDER TRAJECTORY OF INFECTIVES; GAUSSIAN LIMIT THEOREMS; EFFECT OF VARIABILITY

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1. Introduction

A problem which has recently attracted a great deal of attention in the epidemic modelling literature is that of showing that for various different epidemic models, when the initial population is allowed to become large, then in the case of a major outbreak of infection the number of individuals to become infected during the epidemic is asymptotically normally distributed. Thus von Bahr and Martin-Löf (1980) used a martingale argument to establish such a result for the Reed–Frost discrete-time epidemic model, and for a generalisation which they referred to as the randomised Reed–Frost process. Their methods were extended by Scalia-Tomba (1986) to apply to a multitype version of the Reed–Frost process, in which the population is divided into several groups, with the probability of contact between an infective and a susceptible individual depending

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upon the groups of each. The technique used by both of these papers was rather technical, and more intuitively appealing ideas, based on weak convergence of appropriately defined random processes to continuous Gaussian processes, were applied by Scalia-Tomba (1985), (1990) and Martin-Löf (1986), (1990) to prove corresponding results for various single population models, their results applying in both discrete-time and continuous-time settings. These methods were then extended by Andersson (1993) and Ball and Clancy (1993) to multitype versions of the models. Similar multitype results have been derived by Svensson (1993).

In this paper, we show how the methods of Scalia-Tomba (1990) can be extended in a rather different direction. The model which we consider is based on the carrier-borne model of Downton (1968), as extended by Picard and Lefèvre (1990). Thus we have a single closed, homogeneously mixing population divided into susceptibles, infectives and removed individuals, but now the infectives are themselves divided into several different types. When a susceptible becomes infected, it chooses its type at random from those available. The model will be defined more precisely in the next section. We then show that in the case of a major outbreak of infection, the final size of the epidemic is asymptotically normally distributed as the initial population becomes large. We also show that the final severity, or area under the trajectory of infectives, is likewise asymptotically normally distributed. Previous work has generally concentrated on final size alone, though Ball and Clancy (1993) considered final severity also, Svensson (1993) considered the joint distribution of final size and severity, and Andersson and Djehiche (1994) were solely concerned with the asymptotic final severity, using arguments similar to those of Andersson (1993). Finally, we use our limit theorem to compare the asymptotic final size distribution of our model with that of a related multigroup model, in which the type of each infective is assigned deterministically.

2. The model and fundamental construction

We consider a closed, homogeneously mixing population divided into susceptibles, infectives and removed individuals, the infectives being further subdivided into ρ types. The population initially consists of N susceptibles and a infectives, with a_i of the infectives being of type i for $i=1, 2, \cdots, \rho$. Each initial infective of type i remains infectious for a random time distributed as a non-negative random variable $I^{(i)}$, and is then removed. During this time, the infective makes contacts at the points of a homogeneous Poisson process of rate β_i. We assume that $\beta_i \boldsymbol{E}[I^{(i)}] > 0$ for some $i=1, 2, \cdots, \rho$, since otherwise there would almost surely be no spread of infection. Each contact is with an individual chosen uniformly at random from the N initial susceptibles, independently of all other events. When an individual is contacted, if it is still susceptible, then it becomes infected. Otherwise the contact has no effect. On becoming infected, an individual chooses its type at random from the set $\{1, 2, \cdots, \rho\}$, the probability of choosing type i being taken to be π_i, where $\pi_1 + \pi_2 + \cdots + \pi_\rho = 1$, and we assume that $\pi_i > 0$ for $i=1, 2, \cdots, \rho$. This choice is made independently of all other events. Each newly infected type i infective remains infectious for a time distributed as $I^{(i)}$, during which it makes contacts at the

points of a homogeneous Poisson process of rate β_i, and is then removed. The infectious period and contact process of a particular infective are independent of one another, and the infectious periods and contact processes of distinct individuals are also all mutually independent. The epidemic process continues until no infectives are left in the population. For future reference, we write $\boldsymbol{\pi}=(\pi_1, \pi_2,\cdots, \pi_\rho)$, $\boldsymbol{\beta}=(\beta_1, \beta_2,\cdots, \beta_\rho)$ and $\boldsymbol{a}=(a_1, a_2,\cdots, a_\rho)$. Note that, throughout the paper, all vectors are row vectors.

If we take $\rho=2$, $\Pr(I^{(2)}=0)=1$ and $a_2=0$ in the above model, then we have the generalisation of Downton's carrier-borne model studied by Ball (1990). Type 1 infectives now correspond to carriers, and type 2 infectives to directly removed individuals. If we further set $I^{(1)}$ to be exponentially distributed with mean γ^{-1} then our model reduces to Downton's original model.

The statistics in which we are interested are $\boldsymbol{N}^*$, the final size vector, and $\boldsymbol{\alpha}$, the final severity vector, defined as follows. For $i=1, 2,\cdots, \rho$, let N_i^* be the number of initially susceptible individuals which become infectives of type i during the course of the epidemic, and let α_i be the sum of the infectious periods of all $a_i+N_i^*$ type i infectives to exist during the epidemic. Then $\boldsymbol{N}^*=(N_1^*, N_2^*,\cdots, N_\rho^*)$ and $\boldsymbol{\alpha}=(\alpha_1, \alpha_2,\cdots, \alpha_\rho)$.

In order to find the limiting distributions of $\boldsymbol{N}^*$ and $\boldsymbol{\alpha}$ when the population is large, we use a construction based on that of Sellke (1983). First, attach to each individual a label (i, r) as follows. For those individuals which are initially infective, $i=1, 2,\cdots, \rho$ gives the type of individual (i, r) and $r=-(a_i-1), -(a_i-2),\cdots, 0$ gives the individual's number amongst initial infectives of type i. For the initial susceptibles, we take $i=0$, and $r=1, 2,\cdots, N$ gives the number of individual $(0, r)$ amongst the initial susceptibles. Now for $i=1, 2,\cdots, \rho$, let $\{I^{(i,r)} : r=-(a_i-1), -(a_i-2),\cdots, N\}$ be a set of independent, identically distributed random variables such that $I^{(i,r)} \stackrel{\mathcal{D}}{=} I^{(i)}$, and suppose that these sets of random variables are mutually independent. Let $\{Q^{(r)} : r=1, 2,\cdots, N\}$ be an independent set of independent random variables, each exponentially distributed with mean N, and let $\{i^{(r)} : r=1, 2,\cdots, N\}$ be a set of independent random variables taking values in $\{1, 2,\cdots, \rho\}$, independently of the $Q^{(r)}$ and $I^{(i,r)}$, such that $\Pr(i^{(r)}=i)=\pi_i$ for $i=1, 2,\cdots, \rho$. The epidemic process is then constructed as follows. For $i=1, 2,\cdots, \rho$ and $r=-(a_i-1), -(a_i-2),\cdots, 0$ the initial infective (i, r) remains infectious for a time $I^{(i,r)}$ and is then removed. At any time $t \geqq 0$, the rth susceptible accumulates exposure to infection at rate $\Sigma_{i=1}^{\rho} \beta_i Y_i(t)$, where $Y_i(t)$ is the number of type i infectives in the population at time t. When the total exposure to infection of individual $(0, r)$ reaches $Q^{(r)}$, it becomes an infective of type $i^{(r)}$, remains so for a time $I^{(i^{(r)},r)}$, and is then removed. The epidemic terminates as soon as there are no infectives present in the population. The epidemic thus defined is easily shown to be equivalent to our model as defined earlier (see Sellke (1983)).

A deterministic version of our model can be obtained by supposing that for $i=1, 2,\cdots, \rho$ a proportion π_i of infected susceptibles become type i infectives, and that each type i infective remains infectious for a time $l_i=\boldsymbol{E}[I^{(i)}]$ before being removed. For this deterministic model, let $x(t)$ be the number of susceptibles in the population at time t, and $y_i(t)$ the number of type i infectives. Then during the course of the epidemic, $a+N-x(\infty)$

individuals become infective, and of these, $a_i+(N-x(\infty))\pi_i$ are of type i. Since each type i infective remains so for a time l_i, we have

$$\int_0^\infty y_i(t)dt=(a_i+(N-x(\infty))\pi_i)l_i \qquad (i=1, 2,\cdots, \rho). \tag{2.1}$$

Now each type i infective makes contact with each susceptible at constant rate β_i/N, and so

$$\frac{dx}{dt}=-\left(\sum_{i=1}^{\rho}\frac{\beta_i}{N}y_i\right)x,$$

which on integration yields

$$\ln(x(\infty)/x(0))=-\sum_{i=1}^{\rho}\frac{\beta_i}{N}\int_0^\infty y_i(t)dt.$$

Substituting from (2.1) and defining $\mu_i=a_i/N$, $\sigma=x(\infty)/N$, this becomes

$$\ln\sigma=-\sum_{i=1}^{\rho}\beta_i(\mu_i+(1-\sigma)\pi_i)l_i. \tag{2.2}$$

Finally, setting $\lambda_i=\beta_i l_i$ and writing $\boldsymbol{\lambda}=(\lambda_1, \lambda_2,\cdots, \lambda_\rho)$, $\boldsymbol{\mu}=(\mu_1, \mu_2,\cdots, \mu_\rho)$, then (2.2) can be rewritten as

$$\sigma=\exp\{-(\boldsymbol{\mu}+(1-\sigma)\boldsymbol{\pi})\boldsymbol{\lambda}^{\mathrm{T}}\}. \tag{2.3}$$

From Lemma 3 of von Bahr and Martin-Löf (1980), we know that when $\boldsymbol{\mu}\boldsymbol{\lambda}^{\mathrm{T}}\neq 0$, equation (2.3) has a unique root σ in the interval [0, 1]. Defining

$$A=\{i=1, 2,\cdots, \rho : \lambda_i>0\},$$

so infectives of type i contribute to the further spread of infection precisely when $i\in A$, then $\boldsymbol{\mu}\boldsymbol{\lambda}^{\mathrm{T}}\neq 0$ if there is some $i\in A$ such that $\mu_i\neq 0$.

If we let $\boldsymbol{\mu}\boldsymbol{\lambda}^{\mathrm{T}}\to 0$, Lemma 3 of von Bahr and Martin-Löf (1980) further tells us that in the case $\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}\leqq 1$, then $\sigma\to 1$, whereas if $\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}>1$, then σ converges to the unique root $\sigma^{(\infty)}$ in [0, 1) of (2.3) with $\boldsymbol{\mu}=\mathbf{0}$, and $\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}<1$.

As the number of initial susceptibles which become type i infectives during the epidemic is $N(1-\sigma)\pi_i$, (2.3) completely determines the final outcome of our deterministic epidemic model.

3. An epidemic initiated by a large number of infectives

We consider a sequence of epidemics indexed by v, such that as $v\to\infty$ we have $N^{(v)}\to\infty$ and $\boldsymbol{\mu}^{(v)}\to\boldsymbol{\mu}^{(\infty)}\geqq\mathbf{0}$ (inequalities between vectors being interpreted componentwise). The vectors $\boldsymbol{\pi}$ and $\boldsymbol{\beta}$ are assumed to be independent of v, as is the distribution of $I^{(i)}$, $i=1, 2,\cdots, \rho$. The index v will generally be omitted for simplicity of notation.

We first define the infectivity processes $\{\xi_i(t) : t \geqq 0\}$ and the susceptibility processes $\{X_i(t) : t \geqq 0\}$ as follows. For $i = 1, 2, \cdots, \rho$,

$$\xi_i(t) = \sum_{r=-(a_i-1)}^{[t]-a_i} \beta_i I^{(i,r)} \qquad (1 \leqq t \leqq N + a_i),$$

$$X_i(t) = \sum_{r=1}^{N} 1_{\{Q^{(r)} \leqq t,\, i^{(r)} = i\}} \qquad (t \geqq 0),$$

where $1_{\{\cdot\}}$ denotes the indicator function of the event $\{\cdot\}$. We relabel the infectious periods $I^{(i,r)}$ so that $I^{(i,r)}$ is the infectious period of the rth type i infective to become infected. Then $\xi_i(t)$ gives the infective pressure exerted by the first $[t]$ type i infectives to exist during the course of the epidemic, and $X_i(t)$ is the number of initial susceptibles which will have become type i infectives when the total infectious pressure reaches t.

Define normalised infectivity and susceptibility processes by

$$\bar{\xi}_i(t) = \frac{1}{N}\xi_i(Nt) \qquad (1/N \leqq t \leqq 1 + \mu_i),$$

$$\bar{X}_i(t) = \frac{1}{N}X_i(Nt) \qquad (t \geqq 0).$$

Let $K_i = \mathrm{Var}[\beta_i I^{(i)}]$, and assume that $\lambda_i = \boldsymbol{E}[\beta_i I^{(i)}]$ and K_i are both finite $(i = 1, 2, \cdots, \rho)$. Then by Donsker's theorem we have that, as $v \to \infty$,

$$\frac{(\bar{\xi}_i(t) - \lambda_i t)\sqrt{N}}{\sqrt{K_i}} \Rightarrow W_i(t) \qquad (i = 1, 2, \cdots, \rho), \tag{3.1}$$

where the processes $W_1, W_2, \cdots, W_\rho$ are independent standard Brownian motions. Letting $\bar{\zeta}(\boldsymbol{t}) = \Sigma_{i=1}^{\rho} \bar{\xi}_i(t_i)$ for $\boldsymbol{t} = (t_1, t_2, \cdots, t_\rho) \in (\mathbb{R}^+)^\rho$ and summing (3.1) over i now yields

$$(\bar{\zeta}(\boldsymbol{t}) - \boldsymbol{t}\boldsymbol{\lambda}^{\mathrm{T}})\sqrt{N} \Rightarrow \sum_{i=1}^{\rho} W_i(t_i)\sqrt{K_i}\,. \tag{3.2}$$

Turning to the susceptibility processes, and writing $\bar{\boldsymbol{X}}(t) = (\bar{X}_1(t), \bar{X}_2(t), \cdots, \bar{X}_\rho(t))$, we define $\boldsymbol{M}(t) = \boldsymbol{E}[\bar{\boldsymbol{X}}(t)] = (1 - e^{-t})\boldsymbol{\pi}$ and $C(s, t) = N\,\mathrm{Cov}\,[\bar{\boldsymbol{X}}(s), \bar{\boldsymbol{X}}(t)]$, so using $\wedge$ to denote minimum, and writing $\delta_{ij} = 1_{\{i=j\}}$, $C(s, t)$ is a $\rho \times \rho$ matrix with (i, j)th element $C_{ij}(s, t) = (1 - e^{-(s \wedge t)})\pi_i \delta_{ij} - (1 - e^{-s})(1 - e^{-t})\pi_i \pi_j$. In vector notation, $C(s, t) = (1 - e^{-(s \wedge t)})\,\mathrm{diag}(\boldsymbol{\pi}) - (1 - e^{-s})(1 - e^{-t})\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\pi}$, where $\mathrm{diag}(\boldsymbol{\pi})$ denotes the diagonal matrix with diagonal entries $\pi_1, \pi_2, \cdots, \pi_\rho$. Now $\bar{\boldsymbol{X}}(t)$ is a Markov process with initial condition $\Pr(\bar{\boldsymbol{X}}(0) = \boldsymbol{0}) = 1$, transition rates

$$\Pr\left(\bar{X}_i(t + \delta t) = x_i + \frac{1}{N},\ \bar{X}_j(t + \delta t) = x_j \text{ for } j = 1, 2, \cdots, \rho, j \neq i \,\middle|\, \bar{\boldsymbol{X}}(t) = \boldsymbol{x}\right)$$

$$= N\pi_i \left(1 - \sum_{j=1}^{\rho} x_j\right)\delta t + o(\delta t).$$

This clearly satisfies the conditions for Theorem 2.3 of Ethier and Kurtz (1986), Chapter 11, and so we have

$$(\bar{\boldsymbol{X}}(t)-\boldsymbol{M}(t))\sqrt{N}\Rightarrow \boldsymbol{Z}^{(\infty)}(t),$$

where $\boldsymbol{Z}^{(\infty)}$ is a Gaussian process on $D_{\mathbb{R}^\rho}[0,\infty)$, the space of right-continuous functions from $[0,\infty)$ to $\mathbb{R}^\rho$, with almost surely continuous trajectories and $\boldsymbol{E}[\boldsymbol{Z}^{(\infty)}(t)]=\boldsymbol{0}$. In their notation, the $\rho\times\rho$ matrices $\Phi(t,s)$ $(t,s\in\mathbb{R}^+)$ and $G(\boldsymbol{x})$ $(\boldsymbol{x}\in\mathbb{R}^\rho)$ have (i,j)th elements $\Phi_{ij}(t,s)=\delta_{ij}-(1-e^{-(t-s)})\pi_i$ and $G_{ij}(\boldsymbol{x})=(\pi_i-x_i)\delta_{ij}$, and so

$$\mathrm{Cov}[\boldsymbol{Z}^{(\infty)}(s),\boldsymbol{Z}^{(\infty)}(t)]=\int_0^{s\wedge t}\Phi(s,u)G(\boldsymbol{M}(u))(\Phi(t,u))^{\mathrm{T}}du=C(s,t).$$

Defining

$$\tilde{\boldsymbol{Z}}(t)=(\bar{\boldsymbol{X}}(\bar{\zeta}(t))-\boldsymbol{M}(\bar{\zeta}(t)))\sqrt{N},$$

$$\boldsymbol{D}(t)=(\boldsymbol{M}(\bar{\zeta}(t))-\boldsymbol{M}(t\boldsymbol{\lambda}^{\mathrm{T}}))\sqrt{N},$$

$$\boldsymbol{V}(t)=(\bar{\boldsymbol{X}}(\bar{\zeta}(t))-\boldsymbol{M}(t\boldsymbol{\lambda}^{\mathrm{T}}))\sqrt{N},$$

and writing $\bar{\boldsymbol{T}}=(\bar{T}_1,\bar{T}_2,\cdots,\bar{T}_\rho)$, where $\bar{T}_i=(N_i^*+a_i)/N$ $(i=1,2,\cdots,\rho)$, then since $N_i^*=X_i(\Sigma_{i=1}^{\rho}\xi_i(a_i+N_i^*))$, we have that $\bar{\boldsymbol{T}}$ satisfies the equation

$$\bar{\boldsymbol{T}}-\boldsymbol{\mu}-\boldsymbol{M}(\bar{\boldsymbol{T}}\boldsymbol{\lambda}^{\mathrm{T}})=\boldsymbol{V}(\bar{\boldsymbol{T}})/\sqrt{N}. \tag{3.3}$$

Now since $\zeta(t)\Rightarrow t\boldsymbol{\lambda}^{\mathrm{T}}$, applying the mean value theorem to $\boldsymbol{D}(t)$ and using (3.2) gives

$$\boldsymbol{D}(t)\Rightarrow\sum_{i=1}^{\rho}W_i(t_i)\sqrt{K_i}\,\boldsymbol{M}'(t\boldsymbol{\lambda}^{\mathrm{T}}),$$

where $\boldsymbol{M}'(t)=(M_1'(t),M_2'(t),\cdots,M_\rho'(t))=e^{-t}\boldsymbol{\pi}$.

The fact that $\zeta(t)\Rightarrow t\boldsymbol{\lambda}^{\mathrm{T}}$ also implies that $\tilde{\boldsymbol{Z}}(t)\Rightarrow\boldsymbol{Z}^{(\infty)}(t\boldsymbol{\lambda}^{\mathrm{T}})$, and so finally

$$\boldsymbol{V}(t)\Rightarrow\boldsymbol{V}^{(\infty)}(t),$$

where $\boldsymbol{V}^{(\infty)}(t)=\boldsymbol{Z}^{(\infty)}(t\boldsymbol{\lambda}^{\mathrm{T}})+\boldsymbol{M}'(t\boldsymbol{\lambda}^{\mathrm{T}})\,\Sigma_{i=1}^{\rho}W_i(t_i)\sqrt{K_i}$. Thus $\boldsymbol{V}^{(\infty)}(t)$ is a Gaussian random function from $(\mathbb{R}^+)^\rho$ to $\mathbb{R}^\rho$ with almost surely continuous trajectories, $\boldsymbol{E}[\boldsymbol{V}^{(\infty)}(t)]=\boldsymbol{0}$, and writing $\boldsymbol{K}=(K_1,K_2,\cdots,K_\rho)$, $\boldsymbol{s}\wedge\boldsymbol{t}=(s_1\wedge t_1,s_2\wedge t_2,\cdots,s_\rho\wedge t_\rho)$, then

$$\mathrm{Cov}[\boldsymbol{V}^{(\infty)}(\boldsymbol{s}),\boldsymbol{V}^{(\infty)}(\boldsymbol{t})]=C(\boldsymbol{s}\boldsymbol{\lambda}^{\mathrm{T}},\boldsymbol{t}\boldsymbol{\lambda}^{\mathrm{T}})+e^{-(\boldsymbol{s}+\boldsymbol{t})\boldsymbol{\lambda}^{\mathrm{T}}}((\boldsymbol{s}\wedge\boldsymbol{t})\boldsymbol{K}^{\mathrm{T}})\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\pi}. \tag{3.4}$$

We now suppose that $\boldsymbol{\mu}^{(\infty)}$ is such that for some $i\in A$, $\mu_i^{(\infty)}\neq 0$. Then letting $\boldsymbol{\tau}=\boldsymbol{\mu}+(1-\sigma)\boldsymbol{\pi}$, Equation (2.3) becomes

$$\boldsymbol{\tau}-\boldsymbol{\mu}-\boldsymbol{M}(\boldsymbol{\tau}\boldsymbol{\lambda}^{\mathrm{T}})=\boldsymbol{0}, \tag{3.5}$$

and we know from Lemma 3 of von Bahr and Martin-Löf (1980) that the equation corresponding to (3.5) with $\boldsymbol{\mu}^{(\nu)}$ replaced by $\boldsymbol{\mu}^{(\infty)}$ has a unique solution $\boldsymbol{\tau}^{(\infty)}$ in $[\boldsymbol{\mu}^{(\infty)},\boldsymbol{1}+\boldsymbol{\mu}^{(\infty)}]=\{\boldsymbol{u}\in\mathbb{R}^\rho:\boldsymbol{\mu}^{(\infty)}\leqq\boldsymbol{u}\leqq\boldsymbol{1}+\boldsymbol{\mu}^{(\infty)}\}$. So as $\nu\to\infty$ we have $\boldsymbol{\tau}^{(\nu)}\to\boldsymbol{\tau}^{(\infty)}$. Furthermore,

it follows from (3.3) that $\bar{\boldsymbol{T}}^{(\nu)} \xrightarrow{\mathrm{p}} \boldsymbol{\tau}^{(\infty)}$, and so $\boldsymbol{V}^{(\nu)}(\bar{\boldsymbol{T}}^{(\nu)}) \xrightarrow{\mathscr{D}} \boldsymbol{V}^{(\infty)}(\boldsymbol{\tau}^{(\infty)})$. Now using (3.5), Equation (3.3) can be rewritten as

$$\boldsymbol{V}^{(\nu)}(\bar{\boldsymbol{T}}^{(\nu)}) = ((\bar{\boldsymbol{T}}^{(\nu)} - \boldsymbol{M}(\bar{\boldsymbol{T}}^{(\nu)}\boldsymbol{\lambda}^{\mathrm{T}})) - (\boldsymbol{\tau}^{(\nu)} - \boldsymbol{M}(\boldsymbol{\tau}^{(\nu)}\boldsymbol{\lambda}^{\mathrm{T}})))\sqrt{N^{(\nu)}},$$

and so by the mean value theorem,

$$\boldsymbol{V}^{(\nu)}(\bar{\boldsymbol{T}}^{(\nu)}) = (\bar{\boldsymbol{T}}^{(\nu)} - \boldsymbol{\tau}^{(\nu)})S^{(\nu)}\sqrt{N^{(\nu)}},$$

where $S^{(\nu)}$ is a $\rho \times \rho$ matrix with elements $S_{ij}^{(\nu)} = \delta_{ij} - \lambda_i M_j'(\boldsymbol{s}^{(\nu)}\boldsymbol{\lambda}^{\mathrm{T}})$, some $\boldsymbol{s}^{(\nu)}$ between $\bar{\boldsymbol{T}}^{(\nu)}$ and $\boldsymbol{\tau}^{(\nu)}$. Thus as $\nu \to \infty$, $\boldsymbol{s}^{(\nu)} \xrightarrow{\mathrm{p}} \boldsymbol{\tau}^{(\infty)}$, so $S_{ij}^{(\nu)} \xrightarrow{\mathrm{p}} S_{ij}^{(\infty)} = \delta_{ij} - \lambda_i \pi_j \sigma^{(\infty)}$, and hence

$$(\bar{\boldsymbol{T}}^{(\nu)} - \boldsymbol{\tau}^{(\nu)})\sqrt{N^{(\nu)}} \xrightarrow{\mathscr{D}} \boldsymbol{V}^{(\infty)}(\boldsymbol{\tau}^{(\infty)})(S^{(\infty)})^{-1}, \tag{3.6}$$

provided that the matrix $S^{(\infty)}$ is invertible. But denoting by I the $\rho \times \rho$ identity matrix, we have that $S^{(\infty)} = I - \sigma^{(\infty)}\boldsymbol{\lambda}^{\mathrm{T}}\boldsymbol{\pi}$, and the only eigenvalues of $S^{(\infty)}$ are 1 and $1 - \sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}$. It is clear from the proof of Lemma 3 of von Bahr and Martin-Löf (1980) that when $\boldsymbol{\mu}^{(\infty)}\boldsymbol{\lambda}^{\mathrm{T}} \neq 0$, then $\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}} < 1$, and so $S^{(\infty)}$ is indeed non-singular. In fact, the inverse matrix of $S^{(\infty)}$ is given by

$$S^{(\infty)-1} = I + \frac{\sigma^{(\infty)}\boldsymbol{\lambda}^{\mathrm{T}}\boldsymbol{\pi}}{1 - \sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}}.$$

Letting $\Xi = \mathrm{Var}[\boldsymbol{V}^{(\infty)}(\boldsymbol{\tau}^{(\infty)})]$, we now have that as $\nu \to \infty$,

$$(\bar{\boldsymbol{T}}^{(\nu)} - \boldsymbol{\tau}^{(\nu)})\sqrt{N^{(\nu)}} \xrightarrow{\mathscr{D}} N(\boldsymbol{0}, (S^{(\infty)-1})^{\mathrm{T}}\Xi S^{(\infty)-1}). \tag{3.7}$$

But from (3.4), we know that $\Xi = (1 - \sigma^{(\infty)})\mathrm{diag}(\boldsymbol{\pi}) + (\sigma^{(\infty)2}\boldsymbol{\tau}^{(\infty)}\boldsymbol{K}^{\mathrm{T}} - (1 - \sigma^{(\infty)})^2)\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\pi}$. Thus the variance matrix of the limiting distribution of $(\bar{\boldsymbol{T}}^{(\nu)} - \boldsymbol{\tau}^{(\nu)})\sqrt{N^{(\nu)}}$ is given by

$$\begin{aligned}(S^{(\infty)-1})^{\mathrm{T}}\Xi S^{(\infty)-1} &= (1 - \sigma^{(\infty)})\mathrm{diag}(\boldsymbol{\pi}) \\ &+ \left(\frac{\sigma^{(\infty)}(1 - \sigma^{(\infty)})}{1 - \sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}}\right)(\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\lambda}\,\mathrm{diag}(\boldsymbol{\pi}) + \mathrm{diag}(\boldsymbol{\pi})\boldsymbol{\lambda}^{\mathrm{T}}\boldsymbol{\pi}) \\ &+ \left(\frac{\sigma^{(\infty)2}\boldsymbol{\tau}^{(\infty)}\boldsymbol{K}^{\mathrm{T}} - (1 - \sigma^{(\infty)})^2 + \sigma^{(\infty)2}(1 - \sigma^{(\infty)})\boldsymbol{\lambda}\,\mathrm{diag}(\boldsymbol{\pi})\boldsymbol{\lambda}^{\mathrm{T}}}{(1 - \sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}})^2}\right)\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\pi}.\end{aligned} \tag{3.8}$$

4. An epidemic initiated by a trace of infection

Suppose now that we have $\boldsymbol{a}^{(\nu)} = \boldsymbol{a}$ for all ν. Then the coupling argument of Ball (1983) shows that as $\nu \to \infty$, the process of infectives in our epidemic model converges almost surely to a ρ-type continuous-time branching process, in which individuals born in group i have lifetimes distributed as $I^{(i)}$, and during their lifetime give birth in group j at the points of a homogeneous Poisson process of rate $\beta_i \pi_j$. The total size of the epidemic also converges almost surely to that of the branching process, which has the same distribution as the total size of the embedded Galton–Watson process, initiated by $\boldsymbol{a}$

ancestors, in which each group i individual has offspring distributed as $\boldsymbol{G}^{(i)}=(G_1^{(i)}, G_2^{(i)},\cdots, G_\rho^{(i)})$, where, writing $\phi_i(\theta)=\boldsymbol{E}[\exp\{-\theta I^{(i)}\}]$,

$$f_i(\boldsymbol{s})=\boldsymbol{E}\left[\prod_{j=1}^{\rho} s_j^{G_j^{(i)}}\right]=\phi_i(\beta_i(\mathbf{1}-\boldsymbol{s})\boldsymbol{\pi}^{\mathrm{T}}).$$

The mean number of type j offspring produced by each type i individual in the Galton–Watson process is $\lambda_i\pi_j$, and it is easy to see that the matrix of mean values $\boldsymbol{\lambda}^{\mathrm{T}}\boldsymbol{\pi}$ has only one non-zero eigenvalue, $R_0=\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}$. This value has the natural interpretation that whenever a new individual is born, the expected number of offspring which it will produce is $\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}$. If $R_0\leqq 1$ then the total number of progeny of the Galton–Watson process is almost surely finite. If $R_0>1$, then there is a probability $1-\Pi_{i=1}^{\rho} q_i^{a_i}$ that the Galton–Watson process will produce an infinite number of progeny, where $\boldsymbol{q}=(q_1, q_2,\cdots, q_\rho)$ is the unique solution of $\boldsymbol{q}=\boldsymbol{f}(\boldsymbol{q})$ with $0\leqq q_i<1$ for $i\in A$, $q_i=1$ for $i\notin A$. In this case, if the limiting Galton–Watson process does not become extinct then it can be shown that the asymptotic distribution of the final size of the corresponding epidemic is given by (3.7), but with $\boldsymbol{\mu}^{(\infty)}$ set equal to $\mathbf{0}$ and $\boldsymbol{\tau}^{(\nu)}$ replaced by $\boldsymbol{\tau}^{(\infty)}=\lim_{\nu\to\infty}\boldsymbol{\tau}^{(\nu)}$. From Lemma 3 of von Bahr and Martin-Löf (1980), we again have that the matrix $S^{(\infty)}$ is non-singular. Details of the proof, which parallels that of Scalia-Tomba (1985), (1990), appear in Clancy (1993).

5. Final severity limit

To find the limiting distribution of the final severity vector $\boldsymbol{\alpha}$, we use essentially the same method as for the final size vector $\boldsymbol{N}^*$. Letting $\bar{\boldsymbol{\alpha}}=(1/N)\boldsymbol{\alpha}$ and writing $\bar{\boldsymbol{I}}(t)=(\bar{I}_1(t_1), \bar{I}_2(t_2),\cdots, \bar{I}_\rho(t_\rho))$, where

$$\bar{I}_i(t)=\frac{1}{N}\sum_{r=-(a_i-1)}^{[Nt]-a_i} I^{(i,r)},$$

then $\bar{\boldsymbol{\alpha}}$ satisfies

$$\bar{\boldsymbol{\alpha}}=\bar{\boldsymbol{I}}(\boldsymbol{\mu}+\bar{\boldsymbol{X}}(\bar{\boldsymbol{\alpha}}\boldsymbol{\beta}^{\mathrm{T}})).$$

Now let $J_i=\mathrm{Var}[I^{(i)}]$. Then by Donsker's theorem,

$$(\bar{I}_i(t)-tl_i)\sqrt{N}\Rightarrow B_i(t)\sqrt{J_i},$$

where $B_1, B_2,\cdots, B_\rho$ are independent standard Brownian motions. Thus if we write $\boldsymbol{J}=(J_1, J_2,\cdots, J_\rho)$ and $\boldsymbol{B}(\boldsymbol{t})=(B_1(t_1), B_2(t_2),\cdots, B_\rho(t_\rho))$, we have

$$(\bar{\boldsymbol{I}}(\boldsymbol{t})-\boldsymbol{t}\,\mathrm{diag}(\boldsymbol{l}))\sqrt{N}\Rightarrow\boldsymbol{B}(\boldsymbol{t})\sqrt{\mathrm{diag}(\boldsymbol{J})},$$

where $\boldsymbol{l}=(l_1, l_2,\cdots, l_\rho)$. Defining $\boldsymbol{U}(t)=(\bar{\boldsymbol{I}}(\boldsymbol{\mu}+\boldsymbol{X}(t))-(\boldsymbol{\mu}+\boldsymbol{M}(t))\mathrm{diag}(\boldsymbol{l}))\sqrt{N}$, then since $\bar{\boldsymbol{X}}(t)\Rightarrow\boldsymbol{M}(t)$,

$$\boldsymbol{U}(t)\Rightarrow\boldsymbol{U}^{(\infty)}(t),$$

where

$$\boldsymbol{U}^{(\infty)}(t)=\boldsymbol{B}(\boldsymbol{\mu}^{(\infty)}+\boldsymbol{M}(t))\sqrt{\operatorname{diag}(\boldsymbol{J})}+\boldsymbol{Z}^{(\infty)}(t)\operatorname{diag}(\boldsymbol{l}).$$

In the case where $\mu_i^{(\infty)}\neq 0$ for some $i\in A$, then $\bar{\boldsymbol{\alpha}}\xrightarrow{\mathrm{p}}\boldsymbol{\tau}^{(\infty)}\operatorname{diag}(\boldsymbol{l})$ as $v\to\infty$, and so proceeding as in Section 3, we find that

$$\boldsymbol{U}(\bar{\boldsymbol{\alpha}}\boldsymbol{\beta}^{\mathrm{T}})=(\bar{\boldsymbol{\alpha}}-\boldsymbol{\tau}\operatorname{diag}(\boldsymbol{l}))\Upsilon\sqrt{N},$$

where $\Upsilon^{(v)}$ is a $\rho\times\rho$ matrix with elements $\Upsilon_{ij}^{(v)}=\delta_{ij}-\beta_i M_j'(\boldsymbol{\theta}^{(v)}\boldsymbol{\beta}^{\mathrm{T}})l_j$, some $\boldsymbol{\theta}^{(v)}$ between $\bar{\boldsymbol{\alpha}}^{(v)}$ and $\boldsymbol{\tau}^{(v)}\operatorname{diag}(\boldsymbol{l})$. Thus as $v\to\infty$, $\boldsymbol{\theta}^{(v)}\xrightarrow{\mathrm{p}}\boldsymbol{\tau}^{(\infty)}\operatorname{diag}(\boldsymbol{l})$, so $\Upsilon_{ij}^{(v)}\xrightarrow{\mathrm{p}}\Upsilon_{ij}^{(\infty)}=\delta_{ij}-\beta_i l_j\pi_j\sigma^{(\infty)}$, and hence

$$(\bar{\boldsymbol{\alpha}}-\boldsymbol{\tau}\operatorname{diag}(\boldsymbol{l}))\sqrt{N}\xrightarrow{\mathscr{D}}\boldsymbol{U}^{(\infty)}(\boldsymbol{\tau}^{(\infty)}\boldsymbol{\lambda}^{\mathrm{T}})(\Upsilon^{(\infty)})^{-1},$$

provided that the matrix $\Upsilon^{(\infty)}$ is invertible. But since $\Upsilon^{(\infty)}=I-\sigma^{(\infty)}\boldsymbol{\beta}^{\mathrm{T}}\boldsymbol{\pi}\operatorname{diag}(\boldsymbol{l})$, the only eigenvalues of $\Upsilon^{(\infty)}$ are 1 and $1-\sigma^{(\infty)}\boldsymbol{\pi}\operatorname{diag}(\boldsymbol{l})\boldsymbol{\beta}^{\mathrm{T}}=1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}$, so exactly as for $S^{(\infty)}$, we know that $\Upsilon^{(\infty)}$ is non-singular. In fact,

$$\Upsilon^{(\infty)-1}=I+\frac{\sigma^{(\infty)}\boldsymbol{\beta}^{\mathrm{T}}\boldsymbol{\pi}\operatorname{diag}(\boldsymbol{l})}{1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}}.$$

Letting $\Omega=\operatorname{Var}[\boldsymbol{U}^{(\infty)}(\boldsymbol{\tau}^{(\infty)}\lambda^{\mathrm{T}})]$, we thus have that

$$(\bar{\boldsymbol{\alpha}}-\boldsymbol{\tau}\operatorname{diag}(\boldsymbol{l}))\sqrt{N}\xrightarrow{\mathscr{D}}\mathrm{N}(\mathbf{0},(\Upsilon^{(\infty)-1})^{\mathrm{T}}\Omega\Upsilon^{(\infty)-1}), \tag{5.1}$$

where

$$\begin{aligned}\Omega=&(1-\sigma^{(\infty)})\operatorname{diag}(\boldsymbol{l})\operatorname{diag}(\boldsymbol{\pi})\operatorname{diag}(\boldsymbol{l})-(1-\sigma^{(\infty)})^2\operatorname{diag}(\boldsymbol{l})\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\pi}\operatorname{diag}(\boldsymbol{l})\\&+\operatorname{diag}(\boldsymbol{\tau}^{(\infty)})\operatorname{diag}(\boldsymbol{J}).\end{aligned} \tag{5.2}$$

In the case $\boldsymbol{a}^{(v)}=\boldsymbol{a}$ for all v, then exactly as for final size the coupling argument of Ball (1983) shows that the distribution of $\boldsymbol{\alpha}^{(v)}$ converges to that of the corresponding quantity for the ρ-type continuous time branching process described in Section 4. The limiting distribution of $\boldsymbol{\alpha}^{(v)}$ thus has total probability mass $\Pi_{i=1}^{\rho}q_i^{a_i}$, and there remains the mass $1-\Pi_{i=1}^{\rho}q_i^{a_i}$ to account for. As before, we can show that if the limiting branching process never becomes extinct, then the asymptotic distribution of final severity is simply given by (5.1) with $\boldsymbol{\mu}^{(\infty)}$ set equal to $\mathbf{0}$ and $\boldsymbol{\tau}^{(v)}$ replaced by $\boldsymbol{\tau}^{(\infty)}$.

6. An equivalent multigroup model

Suppose that instead of each newly infected individual choosing its type at random from $\{1,2,\cdots,\rho\}$, each initial susceptible is deterministically assigned a label giving the type of infective which it will become if ever infected. Thus we now consider an epidemic in a closed population divided into ρ groups. For $i=1,2,\cdots,\rho$, group i initially consists of a_i infectives and $N\pi_i$ susceptibles. (We are now assuming that for all v, $N^{(v)}$ is such that $N^{(v)}\pi_i$ is an integer, $i=1,2,\cdots,\rho$.) Each initial group i infective remains so for a time distributed as $I^{(i)}$ and is then removed. During this time, the infective makes contacts at the points of a homogeneous Poisson process of rate β_i, each contact being with an individual chosen uniformly at random from the N initial

susceptibles, independently of all other events. If the individual contacted is still susceptible, then it becomes infected. Each newly infected group i individual remains so for a time distributed as $I^{(i)}$, during which it makes contacts at the points of a homogeneous Poisson process of rate β_i, and is then removed. All infectious periods and contact processes are independent of one another, and the epidemic terminates when no infectives remain. (Note that our earlier model could be regarded as a multigroup model in which the numbers of initial susceptibles in each group are random, as suggested by Ball (1990).)

The deterministic version of this model is clearly identical to the deterministic version of our previous model, with final outcome determined by (2.3). The stochastic version, on the other hand, is a special case of the generalised stochastic multitype epidemic model of Ball and Clancy (1993), for which asymptotic results were derived corresponding to those which we have found for our model with several types of infective. Thus if in the multigroup model we write $\bar{\boldsymbol{T}}'$ for the quantity corresponding to $\bar{\boldsymbol{T}}$, then in the case of an epidemic initiated by a large number of infectives we find that as the initially susceptible population becomes large,

$$(\bar{\boldsymbol{T}}' - \boldsymbol{\tau})\sqrt{N} \xrightarrow{\mathscr{D}} \mathrm{N}(\mathbf{0}, (S^{(\infty)-1})^{\mathrm{T}}\Xi' S^{(\infty)-1}),$$

where the matrix $S^{(\infty)}$ is as before, while Ξ' is given by

$$\Xi' = \sigma^{(\infty)}(1-\sigma^{(\infty)})\mathrm{diag}(\boldsymbol{\pi}) + \sigma^{(\infty)2}(\boldsymbol{\tau}^{(\infty)}\boldsymbol{K}^{\mathrm{T}})\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\pi}.$$

Thus we have

$$(S^{(\infty)-1})^{\mathrm{T}}\Xi' S^{(\infty)-1} = \left(\frac{\sigma^{(\infty)2}(1-\sigma^{(\infty)})}{1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}}\right)(\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\lambda}\,\mathrm{diag}(\boldsymbol{\pi}) + \mathrm{diag}(\boldsymbol{\pi})\boldsymbol{\lambda}^{\mathrm{T}}\boldsymbol{\pi})$$

(6.1)

$$+\left(\frac{\sigma^{(\infty)2}\boldsymbol{\tau}^{(\infty)}\boldsymbol{K}^{\mathrm{T}} + \sigma^{(\infty)3}(1-\sigma^{(\infty)})\boldsymbol{\lambda}\,\mathrm{diag}(\boldsymbol{\pi})\boldsymbol{\lambda}^{\mathrm{T}}}{(1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}})^2}\right)\boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\pi} + \sigma^{(\infty)}(1-\sigma^{(\infty)})\mathrm{diag}(\boldsymbol{\pi}).$$

In the case of an epidemic initiated by a trace of infection, the branching process part of the asymptotic final size distribution is the same for the multigroup model as for the model with several types of infective, while in the case of a major outbreak of infection the limiting distribution is the same as for an epidemic initiated by a large number of infectives, but with $\boldsymbol{\mu}^{(\infty)}$ set equal to $\mathbf{0}$ and $\boldsymbol{\tau}^{(\nu)}$ replaced by $\boldsymbol{\tau}^{(\infty)}$.

Notice that although the multigroup result given in Ball and Clancy (1993) strictly only applies when $\lambda_i > 0$ for all i, due to the particular structure of our model, the proof goes through even when this is not the case.

Now for the multigroup model, if $\bar{T}_i'$ is large for some i, then this suggests that the infective pressure in the population is large, and thus that $\bar{T}_j'$ will be large for all j. That is, we expect components of $\bar{\boldsymbol{T}}'$ to be positively correlated. From (6.1) it is clear that all elements of the limiting covariance matrix are indeed positive. (The correlation structures of certain epidemic models have been studied in rather greater detail by

Donnelly (1993).) In the case of our model with several types of infective, the situation is more complicated. The creation of a large number of type i infectives now implies that there are less susceptibles available to become infectives of other types, and so there will be both positive and negative contributions to the correlation between $\bar{T}_i$ and $\bar{T}_j$. This interference effect will clearly be more significant when the epidemic is large, so the negative contribution to correlation should be a decreasing function of $\sigma^{(\infty)}$. Now comparing variance matrices for the two models, we have first of all that

$$\Xi - \Xi' = (1-\sigma^{(\infty)})^2(\mathrm{diag}(\boldsymbol{\pi}) - \boldsymbol{\pi}^{\mathrm{T}}\boldsymbol{\pi}),$$

so the difference between the two models does indeed decrease as σ increases.

It also seems likely that the final size of the epidemic with several tyes of infective will be more variable than that of the equivalent multigroup model. Writing $\bar{T} = \bar{T}_1 + \bar{T}_2 + \cdots + \bar{T}_\rho$, $\tau = \tau_1 + \tau_2 + \cdots + \tau_\rho$, then we have

$$(\bar{T} - \tau)\sqrt{N} \xrightarrow{\mathscr{D}} \mathrm{N}(0, v),$$

where from (3.8),

$$v = (1-\sigma^{(\infty)}) + \frac{2\sigma^{(\infty)}(1-\sigma^{(\infty)})\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}}{1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}} + \frac{\sigma^{(\infty)2}\boldsymbol{\tau}^{(\infty)}\boldsymbol{K}^{\mathrm{T}} - (1-\sigma^{(\infty)})^2 + \sigma^{(\infty)2}(1-\sigma^{(\infty)})\boldsymbol{\lambda}\,\mathrm{diag}(\boldsymbol{\pi})\boldsymbol{\lambda}^{\mathrm{T}}}{(1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}})^2}.$$

For the equivalent multigroup model, we find similarly that $(\bar{T}' - \tau)\sqrt{N} \xrightarrow{\mathscr{D}} \mathrm{N}(0, v')$, where

$$v' = \sigma^{(\infty)}(1-\sigma^{(\infty)}) + \frac{2\sigma^{(\infty)2}(1-\sigma^{(\infty)})\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}}{1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}} + \frac{\sigma^{(\infty)2}\boldsymbol{\tau}^{(\infty)}\boldsymbol{K}^{\mathrm{T}} + \sigma^{(\infty)3}(1-\sigma^{(\infty)})\boldsymbol{\lambda}\,\mathrm{diag}(\boldsymbol{\pi})\boldsymbol{\lambda}^{\mathrm{T}}}{(1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}})^2}.$$

Hence we have

$$v - v' = \left(\frac{\sigma^{(\infty)}(1-\sigma^{(\infty)})}{1-\sigma^{(\infty)}\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}}}\right)^2 \{\boldsymbol{\lambda}\,\mathrm{diag}(\boldsymbol{\pi}) - (\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}})^2\}.$$

But now $\boldsymbol{E}[I^{(i^{(r)},r)} \mid i^{(r)} = i] = \lambda_i$ and $\Pr(i^{(r)} = i) = \pi_i$, and so

$$\mathrm{Var}[\boldsymbol{E}[I^{(i^{(r)},r)} \mid i^{(r)} = i]] = \boldsymbol{\lambda}\,\mathrm{diag}(\boldsymbol{\pi})\boldsymbol{\lambda}^{\mathrm{T}} - (\boldsymbol{\pi}\boldsymbol{\lambda}^{\mathrm{T}})^2.$$

Thus as expected, $v \geqq v'$. In fact $v - v'$ is proportional to $\mathrm{Var}[\boldsymbol{E}[I^{(i^{(r)},r)} \mid i^{(r)} = i]]$, which is a measure of the variation in the infective pressure exerted by a particular infective caused by that infective choosing its type at random.

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