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An SIS epidemiology game with two subpopulations

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There is significant current interest in the application of game theory to problems in epidemiology. Most mathematical analyses of epidemiology games have studied populations where all individuals have the same risks and interests. This paper analyses the rational-expectation equilibria in an epidemiology game with two interacting subpopulations of equal size where decisions change the prevalence and transmission patterns of an infectious disease. The transmission dynamics are described by an SIS model and individuals are only allowed to invest in daily prevention measures like hygiene. The analysis shows that disassortative mixing may lead to multiple Nash equilibria when there are two interacting subpopulations affecting disease prevalence. The dynamic stability of these equilibria is analysed under the assumption that strategies change slowly in the direction of self-interest. When mixing is disassortative, interior Nash equilibria are always unstable. When mixing is positively assortative, there is a unique Nash equilibrium that is globally stable.

Keywords: infectious disease; multiple populations; mixing patterns; differential inclusions

1. Introduction

The application of game theory to problems at the intersections of economics, public policy, and epidemiology is a relatively new area of study. While a handful of papers addressed the problem before the turn of the century [4,9,10,12,23], interest in games coupled to epidemiology has spread in the last 5 years. One of the central research tasks in this young field is an exploration of how different aspects of demography and epidemiology change the nature of solutions to the simple vaccination game and related models.

In the simple vaccination game for an endemic disease in an homogeneous population, there is a unique equilibrium behaviour. If the vaccine is expensive or unsafe and risk from infection is small, the equilibrium behaviour for individuals is to avoid vaccination. If vaccine is safe and cheap while the risks from infection are large, the equilibrium behaviour is to vaccinate at rates almost sufficient to eradicate disease. As the cost of vaccine varies continuously between these extremes, the equilibrium behaves in the same way [2,9]. At the extremes of very cheap and very expensive vaccination, the equilibrium solution for individuals is approximately the same as the Utopian ideal where everybody cooperates for the best interest of the community-at-large, but for

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intermediate costs of vaccine, there can be large differences between the Nash equilibrium and the community's ideal. Similar results hold for models where transmission is blocked by changes in behaviour [5].

This cartoon of the equilibrium dependence on the parameters can change in important ways as various generalizations are incorporated into the simple vaccination game and its variants to make them more realistic. Age-dependent virulence, for instance, may change the game such that instead of there being a unique equilibrium, there are three equilibria and two of them are locally stable [25]. Both fast and slow rates of vaccination may be equilibria when vaccine provides imperfect protection [7,20]. Other effects are likely to be discovered in the coming years, as the depth of our exploration of this field grows. One under-explored generalization is the subdivision of populations into two or more interacting subpopulations. Subpopulation structure, and interacting populations in general, will be our focus in this paper.

Several articles have studied epidemiology games with subpopulation structure. Some models have been structured solely in terms of the costs faced by individuals [4,5,7,24]. In these cases, the equilibrium solutions to the games are consistent with the cartoon of the simple vaccination game. Two papers have explicitly addressed epidemiology games where the populations are also biologically structured. In Galvani *et al.* [11], the authors study an influenza vaccination in an age-structured population, but with the age groups treated as distinct subpopulations rather than life stages in a single population. Galvani *et al.* find unique equilibria for the parameter ranges studied, but the numerical results provide limited insight into the general case. A more tractable model is studied by Chen [6]. In that paper, the author studies an SI model with varying degrees of positive assortative mixing, where individuals can invest in self-protective behaviour. The author finds that for all the parameter values investigated, there is a unique equilibrium solution to the game.

The results of these papers suggest that population structure, per say, does not qualitatively change the solution set of epidemiology games. But the various extremes of simplicity and complexity in the studies to date leave the reasons for this pattern murky. This paper is intended as a start to clearing the waters with regards to the effects of subpopulation structure. First, we describe the generalization of the approach of Reluga *et al.* [25] for modelling rational decisions of individuals in games with multiple interacting populations. We will then adapt the SIS model studied in [5] to describe two interacting populations with a full range of mixing patterns and study the equilibrium solutions to a population game of self-protective behaviours. A graphical method for the analysis is presented. Consistent with the results of Chen [6], there is always a unique Nash equilibrium when mixing is positively assortative. However, in the case of disassortative mixing, there may be up to three isolated Nash equilibria. Thus, population structure can qualitatively change the behaviour of equilibria in epidemiology games. We then propose a differential inclusion for strategy dynamics and show that interior Nash equilibria can be unstable in the case of disassortative mixing, but are stable in the case of assortative mixing.

2. Methods

We begin with the extension of the methods used in Reluga *et al.* [25] to population games with multiple interacting populations. A population game contains both population-scale components that describe the states of the populations and individual-scale components that describe the states of individuals within these populations. Suppose we have a set of n populations labelled $i = 1, \ldots, n$. Within a population, an individual occupies one of many possible states. The number of individuals in population i occupying each state at time t is represented by a vector $\boldsymbol{x}_i(t)$ (vectors and matrices will be typeset in bold). We refer to $\boldsymbol{x}_i(t)$ as population i's state, with $x_{ij}(t)$ representing the number of individuals from population i in state j at time t. The state of an

individual in population i at time t is represented by a probability measure $\boldsymbol{p}_i(t)$ over the possible states. We refer to $\boldsymbol{p}_i(t)$ as the individual's state.

We wish to study the structure of a set of functions

$$U_i(\boldsymbol{\pi}_i, \overline{\boldsymbol{\pi}}_1 \ldots \overline{\boldsymbol{\pi}}_n) \tag{1}$$

representing expected utility to an individual from population i playing strategy $\boldsymbol{\pi}_i$ if all other individuals are playing the resident strategies $\overline{\boldsymbol{\pi}}_1, \ldots, \overline{\boldsymbol{\pi}}_n$. We call the individual's strategy $\boldsymbol{\pi}_i$ an invading strategy to distinguish it from each population's resident strategy $\overline{\boldsymbol{\pi}}_i$.

Before calculating the utilities U_i, we must first study the dynamics of the processes of interest. The population-scale and individual-scale dynamics are driven by separate but related processes. Suppose the populations exist in an environment with state vector $\boldsymbol{e}(t)$. The population-scale dynamics satisfy a system of differential equations where the rates of change in the states of population i depend on the states of the other populations, the resident strategies, the environmental state, and the time;

$$\frac{\mathrm{d}\boldsymbol{x}_i}{\mathrm{d}t} = G_i(\boldsymbol{x}_1, \ldots, \boldsymbol{x}_n, \overline{\boldsymbol{\pi}}_1, \ldots, \overline{\boldsymbol{\pi}}_n, \boldsymbol{e}, t). \tag{2}$$

The rate of change in the environment's state also depends on the states of the populations, the resident strategies, the environment's current state, and time, or

$$\frac{\mathrm{d}\boldsymbol{e}}{\mathrm{d}t} = G_{\boldsymbol{e}}(\boldsymbol{x}_1, \ldots, \boldsymbol{x}_n, \overline{\boldsymbol{\pi}}_1, \ldots, \overline{\boldsymbol{\pi}}_n, \boldsymbol{e}, t). \tag{3}$$

We assume the populations are so large that the population dynamics are insignificantly affected by the few individuals with invading strategies that differ from the resident strategies.

While the large population sizes let us describe the population-scale dynamics deterministically, the individual-scale dynamics for an individual in population i with invading strategy $\boldsymbol{\pi}_i$ are stochastic. Each individual has risks and opportunities, but these can differ between individuals depending on the course of their life. Suppose that the state of an individual is governed by a continuous-time Markov process. The individual-scale dynamics describe changes in the probability that an individual will be found in any given state using a linear differential equation where the transition rates depend on the population states, the environment's state, time, and the individual's strategy, or

$$\frac{\mathrm{d}\boldsymbol{p}_i}{\mathrm{d}t} = \mathbf{Q}_i(\boldsymbol{x}_1, \ldots, \boldsymbol{x}_n, \boldsymbol{e}, t, \boldsymbol{\pi}_i)\boldsymbol{p}_i, \tag{4}$$

where $\mathbf{Q}_i$ is a matrix of transition rates. We have allowed the transition rate to only depend on the resident strategies through the population's states, but this is easily relaxed.

Individuals may choose behaviour strategies based on several different criteria. One way of quantifying an individual's decision process is to define an 'expected utility' function that associates a payoff to each strategy choice and to then assume individuals make decisions to maximize this payoff. This approach is commonly referred to as 'rational-expectation' theory in economics. The expected utility for an individual with an invading strategy can be calculated based on the individual-scale dynamics. Given an initial time $t_0 = 0$ and an infinite horizon, the utility of an invading strategy $\boldsymbol{\pi}_i$ to an individual in population i given resident population strategies $\overline{\boldsymbol{\pi}}_1, \ldots, \overline{\boldsymbol{\pi}}_n$ is

$$U_i(\boldsymbol{\pi}_i, \overline{\boldsymbol{\pi}}_1, \ldots, \overline{\boldsymbol{\pi}}_n) = \int_0^\infty \mathrm{e}^{-h_i t}\boldsymbol{v}_i^{\mathrm{T}}\boldsymbol{p}_i(t)\mathrm{d}t, \tag{5}$$

where h_i is the discount rate in population i and $\boldsymbol{v}_i$ is a vector of felicities, a.k.a. utility gains, for residence in each state in population i. Equation (5) can be interpreted as a path integral over an individual's possible life-histories and can be derived from the Markov decision process theory

developed by Howard [16]. The felicities have two parts: a vector of expected gains per unit time $\boldsymbol{f}_i$ for each state, and a matrix $\mathbf{F}_i$ of instantaneous utility gains associated with the transitions between states, where $F_{i,jk}$ is the expected instantaneous gain of an individual in population i jumping from state k to state j. Using a result of Howard [16], it is shown that

$$\boldsymbol{v}_i^{\mathrm{T}} = \boldsymbol{f}_i^{\mathrm{T}} + \mathbf{1}^{\mathrm{T}}(\mathbf{F}_i \circ \mathbf{Q}_i), \tag{6}$$

where $\mathbf{1}$ is a vector of 1's and $\circ$ represents the Hadamard product of two matrices. The Hadamard product of two matrices is the matrix of the product of the components: $(\mathbf{F}_i \circ \mathbf{Q}_i)_{jk} = F_{i,jk}Q_{i,jk}$.

While population games can be solved and studied in the general form we have so far presented, it is often useful to focus on cases where the population-scale dynamics have a simple attractor. If all dynamics are autonomous and $(\boldsymbol{x}_1^*, \ldots, \boldsymbol{x}_n^*, \boldsymbol{e}^*)$ is a stationary solution of the population-scale dynamics, the transition-rate matrix

$$\mathbf{Q}_i^* = \mathbf{Q}_i(\boldsymbol{x}_1^*, \ldots, \boldsymbol{x}_n^*, \boldsymbol{e}^*, \boldsymbol{\pi}_i) \tag{7}$$

is also stationary and

$$\boldsymbol{p}_i(t) = \mathrm{e}^{-t\mathbf{Q}_i^*}\boldsymbol{p}_i(0). \tag{8}$$

If there is a positive discount rate $h_i > 0$ or the Markov process is transient, e.g. the dominate eigenvalue $\lambda_0(\mathbf{Q}_i^*) < 0$, then over the infinite time horizon, the expected utility has a closed form

$$U_i = \boldsymbol{v}_i^{\mathrm{T}}(h_i\mathbf{I} - \mathbf{Q}_i^*)^{-1}\boldsymbol{p}_i(t_0), \tag{9}$$

where $\mathbf{I}$ is the identity matrix.

3. Application in an SIS model

We now study the rational-expectation equilibria of utility functions calculated using the methods described above when the dynamics are governed by a standard epidemic model. We will first describe a stochastic model at the individual scale and use the result to motivate a population-scale model that is used in turn to close the equations for the utility calculation. To study the equilibrium structure of an epidemic population game with interacting populations, we will focus on a 2-population SIS model that is a variation of the work of Chen [5]. In this model, individuals can reduce their risk of infection by adopting costly behaviours that prevent transmission and in-turn change the prevalence of the infectious agent.

Take a population of individuals of a single species that is split into two distinct subpopulations. All individuals of this species can be infected by the same pest or pathogen. The situation can be interpreted as representing, for example, the transmission of lice within and between two classrooms of students in an elementary school or the sexual transmission of gonorrhoea between men and women. In order to conduct our analysis, we are omitting the biological details like stochasticity needed in practical models of lice or gonorrhoea transmission, so our results should not be applied 'out-of-the-box' to either of these cases. Rather, the results should guide future research and applications in disease-specific studies.

The duration of infectiousness is assumed to be exponentially distributed with expectation $1/\gamma$, independent of an individual's subpopulation. When individuals recover, they do not gain any form of immunity against reinfection, so they immediately return to a susceptible state. The risk of infection to a susceptible individual in subpopulation i is proportional to the force of infection λ_i. Individuals may choose to reduce their personal risk to a fraction σ_i relative to the

background risk per day by changing behaviours. Throughout the paper, the relative risk σ_i will be referred to as the individual's strategy, since its value is determined by an individual's choices.

Under these assumptions, the changes in an individual's state are described by a continuous-time Markov process

$$\frac{\mathrm{d}\boldsymbol{p}_i}{\mathrm{d}t} = \mathbf{Q}_i \boldsymbol{p}_i, \tag{10a}$$

where $\boldsymbol{p}_i(t)$ is the probability that an individual in population i is susceptible or infected at time t and the transition-rate matrix

$$\mathbf{Q}_i = \begin{bmatrix} -\lambda_i\sigma_i & \gamma \\ \lambda_i\sigma_i & -\gamma \end{bmatrix}. \tag{10b}$$

Every individual in the population starts in the susceptible state, so

$$\boldsymbol{p}_i(0) = [1 \;\; 0]^{\mathrm{T}}. \tag{10c}$$

Equation (10) is non-autonomous and cannot be solved in closed form without further assumptions because the force of infection λ_i is time-dependent.

The force of infection seen by an individual is determined at the population-scale [22]. In a population with sufficiently strong mixing, we can represent the transmission dynamics using a standard SIS epidemic model. In subpopulation i, let $S_i(t)$ represent the number of susceptible individuals at time t and $I_i(t)$ represent the number infected individuals at time t. To simplify our analysis, let us assume that the two subpopulations have the same sizes which we have normalized to 1, so that $S_1 + I_1 = 1$ and $S_2 + I_2 = 1$.

The forces of infection λ_i will be modelled as a weighted sum of the number of infected individuals in each subpopulation:

$$\lambda_1 = \beta_{11} I_1 + \beta_{21} I_2, \tag{11a}$$

$$\lambda_2 = \beta_{12} I_1 + \beta_{22} I_2, \tag{11b}$$

where β_{ij} represents the background transmission rate from individuals of subpopulation i to individuals of subpopulation j. Since there are no temporal changes in the total population sizes, this may be interpreted as either standard incidence or bilinear incidence. Unless otherwise specified, we will assume the matrix $[\beta_{ij}]$ is positive.

To scale up the effects of individual choices to the population dynamics, we make the simplest reasonable supposition. In subpopulation i, almost all individuals use the behaviours that reduce their infection rate to a fraction $\overline{\sigma}_i$ of the background infection rate. We call $\overline{\sigma}_1$ and $\overline{\sigma}_2$ the resident strategies and for shorthand we write $\overline{\sigma} = (\overline{\sigma}_1, \overline{\sigma}_2)$. Under this postulate, population-scale dynamics of infection are governed by the system of differential equations

$$\dot{S}_1 = \gamma I_1 - \lambda_1 \overline{\sigma}_1 S_1, \tag{12a}$$

$$\dot{I}_1 = \lambda_1 \overline{\sigma}_1 S_1 - \gamma I_1, \tag{12b}$$

$$\dot{S}_2 = \gamma I_2 - \lambda_2 \overline{\sigma}_2 S_2, \tag{12c}$$

$$\dot{I}_2 = \lambda_2 \overline{\sigma}_2 S_2 - \gamma I_2. \tag{12d}$$

The SIS model described by Equation (12) and it variants have been studied extensively in the past [3,14].

4. Game equilibria

To formulate a population game, we must determine utilities for various strategies. As described above, we can calculate utilities by inspecting the costs and benefits of isolated events in an individual's life and adding them all up under appropriate discounting.

For the purpose of our analysis, we propose that individuals must balance two simple costs. One cost, $c_i(I)$, is the cost paid per day of infection by an individual of subpopulation i. The other cost, $c_i(B)$, is the cost of self-protecting behaviours per day per percent reduction in infection risk. For a fixed force of infection, we might expect an individual's risk of infection to be a monotone decreasing function of their investment in self-protection. It will be convenient to assume that the daily risk of infection is a convex function reaching 0 at a finite value of protection investment. There are economic arguments why risk may not be a convex function of pure strategies and good biological reasons why risk may never reach 0. However, these issues would complicate our analysis. In this model, we will assume that an individual must invest $(1-\sigma_i)c_i(B)$ per day to reduce one's risk to a fraction σ_i of the background risk in population i. Thus, the vectors of felicities for the susceptible or infected states in each subpopulation i will be

$$\boldsymbol{v}_i = [-(1-\sigma_i)c_i(B), -c_i(I)]^{\mathrm{T}}. \tag{13}$$

Our model places some important restrictions on the possible applications of the results. For instance, we assume that the prevention is a daily hygiene activity like hand-washing and does not change the individual's biological state. Preventative measures like vaccination cannot be represented here because they introduce a long-term change in an individual's state for a one-time cost, which we have not allowed (but see [1,11,24]). Similarly, this system does not immediately apply to antiviral prevention, since antivirals change an individual's state in a way that affects not only susceptibility but also recovery. The costs of infection are also daily costs that persist for exactly the duration of infection. This does not allow for treatment, since treatment would involve a choice that could shorten the duration of infection and alter daily costs. Nor have we allowed for long-term disabilities incurred from infection because the SIS theory assumes recovered individuals are indistinguishable from naive individuals. All of these events and others could be incorporated into our system with the addition of appropriate states, transitions, costs, and control variables, but would lengthen and complicate our analysis.

Given individual-scale and population-scale models (Equations (10) and (12), respectively), together with felicities of possible events and behaviours, we can now investigate the equilibria available. For an equilibrium analysis, we assume system (12) is at the globally attracting stationary solution, with stationary transition rates $\mathbf{Q}_i^*$ and stationary forces of infection λ_i^*. Calculated over an infinite time horizon, the utility of individual strategy σ_i is

$$U_i(\sigma_i, \overline{\sigma}_1, \overline{\sigma}_2) = \boldsymbol{v}_i^{\mathrm{T}}(h\mathbf{I} - \mathbf{Q}_i^*)^{-1}\boldsymbol{p}_i(0) = \frac{-c_i(B)(1-\sigma_i)(h+\gamma) - c_i(I)\lambda_i^*\sigma_i}{h(h+\gamma+\lambda_i^*\sigma_i)}, \tag{14}$$

where λ_i^* is an implicit function of $\overline{\sigma}_1$ and $\overline{\sigma}_2$.

The discount rate $h = h_1 = h_2$ acts as a catch-all for independent environmental risks that reduce incentives for planning ahead. In the limit of large discount rates, the future risks from infection are very small compared with the current costs of prevention. In the limit of small discount rates, the utility diverges because every future illness has the same costs as a current illness. Thus, tuning h allows us to account for some risks external to our epidemic model.

Since the subpopulation sizes equal 1 by assumption, $S_i = 1 - I_i$ at all times. After this substitution, determining the stationary solutions to Equation (12) reduces to simultaneously solving

the pair of quadratic equations

$$\overline{\sigma}_1(1 - I_1^*)(\beta_{11}I_1^* + \beta_{21}I_2^*) - \gamma I_1^* = 0, \tag{15a}$$

$$\overline{\sigma}_2(1 - I_2^*)(\beta_{12}I_1^* + \beta_{22}I_2^*) - \gamma I_2^* = 0. \tag{15b}$$

One stationary solution is the disease-free stationary solution $(S_1, I_1, S_2, I_2) = (1, 0, 1, 0)$. The disease-free stationary solution is globally attracting so long as the reproductive number $\mathcal{R} < 1$ [14] (see Appendix A). If $\mathcal{R} > 1$, there is exactly one stationary solution with positive infection prevalence, and it is globally attracting [3]. We will refer to this solution as the endemic stationary solution.

The role of between-subpopulation transmission relative to within-subpopulation transmission in asymptotic disease prevalences can be partially understood in terms of source-sink concepts. A subpopulation is a source of infections if the infection can persist in that population in isolation. A subpopulation is a sink population if infection cannot persist in the subpopulation in isolation. Subpopulation i is a source if $\beta_{ii}/\gamma > 1$ and a sink if $\beta_{ii}/\gamma < 1$. If either subpopulation is a source, then $\mathcal{R} > 1$. But in some cases, between-subpopulation transmission can be strong enough such that $\mathcal{R} > 1$ even when both subpopulations alone are sinks.

The endemic stationary solution has the property that it is decreasing in the resident strategies $\overline{\sigma}_i$, and strictly decreasing so long as $\mathcal{R} > 1$, $\overline{\sigma}_1\beta_{11}/\gamma > 1$, and $\overline{\sigma}_2\beta_{22}/\gamma > 1$ (see Appendix A and Figure 1). The monotonicity of the endemic stationary solution makes it easier for us to calculate the game equilibria. As the resident strategies $\overline{\sigma}$ are varied, there is a region of reachable stationary disease prevalences. The shape of this region depends on whether the subpopulations are sources or

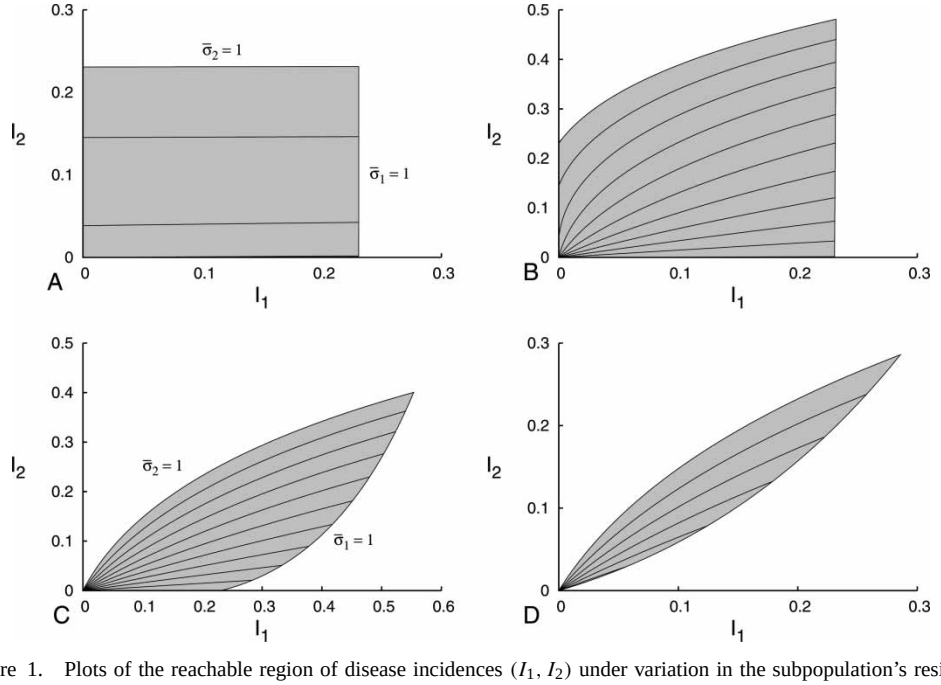

Figure 1. Plots of the reachable region of disease incidences (I_1, I_2) under variation in the subpopulation's resident strategies $\overline{\sigma}_1$ and $\overline{\sigma}_2$. Each plot corresponds to a different pattern of transmission rates, given $\gamma = 1$. The black curves within each region are curves of constant $\overline{\sigma}_2$. The right boundary of the reachable region has $\overline{\sigma}_1 = 1$ and the top boundary of the reachable region has $\overline{\sigma}_2 = 1$. (A) No transmission between subpopulations. $\beta_{11} = 1.3$, $\beta_{12} = 0$, $\beta_{21} = 0$, $\beta_{22} = 1.3$. (B) Unidirectional transmission between two source subpopulations. $\beta_{11} = 1.3$, $\beta_{12} = 0$, $\beta_{21} = 1.3$, $\beta_{22} = 1.3$.(C) Subpopulation 1 is a source, subpopulation 2 is a strong sink. $\beta_{11} = 1.3$, $\beta_{12} = 1.3$, $\beta_{21} = 0.7$, $\beta_{22} = 0.7$. (D) Both subpopulations are sinks and cross-transmission is necessary for persistence. $\beta_{11} = 0.7$, $\beta_{12} = 0.7$, $\beta_{21} = 0.7$, $\beta_{22} = 0.7$.

sinks (Figure 1). For each point in the reachable region of prevalences, we can uniquely determine the forces of infection exerted on individuals in each subpopulation. Individual best responses are correspondences depending on these forces (a 'correspondence' maps each element of the domain to a subset of the range). From the individual utilities, we can see that the best responses are step functions. Differentiating the utility with respect to σ_i, we find that the best-response correspondence is given by

$$\underset{\sigma_i}{\operatorname{argmax}}\, U_i(\sigma_i, \overline{\sigma}_1, \overline{\sigma}_2) = \begin{cases} 0 & \text{if } \lambda_i^*(\overline{\sigma}_1, \overline{\sigma}_2) > \lambda_i^+, \\ [0,1] & \text{if } \lambda_i^*(\overline{\sigma}_1, \overline{\sigma}_2) = \lambda_i^+, \\ 1 & \text{if } \lambda_i^*(\overline{\sigma}_1, \overline{\sigma}_2) < \lambda_i^+, \end{cases} \tag{16}$$

where the threshold forces of infection

$$\lambda_i^+ = \begin{cases} \dfrac{c_i(B)(h+\gamma)}{c_i(I) - c_i(B)} & \text{if } c_i(I) > c_i(B), \\ +\infty & \text{otherwise.} \end{cases} \tag{17}$$

The bang-bang form of this best-response correspondence is reminiscent of linear optimal control theory and is a direct consequence of Theorem B.1 in Appendix B. If the force of infection is below the threshold λ_i^+ in subpopulation i, individuals prefer no self-protection ($\sigma_i = 1$). If the force of infection is above the threshold value λ_i^+, individuals prefer complete self-protection ($\sigma_i = 0$). If the force of infection is exactly the threshold value, individuals will have the same utility for all self-protection strategy choices. This last condition implies that if the threshold forces of infection intersect within the reachable region, the strategy pair $\sigma^+ = (\sigma_1^+, \sigma_2^+)$ for the disease prevalences corresponding to the intersection ($\lambda_1^* = \lambda_1^+, \lambda_1^* = \lambda_1^+$) must constitute a Nash equilibrium.

In general, a strategy pair (σ_1^*, σ_2^*) is a Nash equilibria of the utility functions (U_1, U_2) if the strategies are best-responses to themselves for all individuals in all subpopulations. The variational-inequality approach described in Cojocaru *et al.* [8] supplies one approach to solving these equations. Instead, we will adopt a graphical approach. The Nash equilibria to the population game can be determined by plotting the threshold forces of infection over the reachable region (Figure 2). The best-response correspondences are step functions, so all equilibria of the game must lie on the boundaries of the feasible region and at threshold values of the forces of infection. If the stationary prevalences in the absence of self-protection are below both threshold forces of infection for both subpopulations ($\lambda_i^*(\overline{\sigma}_1 = 1, \overline{\sigma}_2 = 1) < \lambda_i^+$ for each i), then the only Nash equilibrium will be no self-protection in either subpopulation ($\sigma_1^* = \sigma_2^* = 1$) (Figure 2A). If the threshold forces of infection are not positive ($\lambda_1^+ \leq 0, \lambda_2^+ \leq 0$), then complete self-protection $\sigma_1^+ = \sigma_2^+ = 0$ is the best response under all stationary prevalences, and thus is the unique Nash equilibrium.

In between these extremes, one or both subpopulations will prefer some intermediate investment in self-protective behaviours. At this point, it is convenient to define some terminology. Let the set

$$T_j = \{(I_1^*, I_2^*) : \beta_{1j} I_1^* + \beta_{2j} I_2^* \geq \lambda_j^+\}. \tag{18}$$

We say that subpopulation i has a lower threshold than subpopulation j if and only if $T_j \subsetneq T_i$ and that subpopulation i has a higher threshold than subpopulation j if and only if $T_i \subsetneq T_j$.

If the endemic stationary solution in the absence of self-protection ($\overline{\sigma} = (1,1)$) generates a force of infection greater than the threshold force of infection in atleast one subpopulation and no disease prevalences in the feasible region impose forces of infection at threshold levels for both subpopulations (the thresholds plotted in Figure 2 do not cross), there will be a unique Nash

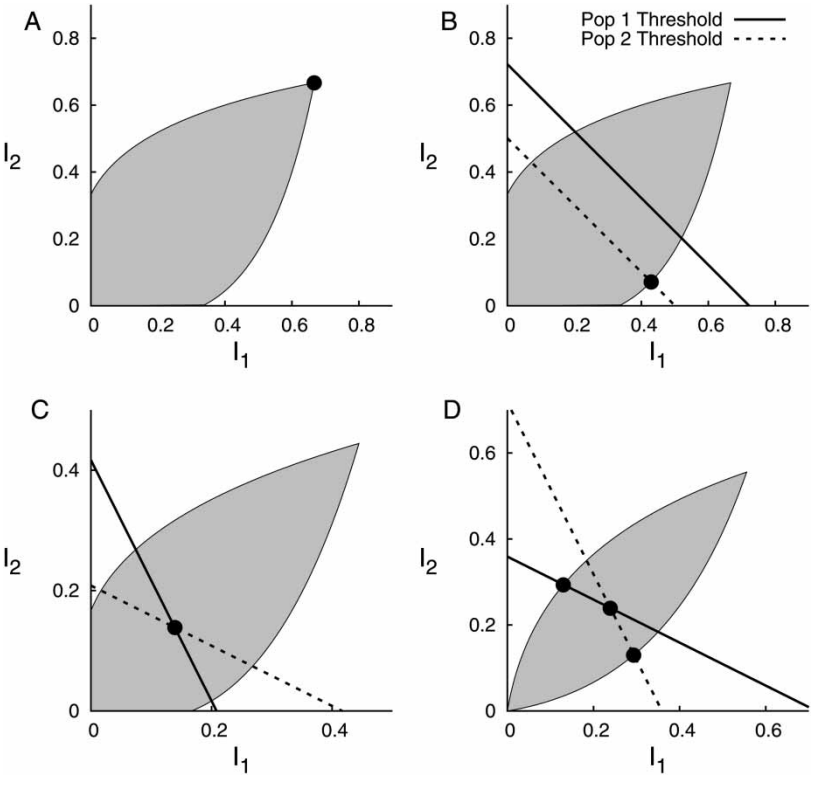

Figure 2. Depending on the transmission patterns and the relative costs of protection to infection, there may be one or three solutions to the population game. The threshold forces of infection are plotted as lines over the reachable regions of disease incidence and the dots mark game equilibria. If the costs of protection are sufficiently large for both populations, the endemic prevalences will be an equilibrium state (A). If the costs of protection are lower, but do not cross with the reachable region of prevalences, the costs of protection are solely carried by the population with the lower threshold (B). If the thresholds cross within the reachable region and the direct transmission risks are greater than the indirect transmission risks ($\beta_{11}\beta_{22} > \beta_{12}\beta_{21}$), there is a single internal equilibrium (C). If the thresholds cross within the reachable region and the direct transmission risks are less than the indirect transmission risks ($\beta_{11}\beta_{22} < \beta_{12}\beta_{21}$), there will be three equilibria (D). Parameter values: $h = 0$, $\gamma = 1$, $c_1(I) = c_2(I) = 1$.

equilibrium with some self-protection in the subpopulation with the lower threshold and less-than-complete self-protection in the subpopulation with the higher threshold. To illustrate this, let us assume that, as in Figure 2B, subpopulation 2 has a lower threshold than subpopulation 1. If self-protection within subpopulation 2 alone can reduce the force of infection to the threshold value λ_2^+ of subpopulation 2, then there is a unique Nash equilibrium $\sigma^{\lrcorner}$ with $\sigma_1^{\lrcorner} = 1, 0 < \sigma_2^{\lrcorner} < 1$, where $\lambda_1^* < \lambda_1^+$ and $\lambda_2^* = \lambda_2^+$. If self-protection within subpopulation 2 alone cannot reduce the force of infection to the threshold of subpopulation 2 but can reduce the force of infection below the threshold of subpopulation 1, then there is a unique Nash equilibrium $\sigma^{\lrcorner}$ with $\sigma_1^{\lrcorner} = 1$, $\sigma_2^{\lrcorner} = 0$. If self-protection within subpopulation 2 alone cannot reduce the force of infection below the thresholds of either subpopulation, then there is a unique Nash equilibrium $\sigma^{\lrcorner}$ with $\sigma_2^{\lrcorner} = 0$, $0 < \sigma_1^{\lrcorner} < 1$, where $\lambda_1^* = \lambda_1^+$. There is a symmetrically defined set of rules for a unique Nash equilibrium $\sigma^{\ulcorner}$ if subpopulation 1 has a lower threshold than subpopulation 2.

If there are disease prevalences within the reachable region such that the stationary forces of infection equal their threshold values for both subpopulations simultaneously (as in Figure 2C and D), then, as stated above, the corresponding strategy pair σ^+ is a Nash equilibrium. The uniqueness of this equilibrium depends on the sign of the threshold intersection.

If $\beta_{11}\beta_{22} - \beta_{12}\beta_{21} > 0$, then the mixing pattern for transmission is assortative and self-protective behaviours preferentially reduce transmission within the same subpopulation. When the intersection of the thresholds occurs within the reachable region, self-protection that reduces the force of infection to threshold levels within a single subpopulation is insufficient to reduce the forces of infection below threshold levels in the other subpopulation. There will be no Nash equilibria on the boundaries of the reachable region, and the interior equilibrium is the unique Nash equilibrium (Figure 2C). This is a minor variation of the special case considered in [6].

If $\beta_{11}\beta_{22} - \beta_{12}\beta_{21} < 0$, then the mixing pattern for transmission is disassortative, and self-protective behaviours preferentially reduce the force of infection in the other subpopulation. When the intersection σ^+ of the thresholds occurs within the reachable region, self-protection within either subpopulation in isolate can reduce the forces of infection in both subpopulations below threshold levels (Figure 2D). The interior equilibrium will not be unique because there will be Nash equilibria on upper left ($\sigma^{\ulcorner}$) and lower right ($\sigma^{\lrcorner}$) boundaries of the reachable region. Thus, while assortative mixing leads to unique equilibria, disassortative mixing can create multiple Nash equilibria. In degenerate cases when the threshold forces of infection coincide in both populations ($\beta_{11}\beta_{22} - \beta_{12}\beta_{21} = 0$ and $\lambda_1^+\beta_{22} - \lambda_2^*\beta_{21} = 0$), there is a whole segment of Nash equilibria.

5. Stability of the game equilibria

A Nash equilibrium is a situation where individuals have no reason to change their minds about strategies if everything starts out just right. But a strategy that lets us 'win' the game no matter how things start out would be even more useful. This leads us to the concepts of convergence and invasion potential. In particular, if the resident strategies are near the equilibrium, can individuals do better by using the equilibrium strategies? As Figure 3 shows, when the resident strategies are perturbed from the internal equilibrium σ^+, the internal equilibrium may or may not be able to re-invade depending on the direction of the perturbation. Local invasion potential can be perturbation-dependent for either orientation of the crossings. In fact, an interior Nash equilibrium can only have universal local invasion potential if the response thresholds for both populations are locally independent. By independent, we mean that if (σ_1^*, σ_2^*) is an internal Nash equilibrium where

$$U_1(\sigma_1^*, \lambda^*(\sigma^*)) = U_1([0,1], \lambda^*(\sigma^*)) \quad \text{and} \quad U_2(\sigma_2^*, \lambda^*(\sigma^*)) = U_2([0,1], \lambda^*(\sigma^*)), \tag{19}$$

then the best-response thresholds $\hat{\sigma}_1(\overline{\sigma}_2)$ and $\hat{\sigma}_2(\overline{\sigma}_1)$ are locally independent when

$$\left.\frac{\partial \hat{\sigma}_1(\overline{\sigma}_2)}{\partial \overline{\sigma}_2}\right|_{\overline{\sigma}_2=\sigma_2^*} = 0 \quad \text{and} \quad \left.\frac{\partial \hat{\sigma}_2(\overline{\sigma}_1)}{\partial \overline{\sigma}_1}\right|_{\overline{\sigma}_1=\sigma_1^*} = 0. \tag{20}$$

So interior Nash equilibria with different orientations of the threshold crossings are not distinguished simply by the local invasion potential. However, it does seem that there should be some fundamental dynamic difference between assortative and disassortative mixing systems (Figure 2C and D, respectively).

To formalize our intuition about dynamics, we adopt a variation of the adaptive-dynamics approach. Assume resident strategies change sufficiently slowly to preserve the stability of the endemic disease equilibria and always change in the direction of *individuals'* best interests, but the relative rates of change are unknown. Can we say anything about stability of stationary solutions? This problem is one of establishing the stability of a stationary solution in a differential inclusion.

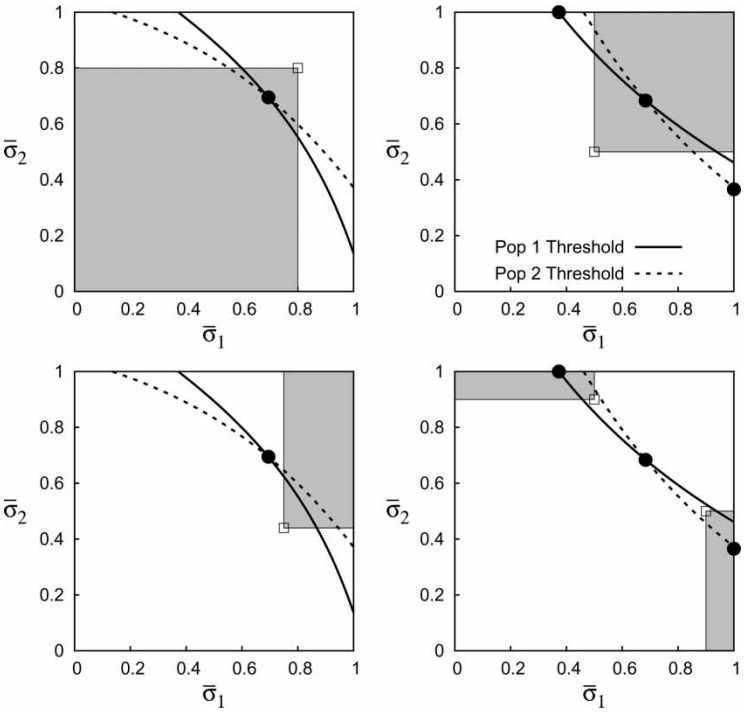

Figure 3. Regions of individual strategy pairs (shaded rectangles) that can invade resident strategies (open squares at the corners). Nash equilibria are marked with a solid dot. Individuals using any strategies within a shaded region can invade against the resident strategy pair marked at the interior corner. The left plots correspond to the parameter values in Figure 2C. The right plots correspond to Figure 2D. In both cases, we see that the interior Nash equilibria invade some nearby resident strategies (top) but not others (bottom). The solid line represents the set of resident strategy pairs solving the threshold condition $\lambda_1^*(\overline{\sigma}_1, \overline{\sigma}_2) = \lambda_1^+$, and the dotted line represents resident strategy pairs solving the threshold condition $\lambda_2^*(\overline{\sigma}_1, \overline{\sigma}_2) = \lambda_2^+$.

Suppose that

$$\dot{\overline{\sigma}}_1 = G_1(t)[A_1(\overline{\sigma}_1, \overline{\sigma}_2) - \overline{\sigma}_1], \tag{21a}$$

$$\dot{\overline{\sigma}}_2 = G_2(t)[A_2(\overline{\sigma}_1, \overline{\sigma}_2) - \overline{\sigma}_2], \tag{21b}$$

where $G_1(t)$ and $G_2(t)$ are piecewise continuous functions with finite upper bounds and positive lower bounds, and

$$A_i(\overline{\sigma}_1, \overline{\sigma}_2) = \begin{cases} 0 & \text{if } \lambda_i^*(\overline{\sigma}_1, \overline{\sigma}_2) > \lambda_i^+, \\ \overline{\sigma}_i & \text{if } \lambda_i^*(\overline{\sigma}_1, \overline{\sigma}_2) = \lambda_i^+, \\ 1 & \text{if } \lambda_i^*(\overline{\sigma}_1, \overline{\sigma}_2) < \lambda_i^+. \end{cases} \tag{21c}$$

Our choice of A_i is dictated by the assumption that resident strategies will change in the direction of individual self-interest. Under this description of strategy dynamics, the resident strategy of each population changes slowly in the direction preferred by the self-interest of individuals, but the relative rates of change can be arbitrary. Substitution shows the Nash equilibria strategy pairs are stationary solutions of the dynamics. The differential equations are non-autonomous and there may be discontinuities in the coefficients, but we can still obtain some stability results for the equilibria.

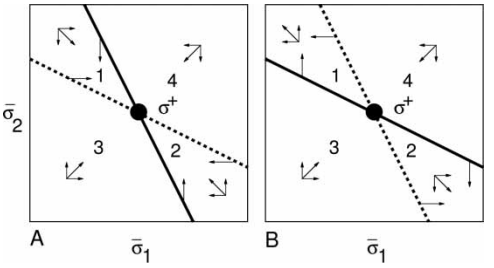

Figure 4. Cartoon of the phase plane for the differential inclusion described by Equation (21) in the neighbourhood of the σ^+ (large dot) when (A) $\beta_{11}\beta_{22} - \beta_{12}\beta_{21} > 0$ and (B) $\beta_{11}\beta_{22} - \beta_{12}\beta_{21} < 0$. The solid line is the threshold for population 1. The dotted line is the threshold for population 2. The arrows clusters represent the cones of available directions within each of the four quadrants labelled 1–4 and along the thresholds.

THEOREM 5.1 *Suppose the Nash equilibrium σ^+ exists for the population game described by Equation* (14)*. If $\beta_{11}\beta_{22} - \beta_{12}\beta_{21} > 0$, then σ^+ is a locally stable solution to Equation* (21)*. If $\beta_{11}\beta_{22} - \beta_{12}\beta_{21} < 0$, then σ^+ is a locally unstable stationary solution to Equation (21).*

Proof It will be helpful to refer to the cartoon shown in Figure 4, where the thresholds divide the resident-strategy phase-space up into quadrants numbered 1–4 in the neighbourhood of an interior Nash equilibrium σ^+. First, we prove that the orientations of the thresholds in strategy space are the same as orientations in incidence space. Then we study stability based on the threshold orientations in strategy space.

For each pair of resident strategies and each time, the velocity of change in system (21) takes on one of a cone of values bounded away from 0 except at the Nash equilibria. These cones can be characterized based on quadrant, as in the examples in Figure 4. The nullclines of system (21) solve $\lambda_i^*(\overline{\sigma}_1, \overline{\sigma}_2) = \lambda_i^+$. Since the stationary incidences I_1^* and I_2^* are decreasing functions of $\overline{\sigma}_1$ and $\overline{\sigma}_2$ (see Appendix A) and λ_i^* is a positively weighted sum of the stationary incidences, then the force of infection λ_i^* is also a decreasing function of the resident self-protection probabilities. It follows that the thresholds in Figure 4 must have non-positive slopes. In addition, the Jacobian determinant of the coordinate transform between $\overline{\sigma}$ and I^*

$$\left|\frac{\mathrm{d}\overline{\sigma}}{\mathrm{d}I^*}\right| = \gamma^2\left(1 + \frac{\beta_{12}I_1^*}{(\lambda_2^*)^2} + \frac{\beta_{21}I_2^*}{(\lambda_1^*)^2}\right) > 0 \tag{22}$$

everywhere when I^* is strictly positive. This ensures that the sign of the threshold crossing in $(\overline{\sigma}_1, \overline{\sigma}_2)$ space is the same as the sign in (I_1^*, I_2^*) space. Thus, the transforms of plots like those in Figure 2 to resident strategy space like the plots in Figure 3 consist only of stretchings (no flips or folds).

Since the threshold curves have non-positive slopes, any trajectories starting initially in quadrants 3 or 4 must eventually be absorbed by quadrants 1 and 2. By inspecting the vector-field cones on the boundaries of quadrants 1 and 2, we find that quadrants 1 and 2 are invariant. Within quadrants 1 and 2, the dynamics are monotone. If $\beta_{11}\beta_{22} - \beta_{12}\beta_{21} > 0$, as in Figure 4A, the dynamics converge to the interior Nash equilibrium σ^+. But if $\beta_{11}\beta_{22} - \beta_{12}\beta_{21} < 0$, as in Figure 4B, the dynamics will diverge from the internal Nash equilibrium σ^+. ■

A similar argument can be used to show that $\sigma^{\ulcorner}$ and $\sigma^{\lrcorner}$ are locally dynamically stable under Equation (21) when they exist. Thus, we find stability properties very reminiscent of the Lotka competition models. If transmission is stronger between populations than within populations, there may be two boundary Nash equilibria that are locally stable, but if transmission is stronger within populations, a unique interior Nash equilibrium is always globally stable.

6. Discussion

In this paper, we have described a modelling approach to games in structured populations and applied the approach to study the rational–expectation equilibria in a two-population game where choices reduce the risk of infection. We find that, depending on the mixing patterns and relative costs, there may not be a unique Nash equilibrium. Nash equilibria appearing on the interior of the reachable region of stationary incidences are stable against slow changes in the resident strategies when mixing is assortative, but unstable against slow changes in the resident strategies when mixing is disassortative. If transmission is stronger between populations than within populations, the game may have an unstable equilibrium and two stable equilibria satisfying the rational-expectation criteria. We conjecture that all Nash equilibria on the boundary of the reachable region are locally stable if there is an unstable interior Nash equilibrium. The graphical analysis we have used to illustrate equilibria solutions is reminiscent of the phase-plane analysis of Lotka's competition model from biology [17] and Cournot's duopoly model from economics [13]. To some extent, this similarity is superficial, since the underlying mechanisms are different from either of these two classical works. The option of having a graphical shorthand supplementing mathematical and verbal reasoning will hopefully be useful to some readers.

In these analyses, we have avoided special cases for the sake of the larger picture. In cases where the transmission rate matrix $[\beta_{ij}]$ is non-negative but not strictly positive, the Perron–Frobenius theory and related results should lead to a natural dissection of the games equilibrium structure. For instance, if there is no cross-transmission between subpopulations, the subpopulations can be treated as independent populations. Cases where $\mathcal{R}o = 1$ or $\lambda^* = 0$ that appear in various calculations can be dealt with piecemeal. Also, if the threshold forces of infection are the same in both populations, e.g. the lines in Figure 2 lie on top of each other, then there is a continuous interval of weak Nash equilibria. If the threshold forces of infection are proportional between the populations, e.g. the lines in Figure 2 are parallel, then the problem reduces to one of heterogeneity in costs [4]. In some circumstances, special cases like these may be very useful as illustrations of specific phenomena, and care should exercised when studying systems near these special cases. Similarly, analysis in cases of unequal subpopulation sizes should be straight-forward.

The practical importance of multiple equilibria in this example of an epidemiological game is unclear. The regions of parameter space for which multiple equilibria occur are relatively narrow because, as the transmission dynamics become more-strongly disassortative, the reachable region of forces of infection becomes narrower. If the precision of information used in individual decisions is too low, actors will not be able to distinguish among the various equilibria and analysis of the distinction in this particular model will be moot. More research is needed to establish whether this narrowness is common or unusual, depending on the economic, demographic, biological, and epidemiological structures involved. In addition, multiple equilibria may be created by other mechanisms. Our results depend strongly on the monotonicity result of Appendix A. Monotonicity does not necessarily hold for generalizations of this model, since there may be multiple stationary solutions for given parameter values and these may not be monotone functions of the resident self-protection strategies. The generalization of our results to other behaviours and mechanisms is also unclear. It seems reasonable to conjecture that this analysis will hold under other control mechanisms like vaccination, but other game-theory results like those of [18] suggest there may be a great diversity of results that depends on the specifics of the models.

This analysis has focused on the very constrained problem managing infectious disease risk, while neglecting external factors. To some extent, independent external risk factors like car crashes are accounted for by the discount rate h. When there are few external risks, discounting is small ($h \approx 0$), but when external risks are many, the discount rate is large. However, discounting cannot account for additional external effects correlated to internal states and behaviours. Greater model specificity would be needed to incorporate such additional external effects. A related issue is the

relative size of costs incurred from prevention and illness. The importance of a rational-expectation equilibrium depends greatly on the size of costs incurred under sub-optimal behaviour. If the expected costs of suboptimal behaviour are small compared with the variance of internal and external costs, it seems unreasonable to expect individuals to put much thought into optimizing behaviour.

The results should generalize to some extent when considering arbitrary numbers of subpopulations, but the best approach to multi-group problems is not obvious. The results in the case of positive assortative mixing [6] rely on a convenient mathematical form for the force of infection from which a generalization is not immediately clear. It seems reasonable to conjecture that multiple equilibria become more likely as the number of subpopulations increases, given May's [21] observations regarding n-species competition models, but narrowing of the windows of reachable disease prevalences suggests that internal equilibria become less likely as the number of subpopulations increases. Another open problem is to investigate the trade-off between these effects.

Our suppositions in the derivation of the population-scale model from the individual-scale can be weakened or reinterpreted in several ways. For instance, $\overline{\sigma}_i$ can be interpreted as an average or aggregate rate of self-protection in a heterogeneous population. This broadens the application of our results, but does not change any of the fundamental results of the analysis. The 'hazard' strategy-space considered in this paper is a subset of larger strategy spaces. A mixed strategy space might be a set of probability measures defined on a subset of functions $\mathbb{R} \mapsto [0, 1]$ mapping a point in time to a specific behaviour. This could include strategies where individuals change their behaviours systematically from day-to-day even when their state has not changed. Correlations among individual behaviours could then lead to significant effects at the scale of the population dynamics, for which we would need to account. But despite its limitations, consideration of a 'hazard' strategy space seems to yield useful results.

While we have found some insight into how population structure can change Nash equilibria in epidemiology games, clearly there are opportunities for further study.

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Appendix A. Uniqueness and monotonicity

For the SIS model considered in this paper, the basic reproductive number of the disease-free state in the absence of self-protective behaviour is

$$\mathcal{R}o = \rho_0\left(\begin{bmatrix} \frac{\beta_{11}}{\gamma} & \frac{\beta_{21}}{\gamma} \\ \frac{\beta_{12}}{\gamma} & \frac{\beta_{22}}{\gamma} \end{bmatrix}\right). \tag{A1}$$

The disease-free reproductive number with possible self-protective behaviour is

$$\mathcal{R} = \rho_0\left(\begin{bmatrix} \overline{\sigma}_1\frac{\beta_{11}}{\gamma} & \overline{\sigma}_2\frac{\beta_{21}}{\gamma} \\ \overline{\sigma}_1\frac{\beta_{12}}{\gamma} & \overline{\sigma}_2\frac{\beta_{22}}{\gamma} \end{bmatrix}\right), \tag{A2}$$

which is the largest solution of the quadratic equation

$$\left(\mathcal{R} - \overline{\sigma}_1\frac{\beta_{11}}{\gamma}\right)\left(\mathcal{R} - \overline{\sigma}_2\frac{\beta_{22}}{\gamma}\right) - \overline{\sigma}_1\overline{\sigma}_2\frac{\beta_{12}\beta_{21}}{\gamma^2} = 0. \tag{A3}$$

The reproductive number is bounded,

$$\mathcal{R}o \geq \mathcal{R} \geq \max\left\{\overline{\sigma}_1\frac{\beta_{11}}{\gamma}, \overline{\sigma}_2\frac{\beta_{22}}{\gamma}, \sqrt{\overline{\sigma}_1\overline{\sigma}_2\frac{\beta_{12}\beta_{21}}{\gamma^2}}\right\}. \tag{A4}$$

We will now provide an alternative proof of the uniqueness of a positive stationary solution when $\mathcal{R} > 1$.

THEOREM A.1 *If $[\beta_{ij}]$ is a positive matrix with a unique inverse and $\mathcal{R} > 1$, then Equation* (12) *has a unique endemic stationary solution. If $\mathcal{R} < 1$, there is no endemic stationary solution.*

Proof An endemic stationary solution is a stationary solution with a positive number of infecteds ($I_1 I_2 \geq 0$, $I_1 + I_2 > 0$). Equation (15), supplying the stationary solution condition, can be rewritten as the matrix equation

$$\begin{bmatrix} \beta_{11} & \beta_{21} \\ \beta_{12} & \beta_{22} \end{bmatrix}\begin{bmatrix} I_1 \\ I_2 \end{bmatrix} = \begin{bmatrix} \frac{\gamma}{\overline{\sigma}_1}\frac{I_1}{1-I_1} \\ \frac{\gamma}{\overline{\sigma}_2}\frac{I_2}{1-I_2} \end{bmatrix}. \tag{A5}$$

The inverse of $[\beta_{ij}]$ is an M-matrix so long as it exists. Equation (A5) can be rewritten as the equation

$$H(I) - I = 0, \tag{A6a}$$

where

$$H_i(I) = \frac{\overline{\sigma}_i}{\gamma}\frac{\sum_j \beta_{ji} I_j}{1 + \sum_j \beta_{ji} I_j}. \tag{A6b}$$

We now apply the theorem of Kennan [19]. The vector-valued function $H(I)$ satisfies the quasi-increasing condition because for every i and $j \neq i$,

$$\frac{\partial H_i}{\partial I_j} \geq 0. \tag{A7}$$

Next, the vector valued function $H(I) - I$ is strictly radially quasi-concave. Suppose there exists a solution I^* to Equation (A6). Let $k \in (0, 1)$.

$$H_i(kI^*) - kI_i^* = \frac{\overline{\sigma}_i}{\gamma} \frac{k \sum_j \beta_{ji} I_j^*}{1 + \sum_j \beta_{ji} k I_j^*} - kI_i^* \tag{A8}$$

$$= k \left(\frac{\overline{\sigma}_i}{\gamma} \frac{\sum_j \beta_{ji} I_j^*}{1 + k \sum_j \beta_{ji} I_j^*} - I_i^* \right). \tag{A9}$$

By inspection,

$$\frac{\overline{\sigma}_i}{\gamma} \frac{\sum_j \beta_{ji} I_j^*}{1 + k \sum_j \beta_{ji} I_j^*} - I_i^* \tag{A10}$$

vanishes at $k = 1$ and is decreasing in k. Thus, for all $k \in (0, 1)$ and any i,

$$\frac{\overline{\sigma}_i}{\gamma} \frac{\sum_j \beta_{ji} I_j^*}{1 + k \sum_j \beta_{ji} I_j^*} - I_i^* > 0, \tag{A11}$$

$$k \left(\frac{\overline{\sigma}_i}{\gamma} \frac{\sum_j \beta_{ji} I_j^*}{1 + k \sum_j \beta_{ji} I_j^*} - I_i^* \right) > 0. \tag{A12}$$

And so, $H(I) - I$ is strictly radially quasi-concave. By [19, Corollary 1], there is no more than one positive solution.

Near $I = 0$,

$$H(I) \approx \begin{bmatrix} \overline{\sigma}_1 \frac{\beta_{11}}{\gamma} & \overline{\sigma}_2 \frac{\beta_{21}}{\gamma} \\ \overline{\sigma}_1 \frac{\beta_{12}}{\gamma} & \overline{\sigma}_2 \frac{\beta_{22}}{\gamma} \end{bmatrix} I. \tag{A13}$$

The matrix in this equation is positive, so it has a positive eigenvalue (namely $\mathcal{R}$) with a corresponding positive eigenvector. Thus, if $\mathcal{R} > 1$, then $H(I) - I$ is initially strictly increasing for I lying in this eigenspace. We can also see from inspection that for all positive I,

$$H_i(I) < \frac{\overline{\sigma}_i}{\gamma}. \tag{A14}$$

By Tarski's theorem [19], there exists one or more positive fixed points.

Thus, $\mathcal{R} > 1$ implies there exists a unique positive fixed point. If $\mathcal{R} < 1$, then $H(I)$ is bounded above by its linearization in Equation (A13), which is a contraction to the origin (since both eigenvalues have modulus less than 1), so the only fixed point is $I = (0, 0)$. ■

Hethcote and Yorke [14] show that the unique positive stationary solution is globally attracting when $\mathcal{R} > 1$ and the disease-free stationary solution is globally attracting when $\mathcal{R} \leq 1$. We now show that more self-protection always decreases disease prevalence. Although this seems intuitive, I am not aware of an earlier proof. The identification of game equilibria depends critically on this result.

THEOREM A.2 *If $\mathcal{R}(\overline{\sigma}) > 1$, then the unique positive stationary disease prevalences I_1^*, I_2^* are strictly increasing in both $\overline{\sigma}_1$ and $\overline{\sigma}_2$.*

Proof From Theorem A.1, we know that the vector-valued function

$$I^*(\overline{\sigma}) = \left(I_1^*(\overline{\sigma}_1, \overline{\sigma}_2), I_2^*(\overline{\sigma}_1, \overline{\sigma}_2) \right), \tag{A15}$$

which maps resident strategies to the disease prevalences of the globally attracting stationary solution, is a well-defined function. $\mathcal{R}(\overline{\sigma}) > 1$ implies the stationary solution $I_1^*(\overline{\sigma}) > 0$ and $I_2^*(\overline{\sigma}) > 0$. The inverse relation

$$\overline{\sigma}_i = \gamma I_i^* \left(1 + \frac{1}{\sum_j \beta_{ji} I_j^*} \right) \tag{A16}$$

derived from Equation (A6) is a smooth function for positive I^*. The derivative only becomes unbounded as $\mathcal{R}$ approaches 1, corresponding to a loss of uniqueness. Thus, $I^*(\overline{\sigma})$ is a bijective function on the domain

$$\{(\overline{\sigma}_1, \overline{\sigma}_2) : \overline{\sigma}_i \in [0, 1] \quad \text{and} \quad \mathcal{R}(\overline{\sigma}_1, \overline{\sigma}_2) > 1\}. \tag{A17}$$

If follows from the bijective property that the Jacobian matrix $\partial I^*/\partial\overline{\sigma}$ exists within the domain and is non-singular. When we differentiate Equation (A16), we find that the Jacobian matrix solves

$$\frac{\partial\overline{\sigma}}{\partial I^*} = \gamma\begin{bmatrix} 1+\dfrac{1}{\lambda_1^*} & 0 \\ 0 & 1+\dfrac{1}{\lambda_2^*}\end{bmatrix} - \gamma\begin{bmatrix} \dfrac{I_2^*\beta_{11}}{(\lambda_1^*)^2} & \dfrac{I_2^*\beta_{21}}{(\lambda_1^*)^2} \\ \dfrac{I_1^*\beta_{12}}{(\lambda_2^*)^2} & \dfrac{I_2^*\beta_{22}}{(\lambda_2^*)^2}\end{bmatrix} \tag{A18}$$

$$= \gamma\begin{bmatrix}1 & 0\\ 0 & 1\end{bmatrix} + \gamma\begin{bmatrix} \dfrac{I_2^*\beta_{21}}{(\lambda_1^*)^2} & -\dfrac{I_2^*\beta_{21}}{(\lambda_1^*)^2} \\ -\dfrac{I_1^*\beta_{12}}{(\lambda_2^*)^2} & \dfrac{I_1^*\beta_{12}}{(\lambda_2^*)^2}\end{bmatrix}. \tag{A19}$$

The eigenvalues are γ and $\gamma(1 + I_2^*\beta_{21}/(\lambda_1^*)^2 + I_1^*\beta_{12}/(\lambda_2^*)^2)$, which are both positive; so the Jacobian matrix $\partial\overline{\sigma}/\partial I^*$ is an M-matrix. [15,p. 114]. From the M-matrix theory, it immediately follows that $\partial I^*/\partial\overline{\sigma}$ is non-negative. The extra condition that $\beta_{ij} > 0$ for all ij ensures $\partial\overline{\sigma}/\partial I^*$ is irreducible and $\partial I^*/\partial\overline{\sigma}$ is positive. We conclude the unique endemic stationary solution is strictly increasing in the population's resident strategies $\overline{\sigma}$. ■

Appendix B. On linear-fractional forms of the utility function

One result that follows for the form of utility function derived in Section 2 is that linearity in a strategy parameter σ implies that the utility function is a linear-fractional transform, a.k.a. Mobius transform, in said strategy parameter.

THEOREM B.1 *Suppose that σ is a component of the strategy vector $\boldsymbol{\pi}$ appearing in exactly one row of the augmented matrix $[(h\boldsymbol{I} - \boldsymbol{Q})^{\mathrm{T}}|\boldsymbol{v}]$ and that row is an affine function of σ. Also, assume that the initial state $\boldsymbol{p}(0)$ is independent of σ. Then the utility function has the Mobius transform form*

$$U(\sigma) = \frac{a\sigma + b}{d\sigma + e} \tag{B1}$$

and

$$\frac{\partial U(\sigma)}{\partial\sigma} = \frac{ae - bd}{(d\sigma + e)^2} \tag{B2}$$

where $a, b, d,$ and e are independent of σ.

Proof For a square $n \times n$ matrix $\mathbf{A}$,

$$\det\mathbf{A} = \sum_{\rho}\operatorname{sign}(\rho)\prod_{j}\mathbf{A}_{\rho(j),j}, \tag{B3}$$

where each ρ is a different permutation of the integers $1, \ldots, n$. Since permutations are bijections, components from each row appear exactly once in each monomial term of the determinant. Thus, if σ occurs only in one row of $\mathbf{A}$, and occurs linearly in that row, then $\det\mathbf{A}$ must be linear in σ also.

Now let w be a solution of $(h\mathbf{I} - \mathbf{Q}^*)^{\mathrm{T}}w = v$. We will use Cramer's rule to solve this. For every k,

$$w_k = \frac{\det\mathbf{M}_k}{\det h\mathbf{I} - \mathbf{Q}^*}, \tag{B4}$$

where $\mathbf{M}_k$ is the same as $(h\mathbf{I} - \mathbf{Q}^*)^{\mathrm{T}}$ accept that the kth column has been replaced by v. From our rule for determinants, both the numerator and denominator must be linear in σ, making w_k a linear-fractional transform in σ. Since $U = w^{\mathrm{T}}p(0)$ and $p(0)$ is independent of σ, then U is a sum of linear-fractional transforms with the same denominator; so U itself is a linear-fractional transform in σ. Equation (B2) is obtained by direct differentiation. ■

This may raise several interesting mathematical ideas for the reader, which, alas, we do not have space to address. Obvious cases where Theorem B.1 will fail are cases in which individuals have imperfect knowledge about their own state, so strategies appear in multiple columns of Q^* and cases in which payoffs are nonlinear functions of strategy.