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A GENERALIZATION OF EPIDEMIOLOGICAL MODELS UNDER RATIONAL EXPECTATIONS

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Dedicated to Professor John R. Graef on the occasion of his 70th birthday.

ABSTRACT. In this paper, we introduce an extended discrete economic epidemiological model based on the most recent work by Aadland et al.. Our generalization is on the consideration of unequal birth and death rates and also the modification of the utility function by using a parametric quadratic function. The new assumptions make it possible to consider the maximum number of contact made by rational individuals as a space variable. It is shown that, if rational individuals have a range of possible contacts to choose from, with the maximum number of contacts allowable for these individuals being dependent on the parameter of the utility function, the variation of this parameter tends to affect the stability properties of the system. We also show that birth and death rates affect the stability of the system for high values of the health gap and the infection parameter. Finally, effects of the parameters on various types of dynamic paths of the system are investigated by numerical simulation.

Keywords. Economic epidemiology, dynamic system, eigenvalue, equilibrium, stability, SIRS model.

AMS (MOS) Subject Classification. 39A30, 39A60, 91D30.

1. INTRODUCTION

Economic Epidemiology (EE) incorporates economic choices in epidemiology models. It is mainly concerned with the study of how the health status of an individual affects his or her responses to a disease outbreak [4]. The commonly held view is that individual behaviour in the presence of an infectious disease is dependent on the disease's prevalence and the danger that it poses [8]. For instance, extended research has been done in trying to understand how the behavioural pattern has affected the spread of the AIDS epidemics [12]. EE models also take into account the role of externalities in disease propagation; how an individual's response to an infectious disease outbreak can have a tremendous effect on the epidemiology of the disease; and finally the cost of curbing it. It is recognized that disease treatment and prevention depend heavily on the behaviour of the afflicted and non-afflicted; sometimes their behaviour

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is volatile during and in the aftermath of the outbreak and these mood swings have a bearing on the rest of the population as well [8]. Therefore, it is imperative to understand the wide variety of individual reactions during and in the aftermath of an epidemic outbreak and how the same affects policy formulation and implementation.

In the earlier work [11], Kaplan considered how the number of sexual contacts by individuals affect HIV infection rate. Later, syphilis cycles were studied in [1] and it was pointed out that the cycles depend heavily on individuals' preferences over their health and sexual activities. Most recently, Aadland et al. introduced an economic version of compartmental models in epidemiology under rational expectations [2, 3]. In the form of difference equations, their models have the key assumptions of constant population and the most commonly applied logarithmic utility function. They assumed that a representative agent makes a finite number of contacts that maximizes his or her expected life time utility. In this paper, we considered generalizations of the models from [2] by allowing dynamical population (unequal birth and death rates) and the replacement of the utility function using a parametric quadratic function. The new assumptions make it possible to consider the maximum number of contacts made by rational individuals as a new space variable. We studied the stability properties of the equilibria for the extended EE models using the linearizing analysis for discrete dynamical systems [5, 13]. It is shown that, if rational individuals have a range of possible contacts to choose from, with the maximum of the number of contacts allowable for these individuals being dependent on the parameter of the utility function, the variation in this parameter tends to affect the stability properties of the system. It is also shown that birth and death rates affect the stability of the system for high values of health gap and the infection parameter.

The rest of the paper is organized as the following: In Section 2, we introduced the modified EE SIRS and EE SIS models as discrete dynamical systems under the new assumptions. Stability analysis of the equilibria is given in Section 3. Simulation results on various types of dynamic paths are demonstrated in Section 4. Finally, Section 5 presents some discussions and conclusions.

2. EE SIRS AND SIS MODEL WITH RATIONAL EXPECTATIONS

2.1. **The EE SIRS Model.** The classical SIRS model consists of three mutually exclusive disease categories: Susceptible(S), Infected(I) and Recovered with immunity(R). The mechanism involved in transitioning from one disease category to the other is as follows: an individual infected by a disease will migrate from the susceptible category to the infected category and then when treated and immune against the disease, will migrate from the infected category to the recovered category and then back to the susceptible category when he or she becomes prone to the disease again.

Let p_t be the probability that susceptible individuals become infected after coming into contact with infected individual(s) and ν be the recovery rate of the infected category. Assume the immunity period is exponentially distributed with the rate γ. Then $\frac{1}{\gamma}$ is the average duration of immunity and $(1-\gamma)$ is the rate at which individuals remain in the recovered category. In this case, the number of individuals entering the susceptible category is γR and those remaining in the recovered category is $(1-\gamma)R$. Finally, assume the birth and death rates are ω and μ respectively. Following the similar approaches as [2], we have the following system:

$$\begin{aligned} S_{t+1} &= \omega N_t + (1 - p_t - \mu)S_t + \gamma R_t, \\ I_{t+1} &= (1 - \nu - \mu)I_t + p_t S_t, \\ R_{t+1} &= (1 - \gamma - \mu)R_t + \nu I_t. \end{aligned} \tag{2.1}$$

Let $N_{t+1} = S_{t+1} + I_{t+1} + R_{t+1}$ be the total population at time $t+1$. We have $N_{t+1} = (1+\omega-\mu)N_t$. Therefore system (2.1) can be written as proportions of N_{t+1}:

$$\begin{aligned} s_{t+1} &= A\omega + A(1 - p_t - \mu)s_t + A\gamma r_t, \\ i_{t+1} &= A(1 - \nu - \mu)i_t + Ap_t s_t, \\ r_{t+1} &= A(1 - \mu - \gamma)r_t + A\nu i_t, \end{aligned} \tag{2.2}$$

where $s_{t+1} = \frac{S_{t+1}}{N_{t+1}}$, $s_t = \frac{S_t}{N_t}$, $i_{t+1} = \frac{I_{t+1}}{N_{t+1}}$, $i_t = \frac{I_t}{N_t}$, $r_{t+1} = \frac{R_{t+1}}{N_{t+1}}$, $r_t = \frac{R_t}{N_t}$ and $A = \frac{1}{1+\omega-\mu}$.

Suppose individuals independently choose x_t contacts and that the probability of an uninfected individual becoming infected follows the Bernoulli process. Let α be the chance of becoming infected with each contact. Then the probability of a susceptible individual becoming infected is

$$p_t = \Pr(\text{infection}) = 1 - (1 - \alpha i_t)^{x_t}. \tag{2.3}$$

The dependence of the probability of infection on the chosen number of contacts differentiates the analysis from the standard (classical) mathematical epidemiology (ME) [1]. For instance, if individuals under study do not take into account the health consequences of their risky behaviour, thus going for the maximum number of contacts $\bar{x}$ each period, then we have the EE model collapsing to the standard ME with infection probability being

$$p_t = 1 - (1 - \alpha i_t)^{\bar{x}}\,. \tag{2.4}$$

2.2. **Rational Expectations on the Number of Contacts.** Suppose the representative agent n maximizes expected lifetime utility by choosing the number of contacts, $x_{n,t}$. Using the utility function $u(x_{n,t}, h_{n,t}) = x_{n,t} - \delta x_{n,t}^2 + h_{n,t}$, where $0 < \delta < 1$ is a fixed parameter, $h_{n,t}$ is a parameter that captures the agent's health

status at time t, we have the following as the objective function for the agent:

$$E_t \sum_{j=0}^{\infty} \beta^j [(x_{n,t+j} - \delta x_{n,t+j}^2) + h_{n,t+j}] \,, \tag{2.5}$$

where $0 < \beta < 1$ is the discount factor, E_t is the individual's expectation operator at time t. The parameter h plays a very important role in the individual's choice of number of contacts. In that, if the individual is infected, the individual experiences low value of h. Because the additional contacts made by an individual bring immediate satisfaction or a risk of getting infected by the disease, an additional contact the individual makes either affect the level of utility positively or negatively. For instance, a contact made by an individual that resulted in contracting the disease will cause a deterioration in the individual's health, thus reducing the value of the parameter h at the given period, hence affecting the utility of the individual inversely.

Under the assumption of all individuals are identical with the exception of having a different disease states and health levels, we analyze the model in terms of a single individual in each of the disease categories [3]. So the subscript n can be dropped. An individual belonging to the susceptible group makes a choice about contacts on the basis of his single-period utility function and expected future utility which depends on infection expectations. This susceptible individual's decision will satisfy the Bellman's equation

$$V_t^s = \max_{x \in X} \{x_t - \delta x_t^2 + h^s + \beta E_t [p_t V_{t+1}^i + (1 - p_t) V_{t+1}^s]\}, \tag{2.6}$$

where V_t^s is the value function associated with being susceptible at time t. The term in the bracket is the expected future utility which depends on expected future infection levels. The present value of the expected future utility is V_{t+1}^s if the individual remains susceptible and V_{t+1}^i if the individual becomes infected after making a choice in period t [8]. X is the range of possible contacts. In our case, we have $X = [0, \frac{1}{2\delta}]$ derived from the quadratic utility function.

Assume that all individuals regardless of infection status maximize the objective function (2.5) without the concern for the general population. Infected and recovered individuals with immunity therefore choose the maximum number of contacts $\bar{x}$ because they do not stand any risk of immediate infection [2]. The value functions for the infected and recovered groups can then be obtained as the following:

$$V_t^i = \bar{x} - \delta \bar{x}^2 + h^i + \beta E_t [\nu V_{t+1}^r + (1 - \nu) V_{t+1}^i], \tag{2.7}$$

$$V_t^r = \bar{x} - \delta \bar{x}^2 + h^s + \beta E_t [\gamma V_{t+1}^s + (1 - \gamma) V_{t+1}^r], \tag{2.8}$$

where $h^s > h^i$ are the health status associated with an individual in the susceptible (or recovered) and infected groups respectively.

The implication of the model is that an infected individual who involved in the maximum possible amount of risky behavior will spread the disease in the population, thus causing the susceptible group to make the number of contacts that is suboptimal [1]. The converse holds if one is dealing with an altruistic population (In [1], syphilis cycles were studied based on this assumption). Suppose an individual in the susceptible group chooses a number of contacts x_t such that the following Euler's equation is satisfied:

$$(2\delta x_t - 1) = -\beta p_{x,t} E_t[V^s_{t+1} - V^i_{t+1}], \tag{2.9}$$

where $p_{x,t} = \frac{\partial p_t}{\partial x_t} = -\frac{(1-p_t)}{x_t}\ln(1-p_t)$ (from (2.3)). The right hand of (2.9) depicts the expected marginal damage costs of increasing current contacts in terms of the discounted expected reduction in future utility due to infection. On the other hand, the left hand term represents the current period benefit as the individual increases contacts. Therefore, condition (2.9) implies that an individual who is in the susceptible group chooses x_t such that his or her marginal benefits and expected marginal cost are equal. The contact level also influences the probability of becoming infected. Furthermore, equation (2.9) shows that the contact rate in the EE model is based on behavioral responses to changes in disease risk as opposed to the classical epidemiology models where the contact rate is considered as being constant or can be varied deterministically. This is exhibited by the expression connecting $p_{x,t}$.

We consider two cases that is dependent on the agent's observance of his or her own immunity [2].

Case (1): Unobservable Host Immunity

Suppose an individual who has recovered with immunity believes he is still susceptible to the disease. We can ignore equation (2.8) and obtain from (2.7) that

$$V^i_t = x_t - \delta x_t^2 + h^i + \beta E_t[\nu V^s_{t+1} + (1-\nu)V^i_{t+1}]. \tag{2.10}$$

Substituting out the value functions V^s_{t+1} and V^i_{t+1} from (2.9), we have

$$(2\delta x_t - 1) = p_{x,t}\beta E_t\left[-[\psi(x_{t+1},\bar{x}) + h] + \frac{(1-\nu-p_{t+1})}{p_{x,t+1}}[2\delta x_{t+1} - 1]\right], \tag{2.11}$$

where $\psi(x_t,\bar{x}) = (x_t - \delta x_t^2) - (\bar{x} - \delta\bar{x}^2), \psi(x_{t+1},\bar{x}) = (x_{t+1} - \delta x^2_{t+1}) - (\bar{x} - \delta\bar{x}^2)$, and $h = h^s - h^i$.

Case (2): Observable Host Immunity

Assume individuals recovered with immunity observe their own immunity and thus rationally choose the maximum number of contacts $\bar{x}$ and have health level h^s. Then (2.6), (2.7) and (2.8) becomes relevant and equation (2.9) can be derived to

$$(2\delta x_t - 1) = \beta p_{x,t} E_t\left[-[\psi(x_{t+1},\bar{x}) + h] + (1-\nu-p_{t+1})\frac{(2\delta x_{t+1} - 1)}{p_{x,t+1}} + \beta\tau_{t+2}\right], \tag{2.12}$$

where

$$\tau_{t+2} = (1-\nu-\gamma)\left[\psi(x_{t+2},\bar{x}) - \frac{(1-p_{t+2})(2\delta x_{t+2}-1)}{p_{x,t+2}}\right] + (1-\gamma)\left[h + \frac{(2\delta x_{t+1}-1)}{\beta p_{x,t+1}}\right] - \nu\gamma\left[\frac{(2\delta x_{t+2}-1)}{p_{x,t+2}}\right],$$

and $\psi(x_{t+2},\bar{x}) = (x_{t+2} - \delta x_{t+2}^2) - (\bar{x} - \delta\bar{x}^2)$.

Equations (2.11) and (2.12) are identical except for τ_{t+2} in (2.12). The term τ_{t+2} captures the expected future "costs" of an individual infected but can observe acquired immunity [2]. If $\tau_{t+2} < 0$, the possibility of future immunity will be a benefit of becoming infected since it will have an adverse effect on the marginal cost. On the other hand, if τ_{t+1} is positive, becoming infected will be a cost even under the possibility of future immunity.

2.3. **The EE SIS Model.** As a special case for the SIRS model, the SIS model considers two mutually exclusive disease categories: Susceptible(S) and Infected(I). An individual in the susceptible category makes a transition to the infected category when he becomes infected and then back to the susceptible category immediately after recovering. That is, the disease does not confer any long lasting immunity so there is no need to create the recovered region. An example is the common cold.

In this case, ν is the rate of migrating from the infected group to the susceptible. Other parameters such as the birth and death rates, probability of infection and the chance of becoming infected with each contact are same as those of the SIRS model. We therefore have the following system explaining the model:

$$s_{t+1} = A\omega + A(1-p_t-\mu)s_t + A\nu I_t, \tag{2.13}$$

$$i_{t+1} = A(1-\nu-\mu)i_t + Ap_t s_t, \tag{2.14}$$

where s_{t+1}, s_t, i_{t+1}, i_t, A and N_{t+1} are same as before.

The economic part of the model also follows the same reasoning discussed in Section 2.2. In this case, the Euler's equation can be obtained as

$$(2\delta x_t - 1) = p_{x,t}\beta E_t\left[-[\psi(x_{t+1},\bar{x}) + h] + \frac{(1-\nu-p_{t+1})}{p_{x,t+1}}[2\delta x_{t+1}-1]\right], \tag{2.15}$$

where $\psi(x_t,\bar{x}) = (x_t - \delta x_t^2) - (\bar{x} - \delta\bar{x}^2), \psi(x_{t+1},\bar{x}) = (x_{t+1} - \delta x_{t+1}^2) - (\bar{x} - \delta\bar{x}^2)$, and $h = h^s - h^i$. This is same as the case that the individual does not observe his immunity against the disease.

3. DYNAMICS OF THE EQUILIBRIA

3.1. **Stability of the EE SIRS Model.** Since an infectious disease can be endemic in or may be eradicated from a population, generally there are two possible steady state equilibria: the endemic equilibrium and the eradication equilibrium. For the

eradication steady state equilibrium, $s = 1$, $i = r = 0$, and $x = \bar{x}$. At the endemic steady state, we assume time is invariant. Therefore we have the following system of equations in four unknown variables (s, i, r, x):

$$\begin{cases} s = \frac{A(\omega+\gamma r)}{1-A(1-p-\mu)}, \\ i = \frac{Asp}{1-A(1-\nu-\mu)}, \\ r = \frac{A\nu i}{1-A(1-\mu-\gamma)}, \\ x = \frac{\beta}{2\delta}\left[p_x[\phi\beta\tau - (\psi(x,\bar{x}) + h)] + (1-\nu-p)(2\delta x - 1)\right] + \frac{1}{2\delta}. \end{cases} \tag{3.1}$$

where the Euler Equation either takes the form (2.11) when the indicator variable $\phi = 0$ or the form (2.12), when $\phi = 1$.

$$\tau = \frac{1}{p_x}\left[(2\delta x - 1)\left[\frac{(1-\gamma)}{\beta} - (1-\nu-\gamma)(1-p) - \nu\gamma\right]\right] + (1-\gamma)h + (1-\nu-\gamma)\psi(x,\bar{x}),$$

where $\psi(x,\bar{x}) = (x - \delta x^2) - (\bar{x} - \delta\bar{x}^2)$. Linearizing around the endemic steady state by employing first-order Taylor series approximation, we have

$$\begin{aligned} \hat{s}_{t+1} &= A(1-p-\mu)\hat{s}_t + A\gamma\hat{r}_t - As\hat{p}_t, \\ \hat{i}_{t+1} &= A(1-\nu-\mu)\hat{i}_t + As\hat{p}_t + Ap\hat{s}_t, \\ \hat{r}_{t+1} &= A(1-\mu-\gamma)\hat{r}_t + A\nu\hat{i}_t, \end{aligned} \tag{3.2}$$

where hat $(\wedge)$ over the variables denotes deviation from the endemic steady state. The linearized Euler equation is below:

$$\begin{aligned} 2\delta p_x\hat{x}_t - (2\delta x - 1)\hat{p}_{x,t} = {} & \beta p_x[p_x(2\delta x - 1) + 2\delta(1-\nu-p)]E_t\hat{x}_{t+1} \\ & - \beta(1-\nu-p)(2\delta x - 1)E_t\hat{p}_{x,t+1} \\ & - \beta p_x(2\delta x - 1)E_t\hat{p}_{t+1} \\ & + \phi\beta^2\Big[\frac{2\delta p_x(1-\gamma)}{\beta}E_t\hat{x}_{t+1} - \frac{(1-\gamma)(2\delta x - 1)}{\beta}E_t\hat{p}_{x,t+1} \\ & + p_x[(1-\nu-\gamma)[p_x(1-2\delta x) - 2\delta(1-p)] - 2\delta\nu\gamma]E_t\hat{x}_{t+2} \\ & + [(1-\nu-\gamma)(1-p) + \nu\gamma](2\delta x - 1)E_t\hat{p}_{x,t+2} \\ & + p_x(2\delta x - 1)(1-\nu-\gamma)E_t\hat{p}_{t+2}\Big], \end{aligned} \tag{3.3}$$

where

$$\begin{aligned} \hat{p}_t &= p_i\hat{i}_t + p_x\hat{x}_t, \\ \hat{p}_{x,t} &= \frac{[1 + \ln[1-p]]}{x}\hat{p}_t - \frac{p_x}{x}\hat{x}_t, \end{aligned}$$

and

$$p_i = \frac{\partial p}{\partial i} = x\alpha(1-\alpha i)^{x-1},$$
$$p_x = -\frac{(1-p)}{x}\ln(1-p).$$

In the case of unobservable immunity, $\phi = 0$. Coupled with the relation $\hat{s}_t = -\hat{r}_t - \hat{i}_t$ and imposing perfect foresight ($E_t\hat{x}_{t+1} = \hat{x}_{t+1}$), we obtain the following system:

$$\underbrace{\begin{bmatrix} 0 & A(1-\nu-\mu-p) & -Ap \\ 0 & A\nu & A(1-\mu-\gamma) \\ 2\delta p_x & 0 & 0 \end{bmatrix}}_{M_1} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \\ \hat{r}_t \end{bmatrix}$$
$$+ \underbrace{\begin{bmatrix} As & 0 \\ 0 & 0 \\ 0 & -(2\delta x - 1) \end{bmatrix}}_{M_2} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} =$$
$$\underbrace{\begin{bmatrix} 0 & 1 & 0 \\ 0 & 0 & 1 \\ \beta p_x[p_x(2\delta x - 1) + 2\delta(1-\nu-p)] & 0 & 0 \end{bmatrix}}_{M_3} \begin{bmatrix} \hat{x}_{t+1} \\ \hat{i}_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} \tag{3.4}$$
$$+ \underbrace{\begin{bmatrix} 0 & 0 \\ 0 & 0 \\ -\beta p_x(2\delta x - 1) & -\beta(1-\nu-p)(2\delta x - 1) \end{bmatrix}}_{M_4} \begin{bmatrix} \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix}$$

and

$$\underbrace{\begin{bmatrix} 1 & 0 \\ -\frac{\left[1+\ln(1-p)\right]}{x} & 1 \end{bmatrix}}_{M_5} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} = \underbrace{\begin{bmatrix} p_x & p_i & 0 \\ -\frac{p_x}{x} & 0 & 0 \end{bmatrix}}_{M_6} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \\ \hat{r}_t \end{bmatrix}. \tag{3.5}$$

Similarly, the case of observable immunity has $\phi = 1$ and (3.3) can be reduced to

$$\begin{aligned} 2\delta p_x\hat{x}_t - (2\delta x - 1)\hat{p}_{x,t} = {} & \beta p_x[p_x(2\delta x - 1) + 2\delta(2-\nu-p-\gamma)]E_t\hat{x}_{t+1} \\ & + \beta^2 p_x[(1-\nu-\gamma)[p_x(1-2\delta x) - 2\delta(1-p)] - 2\delta\nu\gamma]E_t\hat{x}_{t+2} \\ & - \beta p_x(2\delta x - 1)E_t\hat{p}_{t+1} - \beta(2-\nu-p-\gamma)(2\delta x - 1)E_t\hat{p}_{x,t+1} \\ & + \beta^2 p_x(2\delta x - 1)(1-\nu-\gamma)E_t\hat{p}_{t+2} + \beta^2[(1-\nu-\gamma)(1-p) \\ & + \nu\gamma](2\delta x - 1)E_t\hat{p}_{x,t+2}. \end{aligned}$$

Therefore imposing perfect foresight ($E_t x_{t+1} = x_{t+1}$), we have the following as the linearized EE matrix system:

$$\underbrace{\begin{bmatrix} 0 & A(1-\nu-\mu-p) & -Ap & 0 & 0 \\ 0 & A\nu & A(1-\mu-\gamma) & 0 & 0 \\ 2\delta p_x & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix}}_{M_1} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \\ \hat{r}_t \\ \hat{x}_{t+1} \\ \hat{i}_{t+1} \end{bmatrix} + \underbrace{\begin{bmatrix} As & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & -(2\delta x - 1) & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}}_{M_2} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \\ \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix} =$$

$$\underbrace{\begin{bmatrix} 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 \\ \beta p_x[p_x(2\delta x - 1) + 2\delta(2-\nu-p-\gamma)] & 0 & 0 & \beta^2 p_x[(1-\nu-\gamma)[p_x(1-2\delta x) - 2\delta(1-p)] - 2\delta\nu\gamma] & 0 \\ 1 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 \end{bmatrix}}_{M_3} \begin{bmatrix} \hat{x}_{t+1} \\ \hat{i}_{t+1} \\ \hat{r}_{t+1} \\ \hat{x}_{t+2} \\ \hat{i}_{t+2} \end{bmatrix} +$$

$$(2\delta x - 1) \underbrace{\begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -\beta p_x & -\beta(2-\nu-p-\gamma) & \beta^2 p_x(1-\nu-\gamma) & \beta^2[(1-\nu-\gamma)(1-p) + \nu\gamma] \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}}_{M_4} \begin{bmatrix} \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \\ \hat{p}_{t+2} \\ \hat{p}_{x,t+2} \end{bmatrix}$$

and

$$\underbrace{\begin{bmatrix} 1 & 0 & 0 & 0 \\ -\frac{(1+\ln(1-p))}{x} & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & -\frac{(1+\ln(1-p))}{x} & 1 \end{bmatrix}}_{M_5} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \\ \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix} = \underbrace{\begin{bmatrix} p_x & p_i & 0 & 0 & 0 \\ -\frac{p_x}{x} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & p_x & p_i \\ 0 & 0 & 0 & -\frac{p_x}{x} & 0 \end{bmatrix}}_{M_6} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \\ \hat{r}_t \\ \hat{x}_{t+1} \\ \hat{i}_{t+1} \end{bmatrix}.$$

Let $\hat{Z}_t = (\hat{x}_t, \hat{i}_t, \hat{r}_t)^T$ or $\hat{Z}_t = (\hat{x}_t, \hat{i}_t, \hat{r}_t,\ \hat{x}_{t+1}, \hat{i}_t)^T$ and $\hat{P}_t = (\hat{p}_t, \hat{p}_{x,t})^T$ or $\hat{P}_t = (\hat{p}_t, \hat{p}_{x,t}, \hat{p}_{t+1},\ \hat{p}_{x,t+1})^T$, we have

$$M_1 \hat{Z}_t + M_2 \hat{P}_t = M_3 \hat{Z}_{t+1} + M_4 \hat{P}_{t+1}$$

and $M_5 \hat{P}_t = M_6 \hat{Z}_t$. Therefore the EE system reduces to

$$\hat{Z}_t = J \hat{Z}_{t+1}, \tag{3.6}$$

where $J = (M_1 + M_2 M_5^{-1} M_6)^{-1}(M_3 + M_4 M_5^{-1} M_6)$.

The three-variable system (3.6) has one non-predetermined ($\hat{x}_t$) and two predetermined ($\hat{i}_t$ and $\hat{r}_t$) variables. Applying the results of [5], if there are exactly two eigenvalues of J outside the unit circle, the system exhibits a stable saddle-path. On the other hand, the system will exhibit indeterminate multiple stable paths or a sink if all the eigenvalues of J are unstable (outside the unit circle) and explosive paths if the number of forward stable eigenvalues (inside the unit circle) of J are more than one [5]. The five-variable system has three non-predetermined ($\hat{x}_t, \hat{x}_{t+1}$, and $\hat{i}_{t+1}$) and two predetermined (i_t and $\hat{r}_t$) variables. Following the same chain of analysis, the

system will exhibit a saddle-path stability if there are exactly two eigenvalues outside the unit circle, indeterminate multiple path stability if more than three unstable eigenvalues, and explosive paths if less than two unstable eigenvalues.

It is noticed that equation (3.6) can also be written as:

$$\hat{Z}_{t+1} = J^{-1}\hat{Z}_t\,, \tag{3.7}$$

where $J^{-1} = (M_3 + M_4M_5^{-1}M_6)^{-1}(M_1 + M_2M_5^{-1}M_6)$. Stability results can also be determined from the corresponding eigenvalues of J^{-1}[5].

3.2. **Stability of the EE SIS Model.** The EE SIS model discussed in Section 2.3 has the endemic steady state

$$\begin{cases} s = \frac{A(\omega+\nu i)}{1-A(1-p-\mu)}, \\ i = \frac{Asp}{1-A(1-\nu-\mu)}, \\ x = \frac{\beta}{2\delta}\Big[p_x[-(\psi(x,\bar{x})+h)] + (1-\nu-p)(2\delta x-1)\Big] + \frac{1}{2\delta}. \end{cases} \tag{3.8}$$

The linearized system around the endemic steady state is as follows:

$$\hat{s}_{t+1} = A(1-\mu-p)\hat{s}_t + A\nu\hat{i}_t - As\hat{p}_t, \tag{3.9}$$

$$\hat{i}_{t+1} = A(1-\nu-\mu)\hat{i}_t + As\hat{p}_t + Ap\hat{s}_t. \tag{3.10}$$

The linearized Euler equation has the form:

$$\begin{aligned} 2\delta p_x\hat{x}_t - (2\delta x-1)\hat{p}_{x,t} = {} & \beta p_x[p_x(2\delta x-1) + 2\delta(1-\nu-p)]E_t\hat{x}_{t+1} \\ & - \beta(1-\nu-p)(2\delta x-1)E_t\hat{p}_{x,t+1} \\ & - \beta p_x(2\delta x-1)E_t\hat{p}_{t+1}. \end{aligned}$$

From $\hat{s}_t + \hat{i}_t = 0$, (3.10) can be written as

$$\hat{i}_{t+1} = A(1-\nu-\mu-p)\hat{i}_t + A(1-i)\hat{p}_t. \tag{3.11}$$

Follow the same approaches of Section 3.1, we have the following EE matrices:

$$\underbrace{\begin{bmatrix} 0 & A(1-\nu-\mu-p) \\ 2\delta p_x & 0 \end{bmatrix}}_{N_1}\begin{bmatrix}\hat{x}_t \\ \hat{i}_t\end{bmatrix} + \underbrace{\begin{bmatrix} A(1-i) & 0 \\ 0 & -(2\delta x-1)\end{bmatrix}}_{N_2}\begin{bmatrix}\hat{p}_t \\ \hat{p}_{x,t}\end{bmatrix}$$

$$\underbrace{\begin{bmatrix} 0 & 1 \\ \beta p_x[p_x(2\delta x-1)+2\delta(1-\nu-p)] & 0\end{bmatrix}}_{N_3}\begin{bmatrix}\hat{x}_{t+1} \\ \hat{i}_{t+1}\end{bmatrix} + \underbrace{\begin{bmatrix} 0 & 0 \\ -\beta p_x(2\delta x-1) & -\beta(1-\nu-p)(2\delta x-1)\end{bmatrix}}_{N_4}\begin{bmatrix}\hat{p_{t+1}} \\ \hat{p}_{x,t+1}\end{bmatrix}$$

and

$$\underbrace{\begin{bmatrix} 1 & 0 \\ -\frac{(1-\ln(1-p)}{x} & 1\end{bmatrix}}_{N_5}\begin{bmatrix}\hat{p}_t \\ \hat{p}_{x,t}\end{bmatrix} = \underbrace{\begin{bmatrix} p_x & p_i \\ -\frac{p_x}{x} & 0\end{bmatrix}}_{N_6}\begin{bmatrix}\hat{x}_t \\ \hat{i}_t\end{bmatrix}.$$

Let $\hat{Z}_t = (\hat{x}_t, \hat{i}_t)^T$ and $\hat{Q}_t = (\hat{p}_t, \hat{p}_{x,t})^T$ so that the system reduces to

$$\hat{Z}_t = J\hat{Z}_{t+1}. \tag{3.12}$$

where $J = (N_1 + N_2 N_5^{-1} N_6)^{-1}(N_3 + N_4 N_5^{-1} N_6)$.

This system has one non-predetermined $\{\hat{x}_t\}$ and one predetermined$\{\hat{i}_t\}$ variable. If there is exactly one unstable eigenvalue, then it shows saddle-path stability. If there are two unstable eigenvalues, the system will exhibit indeterminate multiple paths stability. Zero unstable eigenvalue implies explosive paths.

4. NUMERICAL SOLUTIONS

In this section, we investigate the effects of the health gap ($h = h^s - h^i$) and the infection parameter α on the dynamics of the system using numerical simulation by Maple. These two parameters are the possible public health policy targets. A high h means the health gap of individuals within the population is high, thus raises concerns. For h to be maintained at a low level, investment could be made into drugs or medication. As well, α can be maintained low by introducing vaccines or a new way of protecting the population from being infected [9]. A high α means the disease can spread quickly among the population.

Using colors red, green, yellow and black to indicate saddle-path equilibria (stability), indeterminate multiple path stability, explosive paths and where individuals are going for maximum contacts respectively, we plot the regions for different dynamic paths by varying the values of δ (determinant of maximum number of contacts), ω and μ. The selection of the values for β, ν and γ (see Table 1) implies that annual discount rate is 4%, 100% recovery rate within a year of infection and an expected 5-year immunity duration respectively. In the experiments, some starting values for h and α did not produce the endemic values for some of the models. Therefore, we tested and chose the starting values that can produce the endemic values. For example, for the SIRS model, the initial values for h and α were 5 and 0.1 and then increased by steps of 0.02 and 0.01 respectively.

TABLE 1. Parameter values

Parameters	β	ν	γ
Values	0.96	1	0.2

4.1. **The EE SIR Model with Unobservable Immunity.** The EE SIR model is a special case of the EE SIRS model when $\gamma = 0$ (confirming permanent immunity). The numerical solution for $\omega > \mu$ and $\omega = \mu$ at $\delta = 0.025$ exhibits saddle-paths equilibria for all combinations for h and α. This is shown by Fig. 1(a). This indicates that, given an initial condition for i, r and x the system converges to the steady state. It also implies that individuals have contact levels less than the maximum allowable $\bar{x}$. Therefore, public policy targeted at reducing the health gap (or improving the health of infected individuals) would not affect stability of the system.

Figs. 1(b) and 1(c) show the cases for $\omega > \mu$ and $\omega = \mu$ at $\delta = 0.05$ respectively. For $\omega > \mu$, at very low values of α, individuals are going for the maximum number of contacts. The rest of the region exhibits saddle-path equilibria. For $\omega = \mu$ (Fig. 1(c)), the system shows similar pattern, but with a smaller maximum-contact region. Thus indicating that, at this level, individuals may be behaving in a fatalistic way. As such, policy direction towards the reduction of the level of contacts may not be effective as individuals will place much importance on the benefit associated with going for maximum number of contacts.

These results show that δ plays a significant role in determining the stability of the system. The only case for which μ and ω has a somewhat significant effect on the EE SIR system is when $\delta = 0.05$. Further increasing in δ, the region for maximum contact becomes wider.

4.2. **The EE SIRS Model with Unobservable Immunity.** Assume an average duration of immunity is five years ($\gamma = 0.2$).

The system exhibits the same dynamic paths for $\omega > \mu$ and $\omega = \mu$ at $\delta = 0.025$ respectively. Similar to the EE SIR case, the system exhibits saddle-paths equilibria for the entire parameter combination of h and α, indicating that, when individuals behave rationally, they will always go for contact levels less than $\bar{x}$. Fig. 2(a) shows this result.

Figs. 2(b) and 2(c) are for the cases for $\omega > \mu$ and $\omega = \mu$ at $\delta = 0.05$ respectively. It is noticed that the system exhibited saddle-path stability for all values of h and low range of α. It also exhibits some indeterminate multiple paths for high values of α and moderate values for h. Finally, when $\omega \geq \mu$ and both α and h are high, explosive paths are shown.

The discussion above implies the birth and death rates do not have effect on the dynamic paths of this system. On the other hand, it is evident that δ has significant effect, therefore, the assumption of $\overline{x}$ depending on a parameter led a different outlook for the dynamic paths.

4.3. **Observable Host Immunity for the SIR(S) Model.** Assume individuals observe their immunity. We obtained the dynamic paths shown in Figs. 3 and 4 respectively.

Figs. 3(a)–3(c) show the dynamic paths for the EE SIR system. For unequal birth and death rates and δ set at 0.05, the system exhibits saddle-paths stability for all the parameter combinations of h and α. The same pattern holds for equal birth and death rates, but with individuals going for $\bar{x}$ at very low values of α. Both cases (i.e $\omega \geq \mu$) indicate that as rational individuals observe their immunity against a particular infectious disease, they are still conscious of the health status of others and

themselves and thus will opt to go for a number of contacts less than the maximum allowable. Figs. 3(b) and 3(c) indicate these dynamics. Fig. 3(a) shows the dynamic for δ set at 0.025. It exhibits the same dynamic paths shown in the case for $\omega > \mu$ with δ set at 0.05.

Figs. 4(a)–4(c) show the dynamic paths for the EE SIRS system. All the parameter combinations yielded the same dynamic paths. That is, they exhibited saddle-paths equilibria for all the possible combinations of h and α given the respective values for ω and μ. These results mean that public policy direction (whether to reduce h or α or both) will not have any bearing on the stability properties of the system.

4.4. **The EE SI Model.** A special case of the EE SIS model is when $\nu = 0$, which indicates that no treatment is available for the disease. The numerical analysis for $\delta = 0.025$ with $\omega = 0.05$ or $\omega = 0.06$ and

Figs. 5(b) and 5(c) have the parameters $\delta = 0.05$ with $\omega > \mu$ and $\omega = \mu$ respectively. Both cases show saddle-path stability for high values of h given the entire range of values for α. This demonstrates that, at a high level of health gap, rational individual are willing to choose a number of contacts less than $\bar{x}$. On the other hand, low values of h yield a case where rational individuals are choosing $\bar{x}$. This indicates that irrespective of the level of infection parameter, they are willing to involve in risky behaviour by going for the $\bar{x}$. It also shows that δ has a significant effect on the properties of the system.

4.5. **The EE SIS model.** As in the case for the EE SI system, the EE SIS system shows the same property for the parameter combination of $\omega > \mu$ and $\omega = \mu$ for $\delta = 0.025$, in that, irrespective of the levels of infection and health gap, rational individuals are going for the maximum number of contacts. Fig. 6(a) demonstrates this result.

Figs. 6(b) and 6(c) show the dynamic paths for the system for $\omega > \mu$ and $\omega = \mu$ for $\delta = 0.05$ respectively. For $\omega > \mu$, the system exhibited the same property for the EE SI system. On the other hand, it is different for $\omega = \mu$, in that, the system exhibited explosive paths for parameter combinations of high values of h and α.

5. CONCLUSION

As the emergence of infectious disease has become a thorn in the flesh of humanity, it is imperative to understand the mechanisms involve in the transmission of these diseases so that health policies targeted at controlling their spread are effective. Classical mathematical epidemiological models provide a fair framework to achieving this purpose [7]. However, it has the limitation of not explicitly modelling the behavioural influence of individuals on the spread of these diseases. Economic

Epidemiology aims to fill this gap since disease treatment and prevention depends heavily on the behaviour of individuals [8].

In this paper, we study a modified version of the EE model that was recently introduced in [2] within an optimization framework. As a new space variable, the maximum number of contacts is introduced. In addition, we extended the previous model by considering the case of dynamic population (different birth and death rates). Our assumption is particular practical at the beginning of the spread of a disease since the maximum number of contacts can be controlled by isolation.

Applying linearizing analysis, we investigated the stability of the EE system. Numerical simulation is also employed to get an insight into the various types of dynamics paths. Our results indicated that the maximum number of contacts have clear effect on the dynamics of the system. On the other hand, the birth and death rates do not have significant effect with the exception of some extreme cases where both the levels of the health gap and the infection parameter have high values.

As future work, it would be interesting to apply real world data on a specific disease to the model so as to ascertain a precise policy recommendations.

6. ACKNOWLEDGMENT

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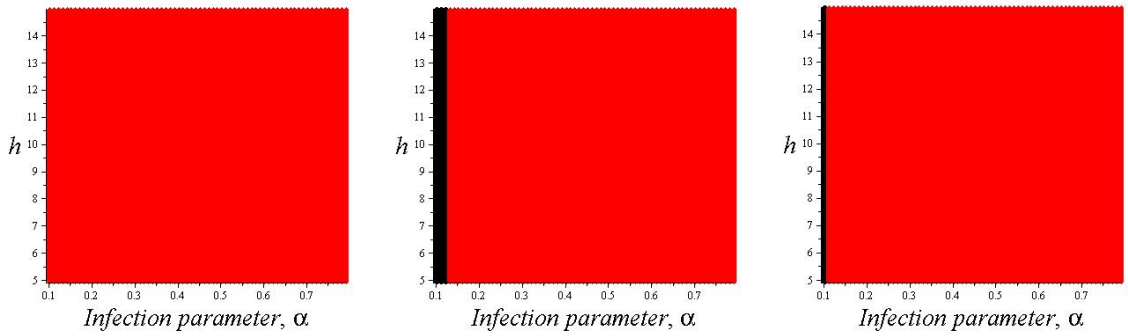
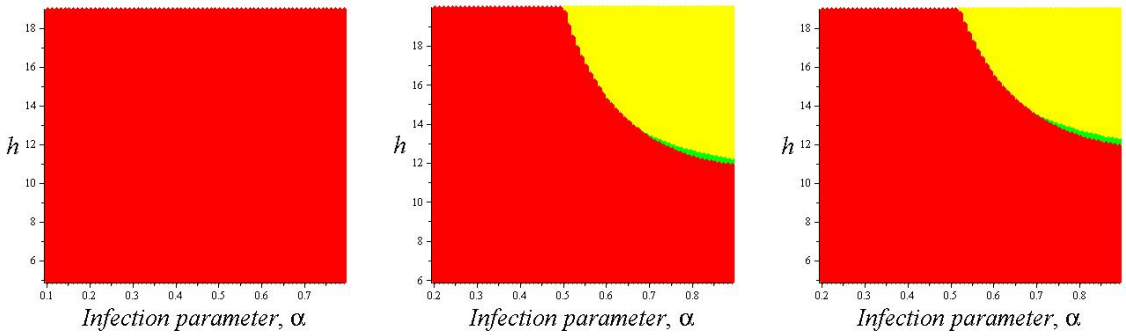


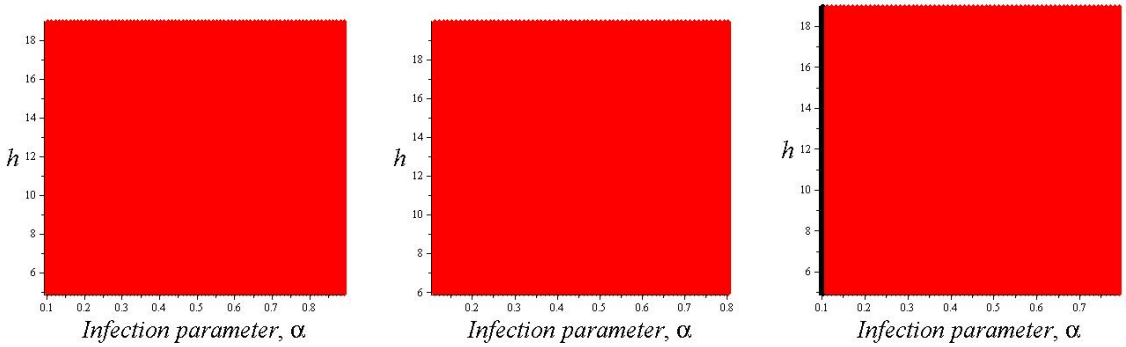
(a) $\omega = 0.05$ or 0.06, $\mu = 0.05$, $\delta = 0.025$ (b) $\omega = 0.06, \mu = \delta = 0.05$ (c) $\omega = \mu = \delta = 0.05$

FIGURE 1. The EE SIR model for unobservable host immunity



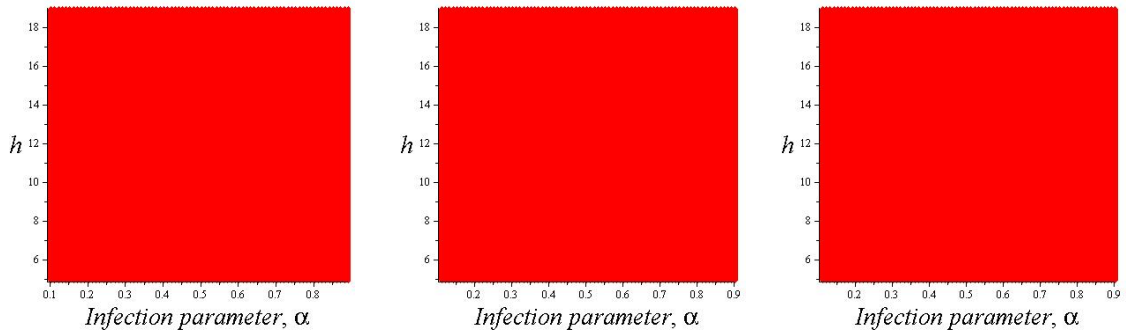
(a) $\omega = 0.05$ or 0.06, $\mu = 0.05$, $\delta = 0.025$ (b) $\omega = 0.06, \mu = \delta = 0.05$ (c) $\omega = \mu = \delta = 0.05$

FIGURE 2. The EE SIRS model for unobservable host immunity



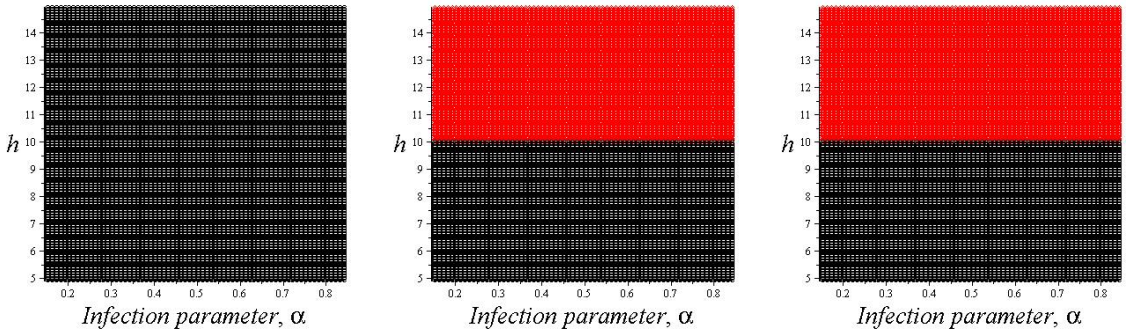
(a) $\omega = 0.05$ or 0.06, $\mu = 0.05$, $\delta = 0.025$ (b) $\omega = 0.06, \mu = \delta = 0.05$ (c) $\omega = \mu = \delta = 0.05$

FIGURE 3. The EE SIR model for observable host immunity



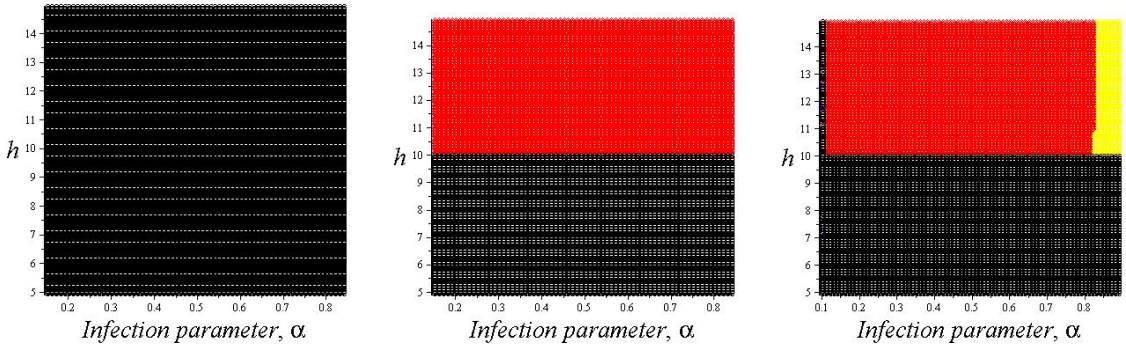
(a) $\omega = 0.05$ or 0.06, $\mu = 0.05$, $\delta = 0.025$ (b) $\omega = 0.06, \mu = \delta = 0.05$ (c) $\omega = \mu = \delta = 0.05$

FIGURE 4. The EE SIRS model for observable host immunity



(a) $\omega = 0.05$ or 0.06, $\mu = 0.05$, $\delta = 0.025$ (b) $\omega = 0.06, \mu = \delta = 0.05$ (c) $\omega = \mu = \delta = 0.05$

FIGURE 5. The EE SI model



(a) $\omega = 0.05$ or 0.06, $\mu = 0.05$, $\delta = 0.025$ (b) $\omega = 0.06$, $\mu = \delta = 0.05$ (c) $\omega = \mu = \delta = 0.05$

FIGURE 6. The EE SIS model

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