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SIR DYNAMICS WITH ECONOMICALLY DRIVEN CONTACT RATES

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Abstract

The susceptible-infected-recovered (SIR) model has greatly evidenced epidemiology despite its apparent simplicity. Most applications of the SIR framework use a form of nonlinear incidence to describe the number of new cases per instant. We adapt theorems to analyze the stability of SIR models with a generalized nonlinear incidence structure. These theorems are then applied to the case of standard incidence and incidence resulting from adaptive behavioral response based on epidemiological-economic theory. When adaptive behavior is included in the SIR model multiple equilibria and oscillatory epidemiological dynamics can occur over a greater parameter space. Our analysis, based on the epidemiological-economic incidence, provides new insights into epidemics as complex adaptive systems, highlights important nonlinearities that lead to complex behavior, and provides mechanistic motivation for a shift away from standard incidence, and outlines important areas of research related to the complex-adaptive dynamics of epidemics.

Keywords

Economic-epidemiology; adaptive behavior; nonlinear incidence

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We dedicate this article to Kenneth Cooke who passed away on August 25, 2007 at the age of 82.

1. Introduction

Adaptive behavioral changes affect interactions and mixing rates among individuals and play a crucial role in determining the level of incidence, rate of spread, and the overall dynamic path of an epidemic. Individuals likely alter their behaviors in response to current epidemic conditions in order to prevent themselves from getting sick, but these behavioral changes are mediated by the individual's assessment of the role of factors that contribute to infection risk (e.g., activity, vaccination status, contact networks). Certain behavioral modifications such as total individual isolation or celibacy, in the case of sexually transmitted diseases, would eliminate the risk of individual infection. However, such behavior is likely to be excessively costly to individuals. In general, individuals value health as an input to generating utility, an index of satisfaction or wellbeing. Most mathematical models of disease transmission do not explicitly incorporate utility, or goal-seeking behavior, which leads to adaptive behavioral changes (Kermack and McK-endrick [1927], Anderson et al. [1989], Hadeler and Castillo-Chavez [1995], Brauer et al. [1998], Chowell et al. [2003], Del Valle et al. [2005], Morin et al. [2010]). By focusing on goal seeking models, we are able to capture truly endogenous adaptive behavioral responses. This level of analysis, within the contact structure of a population, is only beginning to emerge in epidemiological models (Chen [2009], Fenichel et al. [2011], Shim et al. [2012]). Funk, Salathe, and Jansen put together reviews of adaptive behaviors in epidemiological models Funk et al. [2010]; our paper fits neatly into the discourse of models with globally available prevalence information and behavior alters both the model parameters, and, due to the behavior response being based on state variables, the contact structure itself. We are also distinct from the review of economic controls within the work of Gersovitz and Hammer as we are modeling positive behavior rather than normative (e.g., vaccination) Gersovitz and Hammer [2004].

The "co-evolutionary" dynamic between disease prevalence and behavioral response is a quintessential complex adaptive system, systems with nonlinear interactions at multiple scales Mitchell [2009]. In models without the behavioral adaptive dynamic approach, one considers dynamics on a static landscape—the phase dynamics are can be completely characterized irrespective of the initial condition of the system—where the future course of the dynamic flow can be well described without precise knowledge of the initial conditions. Acknowledging adaptive behaviors means acknowledging a feedback where the landscape affects the evolution of the epidemic and the evolution of the epidemic affects the topological landscape and phase dynamics (see Fenichel and Horan [2007], Horan et al. [2011] for more discussion of these types of feedbacks). The implications of this feedback can be profound for empirical work in addition to theory. Geoffard and Philipson note that while estimates of parameters in nonadaptive epidemiological models are quite robust, even with the proliferation of compartments, once adaptive behavior is introduced the robustness of estimation, and identification of mechanism, is lost Geoffard and Philipson [1995].

Recent experiences with epidemics such as SARS Chowell et al. [2003 SARS Chowell et al. [2004a], avian influenza Shim and Galvani [2009], and ebola Chowell et al. [2004b] demonstrate the role of behavior in both the spread and control of the epidemics. Policy response to these outbreaks, aimed at altering pathogen dynamics, resulted in public

interventions that had large impacts on the socio-economic landscape (i.e., the collection of individual social and economic statuses within the population). A rigorous theoretical epidemiological framework for modeling how human decisions, related to intentional and adaptive goal-seeking behavior, shape disease dynamics is needed to capture the influence of the protective behaviors induced by the fear of emergent or re-emergent infectious pathogens (e.g., HIV Anderson et al. [1989], Gupta et al. [1989], Busenberg and Castillo-Chavez [1991], Palmer et al. [1991], Hadeler and Castillo-Chavez [1995], Brauer et al. [1997 , 1998], Kribs-Zaleta et al. [2005], Gumel et al. [2006], Kasseem et al. [2006], Hatchett et al. [2007]) or from the potential deliberate release of biological agents Banks and Castillo-Chavez [2003], Del Valle et al. [2005].

Recently, public health officials have systematically employed travel restrictions and social distancing measures to reduce disease spread (Chowell et al. [2003, 2004a, 2009], Epstein et al. [2007], Barrett and Brown [2008], Córdova-Villalobos et al. [2009]). Policies quarantining or restricting contact among individuals may lead to the greatest reduction in cases but the implementation (particularly over sustained periods in time) of such extreme policies may induce other unforeseen private and social costs Smith et al. [2009]. Enactment of contact related policies may close schools, restrict social and cultural activity, and even shut down major metropolitan areas. These heavy handed policies had serious impact on local and global economies specifically during the H1N1 pandemic Shim and Galvani [2009], Córdova-Villalobos et al. [2009].

Related literature (Geoffard and Philipson [1996], Philipson [2000], Auld [2003], Fenichel et al. [2011], Galvani et al. [2007], Reluga [2010], Chen [2004, 2006, 2009]) posits that forward looking individuals aim to maximize an objective function that includes, but is not limited to, health over a planning horizon. A utility function is specified that includes health status and other goods. Maximization of this utility function induces individuals to make tradeoffs between long term health and the short term costs of avoiding infection. In these studies optimization is used as a way of modeling the goal seeking adaptive decisions of members of society (important to the understanding of behaviorally driven disease dynamics) and not for engineered public health interventions. Such understanding can ultimately help develop better normative (policy driven) disease intervention strategies (e.g. Galvani et al. [2007]). Fenichel et al. [2011] simulate the effects of an individual decision making model and illustrate the implications of adaptive behavior for reproductive number theory and disease dynamics within the compartmental framework. Moreover, they show the potential for policy changes that alter the benefits and cost of disease avoidance lead to oscillatory dynamics (sometimes called waves in the epidemiological literature Chowell et al. [2009], Miller et al. [2009]). In the work by Chen [2004 , 2006] the individuals choose the level of risky behavior they undertake by weighing the costs and benefits of such actions. In these models the individuals within the population are each associated with a parameter describing the measure of preference for self-protection they exercise. Solving the dynamic optimization problem gives the distribution of this parameter at the steady state equilibrium. This is similar to work done by Novozhilov, but the latter did not utilize goal seeking behavior Novozhilov [2008]. Additionally Chen's work finds the stability conditions in the homogeneous case, where the entire population evaluates risky behavior with the same preference.

We provide a general mathematical foundation for including adaptive human behavior in epidemiological models by incorporating work by Blythe et al. [1991] into a behavioral framework where individuals have a short-term payoff from making contacts with others. Individuals trade off between the increase in utility that results from increased contacts with the risk that additional contacts could lead to future utility lose through infection. Forgoing contacts in the present is similar to investing in future health capital as found in Fenichel et al. [2011]. We aim to combine the analyses of the general, nonlinear interactions between individuals during the course of an epidemic with the individual based optimization framework to construct an approach to modeling, that is, mechanistic with respect to social and economic considerations, *economic epidemiology*. Differing from Chen's work, we assume that the rational behavior which has an impact on health state is found only within susceptible population and that the adoption of such behavior is homogeneous within this group. In Chen's papers there is a mechanism for reinfection.[1]

In Section 2 we develop a traditional, and general, model for the transmission of an influenza like disease with nonlinear incidence. The economic considerations are described in detail and introduced into the model to serve as the new *adaptive behavior model*. The major difference between the two models lies in the individual's ability to adjust behavior in the economic model while all contact rates (i.e., behaviors) are fixed in the classical formulation. This serves to highlight the mathematical differences induced by the changing behavioral landscape as well as the possible richness of behavior that may arise when explicitly considering behavior. Section 3 compares outcomes between the two models and details the application of the theorems contained in Blythe et al. [1991] to the adaptive behavior model. Section 4 raises new questions about the implementation of the individual behavior regime and discusses the results of this paper.

2. Mathematical formulations

Incorporating adaptive human behavior into mathematical models introduces a number of nonlinearities thereby drastically increasing the model's complexity. Here we investigate one of the simplest models describing influenza like dynamics in order to outline a basic technique of incorporating adaptive behavior. We divide the population into three compartments based on disease state to describe classic susceptible-infected-recovered (SIR) disease dynamics Kermack and McKendrick [1927]: susceptible to the disease, S; infected and infectious, I; recovered and permanently immune, R. Individuals are added to the susceptible class at the constant rate Λ and are removed from each health class at the per-capita rate μ. Disease recovery is modeled via a constant per capita rate γ (with $1/\gamma$ being the average length of infectiousness). Infection incidence within a population can be described as the product of four terms: the per-capita average number of contacts, c; the probability that a contact between a susceptible and infectious individual results in a new infection, β; the number of susceptible individuals who may become infected, S; and a nonlinear function $F(S, I, R)$ describing how the presence of a disease affects incidence. We assume that all system parameters are strictly positive, and that the population mixes proportionately resulting in an incidence function given by

[1]Recovery does not imply permanent immunity such as in an SIRS or SIS setting.

$$B = c\beta F(S, I, R) S \frac{I}{N}, \quad (1)$$

where $N = S + I + R$ as in Blythe et al. [1991]. This results in a model given by

$$\begin{aligned} \dot{S} &= \Lambda - c\beta S F(S, I, R) \tfrac{I}{N} - \mu S, \\ \dot{I} &= c\beta S F(S, I, R) \tfrac{I}{N} - \gamma I - \mu I, \\ \dot{R} &= \gamma I - \mu R. \end{aligned} \quad (2)$$

The number and stability of equilibrium points and whether oscillatory solutions exist follow from the basic reproductive number paradigm often applied to compartmental disease models Brauer and Castillo-Chavez [2001].

2.1. Nonadaptive, nonlinear theorems

First we characterize the stability of the disease free equilibrium (DFE). Common practice is to characterize the stability of the DFE through an appeal to the concept of the basic reproductive number $\Re_0$. This quantity is interpreted as the average number of secondary infections a typical infectious individual causes in a fully susceptible population. If $\Re_0$; is less than unity, then the disease dies out,[2] and the DFE is locally stable. If $\Re_0$; exceeds unity, in systems with recruitment of new susceptibles, then the rate of infection results in an endemic equilibrium level of infection, and the DFE is locally unstable. For the purpose of our analysis we assume that asymptotically the entire population reaches an equilibrium value[3] of $\frac{\Lambda}{\mu}$.

Theorem 1. (Blythe et al. [1991])—If

$$0 \leq F(S, I, R) \leq F\left(\frac{\Lambda}{\mu}, 0, 0\right) \leq F(\infty, 0, 0) = 1 \quad (3)$$

and

$$\Re_0 := \frac{\beta c}{\gamma + \mu} F\left(\frac{\Lambda}{\mu}, 0, 0\right) < 1, \quad (4)$$

then the DFE of System 2 attracts all local solutions, that is,

$$\lim_{t \to \infty} (S(t), I(t), R(t)) = \left(\frac{\Lambda}{\mu}, 0, 0\right).$$

If $\Re_0$; > 1, then the DFE is locally unstable.

[2]It is known that the biological interpretation of this typical threshold quantity breaks down in more complicated models Heesterbeek and Roberts [2007], Roberts and Heesterbeek [2003].

[3]A result easily arrived at when solving the ordinary differential equation for $\dot{N} = \dot{S} + \dot{I} + \dot{R}$.

The interesting case is when the DFE is unstable. The existence of oscillations of the susceptible and infected populations is possible due to the recruitment mechanism of new susceptible individuals into the population. We can extend previous local stability claims to global ones by ruling out oscillatory behavior. To facilitate analysis of oscillatory solutions, assume that the total population has stabilized[4], for example, $S(t) + I(t) + R(t) = \Lambda/\mu$. Applying Dulac's Criteria (e.g., see Busenberg and Vandendreissche [1993] for some examples and an extension) to the resulting planar system results in

Theorem 2. Blythe et al. [1991]—If $\frac{\partial F}{\partial S} > \frac{\partial F}{\partial I}$ for $S > 0$, $I > 0$, then System 2 has no limit cycles (e.g., oscillatory solutions) in the positive cone.

It is useful to note that the term $\frac{\partial F}{\partial R}$ is absent from this condition on the existence of limit cycles. This is because we are analyzing limiting behavior and thus $R(t) = N(t) - S(t) - I(t) = \frac{\Lambda}{\mu} - S(t) - I(t)$ (all dynamic information is studied by considering $S(t)$ and $I(t)$). Even if limit cycles have been ruled out the autonomous nature of F may enduce endemic levels that are dependent on the initial status of the system. An additional theorem supplies sufficient conditions for unique equilibria.

Theorem 3. (Blythe et al. [1991])—Given System 2 if $\frac{\partial F}{\partial S} \geq 0$, $\frac{\partial F}{\partial I} \leq 0$, $\frac{\partial F}{\partial R} \leq 0$, and $\Re_0$; > 1 then System 2 has a unique endemic equilibrium.

The condition on $\frac{\partial F}{\partial R}$ in Theorem 3 implies that the existence of recovered individuals does not result in increased transmission Blythe et al. [1991]. These theorems may be used to illustrate the effects adaptive behavior can have on the course of an epidemic.

2.2. Incidence functions and the adaptive behavior model

In epidemiological models frequency dependent mixing, or standard incidence, occurs when contact rates are assumed independent of population density. Classical standard incidence can be expressed as $F(S, I, R) = 1$, yielding an incidence function of $B = c\beta S\frac{I}{N}$.

Individuals may alter contact behavior over time with respect to the amount of infectious individuals within the population, I. Adaptive behavior implies that the rates c or β are not constant, but functions of I and potentially other state variables. If people behave adaptively, then the observed population level dynamics of an epidemic emerge from individual decision making. We abstract the measure of wellbeing, or benefit, an individual gains in the process of interacting with others (money, enjoyment, etc …) as utility. Individuals aim to minimize the loss of utility, that comes from becoming infected, during the course of an epidemic. Forgoing contacts reduces the probability of infection, but results in a loss of otherwise beneficial social contacts that may lead to infection. An individual may possibly reduce the intensity or alter the nature of activities during an epidemic to reduce the risk of infection, but in this work we only suppose social distancing, that is, β is left constant.

[4]The assumption that the demographically limited dynamics match those of the original model is made valid in Castillo-Chavez and Thieme [1994]. This does not imply that for the remainder of the paper we assume the demographic limit. Indeed, other than when discussing limiting behavior, N is varying with time.

The tradeoff between gaining utility through current period contacts and gaining utility through avoiding future infection implies an optimal individual utility maximization describing the strategies used by purposeful goal-seeking individuals to adaptively manage the benefit-risk tradeoffs tied to contact activities over a planning horizon with discount rate δ (e.g., Dixit and Pindyck [1994], Mas-Colell et al. [1995], Adda and Cooper [2003]).

Individuals experience a marginal increase in utility up to some point as a result of making contacts within a unit time. We let $f_t := f(c_t)$ denote the utility function associated with the time interval $[t, t + 1]$, a measure of the net benefit one receives from contacts during a given time period where c_t is the number of contacts made within this interval. Assume that f_t is concave where c_t^* is the optimal number of contacts within the selected time window $[t, t + 1]$ when the disease is absent from the population. The cost of making contacts in excess of c_t^* is prohibitive, and we let $f_t(0) = 0$. This discrete time decision making process does not imply that the disease dynamics are no longer continuous as in equations (2). Rather we are modeling a behavior that occurs once, at the beginning of a "day": each individual is aware of their own disease state as well as the current time $S(t)$, $I(t)$, and $R(t)$; the individuals, with knowledge of all demographic and epidemiological parameters, now choose their number of contacts in order to maximize utility over a planning horizon; this is repeated each "day." Updating the decision in a continuous manner may imply that either there is a continuum of time periods over which different individuals choose their contact number or that individuals are constantly adjusting their contacts to a persistent flow of real time information. The former implication, while it may be realistic in a setting where different people have different behaviors and even wake up to start their day at different times, this is not within the realm of the homogeneity in this model (see Chen [2004 , 2006] for approaches with heterogeneous behavior patterns within disease state groups). We feel that the latter implication of a fully connected population with enough social agility to constantly affect their contacts is unrealistic.

We assume the health status of an individual influences his utility function directly in situations where the population faces an epidemic outbreak. An individual's status is indicated via the subscript $m \in \{s, i, r\}$ (susceptible, infected, and recovered) and it is assumed that individuals with different statuses benefit from contacts differentially. We allow the status of individuals to be an argument in the utility function, $f_t(c_{t,m}, m)$, and assert that for a given number of contacts, $c_{t,m}$, that $f_t(c_{t,i}, i) < f_t(c_{t,r}, r) \leq f_t(c_{t,s}, s)$ where f is strictly a measure of the net benefit one receives from contacts during a given time period.

In this framework all individuals are perfectly informed about the current state of the epidemic and seek to maximize their individual utility. Moreover, we assume that individuals do not care about the health of others and take the behavior of others as given; there is an absence of strategic behavior among individuals who are not susceptible to infection. Utility maximization provides a parsimonious model of goal-seeking behavior.[5] Infected or recovered individuals do not have incentives to modify behavior away from the behavior under disease free conditions as recovered individuals are immune to the disease,

[5]We are not making normative judgements about what that goal should be or what the individual should do to maximize his utility. Rather we are asserting that optimization provides a positive model of individuals making tradeoffs.

and we assume contacts do not affect recovery from infection. Individuals in classes *i* and *r* choose the number of contacts per day that maximizes individual utilities, $c^*_{t,i}$ and $c^*_{t,r}$, respectively. Only susceptible individuals modify their behavior in response to changes in disease prevalence. These individuals face incentives to reduce contacts below the optimal disease free contact level in order to mitigate infection risk. To analyze the intertemporal tradeoff, we construct the expected utility function, a measure of the present and the future value of contacts, over the time horizon *T*. Future utility is discounted by a factor $\delta \in [0, 1]$ to account for the rate of time preference, an individual's preference for goods today relative to tomorrow. Thus over a planning horizon [0, *T*] the expected utility for a susceptible, conditioned on the individual's future state, is $\mathbb{E}(U)=f_0(c_{0,s}, s)+\sum_{t=1}^{T}\delta^t\mathbb{E}^m(f_t(c_{t,m}, m_t))$.

In order to arrive at an expression for the expected utility for an infected individual, we let *v* denote the time of infection and $\rho := \lceil 1/\gamma \rceil$ be the expected length of infection in the relevant time units rounded to the nearest integer above $1/\gamma$. Thus the expected utility for an infected individual is $\mathbb{E}(U)=\sum_{t=v}^{v+\rho} f_t(c^*_{t,i}, i)$, where $c^*_{t,i}$ is used because the infection does not create incentives to decrease contacts. Furthermore, for a recovered individual the utility for every time interval after recovery is $f_t(c^*_{t,r}, r)$.

The differentiated contact structure induced by the adaptive behavior grants a more complicated form to the standard incidence. Over the time interval [*t*, *t* + 1] the incidence is

$$B=c_{t,s}\beta S\frac{c^*_{t,i}I}{c^*_{t,s}S+c^*_{t,i}I+c^*_{t,r}R}, \quad (5)$$

where $c_{t,s}$ denotes the number of contacts of the representative susceptible agent and $\frac{c^*_{t,i}I}{c^*_{t,s}S+c^*_{t,i}I+c^*_{t,r}R}$ is the proportion of these contacts with an infected individual in randomly mixing population. We emphasize that $c_{t,s}$ is a *choice* made by the susceptible individuals, made in this context through optimizing expected utility, and $c^*_{t,h}$ is the expected number of contacts made by an individual of type *h*. Consequently, we may define the state-dependent function *F* as

$$F(S, I, R)=\frac{c_{t,s}c^*_{t,i}}{c^*_0}\frac{S+I+R}{c^*_{t,s}S+c^*_{t,i}I+c^*_{t,r}R}, \quad (6)$$

where *c* and c^*_0 have contextually analogous definitions (i.e., the average number of contacts at time zero of the epidemic). We therefore have two examples of the model described in System (2): one where *F* (*S*, *I*, *R*) = 1 and a second where *F* is defined by equation (6). Noting that $c=c^*_0$ we may rewrite System (2) to reflect the inclusion of adaptive behavior via

$$\begin{aligned}\dot{S} &= \Lambda - c_0^* \beta F(S,I,R) S \tfrac{I}{N} - \mu S,\\ \dot{I} &= c_0^* \beta F(S,I,R) S \tfrac{I}{N} - (\gamma+\mu) I, \quad (7)\\ \dot{R} &= \gamma I - \mu R,\end{aligned}$$

with the important distinction that the number of contacts a susceptible chooses to make is an unspecified nonlinear function of the state parameters and system parameters which could be found explicitly if the optimization problem were analytically solvable, that is, $c_{t,s}^* = c_{t,s}^*(S, I, R, \Lambda, \beta, \mu, \gamma, b, \nu, T)$.

3. Results

Theorem 1 implies

$$\Re_0 := \frac{\beta c_0^* F(N,0,0,)}{\gamma+\mu} = \frac{\beta c_{0|i}}{\gamma+\mu},$$

where time $t = 0$ is the time of introduction of the first primary case. Basic reproductive numbers do not account for the behavioral adaptation of the susceptible population, or any other autonomous effect for that matter. If the risk of infection, β, and/or the period of infection, $\frac{1}{\gamma}$, were large enough, then a single infected individual may cause all susceptibles to reduce contacts to 0. In this situation of extreme behavioral change the epidemic threshold of $\Re_0$; is not reflective of the system dynamics. In order to rule out oscillatory solutions in a system with adaptive behavior through application of Theorem 2 we must show that $\frac{\partial F}{\partial S} > \frac{\partial F}{\partial I}$. Let $D := c_{t,s} S + c_{t,i} I + c_{t,r} R$ and compute the difference

$$\frac{\partial F}{\partial S} - \frac{\partial F}{\partial I} = \frac{c_{t,i}^*}{c_0^* D^2} \left\{ \left[\frac{\partial c_{t,s}}{\partial S} - \frac{\partial c_{t,s}}{\partial I} \right] (c_{t,i}^* I + c_{t,r}^* R) N + c_{t,s}(c_{t,s} - c_{t,i}^*)(S - I - R) \right\}.$$

The first term in brackets is positive by the properties of $c_{t,s}$, the number of susceptible contacts increase with S and decrease with I. The sign of the second term, $(c_{t,s} - c_{t,i})(S - I - R)$, is ambiguous and requires further specification of the utility functions. A sufficient condition for $\frac{\partial F}{\partial S} - \frac{\partial F}{\partial I} > 0$ is that $S < I + R$ if and only if $c_{t,s} < c_{t,i}^*$. That is, in order to rule out oscillatory dynamics, it is sufficient to note that when the total population that is infected, or has been infected, exceeds the susceptible population then the susceptible individuals must each make fewer contacts than infected individuals. Heuristically this requires that in the presence of a large recovered population, very small infection levels would induce susceptible individuals to make very few contacts. This contradicts intuition associated with the utility maximization problem. The preceding description of individual behavioral does not allow for the presence of recovered individuals to have a negative impact on susceptible behavior.

Alternatively, let $\hat{F}(S, I, R) = \frac{c_{t,s}}{c_0^*}$, and apply Dulac's Criteria directly to the planar system

$$\begin{aligned}\dot{S} &= \Lambda - c_0^* \beta \hat{F}(S,I,R) S c_{t,i}^* \tfrac{I}{G} - \mu S, \\ \dot{I} &= c_0^* \beta \hat{F}(S,I,R) S c_{t,i}^* \tfrac{I}{G} - (\mu+\gamma) I, \end{aligned} \quad (8)$$

where $G = c_{t,s} S + c_{t,i}^* I + c_{t,r}^* (\frac{\Lambda}{\mu} - S - I)$. Thus limit cycles do not exist provided that for all t greater than a finite t^* one has that $c_{t,s} < c_{t,i}^*$, $\frac{\partial \hat{F}}{\partial S} > 0$, and $\frac{\partial \hat{F}}{\partial I} < 0$. To better understand this condition consider two cases: a large endemic population and a small endemic population. If the steady state for the infected class, I_∞, were large then requiring $c_{t,s} < c_{t,i}^*$ for all t larger than some t^* is reasonable, because the susceptible individuals would be actively trying to avoid becoming infected. However, for a small I_∞ this requirement would imply some memory (a non-Markov behavioral model) linked to the infection that involves the susceptible population avoiding a second epidemiological peak (i.e., $\frac{dI}{dt} > 0$ after a period where $\frac{dI}{dt} < 0$). Therefore, Theorem 2 does not rule out oscillatory behavior for the adaptive system in general, particularly at low endemic infected levels. This result provides a more mechanistic insight to Brauer et al. [1997] result that information may destabilize a system (induce oscillatory behavior) when dynamics would otherwise be stable in the absence of information.

To illustrate adaptive behavior's effect on the epidemiological system we numerically solved System (2) using a standard fourth order Runge-Kutta method. This was done with $F(S, I, R) = 1$ to give results from a traditional epidemiological model and with F as defined in Equation (6) to compute solution curves $S_e(t)$, $I_e(t)$, and $R_e(t)$ (e.g., the solution curves incorporating behavior). The utility functions are defined by the expressions $f_t(c_{t,s}, s) = f_t(c_{t,r}, r) = (b c_{t,m} - c_{t,m}^2)^\nu$ with $b = 24$ and $\nu = 0.2$ and $f_t(c_{t,i}, i) = 0$. The adaptive model was solved by halting the numerical integration at each time step (day) and then adjusting the contacts of the susceptible population in order to produce the maximum sum of utility from the current time over the time horizon T with the discounted, $\delta = 0.9986$, utility weighted with the probability of being in each of the health states for the next time step. The epidemic parameters used are $\beta = \gamma = 0.2$; and the initial conditions are set at $(S(0), I(0), R(0)) = (9999, 1, 0)$. We varied several quantities: the planning horizon T from 1 to 20 days in single day increments; the birthrate Λ from 0 to 100 individuals per day in increments of 10; and the utility shape parameter ν from 0 to 1 in 0.01 increments. In any case we set the removal rate of individuals to $\mu = \Lambda/10000$ in order to keep the total population consistent throughout changes to the model parameters. The optimization problem was solved using dynamic programming as in Adda and Cooper [2003], but assuming only integer values for $c_{t,s}$.

To capture the effect of the planning horizon on the behavior we consider the effect it has on the minimum number of contacts, the duration of effected behavior, the average number of contacts over the interval of time of effected behavior, and the peak of the prevalence curve (summarized in Figures 1 and 2). As shown in these Figures changing the planning horizon from 8 to 9 days produces the most dramatic changes to the epidemic: the duration decreases by 10 days, the peak prevalence increases by nearly 1185 individuals, and the minimum number of contacts chosen increases by 2.

By grouping all planning horizons that generate behavior identical to $T = 8$, that is, $T \in \{3, 4, 5, 6, 7, 8\}$, we may then chose the planning horizon with the presumably smallest impact on behavior as the optimal planning horizon for closed populations with $\nu = 0.2$, that is, the one that generates the shortest duration of effected behavior, $T = 3$.

We next review the effect of opening the population via $\Lambda > 0$, with $T = 3$. For $\Lambda > 10$ the effect on behavior is persistent for all time, that is, $c_{\infty,s} < 12$. Thus for small Λ the adaptive model returns to the nonadaptive behavior, however in such a case the persistent level of infection is not equivalent to the nonadaptive model, shown in Figure 3. Once behavior returns to a persistent pre-epidemic level, at day 469, the model should achieve the endemic level of the model without behavior. The presence of S_∞ for the adaptive model not equivalent to that of the nonadaptive model (which is simply a special case of the adaptive model) is an example of nonuniqueness (for other examples see Castillo-Chavez et al. [1989], Huang et al. [1990], Blythe et al. [1991], Hadeler and Castillo-Chavez [1995], Dushoff et al. [1998], Feng et al. [2000], Huang and Castillo-Chavez [2002]). Also, in the case that $\Lambda = 80$ for planning horizon $T = 3$, shown in Figure 4, persistent oscillations occur presumably as a result of having I_∞ within a "sweet spot." Within this "sweet spot" the level of infection is not so high as to produce persistent, and very low, constant choices of $c_{t,s}$ or so low as to have the infection process dominated by the entry/removal of individuals, but it is just right to produce low infection levels where the epidemiological process is not dominated by the demographics.

This "sweet spot" may be avoided by not only adjusting T but also via the utility shape parameter ν. For $\Lambda = 80$, $T = 3$, and $\nu = 0.19$ (instead of 0.2) the oscillating behavior gives way to a persistent $c_{t,s} = 9$. If one considers $\Lambda = 10$ and $T = 3$ we can induce oscillatory behavior via $\nu = 0.1375$ where $c_{t,s}$ bounces between 11 and 12 every day.

4. Discussion

Infection within a population creates economic incentives that result in adaptive decisions at multiple levels of social organization and over various temporal scales Fenichel et al. [2011]. Individuals gain utility from making contacts with others, but each contact incurs additional risk or exposure to disease. Tradeoffs between increased utility in the present and the risk that such contact could lead to future utility lose through infection occur on the individual level. These decisions manifest work time, reduced productivity and health care expenses that add to the social cost of disease. In a sense individuals are involved in a dynamic game choosing strategies comprised of current and future contacts with payouts described by expected utility Reluga [2010]. The actual strategies employed may involve a degree of commitment (i.e., open-looped in the short-term) or may involve regularly updated reaction function (i.e., closed looped, Markov perfect strategies) Tsutsui and Mino [1990]. This perspective is different than viewing the control process as an effort to reduce the total number of infected.

Central to the difficulties with implementing the decision making process into the epidemic model, and thus applying the theory of nonlinear incidence, is that we lose a consistency of mixing, an important assumption in most analyses. However, the assumption that

individuals of different health statuses behave identically seems overly strong. The typical dynamic programming Adda and Cooper [2003], solving backwards along an “optimal” path to arrive at the decision to be made in the now, implemented into epidemic systems is difficult because there are at least three time scales to consider: the epidemic scale, the decision scale, and the information arrival rate. As was shown in the contact plot within Figures 3 and 4 the period over which strategies changed were not necessarily days and did not adhere to rates of change of the system in directly identifiable ways. An appropriate and realistic time scale may be a time period of t (that may be random) that models how long it takes to disperse information as a function of factors such as disease prevalence and severity. In this paper, we numerically solved the ODE system 2 stopping at each integer time value and recalculate the expected utility over the next T unit times. Simulating in this fashion has shown that the economic behavior can induce oscillation, in both the behavior and the epidemic trajectory, and may maintain a susceptible population at a much higher level than without the behavioral adjustment (with appropriate parameter values our simulation models have generated infection level a whole order of magnitude less). Longer time scales, likely the case with the proposed random interval model, are expected to induce similar behavior over a broader parameter range.

A time step with biological significance is that of *event times*. The process then has stochastic time steps and the susceptible individuals would update their behavior as events (new infection, new recovery, new individual enters the system or an individual leaves the system) occur. Without exploring this method here it should be clear that this would produce far fewer infections than the methods in this paper because there would be a more rapid dissemination of information implied. This illustrates a critical new frontier yet to be resolved in epidemiology or the study of complex systems more generally—the need for a general way to address temporal scaling issues.

The complex adaptive system generated by the introduction of the economic behavior described here may reduce an epidemic’s forecasted size and alters forecasts to suggest a spreading out of the peak of the epidemic over time while lessening its severity. This implies that individual pathogens may actually be more biologically infectious than currently believed. To strengthen the results, in order to make policy decisions using the ideas of utility maximization, a great deal of work should be put into estimating the form of f_t ($c_{t,m}$, m) as the numerical results are sensitive to its shape. The introduction of differential contact qualities with different payouts and risks would add a level of realism and applicability (e.g., family contacts versus work contacts or monogamous versus polygamous). In addition, population wide policy decisions, such as closing public transportation, may also affect the tradeoffs with respect to $c_{t,s}$. Such policies could have unintended consequences (i.e., forming reservoir susceptible populations that may produce second epidemic peaks) if we do not explicitly consider the adaptive nature of human behavior. Despite all the challenges involved in such a complicated model we have been able to use previous techniques to prove stability and complexity of fixed points for the system, and proofs of qualitative behavior under a delay in information is underway again using theory from Blythe et al. [1991]; for completeness we have outlined the relevant theory here in the appendices.

Numerically we've been able to show that the entry and removal in the system may be used as a control, with the economic behavior structure, to destabilize, and induce oscillatory behavior. Additionally, we've demonstrated that for a given entry/removal rate there exist combinations of planning horizon, T, and utility shape parameter, ν, that may be utilized individually or in tandem to induce/remove oscillatory behavioral response and to affect the persistent (nonoscillatory) behavior level. We state this result in an unproven proposition motivated by our numerical results.

Proposition 1. [Oscillatory Control]

For $\Lambda > 0$ a pair of planning horizon, T, and utility shape parameter, ν, values may be chosen such that oscillation does exist for one and does not exist for the other. In other words $\forall \Lambda > 0$, $\exists(T_1, \nu_1)$, and (T_2, ν_2) such that for one pair oscillatory behavior exists and for the other it does not. Furthermore, it is not necessary that both $T_1 \neq T_2$ and $\nu_1 \neq \nu_2$.

To advance the applicability of epidemiological models it is imperative that we move from thinking of individuals as passive particles to beings that actively attempt to shape their own futures. In so doing, the mathematics becomes more challenging, but we enhance our chance of explaining complex disease dynamics with parsimonious models.

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Appendix

Proof of Theorem 1

Suppose the conditions of the theorem hold (e.g., Equations (3) and 4). If $I(0) = 0$, the solution has $I(t) = 0$ for all $t \geq 0$ as we see from (7), and therefore $R(t) \to 0$, $N(t) \to \Lambda/\mu$, and $S(t) \to \Lambda/\mu$. If $I(0) > 0$ then $S(t) \leq N(t)$ and

$$\begin{aligned}\frac{1}{\gamma+\mu}\frac{dI}{dt} &= \left[\frac{\beta c}{\gamma+\mu}F(S,I,R)\frac{S}{N}-1\right] I \\ \leq \left[\frac{\beta c}{\gamma+\mu}F(S,I,R)-1\right] I &\leq \left[\frac{\beta c}{\gamma+\mu}F\left(\frac{\Lambda}{\mu},0,0\right)-1\right] I \\ &= (\Re_0-1)\, I<0.\end{aligned}$$

Since $I(t)$ is decreasing, $\lim_{t\to\infty} I(t) = 0$. Then the variation of parametric formula gives

$$R(t)=R(0)e^{-(\delta+\mu)t}+\gamma\int_0^t I(s)e^{-(\delta+\mu)(t-s)}ds,$$

and it follows that $R(t) \to 0$ as $t \to 0$. And since $N(t)$ tends to Λ/μ, we deduce that $S(t) \to \Lambda/\mu$.

The Jacobian matrix of 7, with derivatives evaluated at $I = R = 0$, $S = N = \Lambda/\mu$, is

$$\begin{bmatrix} -\mu & -\beta cF_0 & 0 \\ 0 & \beta cF_0-(\gamma+\mu) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}.$$

The eigenvalues are the diagonal entries. Hence, the DFE is unstable if $\beta cF_0 > (\gamma + \mu)$ or equivalently when $\Re_0 > 1$. This completes the proof of Theorem 1.

Proof of Theorem 2

Let g_1 (S, I) and g_2 (S, I) be the functions in the right members of 7 with $N=\frac{\Lambda}{\mu}$ (i.e., the autonomous version), that is

$$\frac{dS}{dt}=g_1(S,I), \quad \frac{dI}{dt}=g_2(S,I).$$

Then

$$\begin{aligned}\frac{\partial}{\partial S}\left\{\frac{g_1(S,I)}{SI}\right\}+\frac{\partial}{\partial I}\left\{\frac{g_2(S,I)}{SI}\right\}&=-\frac{\Lambda\mu}{\mu S^2 I}-\frac{\beta c\mu}{\Lambda}(F_S-F_R)+\frac{\beta c\mu}{\Lambda}(F_I-F_R)\\&=-\frac{\Lambda}{IS^2}+\frac{\beta c\mu}{\Lambda}(F_I-F_S).\end{aligned}$$

Clearly, the first term is negative. The second term is negative by hypothesis. Thus the expression is of fixed sign in the region $S > 0$, $I > 0$, $S + I \leq \Lambda/\mu$, and it follows from Dulac's Criterion test that 7 has no limit cycles in the region.

Proof of Theorem 3

Define

$$G(\eta):=\Re_0 F(h_1(\eta),h_2(\eta),h_3(\eta))-F_0\eta, \quad \text{(A1)}$$

where F is the nonlinear incidence function, $\Re_0=\frac{\beta c}{\gamma+\mu}F(\frac{\Lambda}{\mu},0,0)$ is the basic reproduction number, $F_0=F(\frac{\Lambda}{\mu},0,0)$, $\eta=\Re_0\frac{F(S^*,I^*,R^*)}{F(\frac{\Lambda}{\mu},0,0)}$, and h_i (η) are expressions of the equilibrium in terms of η. More specifically consider the equilibria of 2 which satisfy

$$\begin{aligned}\Lambda-\mu S^*&=c\beta F^* S^*\frac{I^*}{N^*},\\ c\beta F^* S^*\frac{I^*}{N^*}&=(\gamma+\mu)I^*,\\ \gamma I^*&=\mu R^*,\end{aligned}$$

where $F^* = F(S^*, I^*, R^*)$ and $N^*=\frac{\Lambda}{\mu}$. Supposing that $I^* \neq 0$ we have $\frac{S^*}{N^*}=\frac{\gamma+\mu}{c\beta F^*}=\frac{1}{\eta}$. Letting $q=\frac{\gamma}{\mu}$ it also follows that $\frac{I^*}{N^*}=\frac{1}{1+q}(1-\frac{1}{\eta})$ and $\frac{R^*}{N^*}=\frac{q}{1+q}(1-\frac{1}{\eta})$.

We therefore may write that

$$G(\eta)=\Re_0 F\left(\frac{N^*}{\eta},\frac{N^*}{1+q}\left(1-\frac{1}{\eta}\right),\frac{N^* q}{1+q}\left(1-\frac{1}{\eta}\right)\right)-F_0\eta.$$

As $\eta \to 1$ it is easy to see that $F \to F_0$ and thus $G(\eta) = F_0 (\Re_0 - 1) > 0$. Similarly, given the conditions of F given in Theorem 1 we have that for $\eta > \Re_0$ that $G(\eta) < 0$. Thus by

continuity there is at least one equilibrium. For uniqueness we may assume that $\Re_0 > 1$ and look for conditions for which $\frac{dG}{d\eta} < 0$. Straightforwardly one may show that

$$\frac{dG}{d\eta} = \Re_0 \frac{N^*}{\eta^2} \left(-\left(\frac{\partial F}{\partial S}\right)^* + \frac{1}{1+q}\left(\frac{\partial F}{\partial I}\right)^* + \frac{q}{1+q}\left(\frac{\partial F}{\partial R}\right)^* \right) - F_0, \quad \text{(A2)}$$

which clearly illustrates the sufficient conditions

$$\frac{\partial F}{\partial S} \geq 0, \quad \frac{\partial F}{\partial I} \leq 0, \quad \frac{\partial F}{\partial R} \leq 0,$$

for uniqueness.

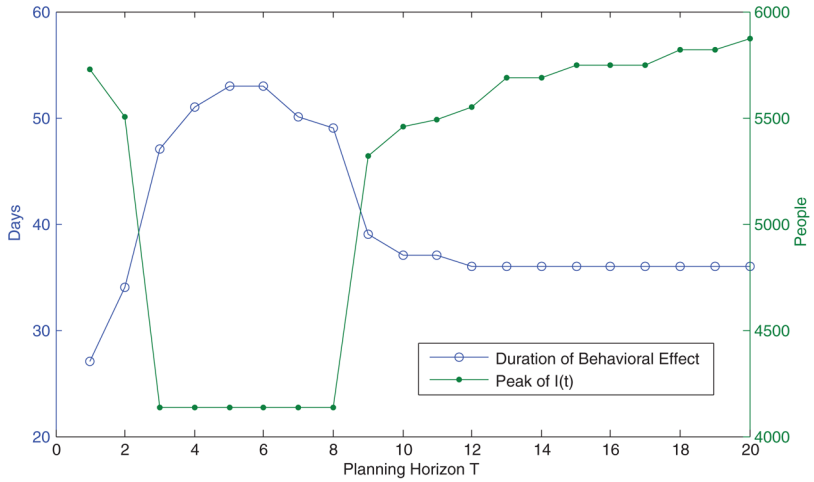

FIGURE 1.

Pictured here we see a comparison of the planning horizon's effect on both the duration of behavior change and the peak of the epidemic. Planning horizons from 3 days to 8 days all produce the same, and relatively small, epidemic peak with $T = 3$ producing the shortest (and thus least taxing on utility) duration of 47 days. Planning horizons shorter than 15 days produce epidemic peaks smaller than the $T = 1$ case.

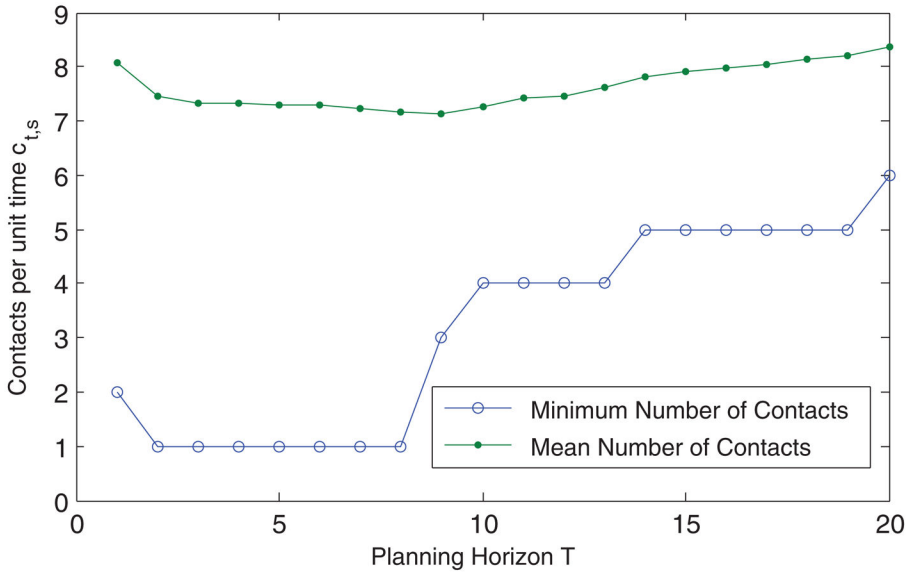

FIGURE 2.

Pictured here is a comparison of the minimum number of contacts chosen during the epidemic and the mean number chosen during the time that behavior was effected (does not include when the number of contacts was chosen to be 12) as functions of the length of the planning horizon. We see that horizons from 3 to 8 days offer no difference in the minimum and marginal difference in the mean. The change from 8 to 9 days offers the greatest change in minimum (2 contacts).

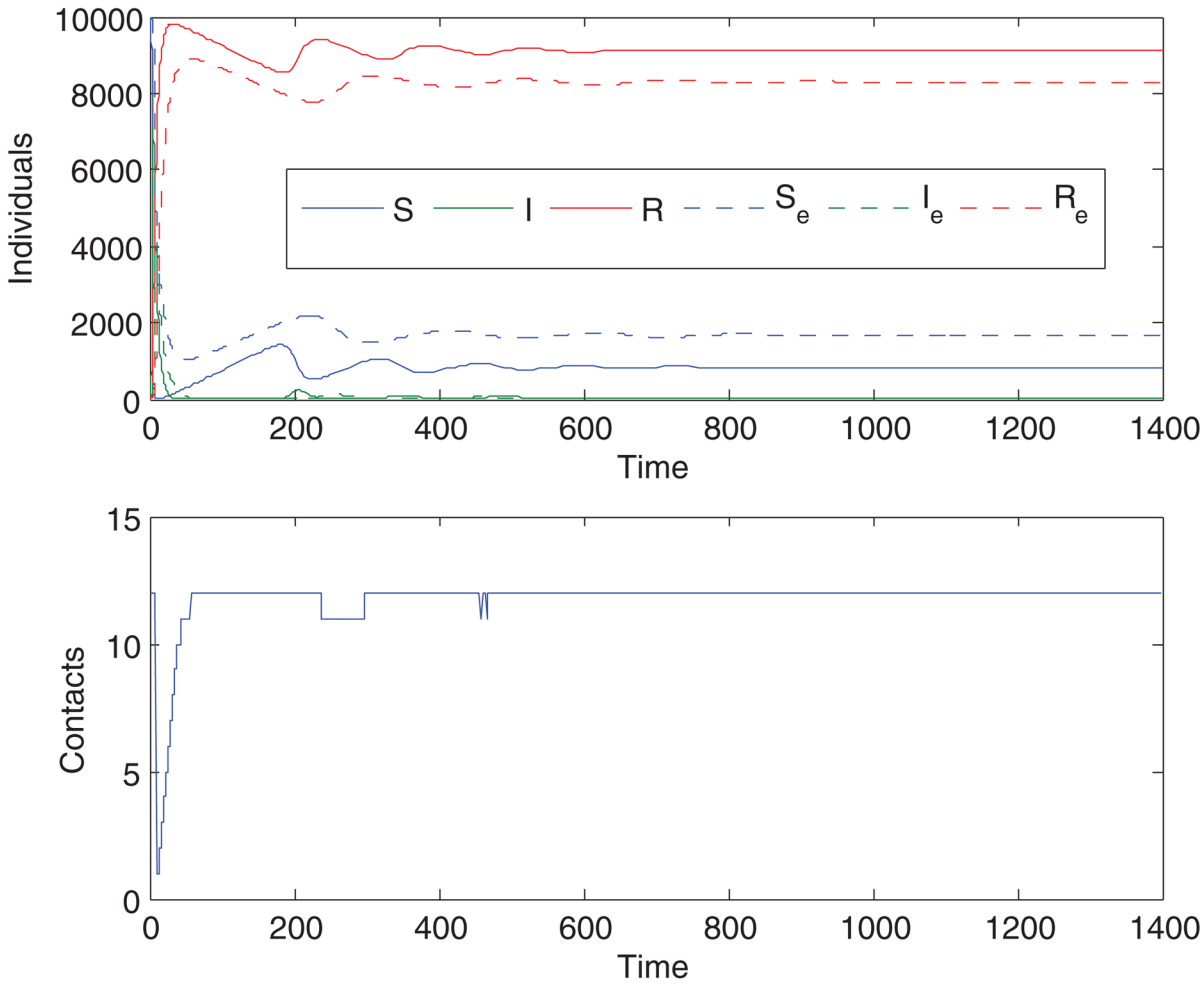

FIGURE 3.

The effect of behavior for $\Lambda = 10$, $T = 3$, and $\nu = .2$ is shown via S_e, I_e, and R_e with the nonadaptive model (S, I, and R). It is clear that although the behavior, shown in the lower subplot, returns to pre-epidemic levels the persistent values for S and R are not equivalent between the two models.

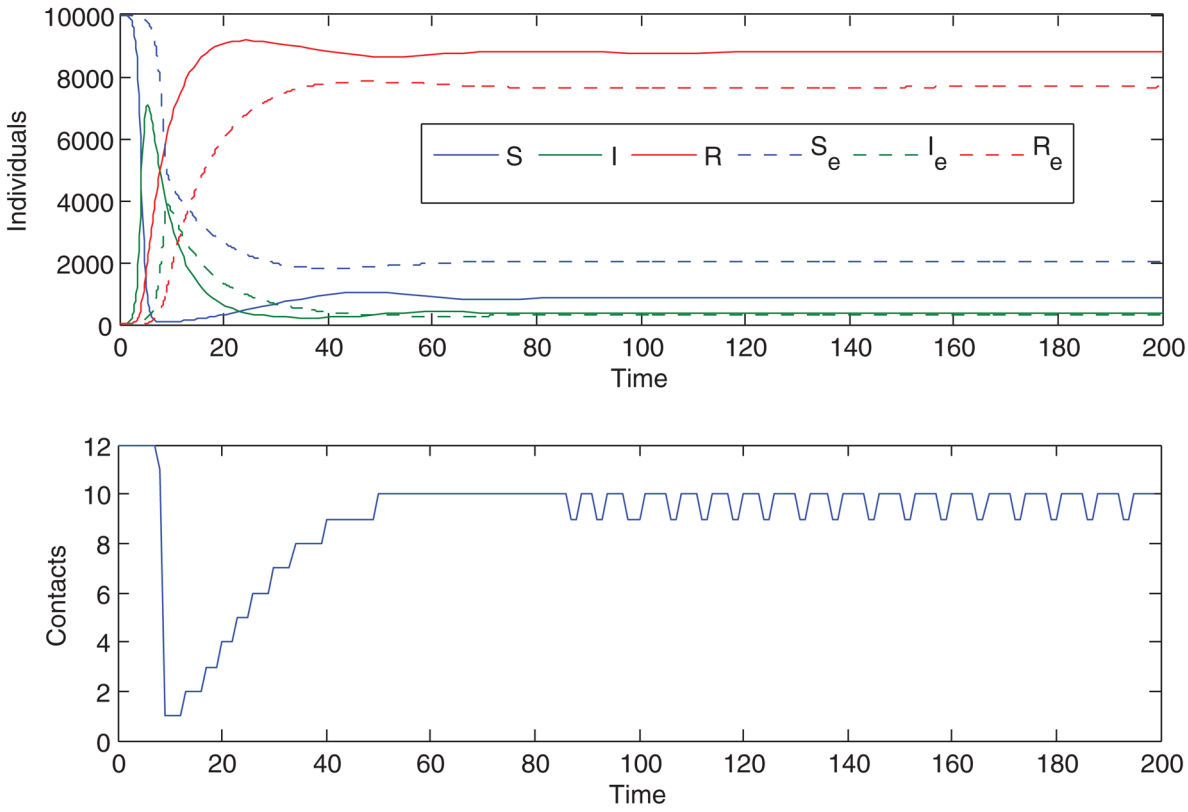

FIGURE 4.

The effect of behavior for $\Lambda = 80$, $T = 3$, and $\nu = .2$ is shown via S_e, I_e, and R_e with the nonadaptive model (S, I, and R). The oscillation in behavior continues for all time with a period of 9 days. Although the oscillations are unobservable in the state variable in this graphic, they are present.