

Selfish Response to Epidemic Propagation

George Theodorakopoulos, Jean-Yves Le Boudec, *Fellow, IEEE*, and John S. Baras, *Fellow, IEEE*

Abstract—An epidemic that spreads in a network calls for a decision on the part of the network users. They have to decide whether to protect themselves or not. Their decision depends on the tradeoff between the perceived infection and the protection cost. Aiming to help users reach an informed decision, security advisories provide periodic information about the infection level in the network. We study the best-response dynamic in a network whose users repeatedly activate or de-activate security, depending on what they learn about the infection level. Our main result is the counterintuitive fact that the equilibrium level of infection increases as the users' learning rate increases. The same is true when the users follow smooth best-response dynamics, or any other continuous response function that implies higher probability of protection when learning a higher level of infection. In both cases, we characterize the stability and the domains of attraction of the equilibrium points. Our finding also holds when the epidemic propagation is simulated on human contact traces, both when all users are of the same best-response behavior type and when they are of two distinct behavior types.

Index Terms—Communication networks, differential inclusions, nonlinear systems, security, switched systems.

I. INTRODUCTION

A COMPUTER worm is a program that self-propagates across a network, exploiting security or policy flaws in widely used services [1]. Worms have the potential to infect a large number of computers, due to the high level of interconnection of the telecommunication infrastructure. Indeed, a relatively recent outbreak (Conficker/Downadup worm) infected more than 9 million computers [2]. Countermeasures to an infection can be centrally enforced, or the decision for their adoption can be left to individual agents such as individual home computer users or companies.

Centralized enforcing is more likely to work in tightly controlled environments, such as within a company network where the users are obliged to abide by the company security policy. In the wireless network setting, controlling the infection has been studied [3] under the assumption that the network operator is

able and willing to control the communication range of each device. Optimal control policies are found, which strike a balance between slowing the spread of the infection and increasing the end-to-end traffic delay in the network.

However, when it is up to individual agents to invest in protection against infection [4]–[7], contradicting incentives appear. Although agents want to be safe against viruses, they would prefer to avoid paying the cost of security. Security may not only cost money, it may also reduce the utility of the network, for example, by isolating the agent from the rest of the network. Moreover, it may reduce the utility of the device, for example, by slowing it down [8].

The risk-seeking attitude of humans when faced with potential losses [9] might shed some light on user incentives. In psychological experiments, Tversky and Kahneman [10] observe that their subjects prefer risking a large loss (in our case, becoming infected) if they also have the chance of losing nothing, rather than taking a sure but smaller loss (in our case, activating security). Therefore, if the threat is not absolutely certain nor imminent, users will resist spending any resources on security (“One solution [for selling security] is to stoke fear.” [9]).

To complicate things further, user incentives may change with time, as the information available to them changes. There exist security advisories that provide information about current and newly emerging threats in popular technology products [11], [12]. Such information influences user decisions and incentives by changing their perception of the risks involved. On the one hand, if users receive news of an ongoing epidemic, they are much more willing to protect themselves. On the other hand, when the infection has subsided and there is no clear danger, complacency may set in with a consequent reduction in the time and resources spent to ensure safety.

To the best of our knowledge, only static incentives of agents have been studied [4], [5]. Users have only been modeled as deciding once and for all whether or not to install a security product. The once-and-for-all approach applies to installing a patch, but other countermeasures exist that can be later revoked by the user: doing background scanning with an antivirus software, setting up traffic-blocking filters on firewalls, disconnecting networks or computers, etc.

In this paper, we study *myopic decision-makers* who receive *dynamically updated information* about the level of infection in the network. We model agents as more likely to activate countermeasures when the infection level is high and, when the infection level is low, as less likely to activate them, or more likely to de-activate them. We combine the epidemic propagation of the worm with a game-theoretic description of the user behavior into a nonlinear dynamical system. Similar to other papers on security investments [4], [5], we do not collect observations to estimate the precise shape of real-user response functions. In this sense, clearly, our conclusions are of a qualitative nature. If

Manuscript received September 28, 2010; revised July 10, 2011 and January 24, 2012; accepted July 04, 2012. Date of publication August 02, 2012; date of current version January 19, 2013. The third author's research was supported in part by the U.S. AFOSR MURI Grants FA9550-10-1-0573 and FA9550-09-1-0538, and by the National Science Foundation Grant CNS-1035655. Recommended by Associate Editor Hideaki Ishii.

G. Theodorakopoulos is with the School of Computer Science and Informatics, Cardiff University, Cardiff CF24 3AA, U.K. (e-mail: g.theodorakopoulos@cs.cardiff.ac.uk).

J.-Y. Le Boudec is with the School of Computer and Communication Sciences, Ecole Polytechnique Federale de Lausanne (EPFL), Lausanne CH-1015, Switzerland (e-mail: jean-yves.leboudec@epfl.ch).

J. S. Baras is with the Department of Electrical and Computer Engineering, University of Maryland, College Park, MD 20742 USA (e-mail: baras@umd.edu).

Digital Object Identifier 10.1109/TAC.2012.2209949

practitioners wish to derive quantitative results from our model, in Section II-D-7, we provide guidance on how to find approximate estimates of the parameter values.

Our main scenario comprises the following three elements: 1) a homogeneous mixing network (random pairwise contacts); 2) homogeneous and best-response (discontinuous) user behavior, with a single threshold that determines whether they activate or deactivate their protection; and 3) users learn what the infection level is at a rate called the *learning rate*. We also test alternative scenarios for user behavior (smooth best-response; two user classes instead of homogeneous user behavior) and for network mixing (simulations on human mobility traces).

Our contributions are as follows.

- The network reaches an endemic equilibrium,¹ that is, an equilibrium where the infection persists. Our main conclusion is the following counterintuitive result: **the higher the learning rate, the higher the infection level at the equilibrium.**
- We confirm our main conclusion in three cases: 1) in our main scenario (Section III); 2) in a scenario with smooth best-response user behavior (Section IV-A); and 3) in a scenario with best-response behavior, with either one or two classes of users simulated on real mobility traces (human pairwise contacts) (Section IV-B).
- In the best-response and smooth best-response user behavior scenarios, we identify the equilibrium points (Sections III-C and IV-A-1), we show under which conditions they are locally stable (Sections III-D and IV-A-2), we rule out the possibility of closed trajectories, and we characterize the domains of attraction for each equilibrium point (Sections III-E and IV-A-3).

II. SIPS MODEL FOR EPIDEMIC PROPAGATION AND USER BEHAVIOR

A. Epidemic Propagation

There are N users in the network. Each user can be in one of three states:

- *Susceptible*, denoted by S . The user does not have any countermeasures in place and is not infected.
- *Infected*, denoted by I . The user is infected by the virus and will spread it to any susceptible user he makes contact with.
- *Protected*, denoted by P . The user has countermeasures in place. As long as the user is in this state, he is immune to the virus.

The number and fraction of users in each state are denoted, respectively, by N_S, N_I, N_P , and S, I, P . It follows that $N_S + N_I + N_P = N$ and $S + I + P = 1$. The state of the network is $x = (S, I, P)$, and the set of possible states is $X = (1/N)\mathbb{N}^3 \ni \{N_S/N, N_I/N, N_P/N\}$.

The evolution of the network state x is described as a Continuous Time Markov Process, as follows. A Poisson process of rate $\beta + \gamma + \delta$ is associated with each user. At an epoch of the Poisson process of user i —say at time t —one of three events occurs as follows.

¹The equilibrium notion that we use is in the long-term sense (i.e., as $t \rightarrow \infty$).

TABLE I
TABLE OF IMPORTANT NOTATIONS

δ	rate of disinfection (average infection duration is $\frac{1}{\delta}$)
β	contact rate of network users
γ	rate of informing users about the infection level
I^*	infection threshold, in the best-response model, above which users switch to protected

TABLE II
EVENTS THAT CAN HAPPEN IN THE NETWORK, THEIR RESULTS, AND HOW EACH EVENT CHANGES THE NETWORK STATE $x = (S, I, P)$

Event :	Meeting between an S user and an I user
Result :	S user becomes infected
Change:	$\frac{1}{N}(-1, +1, 0)$
Event :	Update received by S user
Result :	S user becomes protected with probability $p_{SP}(x)$
Change:	$\frac{1}{N}(-p_{SP}(x), 0, +p_{SP}(x))$
Event :	Update received by P user
Result :	P user becomes susceptible with probability $p_{PS}(x)$
Change:	$\frac{1}{N}(+p_{PS}(x), 0, -p_{PS}(x))$
Event :	Disinfection of I user
Result :	I user becomes protected
Change:	$\frac{1}{N}(0, -1, +1)$

- With probability $\beta/(\beta + \gamma + \delta)$, user i has a *meeting* with another user, chosen uniformly at random. If the meeting is between a susceptible and an infected user, the susceptible user becomes infected. Otherwise, nothing happens.
- With probability $\gamma/(\beta + \gamma + \delta)$, user i receives an *update* about the network state x , and he has the opportunity to revise his current strategy if his state is S or P . If i 's state is S , he switches to P with probability $p_{SP}(x)$. If i 's state is P , he switches to S with probability $p_{PS}(x)$. If i is infected, nothing happens.
- With probability $\delta/(\beta + \gamma + \delta)$, user i has a *disinfection* opportunity. That is, if i is infected, he becomes disinfected, and we assume he becomes protected. If i is not infected, nothing happens.

Table II summarizes the possible events and how each event changes the network state.

B. User Behavior—Best-Response Dynamics

As can be seen from the epidemic propagation model, the only point at which the users can make a choice is at an update event. There is a cost c_I associated with becoming infected, and a cost c_P associated with becoming protected. It holds that $c_I > c_P > 0$, because if $c_I < c_P$ there is no incentive for a susceptible user to become protected. There is no cost for being susceptible. Note that these costs need not be the actual costs; user decisions are influenced by the costs as *perceived* by the users.

The choice between susceptible and protected depends on which state minimizes the user's expected cost. The cost of protection is always c_P . If the user chooses to remain susceptible when the network state is $x = (S, I, P)$, the user's expected cost is Ic_I , as 1) the cost of infection is c_I ; 2) there is a fraction

I of infected users; and 3) the homogeneous mixing assumption implies that the meeting between any given pair of users is equiprobable to any other pair. That is, the probability that the next meeting of the user is with an infected user is equal to I . See also the last paragraph of this section, the discussion in Section II-D and, in particular, II-D-6 and II-D-4.

Therefore, the user's decision would be S if $Ic_I < c_P$, and P if $Ic_I > c_P$. If $Ic_I = c_P$, then both choices are optimal, and any randomization between them is also optimal. So, when $Ic_I = c_P$, the functions $p_{SP}(x)$ and $p_{PS}(x)$ are multivalued. Note that the user's decision depends only on the value of I , rather than the whole state x of the network, so we will slightly abuse the notation and use $p_{SP}(I)$ and $p_{PS}(I)$ in what follows. We denote by I^* the value of I that equalizes the (perceived expected) cost of infection with the cost of protection

$$I^* \triangleq \frac{c_P}{c_I}.$$

The two functions become

$$p_{SP}(I) = \begin{cases} 0, & I < I^* \\ [0, 1], & I = I^* \\ 1, & I > I^* \end{cases} \quad p_{PS}(I) = \begin{cases} 1, & I < I^* \\ [0, 1], & I = I^* \\ 0, & I > I^* \end{cases}$$

Note that it is possible to generalize the users' perceived expected cost of infection at infection level I . Instead of Ic_I , the perceived expected cost can be any increasing function $c_I(I)$ of the infection level I . The only difference in this case is that I^* is such that $c_I(I^*) = c_P$. The rest of the analysis proceeds with no change.

C. Putting Everything Together

We consider the large population scenario (i.e., the limit as $N \rightarrow \infty$). Gast and Gaujal [13] show that when $N \rightarrow \infty$, the trajectory of the stochastic system converges in probability to a solution of a differential inclusion. If the solution is unique, the stochastic system converges to it. If there are multiple solutions for the same initial conditions, then the stochastic system can converge to any of them.

In our case, the Continuous Time Markov Process described previously converges to a solution of the following system of differential inclusions (for brevity, the dependence of S, I, P on t is not explicitly shown)

$$\begin{aligned} \frac{d}{dt}S &\in -\beta SI - \gamma S p_{SP}(x) + \gamma P p_{PS}(x) \\ \frac{d}{dt}I &= \beta SI - \delta I \\ \frac{d}{dt}P &\in \delta I + \gamma S p_{SP}(x) - \gamma P p_{PS}(x) \end{aligned}$$

Since $S + I + P = 1$, we can eliminate one of the three state variables. We eliminate P , and the system becomes

$$\frac{d}{dt}S \in -\beta SI - \gamma S p_{SP}(x) + \gamma(1 - S - I)p_{PS}(x) \quad (1a)$$

$$\frac{d}{dt}I = \beta SI - \delta I, \quad (1b)$$

together with $P = 1 - S - I$. The state space $D := \{(S, I) | 0 \leq S \leq 1, 0 \leq I \leq 1, S + I \leq 1\}$ is bounded. This system² is 2-D and autonomous.³

We denote the right-hand side of system (1) by $F(x)$, and we slightly abuse the notation for x to be $x = (S, I)$, $x \in D$. So system (1) becomes

$$\frac{d}{dt}x \in F(x).$$

D. Discussion of Model Assumptions

1) *Worm Lifecycle* $S \rightarrow I \rightarrow P$: The $S \rightarrow I \rightarrow P$ is a normal lifecycle of a worm. Susceptible users become infected when compromised by the worm. Infected users stop being infected after some time; they then become protected as a result of the action that they take to fight the infection.

Many other potential life cycles are conceivable [1]. $S \rightarrow I \rightarrow S$ is another model that has been considered [15]. It applies, for instance, when the users reboot their machines to delete the worm. Not all worms are deleted in this way (e.g., they might be in the Master Boot Record of the computer, and so they might reload themselves upon reboot). But if the worm is deleted, then the machine simply re-enters the susceptible state upon reboot. The SEIR model [14] can be used for modeling worms with a dormant phase (E for Exposed) before becoming actively infectious.

2) *Adoption/Removal of Countermeasures and Their Timing*: In our model, the countermeasures that users can activate or deactivate include the following:

- Using antivirus software or special programs to clean infected computers.
- Setting up firewall filters to block the virus or worm traffic.
- Disconnecting networks or computers when no effective methods are available.

Other countermeasures exist. A popular one, which has received a lot of attention in the research community [16], is to patch or to upgrade susceptible devices in order to make them immune to the virus or worm. Of course, note that if all devices are eventually patched, then there can be no long-term infection.

Adoption and removal of measures is a binary (ON/OFF) choice in our case; users can choose between being either completely susceptible or completely protected. The antivirus software is either active or not; the firewall filter either exists and blocks the worm packets or not. An alternative would be to model a gradual adoption of a security measure. For instance, we could introduce a parameter for the scanning rate of a firewall and allow for the gradual increase of this rate. This would gradually increase the cost of protection and gradually decrease the probability of infection.

We only allow users to adopt/remove the countermeasure at the update epochs. In principle, users are free to do both at any time they want. But in between update epochs, they do not have

²Note that for $\gamma = 0$, the model is identical to the standard SIR epidemic model (R stands for Recovered). For $p_{SP}(x) = 0, \forall x$, and $p_{PS}(x) = 1, \forall x$, it is identical to the SIRS model [14]. Therefore, our SIPS model generalizes SIR and SIRS.

³An autonomous system is a system of ordinary differential equations whose parameters do not explicitly depend on the independent variable (time t in our case).

any new information. So we assume that if they want to act, they will act immediately when they receive the latest update.

3) *Homogeneous Mixing and Network Topology*: Our homogeneous mixing assumption is suitable for worms that perform random uniform scans of the IP address space to find new victims (Code Red (version 2) [17], Slammer [18]). It does not apply to worms whose propagation is topology dependent [19] (e.g., by e-mails to the address-book contacts of the infected user) or mobile phone worms that propagate by proximity (Bluetooth worms). In Section IV-B, we perform simulations on human contact traces, to study different propagation dynamics. We find that our main conclusion persists.

4) *Best-Response Dynamics and User Rationality*: The concept of best-response is a fundamental one in game theory, and it is also one of the most popular dynamics in evolutionary game theory. Hence, it is our main scenario in this paper. Best-response dynamics lead to differential inclusions, which unfortunately increase the complexity of the analysis [20]–[23].

Differential inclusions appear because of the multiplicity of optimal responses. In this paper, the multiplicity appears when $I = I^*$. At that value of I , both protection and no protection are optimal as well as all randomizations among them. It turns out that under certain conditions (3), the equilibrium point of the system is exactly on the line $I = I^*$; second, on a more technical note, the system trajectories that pass from the point $S = \delta/\beta$, $I = I^*$ cannot be uniquely continued. So if we want to study best-response dynamics, we cannot afford to ignore differential inclusions, as the properties of the system under study are intricately affected.

Of course, users might not be perfectly rational. Even in the case of best-response dynamics, we consider the users to be myopic: they do not take into account the long-term effects of their actions, but they rather behave greedily. But the lack of user rationality may go even further. Their perception of the cost may not be clearcut (e.g., they are not sure about the exact values of c_I and c_P). Alternatively, they may take the network state report to be not completely accurate. To account for such cases, we study smooth best-response dynamics (Section IV-A). Briefly, we assume that $p_{SP}(\cdot)$ and $p_{PS}(\cdot)$ can be arbitrary functions of I , as long as $p_{SP}(\cdot)$ is nondecreasing with I and $p_{PS}(\cdot)$ is non-increasing with I .

5) *Uniform User Behavior*: In the model as described, all users behave the same way. To account for users with different response functions, in Section IV-B, we simulate two user classes, each with a different best-response function. An extension to more than two classes is straightforward.

6) *Costs of Infection and Protection*: The cost of infection and protection have been assumed to be constant, but this need not be so. For example, it is reasonable to make the infection cost depend on δ . A larger δ means a shorter infection duration, so the infection cost should presumably decrease with δ . The influence of the contact rate β is less clear. A higher β increases the probability of getting infected within the next dt time units: this probability is $\beta I dt$. On the one hand, the objective cost of infection itself does not change (removing the virus from the computer, lost hours of productivity, lost/compromised data, psychological effect on user, etc.). On the other hand, as we already said, what matters is the cost as *perceived* by the

users. Perhaps a user feels more threatened due to the high contact rate β .

A further issue is the immediacy of the protection cost versus the vagueness of the infection cost. The infection cost is a potential cost that will be paid in the *future*, if the user becomes infected. The protection cost is an immediate loss. Humans heavily discount future losses and rewards.

We argue that none of the aforementioned considerations change our conclusions, unless some cost depends on the update rate γ . Making the infection cost (and even the protection cost) a function of β and δ merely turns I^* into $I^*(\beta, \delta)$; as β and δ are constants, $I^*(\beta, \delta)$ also remains constant with respect to time, so our conclusions do not change. If, for some reason, I^* depends on γ , and it also happens that the equilibrium infection is equal to I^* (which happens in the case of the equilibrium point X_2 , Section III), then our conclusions on the dependency of the equilibrium infection level on γ would be different.

The qualitatively different nature of the infection and protection costs (future versus immediate) can be incorporated in our model by appropriately discounting the value of c_I . The discounting can even depend on β , as β influences the immediacy of the infection.

7) *Parameter Identification*: There are five parameters in our model, $\beta, \delta, \gamma, c_I, c_P$ and two smooth best-response behavior functions $p_{PS}(\cdot)$ and $p_{SP}(\cdot)$. Our main conclusion is qualitative, so it is not influenced by the exact values of these parameters. Any incidental quantitative results of our work, such as the infection level at the equilibrium, are indeed sensitive to the parameter values. Even though it is not directly relevant to our main contributions, we provide some insight on how an interested reader might go about measuring these parameters.

To measure β , we have to estimate the contact rate per infected user for a real worm, that is, the number of infection attempts that the worm initiates each time unit. This can be done by observing the behavior of the worm in the wild, or in a controlled environment, or by analyzing the source code of the worm if it is available. All three methods are used by security researchers.

To measure δ , we have to estimate the time that a device remains infected. The duration of the infection may be due to any number of factors, such as the user not noticing the worm, the user being indifferent, or the worm being inherently difficult to clean because its removal might cause data loss, for example. Measurements for the infection duration can typically be collected by system administrators, or any other person who is called upon to clean the infected device. Although it is easy to establish the end time of the infection at a device, finding the start time might require computer forensics techniques.

The value of γ is likely easier to determine, because γ is the update rate and updates are sent to all of the users in the network.

An approximate estimate for the infection and protection costs can be obtained, for instance, through psychological/sociological tests and interviews, as well as purely technical and economic evaluations. It is sufficient for our best-response model that there be a threshold I^* for I , above which the users switch from S to P and below which they switch from P to S . Similar considerations apply for the derivation of the smooth best-response functions $p_{SP}(I)$ and $p_{PS}(I)$. Our

conclusions depend only on $p_{SP}(I)$ increasing with I and on $p_{PS}(I)$ decreasing with I . We believe that is a very mild assumption on user behavior, but practitioners who wish to use our conclusions should verify it.

The non-necessity of exact estimation of the various parameters/functions adds to the importance of our results, as a practitioner does not need to estimate the exact values of these parameters in order to ascertain whether our conclusions apply. All that is necessary is that our qualitative assumptions be valid (e.g., assumptions on worm state evolution $S \rightarrow I \rightarrow P$, ability of users/devices to activate and deactivate protection).

III. RESULTS FOR BEST-RESPONSE DYNAMICS

In this section, we study the behavior of differential inclusion (1) for various values of parameters β , γ , δ , and I^* . The main finding is that the equilibrium value of the infection increases with γ , but it cannot increase above I^* . The technical results are summarized as follows (see also Fig. 1).

- Solutions exist.
- All solutions can be uniquely continued, except those that start at $(S, I) = (\delta/\beta, I^*)$. These latter solutions all start at the same point and then diverge, but none of them can ever approach that point again. So if we ignore the initial point of those solutions, all solutions can be uniquely continued.
- Point $X_0 = (1, 0)$ is always an equilibrium point. If $\delta < \beta$, one more equilibrium point exists: X_1 if $I^* > (1 - (\delta/\beta))/(1 + (\delta/\gamma))$, or X_2 otherwise ($X_1 = (\delta/\beta, (1 - (\delta/\beta))/(1 + (\delta/\gamma)))$ and $X_2 = (\delta/\beta, I^*)$).
- If $\delta \geq \beta$, X_0 is asymptotically stable.⁴ If $\delta < \beta$, X_0 is a saddle point. If X_1 exists, it is asymptotically stable. Finally, if X_2 exists, it is asymptotically stable.
- There are no solutions that are closed trajectories. All system trajectories converge to one of the equilibrium points. When there is more than one equilibrium point (X_0 and one of X_1 or X_2), a trajectory converges to $X_0 = (1, 0)$ if and only if it starts on line $I = 0$; all other trajectories converge to the other point.

A. Existence of Solutions

We show that the differential inclusion

$$\frac{d}{dt}x \in F(x), x \in D \quad (2)$$

has solutions.

We define a partition of the state space D into three domains $D^- = D \cap \{(S, I), I < I^*\}$, $D^+ = D \cap \{(S, I), I > I^*\}$, and $L = D \cap \{(S, I) : I = I^*\}$. Domain L will also be referred to as the *discontinuity line*.

A solution for this differential inclusion is an absolutely continuous vector function $x(t)$ defined on an interval J for which $dx(t)/dt \in F(x(t))$ almost everywhere on J [24].

Theorem 1: Solutions of (2) exist.

Proof: See [25]. ■

⁴A point x_0 is asymptotically stable, if for every $\epsilon > 0$ there exists a δ such that if $\|x(0) - x_0\| < \delta$, then $\|x(t) - x_0\| < \epsilon$ for every $t \geq 0$, and $\lim_{t \rightarrow \infty} x(t) = x_0$.

B. Uniqueness of Solutions

In general, because the right-hand side of (2) is multivalued, even though two solutions at time t_0 are both at the point x_0 , they may not coincide on an interval $t_0 \leq t \leq t_1$ for any $t_1 > t_0$. If any two solutions that coincide at t_0 also coincide until some $t_1 > t_0$, then we say that *right uniqueness holds* at (t_0, x_0) . Left uniqueness at (t_0, x_0) is defined similarly (with $t_1 < t_0$), and (right and left) uniqueness in a domain holds if it holds at each point of the domain.

Theorem 2: All solutions of (2) can be uniquely continued, except possibly those that start at $(S, I) = (\delta/\beta, I^*)$. The latter ones will stay at that point if $0 \in F(\delta/\beta, I^*)$ (i.e., if $(\delta/\beta, I^*)$ is an equilibrium point); otherwise, they can be continued in multiple ways.

Proof: See the Appendix. ■

C. Equilibrium Points

The equilibrium points are found by solving the inclusion $0 \in F(x)$ for x . If $\delta \geq \beta$, point $X_0 = (1, 0)$ is the only equilibrium point. If $\delta < \beta$, one more equilibrium point exists: X_1 if $I^* > ((1 - (\delta/\beta))/(1 + (\delta/\gamma)))$, or X_2 , otherwise ($X_1 = (\delta/\beta, (1 - (\delta/\beta))/(1 + (\delta/\gamma)))$ and $X_2 = (\delta/\beta, I^*)$).

1) *Equilibrium Points Above the Discontinuity Line:* There can be no equilibrium points in the domain D^+ . The system becomes

$$\begin{aligned} \frac{d}{dt}S &= -\beta SI - \gamma S \\ \frac{d}{dt}I &= \beta SI - \delta I. \end{aligned}$$

From the first equation, S has to be zero. But then, the second equation implies that I also has to be zero, which is not an admissible value for I as $I = 0$ cannot be above the discontinuity line.

2) *Equilibrium Points Below the Discontinuity Line:* There is either one or two equilibrium points in the domain D^- . The system becomes

$$\begin{aligned} \frac{d}{dt}S &= -\beta SI + \gamma(1 - S - I) \\ \frac{d}{dt}I &= \beta SI - \delta I. \end{aligned}$$

This system has the solutions

$$\begin{aligned} X_0 &= (S_0, I_0) = (1, 0) \\ X_1 &= (S_1, I_1) = \left(\frac{\delta}{\beta}, \frac{1 - \frac{\delta}{\beta}}{1 + \frac{\delta}{\gamma}} \right). \end{aligned}$$

The second solution X_1 is admissible if and only if $X_1 \in D^-$, i.e.,

$$\frac{\delta}{\beta} \leq 1, \quad \frac{1 - \frac{\delta}{\beta}}{1 + \frac{\delta}{\gamma}} < I^*, \quad \text{and} \quad \frac{\delta}{\beta} + \frac{1 - \frac{\delta}{\beta}}{1 + \frac{\delta}{\gamma}} \leq 1.$$

The third condition is always satisfied. Note also that if $\delta/\beta = 1$, then X_0 and X_1 coincide.

3) *Equilibrium Points on the Discontinuity Line:* There is, at most, one equilibrium point on the discontinuity line $I = I^*$. To

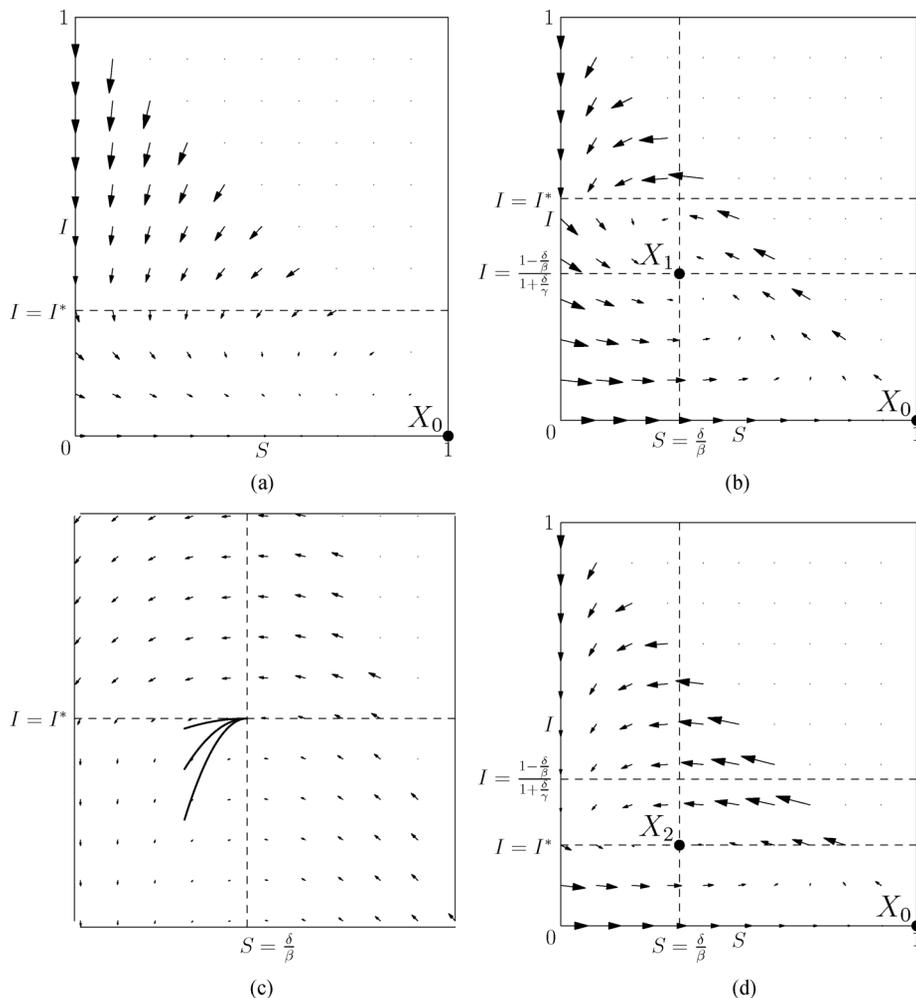


Fig. 1. Vector field of the system and the equilibrium points for all regions of the parameter space. At each point (S, I) , an arrow parallel to $(dS/dt, dI/dt)$ is plotted. In Fig. 1(b) and (d), point $X_0 = (1, 0)$ is also an equilibrium point but it is unstable. All trajectories converge to X_1 or X_2 , respectively, except those that start on the axis $I = 0$, which converge to X_0 . The indicative parameter values used are $\beta = 3.0$, $\delta = 4.0, 1.0, 1.0$, $\gamma = 1.2$, $I^* = 0.3, 0.8, 0.2$. (a) The case $\delta \geq \beta$. The only equilibrium point is $X_0 = (1, 0)$. It is stable and all trajectories converge to it. $\beta = 3.0$, $\delta = 4.0$, $\gamma = 1.2$, $I^* = 0.3$. (b) The case $\delta < \beta$ and $I^* > (1 - (\delta/\beta))/(1 + (\delta/\gamma))$. The point $X_1 = (\delta/\beta, (1 - (\delta/\beta))/(1 + (\delta/\gamma)))$ is a stable equilibrium point. $\beta = 3.0$, $\delta = 1.0$, $\gamma = 1.2$, $I^* = 0.8$. (c) Detail of 1(b) around point $(\delta/\beta, I^*)$. The continuation of a trajectory starting at $(\delta/\beta, I^*)$ is not unique. Three sample trajectories are plotted, corresponding to different elements of set $F(\delta/\beta, I^*)$. (d) The case $\delta < \beta$ and $I^* \leq (1 - (\delta/\beta))/(1 + (\delta/\gamma))$. The point $X_2 = (\delta/\beta, I^*)$ is a stable equilibrium point. $\beta = 3.0$, $\delta = 1.0$, $\gamma = 1.2$, $I^* = 0.2$.

find it, we solve the inclusion $0 \in F(S, I^*)$ for S . The system becomes

$$\begin{aligned} \frac{d}{dt}S &\in -\beta SI^* + [-\gamma S, \gamma(1 - S - I^*)] \\ \frac{d}{dt}I &= \beta SI^* - \delta I^*. \end{aligned}$$

Since $I^* > 0$, dI/dt is zero only when $S = \delta/\beta$. We then have to check if it is possible to make dS/dt equal to zero, that is, if $0 \in F(\delta/\beta, I^*)$. We find that it is possible when I^* is such that

$$I^* \leq \frac{1 - \frac{\delta}{\beta}}{1 + \frac{\delta}{\gamma}}. \quad (3)$$

In that case, the equilibrium point is

$$X_2 = (S_2, I_2) = \left(\frac{\delta}{\beta}, I^* \right).$$

The conditions for admissibility are $S_2 = \delta/\beta \leq 1$ and $S_2 + I_2 = (\delta/\beta) + I^* \leq 1$. The latter is true if (3) holds.

In general, there are many combinations of $p_{SP}(I^*)$ and $p_{PS}(I^*)$ that make dS/dt equal to zero, but there is always one with $p_{SP}(I^*) = 0$. In that case, $p_{PS}(I^*) = \delta I^*/\gamma(1 - (\delta/\beta) - I^*)$.

D. Local Asymptotic Stability

1) Stability of X_0 and X_1 :

Theorem 3: X_0 is asymptotically stable if and only if $\beta \leq \delta$; X_1 is asymptotically stable whenever it exists ($\beta > \delta$) and the trajectories spiral toward X_1 (at least locally). If $\beta = \delta$, X_0 and X_1 coincide, and the resulting point is asymptotically stable.

Proof: We evaluate the Jacobian of the system at X_0 and X_1 . See [25]. ■

Stability of X_2 :

Theorem 4: X_2 is asymptotically stable whenever it exists.

Proof: To show that the equilibrium point on the discontinuity line is asymptotically stable, we will use Theorem 5 below [24, §19, Theorem 3]. To use this theorem, we transform the system so that the line of discontinuity is the horizontal axis,

the equilibrium point is (0,0), and the trajectories have a clockwise direction for increasing t .

We set $x = \delta/\beta - S$ and $y = I - I^*$. Domains D, D^-, D^+ become $G = \{(x, y) | x \leq (\delta/\beta), y \geq -I^*, y - x \leq 1 - I^* - (\delta/\beta)\}$, $G^- = G \cap \{(x, y) | y < 0\}$, and $G^+ = G \cap \{(x, y) | y > 0\}$. Then, the system can be written as

$$\begin{aligned} \frac{dx}{dt} &= P^-(x, y) \\ &= -\beta xy - (\beta I^* + \gamma)x + (\gamma + \delta)y \\ &\quad - \gamma(1 - I^*) + \delta \left(I^* + \frac{\gamma}{\beta} \right) \end{aligned}$$

$$\frac{dy}{dt} = Q^-(x, y) = -\beta x(y + I^*)$$

for $(x, y) \in G^-$, and

$$\begin{aligned} \frac{dx}{dt} &= P^+(x, y) \\ &= -\beta xy - (\beta I^* + \gamma)x + \delta y + \delta \left(I^* + \frac{\gamma}{\beta} \right) \end{aligned}$$

$$\frac{dy}{dt} = Q^+(x, y) = -\beta x(y + I^*)$$

for $(x, y) \in G^+$.

The partial derivatives of P^\pm , that is, of P^+ and of P^- , are denoted by $P_x^\pm, P_{xx}^\pm, P_y^\pm$ etc., and similarly for Q^\pm . We define two quantities A^\pm in terms of functions P^\pm, Q^\pm and their derivatives at (0,0)

$$A^\pm \triangleq \left(\frac{P_x^\pm + Q_y^\pm}{P^\pm} - \frac{Q_{xx}^\pm}{2Q_x^\pm} \right).$$

Theorem 5: Let the conditions

$$Q^- = Q^+ = 0, P^- < 0, P^+ > 0, Q_x^- < 0, Q_x^+ < 0$$

be fulfilled at (0,0). Then, $A^+ - A^- < 0$ implies that the zero solution is asymptotically stable, whereas $A^+ - A^- > 0$ implies that the zero solution is unstable.

All of the conditions of Theorem 5 are satisfied in our case, together with $A^+ - A^- < 0$. The condition $P^- < 0$ is equivalent to (3) (i.e., the condition on I^* that causes the equilibrium point to be on the line of discontinuity). All of the other conditions are straightforward to verify. For example, to prove that $A^+ - A^- < 0$, we can quickly establish that $A^+ < 0$ and $A^- > 0$, again using (3). ■

E. Domains of Attraction

Consider an autonomous system on the plane, as ours is. If a half trajectory T^+ of such a system is bounded, then its ω -limit set⁵ $\Omega(T^+)$ contains either an equilibrium point or a closed trajectory [24, §13, Theor. 6].

The main result for our system is:

⁵The ω -limit set of a half trajectory $T^+(x = \phi(t), t_0 \leq t < \infty)$ is the set of all points q for which there exists a sequence t_1, t_2, \dots tending to ∞ such that $\phi(t_i) \rightarrow q$ as $i \rightarrow \infty$.

Theorem 6: For any half trajectory T^+ , its ω -limit set $\Omega(T)$ can only contain equilibrium points, that is, $X_0 = (1, 0)$, $X_1 = (S_1, I_1) = (\delta/\beta, (1 - (\delta/\beta))/(1 + (\delta/\gamma)))$, or $X_2 = (\delta/\beta, I^*)$.

Proof: If $\delta > \beta$, then only X_0 exists. There are no closed trajectories as $dI/dt < 0$, so all trajectories converge to X_0 . For the remainder of the proof, we assume $\beta \geq \delta$, so either X_1 or X_2 exists and is stable.

The following two functions are useful:⁶

$$E(S, I) \triangleq S - S_1 \ln(S) + I + \frac{\gamma}{\beta} \ln(I), (S, I) \in D^+$$

$$M(S, I) \triangleq S - \left(S_1 + \frac{\gamma}{\beta} \right) \ln \left(S + \frac{\gamma}{\beta} \right) + I - I_1 \ln(I), \\ (S, I) \in D^-.$$

It holds that $E(S, I)$ is constant on trajectories in the area D^+ , and $M(S, I)$ is decreasing along trajectories in the area D^- . Indeed, with some calculations, it can be shown that

$$\begin{aligned} \frac{d}{dt} E(S, I) &= \frac{\partial E}{\partial S} \frac{dS}{dt} + \frac{\partial E}{\partial I} \frac{dI}{dt} = 0 \\ \frac{d}{dt} M(S, I) &= \frac{\partial M}{\partial S} \frac{dS}{dt} + \frac{\partial M}{\partial I} \frac{dI}{dt} = -\frac{(\beta S - \delta)^2}{\beta S + \gamma} \frac{1 + \frac{\gamma}{\beta}}{1 + \frac{\delta}{\gamma}} \leq 0. \end{aligned}$$

Under the assumption $\beta \geq \delta$, we prove:

Lemma 1: A trajectory converges to $X_0 = (1, 0)$ if and only if it starts on line $I = 0$.

Proof: See [25]. ■

From now on, we limit our attention to trajectories that have no common points with the line $I = 0$.

Assume that there exists a half trajectory T^+ whose limit set $\Omega(T)$ contains a closed trajectory Γ . By successively eliminating properties of Γ , we prove that Γ cannot exist.

Lemma 2: Point $(\delta/\beta, I^*)$ cannot be on a closed trajectory.

Proof: See [25]. ■

As $(S, I) = (\delta/\beta, I^*)$ cannot be on Γ , there holds right uniqueness on Γ . Hence, $\Omega(\Gamma) = \Gamma$.

We now prove that Γ cannot have more than two or fewer than two intersection points with L .

Lemma 3: A closed trajectory Γ that does not pass through the point $(\delta/\beta, I^*)$ cannot have either more than two or fewer than two intersection points with the discontinuity line L . If it has two intersection points, they cannot be on the same side of $(\delta/\beta, I^*)$.

Proof: See [25]. ■

Lemma 4: A closed trajectory Γ cannot intersect the discontinuity line L on exactly two points that are on opposite sides of $(\delta/\beta, I^*)$.

Proof: See the Appendix. ■

From the previous lemmata, we conclude that there can be no closed trajectory Γ . Therefore, all trajectories have to converge to equilibrium points. ■

⁶Functions of this form are Lyapunov functions for the SIRS epidemic model [26]. Although they are not Lyapunov functions in our case, we found them using the technique described in [26]: looking for functions of the form $f_1(S) + f_2(I)$ whose time derivatives do not change sign.

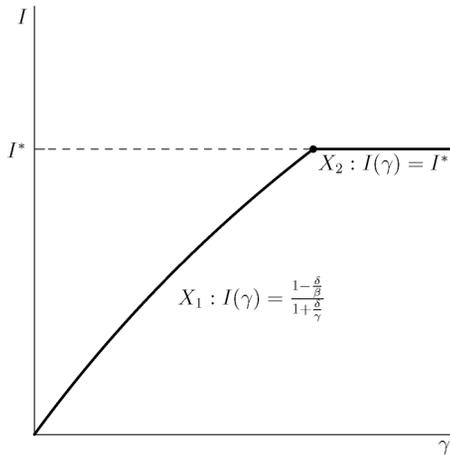


Fig. 2. Total fraction of infected as a function of γ .

F. Discussion of Results

Fig. 2 shows that the total fraction $(1 - \delta/\beta)/(1 + \delta/\gamma)$ of infected at the system equilibrium increases with the update rate γ , until $(1 - \delta/\beta)/(1 + \delta/\gamma)$ becomes equal to the threshold I^* . This increase is due to the combination of two factors. First, when $(1 - \delta/\beta)/(1 + \delta/\gamma) < I^*$, the trajectories will eventually be completely contained in domain D^- (below I^*). In this domain, each time a protected is informed about the fraction of infected, he will choose to become susceptible, thus fueling the infection. Second, no susceptible will choose to become protected. The larger the value of γ , the shorter time a user will spend being protected; thus, the smaller the fraction of protected. However, a smaller fraction of protected implies a larger fraction of infected, as the fraction of susceptible at equilibrium is necessarily δ/β (i.e., it is independent of γ).

When $(1 - \delta/\beta)/(1 + \delta/\gamma) > I^*$, the equilibrium fraction of infected is limited to I^* : further increases of γ have no effect. The explanation is that as soon as the instantaneous value of I exceeds I^* , susceptible users switch to protected, and protected users stay protected, thus bringing the infection level below I^* , into the domain D^- . However, there is no equilibrium point for the system in D^- , so the only possible equilibrium value of I is I^* . For $I = I^*$, there are, in general, many combinations of $p_{SP}(I^*)$ and $p_{PS}(I^*)$ that lead to an equilibrium, including one with $p_{SP}(I^*) = 0$ and $p_{PS}(I^*) > 0$. That combination means that no susceptible users become protected, but some protected become susceptible. Other combinations with $p_{SP}(I^*) > 0$ and $p_{PS}(I^*) > 0$ would be harder to justify, as they imply that at the same value of I^* , users would switch from susceptible to protected and back.

IV. ROBUSTNESS OF RESULTS WITH RESPECT TO MODEL ASSUMPTIONS

In our main scenario (Section II), all users follow best-response (multivalued and discontinuous) dynamics, they contact each other uniformly at random, and they all have the same behavior function.

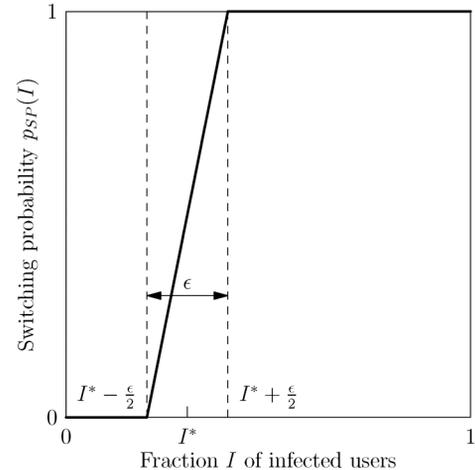


Fig. 3. User response function $p_{SP}(I)$ used in the simulations: the probability that a susceptible user switches to being protected, upon learning the fraction I of infected users in the network.

In the current section, we show that our conclusions do not change if we vary these assumptions. In all cases, we show that the equilibrium infection level increases with the update rate γ . In particular, we prove that this holds even if users follow any arbitrary continuous and single-valued response function, as long as $p_{SP}(I)$ increases with I and $p_{PS}(I)$ decreases with I ; we call this the *smooth best-response* case. We also show that there are no periodic solutions, so all trajectories converge to equilibrium points. Further, we perform simulations on human contact traces, as opposed to assuming uniform contact patterns. We confirm again that the equilibrium infection increases with the update rate, whether the users have a common behavior function or they are split into two types (each with a different best-response threshold).

The more technical results in the smooth best-response case (equilibrium points, conditions for asymptotic stability, absence of closed trajectories) parallel the ones in the best-response case.

A. Smooth Best-Response

The user behavior functions $p_{SP}(I)$ and $p_{PS}(I)$ are continuously differentiable, and we require that $dp_{SP}(I)/dI$ and $dp_{PS}(I)/dI < 0$. Other than that, the two functions are arbitrary. The stochastic system converges to a system of ordinary differential equations [27], [28].

As mentioned before, it is still the case that the equilibrium infection level increases with the update rate γ . Further technical results follow.

- Two equilibrium points may exist X_0 and X_1 . X_0 exists always, X_1 when $\delta/\beta \leq p_{PS}(0)/(p_{SP}(0) + p_{PS}(0))$ [Condition (5)].
- X_0 is asymptotically stable when X_1 does not exist. X_1 is asymptotically stable whenever it exists.
- The trajectories of (4) must converge either to X_0 or to X_1 . When (5) does not hold, all trajectories converge to X_0 . When (5) holds, trajectories starting at points with $I = 0$ approach X_0 along line $I = 0$, whereas all other trajectories converge to X_1 .

1) *Equilibrium Points*: The equilibrium points of the system are found by solving the equation $F(x) = 0$ for x :

$$0 = \frac{d}{dt}S = -\beta SI - \gamma S p_{SP}(I) + \gamma(1 - S - I)p_{PS}(I) \quad (4a)$$

$$0 = \frac{d}{dt}I = \beta SI - \delta I. \quad (4b)$$

From (4b), either $I = 0$ or $S = \delta/\beta$.

- Equilibrium point X_0 .

Substituting $I = 0$ into (4a), we find $X_0 = (S_0, I_0) = (p_{PS}(0)/(p_{SP}(0) + p_{PS}(0)), 0)$. These values of (S_0, I_0) are always admissible as they are always non-negative and, at most, equal to 1.

Recalling the meaning of $p_{PS}(0)$ and $p_{SP}(0)$, we can reasonably expect that $p_{PS}(0) = 1$ and $p_{SP}(0) = 0$. Protected users have no reason to remain protected, and susceptible users have no reason to become protected, when there is no infection in the network. In this case, X_0 is $(1, 0)$.

- Equilibrium point X_1

Substituting $S = \delta/\beta$ into (4a), it follows that I has to satisfy $g(I) = 0$, where

$$g(I) \triangleq -\delta I - \frac{\gamma\delta}{\beta} p_{SP}(I) + \gamma \left(1 - \frac{\delta}{\beta} - I\right) p_{PS}(I).$$

To solve $g(I) = 0$ for I , we need to know the two response functions $p_{SP}(I)$ and $p_{PS}(I)$. But even without knowing them, we can still prove that $g(I) = 0$ has a unique solution for $I \in [0, 1 - (\delta/\beta)]$ under the condition

$$\frac{\delta}{\beta} \leq \frac{p_{PS}(0)}{p_{SP}(0) + p_{PS}(0)}. \quad (5)$$

The proof proceeds in three straightforward steps; we omit the details. The first step is to show that $dg(I)/dI < 0$, so $g(I)$ strictly and monotonically decreases in the interval $[0, 1 - (\delta/\beta)]$. The second step is to show that $g(0) \geq 0$, which leads to (5), and then to show that $g(1 - (\delta/\beta)) < 0$. The final step is to use the Intermediate Value Theorem to conclude that there is exactly one solution of $g(I) = 0$ in $[0, 1 - (\delta/\beta)]$.

Denoting by I_1 the solution of $g(I) = 0$, we conclude that $X_1 = (S_1, I_1) = (\delta/\beta, I_1)$ is uniquely determined under (5). The values S_1, I_1 are admissible as they are both between 0 and 1, and their sum is, at most, equal to 1. Note that if (5) does not hold, then both $g(0) < 0$ and $g(1 - (\delta/\beta)) < 0$, so the monotonicity of g in $[0, 1 - (\delta/\beta)]$ implies that X_1 does not exist. Consequently, (5) is both necessary and sufficient for the existence of X_1 .

- 2) *Asymptotic Stability*:

Theorem 7: X_0 is asymptotically stable when X_1 does not exist. X_1 is asymptotically stable whenever it exists.

Proof: We evaluate the Jacobian of the system at X_0 and X_1 . See [25]. ■

- 3) *Domains of Attraction*:

Theorem 8: The trajectories of (4) must converge either to X_0 or to X_1 . When (5) does not hold, all trajectories converge to

X_0 . When (5) holds, trajectories starting on line $I = 0$ approach X_0 along $I = 0$, whereas all other trajectories converge to X_1 .

Proof: Since the system is 2-D and F is continuously differentiable, we can use Dulac's criterion [29] to show that the system can have no periodic trajectory.

Theorem 9 (Dulac's Criterion): Let A be a simply connected domain. If there exists a continuously differentiable function $h : A \rightarrow \mathbb{R}$ such that $\nabla \cdot (hF)$ is continuous and non-zero on A , then no periodic trajectory can lie entirely in A .

In our case, domain A is the state space excluding line $I = 0$. Note that there can be no periodic trajectory that passes from a point with $I = 0$. We select as function h , the function $h(S, I) = 1/I$. We compute $\nabla \cdot (hF)$ to be

$$\nabla \cdot (hF) = -\beta - \gamma \frac{p_{SP}(I)}{I} - \gamma \frac{p_{PS}(I)}{I} < 0, \quad \forall (S, I) \in A$$

which is continuous and non-zero in A . Then, from Dulac's criterion, no periodic trajectory lies entirely in A and, consequently, the system has no periodic trajectory at all. From the Poincaré–Bendixson theorem, the system can only converge to a periodic trajectory or an equilibrium point; so, we can conclude that every trajectory must converge to an equilibrium point, that is, either to X_0 or to X_1 .

More precisely, when (5) does not hold, only X_0 exists so all trajectories converge to X_0 . When (5) holds, both X_0 and X_1 exist, and X_0 is a saddle point. Trajectories starting on line $I = 0$ approach X_0 along $I = 0$, whereas all other trajectories converge to X_1 . Indeed, if $I(0) > 0$, then the corresponding trajectory will have $I(t) > 0, \forall t > 0$. The reason is that if $I(t_0) = 0$ for some finite $t_0 > 0$, then the uniqueness of solutions would be violated at $(S(t_0), I(t_0))$, because $(S(t_0), I(t_0))$ would be a common point with the trajectories that approach X_0 along the line $I = 0$. If $t_0 = \infty$ (i.e., the trajectory with $I(0) > 0$ converges asymptotically to X_0 while $I(t)$ remains strictly positive), then we reach a contradiction as dI/dt will become positive at points close enough to X_0 . (See (5) and (4b).) ■

4) *Equilibrium Infection Increases With γ* : The no-infection equilibrium point $X_0 = (1, 0)$ is unaffected by γ . We show now that at $X_1 = (\delta/\beta, I_1)$, the equilibrium level of the infected increases with γ .

Theorem 10: The infection I_1 at $X_1 = (\delta/\beta, I_1)$ increases with γ .

Proof: The derivative $dI_1/d\gamma$ is always positive: I_1 satisfies $g(I_1) = 0$, i.e.,

$$-\delta I_1 - \frac{\gamma\delta}{\beta} p_{SP}(I_1) + \gamma \left(1 - \frac{\delta}{\beta} - I_1\right) p_{PS}(I_1) = 0. \quad (6)$$

Differentiating (6) with respect to γ , we have

$$\frac{dI_1}{d\gamma} G(I_1) = H(I_1)$$

where

$$G(I_1) \triangleq -\delta - \frac{\gamma\delta}{\beta} \frac{dp_{SP}(I_1)}{dI_1} - \gamma p_{PS}(I_1) + \gamma \left(1 - \frac{\delta}{\beta} - I_1\right) \frac{dp_{PS}(I_1)}{dI_1}$$

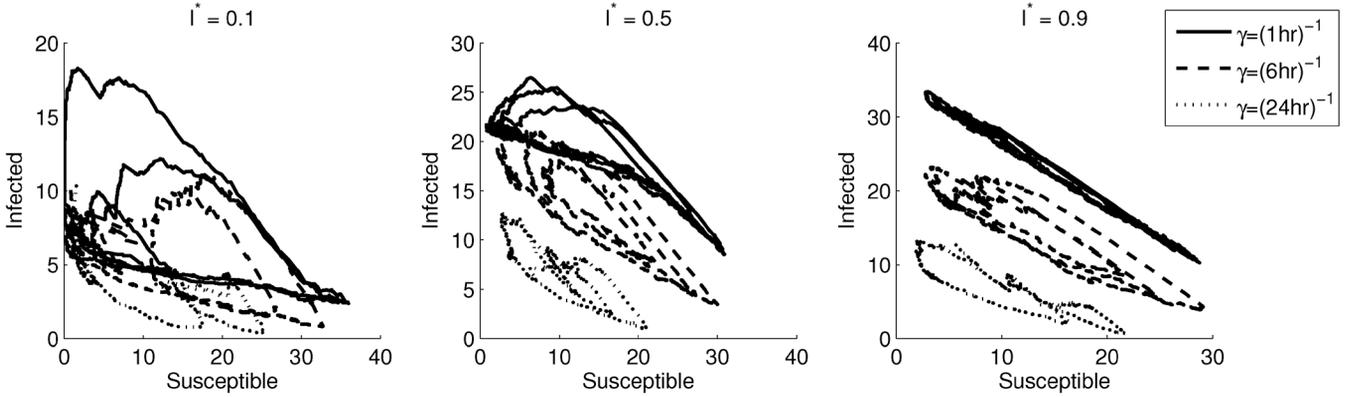


Fig. 4. Single-user behavior class. The trajectory of the system (average of 30 simulations) on the SI plane, when $\delta = (6 \text{ h})^{-1}$ and γ takes the values $(1 \text{ h})^{-1}$, $(6 \text{ h})^{-1}$, and $(24 \text{ h})^{-1}$. The thresholds are $I^* = 0.1, 0.5, 0.9$. The network experiences higher numbers of infected devices for higher values of γ . I^* limits the infection when $I^* = 0.1, 0.5$.

and

$$H(I_1) \triangleq \frac{\delta}{\beta} p_{SP}(I_1) - \left(1 - \frac{\delta}{\beta} - I_1\right) p_{PS}(I_1).$$

But $G(I_1) < 0$ and $H(I_1) < 0$ for all values of I_1 . Therefore, $dI_1/d\gamma$ is positive.

The negativity of $G(I_1)$ is deduced from $dp_{SP}(I_1)/dI_1 > 0$ and $dp_{PS}(I_1)/dI_1 < 0$. The negativity of $H(I_1)$ is deduced from (6), as (6) implies that $(\delta/\beta)p_{SP}(I_1) - (1 - (\delta/\beta) - I_1)p_{PS}(I_1) = -(\delta/\gamma)I_1 < 0$.

Dividing (6) by γ and taking the limit $\gamma \rightarrow \infty$, the limiting value of I_1 is the solution to

$$\frac{\delta}{\beta} p_{SP}(I_1) = \left(1 - \frac{\delta}{\beta} - I_1\right) p_{PS}(I_1).$$

This limiting value is admissible, as it is also a solution of $g(I) = 0$; hence, it lies in the interval $[0, 1 - (\delta/\beta)]$. ■

B. Propagation on Human Contact Traces

In this section, we use human contact traces to simulate the propagation, instead of assuming uniform contact patterns; the objective is to test the robustness of our conclusions with respect to the contact pattern. Moreover, instead of only having a common response function for all users, we now include the case where users are split into two classes, each with a different best-response behavior function. Note that we choose to have two classes to keep the presentation simple, but we believe that our results carry over to multiple user classes. Using enough user classes, it is possible to approximate any arbitrary continuous distribution of user behaviors.

The traces used are Bluetooth contacts among 41 devices given to participants in a conference [30]. These traces were collected over a period of approximately 72 h.

For the single-user class case, a piecewise-continuous response function is used (Fig. 3)

$$p_{SP}(I) = \begin{cases} 0 & I < I^* - \frac{\epsilon}{2} \\ \frac{1}{\epsilon} (I - I^* + \frac{\epsilon}{2}) & I^* - \frac{\epsilon}{2} < I < I^* + \frac{\epsilon}{2} \\ 1 & I > I^* + \frac{\epsilon}{2} \end{cases}$$

and $p_{PS}(I) = 1 - p_{SP}(I)$.

For the two user-class case, we use separate piecewise-continuous response functions for each class. Users in the first class have a low threshold $I^{*1} = 0.1$. Because of their low threshold,

these users become protected easily, and they do not easily switch from protected to susceptible. We call them *responsible* because of the way they behave helps reduce the infection. The second class of users, who we call *selfish*, have a high threshold $I^{*2} = 0.9$. This means that they hardly ever decide to switch from susceptible to protected, whereas they almost always decide to leave the protected state. For both classes, $\epsilon = 0.001$.

We now establish that the fraction of infected indeed increases for larger values of the update rate γ . For the simulations that follow, we set⁷ $\delta = (6 \text{ h})^{-1}$, and we plot the system trajectories on the $S - I$ plane (average of 30 simulations) for three different values of γ , $(1 \text{ h})^{-1}$, $(6 \text{ h})^{-1}$, and $(24 \text{ h})^{-1}$. The initial conditions for all simulations were 1 infected and 40 susceptible. In the case of two user classes, the initially infected user is of class 2 (selfish). Each simulation runs until either there are no infected, or the end of the traces is reached.

In Fig. 4, we plot simulation results for the single-user class case for $I^* = 0.1, 0.5, 0.9$, and $\epsilon = 0.001$, omitting an initial transient phase. The system state oscillates between two equilibrium points X_0 (nighttime, when the contact rate is low) and either X_1 or X_2 , depending on whether I^* is low enough to limit the infection or not. In all cases, the system trajectories go through higher values of I for increasing values of γ , thus confirming our main conclusion that the infection level increases with the update rate. The effect of lowering I^* is that it limits the maximum infection at the equilibrium, so the trajectories are capped at values of I not far above I^* .

In Fig. 5, we plot the system trajectories for the two user-class case. We again omit the initial transient phase, and we show the susceptible and infected of: 1) the total population (first column); 2) the responsible subpopulation (second column); and 3) the selfish subpopulation (third column). Each row corresponds to a different split of the total population into responsible and selfish subpopulations. In the first row, the responsible selfish split is 20%–80%; in the second row, it is 50%–50%; and in the third row, it is 80%–20%.

We again confirm the conclusion that the fraction of infected in the total population increases for larger values of γ . Two secondary conclusions relate to the situation within each

⁷These are indicative values. Our purpose is not to model any specific worm, but to show that our conclusions (dependence of the infection level on γ) hold for worms with a range of characteristics.

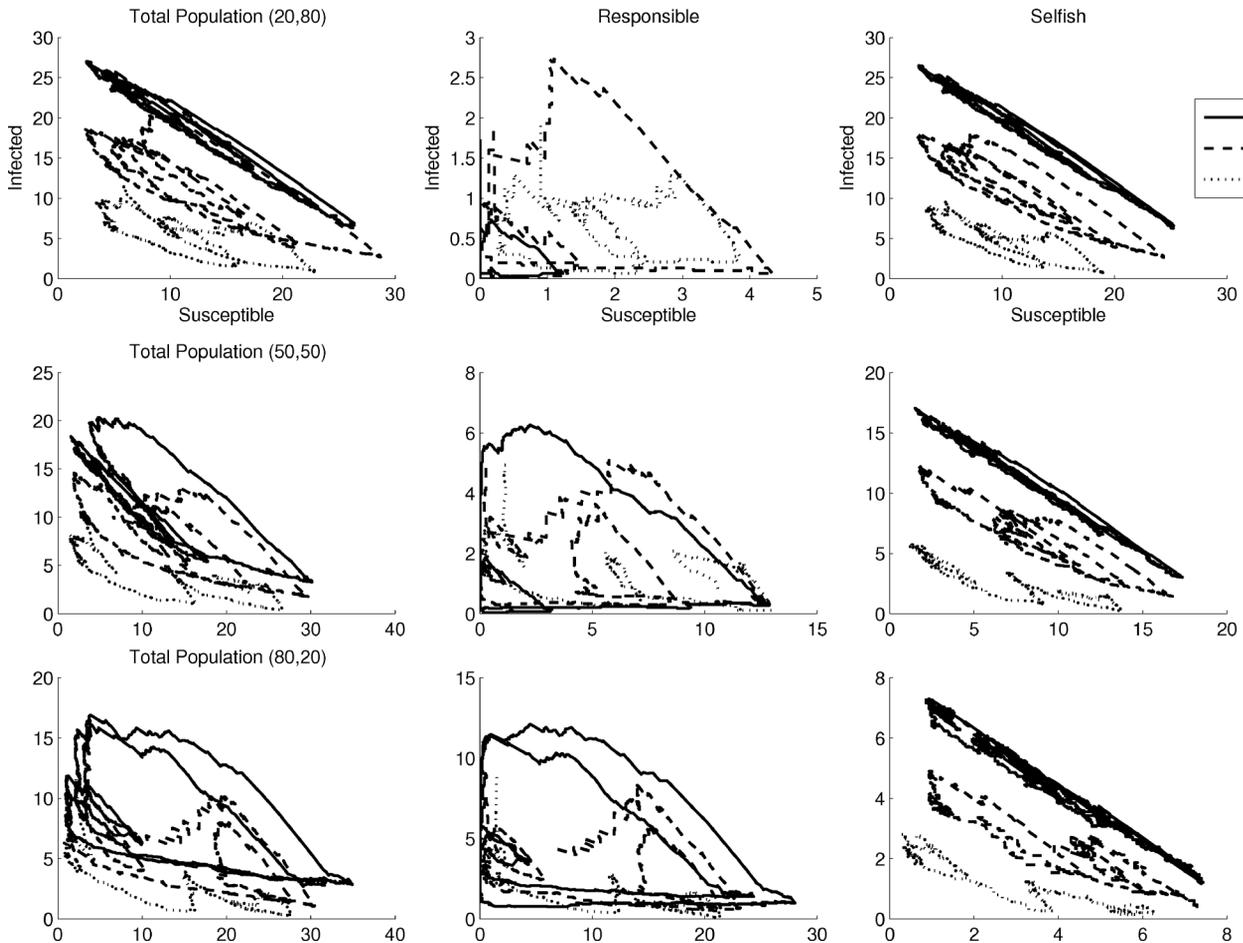


Fig. 5. Trajectory of the system (average of 30 simulations) on the SI plane, when $\delta = (6 \text{ h})^{-1}$ and γ take the values $(1 \text{ h})^{-1}$, $(6 \text{ h})^{-1}$, and $(24 \text{ h})^{-1}$. Users are split into two classes: the responsible, with $I^* = 0.1$, and the selfish, with $I^* = 0.9$. The columns correspond to the total population, the responsible subpopulation, and the selfish subpopulation. The rows correspond to a total population split of 20%–80%, 50%–50%, and 80%–20% into responsible and selfish. As in the case of a single-user class, the network experiences higher numbers of infected devices for higher values of γ . In the current case of multiple user classes, the higher number of infected is mostly due to the selfish users.

subpopulation: the selfish user trajectories seem as if the selfish were isolated. That is, their trajectories are very similar to those they would follow if they were alone in the network (compare with the case $I^* = 0.9$ in Fig. 4). The responsible users, on the contrary, stay mostly in the bottom left region, which means that many of them stay protected. Compared with the case $I^* = 0.9$ in Fig. 4, they now stay a bit closer to the bottom left corner. This means that the selfish-caused infection keeps more of them protected than if they were alone in the network. The observations on the selfish and on the responsible are mutually compatible, as the users that are protected (here, the responsible) do not interact with the rest of the network, so the trajectories of the remaining users (here, the selfish) seem as if they were isolated.

V. POLICY IMPLICATIONS AND POTENTIAL SOLUTIONS

We have confirmed our main conclusion across various scenarios: the higher the learning rate, the higher the infection level at the equilibrium. In order to avoid such an increased infection level, various potential solutions suggest themselves. First, because increasing γ increases the infection, it makes sense to reduce γ , that is, to stop informing users about the current infection level. However, this solution seems a bit radical, throwing out the baby with the bath water. A more moderate solution

would be to inform users only if the infection level exceeds I^* . As long as the infection level is below I^* , the users would not be informed at all, or they would be informed only about the existence (but not the level) of the infection. This solution would indeed decrease the infection, assuming that the users do not start interpreting the absence of information as an indirect notification that the infection level is low.

Another solution is to decrease I^* , as I^* limits the maximum infection level. Decreasing I^* ($I^* \triangleq c_P/c_I$) means decreasing c_P or increasing c_I . On the one hand, increasing c_I (or user perception of c_I) could be achieved by holding users liable⁸ if their devices become infected, or by increasing user awareness for the consequences of an infection. Decreasing c_P , on the other hand, could be done by moving the cost of protection to the mobile operator (or ISP). The operator would have an incentive to shoulder the cost (at least part of it), if it is liable in case of user infection.

Increasing user awareness could also help change the behavior of users. In particular, users should be informed about the long-term consequences of staying unprotected. This might change their myopic behavior.

⁸Penalizing users or software publishers has already been proposed, in the law community, as a potential reform for regulating worms in cyberspace [31].

A synthetic solution that would guarantee a zero-infection level at the equilibrium, is the following: Activate security at the first sign of infection (equivalent to $I^* = 0$) and keep it activated until the infection drops back to zero. That is, the only updates would be at the beginning and at the end of the infection, rather than at a constant rate. However, that would require modifying the behavior of the users, as not all users can be expected to behave voluntarily in such a responsible manner (as $I^* = 0$ implies they would).

VI. CONCLUSIONS AND FUTURE WORK

We have studied the interaction of two factors—myopic decision-makers and dynamic information updates—in the context of security activation decisions in a network with a propagating worm. Our main scenario is the best-response user dynamic in a homogeneous mixing network. We conclude that an increased update rate counterintuitively leads to an increased equilibrium infection level. Our conclusion does not change when the users follow smooth best-response dynamics (arbitrary continuous single-valued function). Our conclusion remains valid in the case of best-response behavior, with one or two classes of users (low and high threshold) simulated on real mobility traces.

In the best-response and smooth best-response dynamics, we identify the equilibrium points, show when they are locally stable, and rule out the possibility of closed trajectories. We also characterize the domains of attraction for each equilibrium point.

In future work, we aim to study the transient behavior of the system. The transient behavior becomes important when the convergence to equilibrium is slow: the time average of the infection cost over some initial finite interval might be quite different from the equilibrium cost.

It may also be worth examining alternative worm models, and testing whether our main conclusion holds for them too. For example, the SEIR model is more suitable for a worm with a dormant phase after infecting a user but before starting to infect others. Alternative countermeasures, such as patching, require different models, such as adding a new state R for the (patched) users that have permanently recovered from the infection.

Worms have multiple spreading patterns (called “target discovery methods” [1]). Depending on the pattern, the epidemic propagation can be faster or slower; it can even be topology dependent (worms that spread to the local network of an infected user, or worms that spread to a user’s e-mail contacts for example). Our conclusions might then need to be modified.

APPENDIX

Proof of Theorem 2: The solution is unique in D^- and in D^+ because F has continuous partial derivatives there.

A solution $x(t)$ of (2) may intersect with the line of discontinuity L , say at time $t = t_0$. We now study when such a solution can be uniquely continued for $t > t_0$ (i.e., we study when right-uniqueness holds).

Formally, let $F^-(x)$ and $F^+(x)$ be the limiting values of F at a point $x \in L$ as F approaches x from D^- and from D^+ , respectively. Let $h(x) = F^+(x) - F^-(x)$, and F_N^-, F_N^+, h_N be the projections of the vectors F^-, F^+, h onto $n = (0, 1)$, the normal to L directed from D^- to D^+ at x .

The values of these vectors and projections are

$$\begin{aligned} F^-(x) &= (-\beta SI^* + \gamma(1 - S - I^*), \beta SI^* - \delta I^*) \\ F^+(x) &= (-\beta SI^* - \gamma S, \beta SI^* - \delta I^*) \\ h(x) &= (-\gamma S - \gamma(1 - S - I^*), 0) \\ F_N^- &= \beta SI^* - \delta I^* \\ F_N^+ &= \beta SI^* - \delta I^* \\ h_N &= 0. \end{aligned}$$

On L , at the points where $F_N^- > 0$, $F_N^+ > 0$ (or $F_N^- < 0$, $F_N^+ < 0$), the solutions pass from D^- into D^+ (correspondingly, from D^+ into D^-) and uniqueness is not violated [24, §10, Corollary 1]. So, at no point of L is uniqueness violated, except possibly at $(\delta/\beta, I^*)$.

For a solution that starts at $(\delta/\beta, I^*)$, there are two possibilities. First, if $0 \in F(\delta/\beta, I^*)$ (i.e., if $(\delta/\beta, I^*)$ is an equilibrium point), then the solution will stay at $(\delta/\beta, I^*)$. But if $0 \notin F(\delta/\beta, I^*)$, the continuation is not unique: There is one continuation for each element of set $F(\delta/\beta, I^*)$, and all of them are tangent to L because $dI/dt = 0$ when $S = \delta/\beta$ [See Fig. 1(c)]. In the proof of Lemma 2, we show that none of them can ever approach $(\delta/\beta, I^*)$ again in the positive direction of time. ■

Proof of Lemma 4, part of Theorem 6: Call $A = (S_A, I^*)$ the point in $\Gamma \cap L$ with $S_A < \delta/\beta$, and call $B = (S_B, I^*)$ the one with $S_B > \delta/\beta$. Let Γ be parameterized by $\phi(t) = (x(t), y(t))$, $t \in [0, T]$; also $\phi(0) = \phi(T)$. Function $\phi(t)$ is a solution of the differential inclusion, that is, $\dot{\phi}(t) = (\dot{x}(t), \dot{y}(t)) \in F(\phi(t))$, $t \in [0, T]$. Let $t_A, t_B \in [0, T]$ be such that $A = \phi(t_A)$ and $B = \phi(t_B)$. Let $\alpha_A, \alpha_B \in [0, 1]$ be such that $\dot{x}(t_A) = -\beta x y - \gamma x + \alpha_A \gamma(1 - y)$ and $\dot{x}(t_B) = -\beta x y - \gamma x + \alpha_B \gamma(1 - y)$.

Define $P(x, y)$ and $Q(x, y)$, $(x, y) \in D \setminus \{y, y > 0\}$ as follows:

$$\begin{aligned} P(x, y) &= -\frac{1}{y}\dot{y} = -\frac{1}{y}(\beta xy - \delta y) = \delta - \beta x \\ Q(x, y) &= \frac{1}{y}\dot{x} \\ &= \begin{cases} \frac{1}{y}(-\beta xy + \gamma(1 - x - y)), & y < I^* \\ \frac{1}{y}(-\beta xy - \gamma x + \alpha_A \gamma(1 - y)), & x \leq \frac{\delta}{\beta}, y = I^* \\ \frac{1}{y}(-\beta xy - \gamma x + \alpha_B \gamma(1 - y)), & x > \frac{\delta}{\beta}, y = I^* \\ \frac{1}{y}(-\beta xy - \gamma x), & y > I^* \end{cases} \end{aligned}$$

We compute $\oint_{\Gamma} P dx + Q dy$ in two ways.

For the first computation, we use the parametrization $\phi(t) = (x(t), y(t))$ of Γ , so $dx = \dot{x} dt$ and $dy = \dot{y} dt$. The result is zero

$$\oint_{\Gamma} P dx + Q dy = \int_0^T -\frac{1}{y}\dot{y}\dot{x} dt + \frac{1}{y}\dot{x}\dot{y} dt = 0. \quad (7)$$

For the second computation, we split $Q(x, y)$ into two functions, one continuous $Q_c(x, y)$ and one discontinuous $Q_d(x, y)$, so that $Q(x, y) = Q_c(x, y) + Q_d(x, y)$

$$Q_c(x, y) = \frac{1}{y}(-\beta xy - \gamma x)$$

$$Q_d(x, y) = \begin{cases} \frac{1}{y}\gamma(1-y), & y < I^* \\ \frac{1}{y}\alpha_A\gamma(1-y), & x \leq \frac{\delta}{\beta}, y = I^* \\ \frac{1}{y}\alpha_B\gamma(1-y), & x > \frac{\delta}{\beta}, y = I^* \\ 0, & y > I^*. \end{cases}$$

So now, the original integral can be split into two $\oint_{\Gamma} P dx + Q dy = \oint_{\Gamma} P dx + (Q_c + Q_d) dy = \oint_{\Gamma} P dx + Q_c dy + \oint_{\Gamma} Q_d dy$. We use Green's theorem to compute the first integral

$$\begin{aligned} \oint_{\Gamma} P dx + Q_c dy &= \int_{\Gamma} \int \frac{\partial Q_c}{\partial x} - \frac{\partial P}{\partial y} dx dy \\ &= \int_{\Gamma} \int -\beta - \frac{\gamma}{y} dx dy < 0. \end{aligned} \quad (8)$$

To compute the second integral $\oint_{\Gamma} Q_d dy$, we define function

$$Q_d^{\text{ext}}(x, y) = \frac{1}{y}\gamma(1-y), \quad (x, y) \in D \setminus \{y, y > 0\}$$

and curves Γ_1 and Γ_2 . Curve Γ_1 is Γ restricted to $y \leq I^*$, with direction from A to B . Curve Γ_2 is the line segment of L joining B and A , with direction from B to A .

Observe that

$$\oint_{\Gamma} Q_d dy = \oint_{\Gamma_1 \cup \Gamma_2} Q_d^{\text{ext}} dy = \int_{\Gamma_1 \cup \Gamma_2} \frac{\partial Q_d^{\text{ext}}}{\partial x} dx dy = 0$$

where the first equality follows from $Q_d \equiv Q_d^{\text{ext}}$ on Γ_1 and $dy = 0$ on Γ_2 , whereas the last equality follows from Green's theorem, because Q_d^{ext} is continuously differentiable.

The result of (7) contradicts the results of (8) and (9). So the trajectory Γ with the assumed properties cannot exist. ■

REFERENCES

- [1] N. Weaver, V. Paxson, S. Staniford, and R. Cunningham, "A taxonomy of computer worms," in *Proc. ACM Workshop Rapid Malcode*, New York, 2003, pp. 11–18.
- [2] F-Secure Corp., Calculating the size of the downadup outbreak. 2009. [Online]. Available: <http://www.f-secure.com/weblog/archives/00001584.html>
- [3] M. Khouzani, E. Altman, and S. Sarkar, "Optimal quarantining of wireless malware through power control," in *Proc. Inf. Theory Appl. Workshop*, Feb. 2009, pp. 301–310.
- [4] L. Jiang, V. Anantharam, and J. Walrand, "How bad are selfish investments in network security?," *Proc. IEEE/ACM Trans. Netw.*, vol. 19, no. 2, pp. 549–560, 2011.
- [5] M. Lelarge and J. Bolot, "Network externalities and the deployment of security features and protocols in the internet," in *Proc. ACM SIGMETRICS Int. Conf. Meas. Model. Comput. Syst.*, New York, 2008, pp. 37–48.
- [6] J. Aspnes, K. Chang, and A. Yampolskiy, "Inoculation strategies for victims of viruses and the sum-of-squares partition problem," *J. Comput. Syst. Sci.*, vol. 72, no. 6, pp. 1077–1093, 2006.
- [7] T. Moscibroda, S. Schmid, and R. Wattenhofer, "When selfish meets evil: Byzantine players in a virus inoculation game," in *Proc. 25th ACM Symp. Principles Distrib. Comput.*, New York, 2006, pp. 35–44.
- [8] W. Yan and N. Ansari, "Why anti-virus products slow down your machine?," in *Proc. IEEE 18th Int. Conf. Comput. Commun. Netw.*, San Francisco, CA, Aug. 2009, pp. 1–6.

- [9] B. Schneier, How to sell security. 2008. [Online]. Available: http://www.schneier.com/blog/archives/2008/05/how_to_sell_sec.html
- [10] D. Kahneman and A. Tversky, "Prospect theory: An analysis of decision under risk," *Economet.: J. Economet. Soc.*, vol. 47, no. 2, pp. 263–291, 1979.
- [11] United States Computer Emergency Readiness Team [Online]. Available: <http://www.us-cert.gov/>
- [12] F-Secure Corp., Mobile malware descriptions. [Online]. Available: <http://www.f-secure.com/v-descs/mobile-description-index.shtml>
- [13] N. Gast and B. Gaujal, "Mean field limit of non-smooth systems and differential inclusions," presented at the Math. Perform. Model. Anal., New York, 2010.
- [14] H. Hethcote, "The mathematics of infectious diseases," *SIAM Rev.*, vol. 42, no. 4, pp. 599–653, 2000.
- [15] J. O. Kephart and S. R. White, "Directed-graph epidemiological models of computer viruses," in *Proc. IEEE Comput. Soc. Symp. Res. Security Privacy*, Oakland, CA, 1991, pp. 343–359.
- [16] M. Vojnovic and A. Ganesh, "On the race of worms, alerts, patches," *IEEE/ACM Trans. Netw.*, vol. 16, no. 5, pp. 1066–1079, Oct. 2008.
- [17] C. C. Zou, W. Gong, and D. Towsley, "Code red worm propagation modeling and analysis," in *Proc. 9th ACM Conf. Comput. Commun. Security*, New York, 2002, pp. 138–147.
- [18] D. Moore, V. Paxson, S. Savage, C. Shannon, S. Staniford, and N. Weaver, "Inside the slammer worm," *IEEE Security Privacy*, vol. 1, no. 4, pp. 33–39, Jul. 2003.
- [19] A. Ganesh, L. Massoulie, and D. Towsley, "The effect of network topology on the spread of epidemics," in *INFOCOM'05: Proc. IEEE 24th Annu. Int. Conf. Comput. Commun.*, Miami, FL, Mar. 2005, vol. 2, pp. 1455–1466.
- [20] J. Weibull, *Evolutionary Game Theory*. Cambridge, MA: MIT Press, 1997.
- [21] J. Hofbauer and K. Sigmund, *Evolutionary Games and Population Dynamics*. Cambridge, U.K.: Cambridge Univ. Press, 1998.
- [22] J. Hofbauer and K. Sigmund, "Evolutionary game dynamics," *Bull. Amer. Math. Soc.*, vol. 40, no. 4, pp. 479–519, 2003.
- [23] W. Sandholm, *Population Games and Evolutionary Dynamics*. Cambridge, MA: MIT Press, 2010.
- [24] A. F. Filippov, *Differential Equations With Discontinuous Righthand Sides*, ser. Mathematics and its applications. Norwell, MA: Kluwer, 1988.
- [25] G. Theodorakopoulos, J.-Y. Le Boudec, and J. S. Baras, EPFL, Selfish response to epidemic propagation, Tech. Rep. 167508, 2011. [Online]. Available: <http://infoscience.epfl.ch/record/167508>
- [26] R. Dolgoarshinnykh, "Law of large numbers for the SIRS epidemic processes," Columbia Univ., New York, 2010. [Online]. Available: <http://www.stat.columbia.edu/~regina/research/>
- [27] T.G. Kurtz, *Approximation of Population Processes*. Philadelphia, PA: SIAM, 1981.
- [28] L. Ljung, "Strong convergence of a stochastic approximation algorithm," *Ann. Stat.*, vol. 6, no. 3, pp. 680–696, 1978.
- [29] H. Dulac, "Recherche des cycles limites," *CR Acad. Sci. Paris*, vol. 204, pp. 1703–1706, 1937.
- [30] J. Scott, R. Gass, J. Crowcroft, P. Hui, C. Diot, and A. Chaintreau, CRAWDAD Data Set Cambridge/Haggle (v. 2006-09-15) Sep. 2006. [Online]. Available: <http://crawdad.cs.dartmouth.edu/cambridge/haggle>
- [31] D. Barnes, "Deworming the internet," *Texas Law Rev.*, vol. 83, pp. 279–329, 2004.



George Theodorakopoulos received the Diploma degree from the National Technical University of Athens, Athens, Greece, in 2002, and the M.S. and Ph.D. degrees in electrical and computer engineering from the University of Maryland, College Park, MD, in 2004 and 2007, respectively.

He is a Lecturer with the School of Computer Science and Informatics, Cardiff University, Cardiff, U.K. From 2007 to 2011, he was a Senior Researcher at the Ecole Polytechnique Fédérale de Lausanne, Lausanne, Switzerland. From 2011 to 2012, he

was a Lecturer at the University of Derby. He co-authored *Path Problems in Networks* (Morgan & Claypool, 2010). His research interests include privacy, network security, trust and reputation systems, game theory, and algebraic combinatorics.

Dr. Theodorakopoulos received the Best Paper award at the ACM Workshop on Wireless Security in 2004, and the 2007 IEEE ComSoc Leonard Abraham Prize.



Jean-Yves Le Boudec (F'09) graduated from Ecole Normale Supérieure de Saint-Cloud, Paris, France. He received the aggregation degree in mathematics and the Ph.D. degree from the University of Rennes, Rennes, France, in 1980 and 1984, respectively.

Currently, he is Full Professor at the Ecole Polytechnique Fédérale de Lausanne, Lausanne, Switzerland. From 1984 to 1987, he was with INSA/IRISA, Rennes, France. In 1987, he joined Bell Northern Research, Ottawa, ON, Canada, as a member of the scientific staff in the Network and Product Traffic Design Department. In 1988, he joined the IBM Zurich Research Laboratory, Zurich, Switzerland, where he was Manager of the Customer Premises Network Department. In 1994, he joined EPFL as Associate Professor. In 1984, he developed analytical models of multiprocessor, multiple bus computers. In 1990, he invented the concept called "MAC emulation" which later became the ATM forum LAN emulation project, and developed the first ATM control point based on OSPF. He also launched public domain software for the interworking of ATM and TCP/IP under Linux. In 1998, he proposed the first solution to failure propagation that arises from common infrastructures in the Internet. He contributed to network calculus, a recent set of developments that forms a foundation to many traffic-control concepts in the Internet, and co-authored a book on this topic. He is also the author of the book *Performance Evaluation* (2010). His interests are in the performance and architecture of communication systems.

Prof. Le Boudec received the IEEE Millennium medal, the Infocom 2005 Best Paper award, the CommSoc 2008 William R. Bennett Prize, and the 2009 ACM Sigmetrics Best Paper award. He is or has been on the program committee or editorial board of many conferences and journals, including Sigcomm, Sigmetrics, Infocom, *Performance Evaluation*, and ACM/IEEE TRANSACTIONS ON NETWORKING.



John S. Baras (F'84) received the B.S. degree in electrical engineering from the National Technical University of Athens, Athens, Greece, in 1970, and the M.S. and Ph.D. degrees in applied mathematics from Harvard University, Cambridge, MA, in 1971 and 1973, respectively.

Prof. Baras was the Founding Director of the Institute for Systems Research (one of the first six National Science Foundation Engineering Research Centers) from 1985 to 1991. Since 1973, he has been with the Electrical and Computer Engineering Department and the Applied Mathematics Faculty, University of Maryland, College Park. In 1990, he was appointed to the Lockheed Martin Chair in Systems Engineering. Since 1991, he has been the Director of the Center for Hybrid and Satellite Communication Networks (a NASA Research Partnership Center). He holds three patents. His research interests include: wireless networks and MANET, wireless network security and information assurance, integration of logic programming and nonlinear programming for tradeoff analysis, multicriteria optimization, non-cooperative and cooperative dynamic games, robust control of nonlinear systems and hybrid automata, mathematical and statistical physics algorithms for control and communication systems, distributed asynchronous control and communication systems, object-oriented modeling of complex engineering systems, satellite and hybrid communication networks, network management, fast Internet services over hybrid wireless networks, stochastic systems, planning and optimization, intelligent control and learning, biologically inspired algorithms for signal processing, and sensor networks.

Prof. Baras is the recipient of the 1980 Outstanding Paper Award, IEEE Control Systems Society; 1978, 1983, and 1993 Alan Berman Research Publication Awards, NRL; 1991 and 1994 Outstanding Invention of the Year Awards, University of Maryland; the Mancur Olson Research Achievement Award, University of Maryland; 2002 Best Paper Award, 23rd Army Science Conference; and 2004 Best Paper Award, 2004 WiSe Conference.