

Mean-field analysis of an inductive reasoning game: Application to influenza vaccination

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Recently we have introduced an inductive reasoning game of voluntary yearly vaccination to establish whether or not a population of individuals acting in their own self-interest would be able to prevent influenza epidemics. Here, we analyze our model to describe the dynamics of the collective yearly vaccination uptake. We discuss the mean-field equations of our model and first order effects of fluctuations. We explain why our model predicts that severe epidemics are periodically expected even without the introduction of pandemic strains. We find that fluctuations in the collective yearly vaccination uptake induce severe epidemics with an expected periodicity that depends on the number of independent decision makers in the population. The mean-field dynamics also reveal that there are conditions for which the dynamics become robust to the fluctuations. However, the transition between fluctuation-sensitive and fluctuation-robust dynamics occurs for biologically implausible parameters. We also analyze our model when incentive-based vaccination programs are offered. When a family-based incentive is offered, the expected periodicity of severe epidemics is increased. This results from the fact that the number of independent decision makers is reduced, increasing the effect of the fluctuations. However, incentives based on the number of years of prepayment of vaccination may yield fluctuation-robust dynamics where severe epidemics are prevented. In this case, depending on prepayment, the transition between fluctuation-sensitive and fluctuation-robust dynamics may occur for biologically plausible parameters. Our analysis provides a practical method for identifying how many years of free vaccination should be provided in order to successfully ameliorate influenza epidemics.

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I. INTRODUCTION

Recently, we have introduced a model that combines an epidemic model of influenza with human cognition and vaccination behavior [1]. Using computational simulations, we found that even without the introduction of pandemic strains, severe influenza epidemics are to be expected. However, we also found that these severe epidemics can be prevented when certain incentive-based vaccination programs are offered to the public. Here, we analyze our model by formulating mean-field equations with added first order fluctuations to describe the dynamics of the collective vaccination behavior. Our analyses explain the numerical results and identify incentive-based vaccination programs that are effective in preventing severe influenza epidemics.

In our model, individuals decide whether or not they should vaccinate against influenza based upon their vaccination experiences. Therefore, individuals adapt to the past influenza epidemiology which, by the action of vaccination, they had helped to determine. This continuous adaptation of one's behavior to predicted future collective behavior is called *inductive reasoning* and applies in many instances where logical deductive reasoning fails, either in principle or due to bounded rationality [2]. Over the past decade, inductive reasoning games have attracted increasing attention from the statistical physics community. In particular, these games have been applied to model financial markets where traders decide to buy or sell a certain asset whose price is determined by their collective action [3–6]. However, until very recently inductive reasoning games have not been applied to theoretical epidemiology. Previous applications of game theory to epidemiology have been based on deductive rea-

soning games. These have helped price vaccines [7] and predict the voluntary *vaccination coverage* (i.e., the proportion of the population that gets vaccinated) for pathogens that provide permanent immunity (e.g., smallpox and measles) [8,9]. However, in the case of pathogens that do not provide permanent immunity (e.g., influenza), individuals need to make repeated vaccination decisions under epidemiological conditions that may not be sufficiently well known. Thus, it may be assumed, in this case, that individuals make vaccination decisions based on their past experiences (i.e., use inductive reasoning) rather than based only on the current epidemiology (i.e., use deductive reasoning).

Since the influenza vaccine is effective only for 1 year, individuals must decide every year whether to vaccinate or not [10]. We assume that individuals act in their own self-interest trying to avoid infection preferably without having to vaccinate. The yearly vaccination coverage is determined by the collective participation of the individuals. Compartmental models of influenza transmission (e.g., [11–13]) have shown that there exists a *critical coverage level* such that: if the coverage is below the critical level, an epidemic will occur, otherwise epidemics will be prevented. In our model, individuals vaccinate to avoid infection, but if the coverage is larger than the critical level, they no longer need to vaccinate. When an individual does not vaccinate and the critical coverage is not reached, they still have a nonzero probability of avoiding infection due to peer vaccination (i.e., they are protected by *herd immunity*). We construct an individual-level model that describes the adaptive dynamics of vaccination decisions in a population of noncommunicating individuals acting in their own self-interest (i.e., *selfish* individuals). Our model tracks individual-level vaccination decisions and behavior as well as the resulting population-

level variables, such as influenza prevalence and vaccination coverage levels.

The outline of the paper is as follows. In Sec. II we describe and analyze our individual-level vaccination model previously introduced in Ref. [1]. Our strategy is to derive a dynamical system for the expected coverage given by the model. Then, we address deviations from this mean-field limit by discussing first order effects of the fluctuations. Using the same approach, in Sec. III we discuss analytical results regarding two cases of the basic model with superimposed vaccination incentives. Our analysis helps explain the coverage dynamics observed in direct numerical simulations of the model previously presented in Ref. [1].

II. BASIC MODEL

A. Description

Deterministic models of influenza transmission, based on ordinary differential equations (e.g., [11–14]), have shown that there exists a *critical coverage level* below which an epidemic is expected, otherwise epidemics are prevented. These models predict that the average number of secondary cases caused by one infectious case at the beginning of an influenza epidemic ranges between 2 and 3 [14]. From these values one obtains that the critical vaccination coverage ranges between 0.50 and 0.67. Our inductive game includes a simple model of this coverage threshold [15]. We denote the coverage by p , the critical coverage by π_c , and the probability of getting infected when the coverage is p by $q(p)$. We assume that if $p < \pi_c$, then $q(p)$ decreases linearly with p , otherwise $q(p) = 0$. This model is consistent with the fact that unvaccinated individuals benefit from herd immunity. Also, it is in qualitative agreement with results found from the analysis of the susceptible-infected-recovered and susceptible-exposed-infected-recovered transmission models [1]. The nature of the results that we describe in this paper does not depend on the details of $q(p)$. It only depends on the fact that $q(p)$ is strictly monotonically decreasing for $p < \pi_c$ and 0 for $p \geq \pi_c$. We now present the assumptions that define our inductive reasoning game which we refer to as the basic model.

(1) We consider a number of N individuals that every year make vaccination decisions [16]. They are assumed to act in their own interest and not to communicate their decisions to each other. The sole interest of the individuals is to avoid getting infected, preferably without having to vaccinate.

(2) To make their vaccination decisions, each individual uses their past experience of vaccination outcomes. Thus, individuals independently decide whether or not to vaccinate using inductive reasoning.

(3) An individual remembers and weights their previous vaccination outcomes with respect to their present vaccination outcome. A parameter s discounts the previous year vaccination outcome with respect to the outcome of the present year ($0 \leq s < 1$). For $s = 0$, individuals completely ignore the outcome of previous seasons and, as a consequence, do not use inductive reasoning. If s were equal to 1, individuals would not discount the previous vaccination seasons; there-

fore, the vaccination outcome of the present season (i.e., season n) would be as important as any of the previous seasons.

(4) We define a vaccination decision as a realization $x_n^{(i)}$ of a Bernoulli variable with parameter $w_n^{(i)}$ that further depends on a variable $v_n^{(i)}$. i and n are positive integers; $i = 1, 2, \dots, N$ labels the individual and $n > 0$ labels the season. If individual i decides to get vaccinated in season n then $x_n^{(i)} = 1$, otherwise $x_n^{(i)} = 0$. $w_n^{(i)}$ is the probability that individual i vaccinates in season n . The variable $v_n^{(i)}$ characterizes the provaccination experience of the i th individual [see details in assumption (7)] and determines $w_n^{(i)}$. If s were 1, $v_n^{(i)}$ would represent the number of years up to year n when individual i has benefited from vaccination. The domains of the variables are as follows: $x_n^{(i)} \in \{0, 1\}$, $w_n^{(i)} \in [0, 1]$, and $v_n^{(i)} \in [0, 1/(1-s)]$.

(5) In year n , a set of N vaccination decisions is made $\{x_n^{(i)}; 1 \leq i \leq N\}$ that determines the provaccination experiences up to year $(n+1)$ of all the individuals $\{v_{n+1}^{(i)}; 1 \leq i \leq N\}$ which further determine $\{w_{n+1}^{(i)}; 1 \leq i \leq N\}$, the parameters of the Bernoulli variables in year $(n+1)$. Then, the set of vaccination decisions in year $(n+1)$ is obtained $\{x_{n+1}^{(i)}; 1 \leq i \leq N\}$. Our inductive reasoning game is an array of sets of vaccination decisions.

(6) The infection event of individual i in year n is described by a variable $z_n^{(i)}$. (If individual i got infected in season n then $z_n^{(i)} = 1$, otherwise $z_n^{(i)} = 0$.) The infection process is as follows. If $x_n^{(i)} = 1$ then $z_n^{(i)} = 0$. If $x_n^{(i)} = 0$, then $z_n^{(i)}$ is a realization of a Bernoulli variable with parameter $q(p_n)$, where $p_n = \sum_{i=1}^N x_n^{(i)} / N$ is the coverage achieved that year. That is, if individuals vaccinate, they are fully protected, otherwise they risk infection with probability $q(p_n)$.

(7) $\{x_n^{(i)}; 1 \leq i \leq N\}$ and $\{z_n^{(i)}; 1 \leq i \leq N\}$ determine $v_{n+1}^{(i)}$ as follows (see Fig. 1). We have four cases: (a1) if $x_n^{(i)} = 1$ and $\pi_c \leq p_n$, then $v_{n+1}^{(i)} = sv_n^{(i)}$; that is, if individual i gets vaccinated in season n and no epidemic occurs, then the individual considers that the vaccination was unnecessary; (a2) if $x_n^{(i)} = 1$ and $p_n < \pi_c$, then $v_{n+1}^{(i)} = sv_n^{(i)} + 1$; which means that if individual i vaccinates in season n and an epidemic occurs, then the individual considers that the vaccination was necessary; (b1) if $x_n^{(i)} = 0$ and $z_n^{(i)} = 1$ then $v_{n+1}^{(i)} = sv_n^{(i)} + 1$; that is, if individual i does not get vaccinated in season n and gets infected, then the individual considers that the vaccination was necessary; and (b2) if $x_n^{(i)} = 0$ and $z_n^{(i)} = 0$ then $v_{n+1}^{(i)} = sv_n^{(i)}$; which means that if individual i does not get vaccinated in season n and they do not get infected, then the individual considers that the vaccination was unnecessary.

(8) The probability that an individual chooses to get vaccinated is updated as follows:

$$w_{n+1}^{(i)} = v_{n+1}^{(i)} / [(1 - s^{n+1}) / (1 - s)]. \quad (1)$$

That is, an individual's probability to get vaccinated in the next season is given by the updated cumulative vaccination experience. We have normalized $v_{n+1}^{(i)}$ by $(1 - s^{n+1}) / (1 - s)$ because this factor is the maximum possible value for $v_{n+1}^{(i)}$ if individual i would have benefited from vaccination in all of the n influenza seasons.

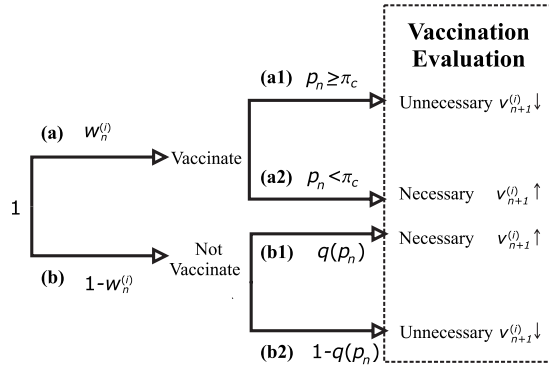


FIG. 1. Diagram illustrating the evaluation tree for each selfish individual. An individual that decides to vaccinate [branch (a)] will judge their choice depending on whether there was an epidemic that season. If the coverage is equal or greater than the critical coverage $p_n \geq \pi_c$ [branch (a1)], they will conclude that their choice to get vaccinated that season was not necessary to prevent infection. Otherwise, if the coverage is lower than the critical coverage $p_n < \pi_c$ [branch (a2)], they will conclude that their choice was beneficial for avoiding infection that season. An individual that decides not to vaccinate that season [branch (b)] will judge their choice based on whether they were infected. If they do get infected [branch (b1)] they will conclude that their choice of not vaccinating was detrimental and that vaccination was necessary for avoiding infection. Instead, if by chance they avoid infection [branch (b2)], they will conclude that vaccination was unnecessary.

B. Numerical results

Figure 2 shows numerics obtained by simulating the basic model. We considered initial conditions that assign a random vaccination probability for the first season to every individual. Specifically, $v_0^{(i)} = 0$ and $w_0^{(i)}$ was uniformly distributed between 0 and 1 for all i . Our initial conditions were chosen to reflect the fact that at the individual level the likelihood of vaccination may have varied considerably. Figures 2(a1,2) show previously obtained dynamics [1] of the coverage and

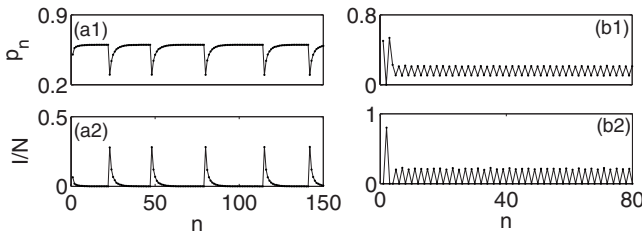


FIG. 2. Coverage (i.e., fraction of vaccinated individuals) and incidence (i.e., fraction of infected individuals) dynamics for a population of $N=10^5$ individuals using a memory parameter $s=0.7$ and a probability $q(0)=0.8$ of getting infected when $p=0$. (a1) For $\pi_c=0.6$, the dynamics of p is approximately cyclic: as p approaches π_c from below, it eventually fluctuates above π_c and then abruptly drops below π_c . (b1) For $\pi_c=0.6$, the dynamics of p is very well approximated by a period two orbit. (a2) and (b2) The dynamics of the incidence I/N mirrors that of the coverage. A low coverage yields a severe incidence, while a high coverage yields a mild incidence or if $p > \pi_c$ epidemics are prevented and therefore the incidence is zero.

the proportion of infected individuals for $\pi_c=0.6$, respectively. It can be seen that as p approaches π_c from below, it eventually fluctuates above π_c , then abruptly drops below π_c , and the dynamics repeat; see [1] for a more in depth discussion of the biological implications of these dynamics. Figures 2(b1,2) show the dynamics of the coverage and the proportion of infected individuals for a lower value of the critical coverage $\pi_c=0.2$. The dynamics is that of a period two orbit and it is qualitatively different from that presented in panels (a1,2). We note however that influenza has a high critical coverage and thus the behavior shown in panels (a1,2) is more realistic. Our analysis aims to provide an understanding of the coverage dynamics in Fig. 2.

C. Analysis

We first derive and analyze a one-dimensional iterated map in the variable π denoting the average coverage over realizations of the game in the limit of large N . Then, we discuss asymptotic aspects of the model that apply as the population is large, yet finite. We thus define $\pi_n \equiv \langle p_n \rangle$; using the definition of p_n , we immediately have $\pi_n = \sum_{i=1}^N \langle w_n^{(i)} \rangle / N$, and, since q is piecewise linear, $\langle q(p) \rangle = q(\pi)$. Following the tree in Fig. 1 which describes the four cases of vaccination evaluation given by assumption (7), we obtain

Branch	Expected population fraction	Average $v^{(i)}$ update
(a1,2)	π_n	$\langle v_{n+1}^{(i)} \rangle = s \langle v_n^{(i)} \rangle + 1 - \theta(\pi_n - \pi_c)$
(b1)	$(1 - \pi_n)q(\pi_n)$	$\langle v_{n+1}^{(i)} \rangle = s \langle v_n^{(i)} \rangle + 1$
(b2)	$(1 - \pi_n)[1 - q(\pi_n)]$	$\langle v_{n+1}^{(i)} \rangle = s \langle v_n^{(i)} \rangle$

(2)

where $\theta(x)$ is the unit step function defined as

$$\theta(x) = \begin{cases} 1 & \text{if } x \geq 0, \\ 0 & \text{if } x < 0, \end{cases} \quad (3)$$

and $q(\pi)$, the probability of an unvaccinated individual getting infected with influenza, is given by

$$q(\pi) = \begin{cases} 0 & \text{if } \pi \geq \pi_c, \\ -\pi q(0)/\pi_c + q(0) & \text{if } \pi < \pi_c. \end{cases} \quad (4)$$

Taking the weighted average over Eqs. (2), we obtain

$$u_{n+1} = s u_n + (1 - \pi_n)q(\pi_n) + \pi_n[1 - \theta(\pi_n - \pi_c)], \quad (5)$$

where u_n denotes the average of $\langle v_n^{(i)} \rangle$ over the entire population. Averaging Eqs. (1) over realizations of the game and over the population, we get

$$\pi_{n+1} = (1 - s)u_{n+1}/(1 - s^{n+1}). \quad (6)$$

Combining Eqs. (5) and (6), we describe the dynamics of the vaccination coverage at the population level in the limit of infinite population, without regards to the individual-level processes

$$\pi_{n+1} = \frac{s(1-s^n)}{1-s^{n+1}}\pi_n + \frac{1-s}{1-s^{n+1}}\{(1-\pi_n)q(\pi_n) + \pi_n[1-\theta(\pi_n-\pi_c)]\}. \quad (7)$$

Our dynamical system is defined on the unit interval $[0, 1]$.

1. Fixed point analysis

As $n \rightarrow \infty$, the map given by Eq. (7) takes the following autonomous asymptotic form:

$$\pi_{n+1} = s\pi_n + (1-s)\{(1-\pi_n)q(\pi_n) + \pi_n[1-\theta(\pi_n-\pi_c)]\}. \quad (8)$$

Due to the discontinuity at $\pi = \pi_c$, we distinguish two complementary domains: $\mathcal{I}_1 = [\pi_c, 1]$ and $\mathcal{I}_2 = [0, \pi_c)$. We emphasize here that the step function $\theta(\pi - \pi_c)$ is due to the structure of the evaluation tree, and thus this discontinuity would still occur in the map even if $q(p)$ were smooth.

Case 1: \mathcal{I}_1 . Equation (8) becomes the following linear dynamical system:

$$\pi_{n+1} = s\pi_n. \quad (9)$$

The above dynamical system has no attractors in \mathcal{I}_1 . However, if we extend the domain to $[0, 1]$, then the system has a fixed point at 0 which is a global attractor that belongs to \mathcal{I}_2 . This fully characterizes the dynamics in \mathcal{I}_1 : orbits in \mathcal{I}_1 will be attracted to 0 until they land in \mathcal{I}_2 . In \mathcal{I}_2 the orbit is iterated with a different smooth map.

Case 2: \mathcal{I}_2 . We now obtain the following nonlinear dynamical system:

$$\pi_{n+1} = \pi_n + (1-s)(1-\pi_n)[- \pi_n q(0)/\pi_c + q(0)], \quad (10)$$

that has no fixed points in \mathcal{I}_2 . However, if we extend the domain to $[0, 1]$, then the system has two fixed points: 1 and π_c ; we denote the fixed point at π_c by π^* . The derivative of the map evaluated at 1 is $1+q(0)(1-s)(\pi_c^{-1}-1) > 1$; thus, this fixed point is unstable for all the parameter values. $\pi^* = \pi_c$ is a potential attractor of the system since the derivative of the map evaluated at π_c is

$$\lambda = 1 - q(0)(1-s)(\pi_c^{-1} - 1) \quad (11)$$

with range $(-\infty, 1]$. It is important to note that π^* lies on the boundary between \mathcal{I}_1 and \mathcal{I}_2 . Thus, even though π^* does not belong to \mathcal{I}_2 , π^* attracts orbits with initial conditions in \mathcal{I}_2 . That is, the basin of attraction of π^* intersects \mathcal{I}_2 . When $0 < \lambda < 1$, orbits starting in \mathcal{I}_2 are immediately attracted from below to π^* , but never reach this fixed point. They only approach arbitrarily close, always remaining in \mathcal{I}_2 . When $-1 \leq \lambda \leq 0$, π^* cannot be an attractor because the orbits would approach π^* through damped oscillations and, since $\pi^* \in \partial\mathcal{I}_2$, they enter \mathcal{I}_1 where they are iterated with a different smooth component of the map.

2. Bifurcation diagram

A bifurcation diagram for $s=0.7$, $q(0)=0.8$ and varying π_c is presented in Fig. 3. For large values of π_c , π^* is the only attractor of the map. A rich dynamical behavior is ob-

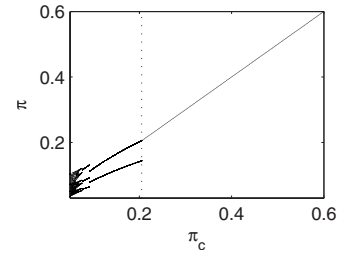


FIG. 3. Bifurcation diagram of the map given by Eq. (7) versus π_c . We have chosen $s=0.7$ and $q(0)=0.8$. The dotted line indicates the approximated position of Π_0 , the π_c value where, due to the discontinuity of the map, a period two orbit is created. We note that numerical noise greatly perturbs the dynamics of the piecewise smooth map since $\pi^* \in \partial\mathcal{I}_2$. To improve the stability of the numerics, we slightly modified the map in order for $\pi^* \in \mathcal{I}_2$; in turn, this slightly changes the threshold value of π_c and reduces the π_c interval for which the period two orbit and the π^* fixed point coexist as attractors.

served for our iterated map with decreasing π_c as λ strictly decreases with decreasing π_c . As expected in piecewise smooth systems, we observe *border-collision bifurcations* [17–23]. A critical period two orbit with elements $\{\pi_c, s\pi_c\}$ is created by a codimension one bifurcation when π_c takes the value $\Pi_0 = \{s + 1/[(1-s)q(0)]\}^{-1} \approx 0.205$; see Fig. 4(a). The fixed point π^* and the period two orbit become coexisting attractors; see Fig. 4(b). With further decreasing π_c , the basin of attraction of the period two orbit increases [24] while the basin of π^* decreases until, when π_c equals $\{1 + 1/[(1-s)q(0)]\}^{-1} \approx 0.194$, λ becomes zero, the π^* attractor is destroyed, and the basin of the period two orbit becomes the entire domain of the map. With further decreasing π_c , period doubling and chaotic behavior is numerically observed.

3. Effects at large finite N

The dynamics of the expected coverage π_n is insufficient to explain the dynamics of p_n . Figure 2(a1) represents a typical realization of our basic model. We observe that the orbit of p_n slowly approaches π^* , abruptly drops to low coverage,

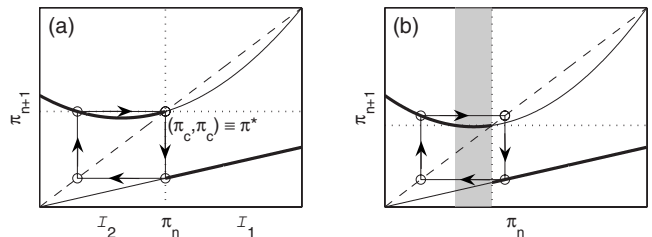


FIG. 4. (a) Schematics of the map when the critical orbit is created. The map is represented by the thick line. The graphical iteration of the critical orbit is represented by a loop. The basin of π^* is the whole domain $[0, 1]$, excluding the points of the period two orbit. (b) Schematics of the map right after the critical orbit is created. The basin of π^* is the open interval in the shaded area, while the basin of the period two orbit consists of all the other points in $[0, 1]$.

and then approaches π^* again; the dynamics continues in a series of such cycles. In contrast, π_n simply stays close to π^* once n is large enough. We thus investigate aspects of the dynamics of p_n which occur due to the fact that the number of individuals N is in fact finite, albeit large.

Since the Lyapunov condition is satisfied for the set of Bernoulli distributions with parameters $\{w^{(i)}; 1 \leq i \leq N\}$, we apply the central limit theorem. We obtain that $p_n = \sum_{i=1}^N x_n^{(i)} / N$ (in the limit of large N) is normally distributed with average π_n and standard deviation $\sqrt{\sum_{i=1}^N \sigma_n^{(i)2} / N^2}$, where $\sigma_n^{(i)}$ is the standard deviation of the distribution of $x_n^{(i)}$ [i.e., $\sigma_n^{(i)2} = w_n^{(i)}(1 - w_n^{(i)})$]. Thus, the dynamics of p_n at large finite N can be described by adding realizations of small amplitude Gaussian noise to the dynamics of π_n . In most of the phase space of π , the noise does not change the qualitative dynamics of the orbit. However, the situation becomes critically different as the noisy orbit asymptotically approaches π^* from \mathcal{I}_2 . Due to the noise (i.e., stochasticity in the mean field due to the finite number of individuals), p_n may jump above (but close to) π^* . According to Eq. (9), in the next iteration the orbit lands (i.e., *drops*) in \mathcal{I}_2 in the vicinity of $s\pi_c$, far from $\pi^* = \pi_c$; see Fig. 2(a1). Then, the orbit is attracted again to π^* and undergoes an apparently periodic dynamics. From the point of view of dynamical systems, this phenomenon may be called a *noise induced border-collision bifurcation* since the presence of arbitrarily small noise transforms an orbit of a piecewise smooth map that is asymptotically approaching π^* into an orbit that is expected to be periodic.

The expected periodicity depends on the number of individuals in the population and can be estimated as follows. At large N , π_n approximates well p_n unless a drop in p_n occurred. For large n and close to π^* , the dynamics of π_n can be approximated as

$$\pi^* - \pi_n \sim \lambda^n; \quad (12)$$

for the parameter values used in Fig. 2(a1), $\lambda \approx 0.840$. Since p_n is normally distributed, we expect a jump of the coverage p_n above π^* when $\pi^* - p_n$ is of the order of the standard deviation of p_n ,

$$\pi^* - p_n \sim \frac{\sqrt{\sum_{i=1}^N \sigma_n^{(i)2} / N}}{N^{1/2}}. \quad (13)$$

For large N , as the orbit of p_n approaches π_c , we expect that the distribution of $w^{(i)}$ becomes asymptotically independent of n just before the drop [25], thus $\sqrt{\sum_{i=1}^N \sigma_n^{(i)2} / N}$ approaches a finite constant. Combining Eqs. (12) and (13) and denoting \tilde{n} as the expected period of the dynamics, we obtain the following scaling result at large finite N :

$$\tilde{n} \sim -\frac{\ln N}{2 \ln \lambda}, \quad (14)$$

which we have successfully verified through numerics (results not shown).

Noise triggers another phenomenon if π_c is less but close enough to Π_0 . Since the right point of the period two orbit

and π^* are close, noise may switch the orbit from the basin of the period two orbit to the basin of π^* , and vice versa. This is expected when the standard deviation of the noise (i.e., stochasticity due to the finite number of individuals) is comparable with the distance between the right point of the period two orbit and π^* . It is straightforward to work out the condition for this switching phenomena analytically. Similar phenomena have been previously studied (e.g., [26–28]) as they are a common occurrence when attractors coexist in noisy dynamical systems. They are called *hopping phenomena* or *noise-induced crises*.

III. MODELS WITH PUBLIC HEALTH INCENTIVES

The basic model predicts that epidemics will not be prevented [1]. Furthermore, severe epidemics are periodically expected. To prevent major influenza epidemics, incentive-based vaccination programs could be offered to the public with the aim of increasing yearly vaccination coverage. Our model allows for the investigation of two major classes of incentive-based programs. The first class uses incentives to correlate vaccination decisions among individuals in the population in one influenza season. The second class uses incentives to correlate vaccination decisions for the same individual over many influenza seasons. Thus, we introduce two additional inductive reasoning games in order to evaluate the potential effects of the following two incentives applied to the basic model:

Incentive 1. If the head of the family (HF) pays to get vaccinated then their family will get vaccinated for free.

Incentive 2. If an individual pays to get vaccinated then that individual will get free vaccinations for a specified number of successive years.

Incentive 1 belongs to the first class of incentive-based vaccination programs, while incentive 2 belongs to the second class of incentive-based vaccination programs. We follow our previous strategy, deriving and analyzing mean-field approximations. Then, we discuss first order deviations from the mean field, exploring first order effects of fluctuations.

A. Public health incentive 1

1. Model description

We consider that the population of N individuals is now divided into F groups representing families. Each family contains C members and one individual in each family acts as it's head. The incentive offers free vaccination to a family if the head of that family paid for his/her vaccination. Only the heads of the families make vaccination decisions and they track the vaccination experience for all of their family members. The sole interest of the HF is to protect their family members from infection, preferably without getting anyone in their family vaccinated. It is very important to note that, as a consequence of this public health program, the vaccination coverage of the HFs equals the population-level vaccination coverage. We specify the model using a set of eight assumptions. The first five assumptions are the same as in the basic model now applied to HFs who are the decision makers, the other three assumptions are as follows.

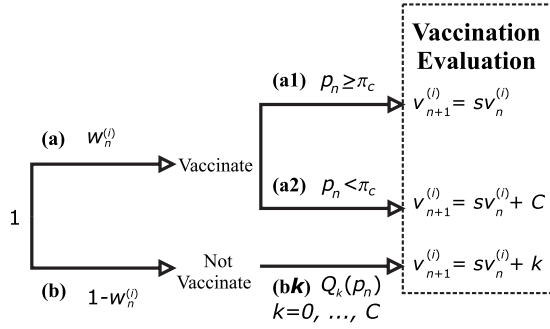


FIG. 5. Schematics illustrating the evaluation tree for each family head. A family head that decides to vaccinate themselves and their family [branch (a)] will judge their choice depending on whether there was an epidemic that season. If the coverage was equal or greater than the critical coverage $p_n \geq \pi_c$ [branch (a1)], they will conclude that their choice to get vaccinated (and have their families vaccinated) that season was unnecessary for preventing infection. Otherwise, if the coverage was lower than the critical coverage $p_n < \pi_c$ [branch (a2)], they will conclude that their choice was beneficial for avoiding infection that season. A family head that decides not to vaccinate themselves and their family [branch (b)] will judge their choice based on how many of their family members were infected. If k members get infected [branch (bk)], they will conclude that vaccination was necessary only for k members of their family.

(6) The infection event of an individual j ($j=1, \dots, N$; i.e., the individual may or may not be a HF) in year n , $z_n^{(j)}$, is the realization of a stochastic variable. (If individual i got infected in season n then $z_n^{(i)}=1$, otherwise $z_n^{(i)}=0$.) If $x_n^{(i)}=1$, then $z_n^{(i)}=0$. If $x_n^{(i)}=0$, then $z_n^{(i)}$ is the realization of a Bernoulli variable with parameter $q(p_n)$, where $p_n = \sum_{j=1}^N x_n^{(j)} / N$ is the coverage achieved that year. That is, if individuals vaccinate, they are fully protected, otherwise they risk infection with probability $q(p_n)$.

(7) $\{x_n^{(i)}; 1 \leq i \leq F\}$ and $\{z_n^{(i)}; 1 \leq i \leq F\}$ determine $v_{n+1}^{(i)}$ as follows (see Fig. 5). We have $C+3$ cases: (a1) if $x_n^{(i)}=1$ and $\pi_c \leq p_n$, then $v_{n+1}^{(i)} = s v_n^{(i)}$; that is, if HF i gets their family (including themselves) vaccinated in season n and no epidemic occurs, then the HF considers that the vaccination was unnecessary; (a2) if $x_n^{(i)}=1$ and $p_n < \pi_c$, then $v_{n+1}^{(i)} = s v_n^{(i)} + C$; which means that if HF i gets their family (including themselves) vaccinated in season n and an epidemic occurs, then the HF considers that the vaccination was necessary for all the family members; (bk) if $x_n^{(i)}=0$ and k family members ($k=0, \dots, C$) have $z_n^{(l,i)}=1$ (where $l=1, \dots, C$ labels the family member), then $v_{n+1}^{(i)} = s v_n^{(i)} + k$; that is, if HF i does not get vaccinated in season n and k members of their family (including themselves) get infected, then the HF adjusts his/her provaccination experience by accounting for the number of their family members that were infected.

(8) The probability that an HF chooses to vaccinate is updated as follows:

$$w_{n+1}^{(i)} = v_{n+1}^{(i)} / [C(1-s^{n+1}) / (1-s)]. \quad (15)$$

That is, an HF's probability to vaccinate (and get their families vaccinated) in the next season is given by the updated

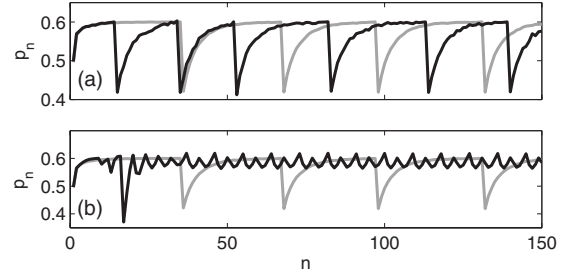


FIG. 6. Coverage dynamics for two public health incentives in a population of $N=10^5$ noncommunicating selfish individuals using a memory parameter $s=0.7$, a critical coverage $\pi_c=0.6$, and a probability $q(0)=0.8$ of getting infected when the coverage $p=0$. (a) The head of the family makes the decision as to whether or not their family vaccinates. The coverage dynamics when the family size is eight ($C=8$) is shown in black; the coverage dynamics when individuals make vaccination decisions independently is shown in gray for comparison. Similar results were obtained for family sizes of two and four. (b) Individuals that pay for one vaccination are given three extra years of vaccination ($y=3$); the coverage dynamics is shown in black and its time average is ameliorated. The coverage dynamics when individuals pay for every year of vaccination is shown in gray for comparison.

cumulative vaccination experience. We have normalized $v_{n+1}^{(i)}$ by $C(1-s^{n+1})/(1-s)$ because this factor is the maximum possible value for $v_{n+1}^{(i)}$ if HF i and their family would have benefited from vaccination in all of the n influenza seasons.

Previously reported numerics of this model are shown in Fig. 6(a); see [1] for details. Apparently, the family incentive increased the frequency of severe epidemics: compared to the basic model the coverage drops below π_c more often. The following analysis aims to provide an understanding of the coverage dynamics shown in Fig. 6(a).

2. Model analysis

Following the HF evaluation tree shown in Fig. 5 we obtain

Branch	Expected population fraction	Average $v^{(i)}$ update
(a1,2)	π_n	$\langle v_{n+1}^{(i)} \rangle = s \langle v_n^{(i)} \rangle + C[1 - \theta(\pi_n - \pi_c)]$
(bk)	$(1 - \pi_n) Q_k(\pi_n)$	$\langle v_{n+1}^{(i)} \rangle = s \langle v_n^{(i)} \rangle + k,$

(16)

where $Q_k(\pi)$ is the probability that k members get infected with influenza in an unvaccinated family when the expected coverage is π . The probability that a single individual gets infected in a season with expected coverage π is $q(\pi)$. Since infection takes place through mass action [29], the probability that k members of a family get infected is binomial

$$Q_k(\pi) = \binom{C}{k} q(\pi)^k [1 - q(\pi)]^{C-k}. \quad (17)$$

Substituting Eq. (17) into (16) and averaging over all branches, we obtain

$$u_{n+1} = su_n + C\pi_n[1 - \theta(\pi_n - \pi_c)] + Cq(\pi_n)(1 - \pi_n). \tag{18}$$

Averaging over Eq. (15) we obtain

$$\pi_{n+1} = u_{n+1}/[C(1 - s^{n+1})/(1 - s)]. \tag{19}$$

The fixed point analysis of the above dynamical system follows similarly to that of the basic model and yields similar results. As in the case of the basic model, $\pi^* = \pi_c$ plays a crucial role in determining the dynamics. The left derivative of the coverage map at π^* (i.e., λ) is the same as in the basic model. The expected periodicity formula is similar to that found for the basic model [Eq. (14)] except that in this case the number of HF ($F=N/C$) determines the periodicity instead of the total number of individuals (N). The expected periodicity can be expressed as a function of the family size C ,

$$\tilde{n}(C) \sim -\frac{\ln F}{2 \ln \lambda}, \tag{20}$$

where for $C=1$ we recover Eq. (14). We now compare the expected periodicity of major epidemics for $C > 1$ to that for $C=1$ (i.e., basic model). We use the same values of s , π_c , and $q(0)$ such that orbits in both models approach π^* from \mathcal{I}_2 . The ratio of the expected periodicities is given by

$$\tilde{n}(C)/\tilde{n}(1) \sim \left(1 - \frac{\ln C}{\ln N}\right) < 1. \tag{21}$$

Hence, the family-based incentive increases the frequency of major influenza epidemics and decreases the time average of the coverage.

B. Public health incentive 2

1. Model description

An individual who pays to participate in the second vaccination program receives an influenza vaccination for the current year and for y successive years. Although the individuals that are enrolled in the program do not make vaccination decisions, they consider the necessity of vaccination every year. At the end of the free vaccination period, they use their evaluations to decide whether to pay for another enrollment. The model uses assumptions (1) through (7) of the basic model. Assumption (8) is modified as follows.

(8) The probability that an individual gets vaccinated is updated as follows:

(i) If the individual decides not to vaccinate in season n (i.e., $x_n^{(i)}=0$), then

$$w_{n+1}^{(i)} = v_{n+1}^{(i)}/[(1 - s^{n+1})/(1 - s)]. \tag{22}$$

(ii) Otherwise, the individual decides to vaccinate in season n (i.e., $x_n^{(i)}=1$). In this case, $w_{n+r}^{(i)}=1$ for $0 < r \leq y$, and, in season $(n+y+1)$,

$$w_{n+y+1}^{(i)} = v_{n+y+1}^{(i)}/[(1 - s^{n+y+1})/(1 - s)]. \tag{23}$$

That is, after vaccinating in season n , and taking advantage of y seasons of free vaccination, the individual resumes

his/her adaptive behavior in season $(n+y+1)$.

Figure 6(b) shows previously reported numerics of the model with the second incentive; see [1] for discussion of the numerics. We note a qualitative change in the orbit of p ; thus, the second incentive can potentially ameliorate epidemics. Our analysis aims to provide an understanding of the coverage dynamics in Fig. 6(b).

2. Model analysis

We now introduce notation to describe the analysis of the model. We use the superscript r to specify the individual-level parameters for individuals that have r vaccinations left. N_n^r ($0 \leq r \leq y$) denotes the expected number of individuals that have r vaccination years left in season n ; $\sum_{r=0}^y N_n^r = N$. The ratio between the number of individuals with r vaccination years that vaccinate in season n and N_n^r is denoted by Φ_n^r . Since we assume that all individuals complete the free vaccination program, $\Phi_n^r=1$ if $r > 0$. The expected population-level coverage can be written as

$$\pi_n = \frac{1}{N} \sum_{r=0}^y N_n^r \Phi_n^r. \tag{24}$$

Given Φ_n^0 we can write a dynamical system for N_n^r

$$N_{n+1}^0 = N_n^1 + (1 - \Phi_n^0)N_n^0,$$

$$N_{n+1}^r = N_n^{r+1} \quad \text{for } 0 < r < y,$$

$$N_{n+1}^y = \Phi_n^0 N_n^0. \tag{25}$$

The number of individuals that will decide to participate in the program the next year (N_{n+1}^0) is given by the number of individuals that finished their vaccination program (N_n^1) and the individuals that did not enroll in the vaccination program in the current year $[(1 - \Phi_n^0)N_n^0]$. The number of individuals with r ($0 < r < y$) years left in the program next year (N_{n+1}^r) is given by the number of individuals with $r+1$ years left in the program in the current year. The number of individuals with y years left in the program in the next year (N_{n+1}^y) is given by the number of individuals that participate in the vaccination program in the current year ($\Phi_n^0 N_n^0$).

The vaccination behavior of the individuals participating in the vaccination program (i.e., $r > 0$) is simple: they vaccinate every year

$$w_n^{r(i)} = 1. \tag{26}$$

However, they still evaluate the necessity of vaccination each year and update their provaccination variable v depending on whether or not there was an influenza epidemic each year

$$v_{n+1}^{r(i)} = sv_n^{r(i)} + 1 - \theta(p_n - \pi_c). \tag{27}$$

The individuals that are not participating in the vaccination program need to decide whether or not to vaccinate each year. The individual-level probabilities for vaccinating get updated as in the basic model

$$w_{n+1}^{0(i)} = v_{n+1}^{0(i)} / [(1 - s^{n+1}) / (1 - s)]. \quad (28)$$

The evaluation of the vaccination decisions is the same as in the basic model; see Fig. 1. Following the evaluation tree in Fig. 1 we obtain

Branch	Expected population fraction	Average $v^{0(i)}$ update
(a1,2)	Φ_n^0	$\langle v_{n+1}^{0(i)} \rangle = s \langle v_n^{0(i)} \rangle + 1 - \theta(\pi_n - \pi_c)$
(b1)	$(1 - \Phi_n^0)q(\pi_n)$	$\langle v_{n+1}^{0(i)} \rangle = s \langle v_n^{0(i)} \rangle + 1$
(b2)	$(1 - \Phi_n^0)[1 - q(\pi_n)]$	$\langle v_{n+1}^{0(i)} \rangle = s \langle v_n^{0(i)} \rangle$

(29)

We now present mean-field equations for our model with the second incentive. Dividing Eqs. (25) by N , we get a set of equations for $\eta_n^r \equiv N_n^r / N$ which are intensive quantities. Furthermore, applying the steps in Sec. II on Eqs. (29), we arrive at the following dynamical system:

$$\eta_{n+1}^0 = \eta_n^1 + (1 - \Phi_n^0)\eta_n^0, \quad (30)$$

$$\eta_{n+1}^r = \eta_n^{r+1} \quad \text{for } 0 < r < y, \quad (31)$$

$$\eta_{n+1}^y = \Phi_n^0 \eta_n^0, \quad (32)$$

$$u_{n+1}^0 = s u_n^0 + (1 - \Phi_n^0)q(\pi_n) + \Phi_n^0[1 - \theta(\pi_n - \pi_c)], \quad (33)$$

$$u_{n+1}^r = s u_n^r + 1 - \theta(\pi_n - \pi_c) \quad \text{for } 0 < r \leq y, \quad (34)$$

$$\Phi_{n+1}^0 = (1 - s)u_{n+1}^0 / (1 - s^{n+1}), \quad (35)$$

$$\Phi_{n+1}^r = 1 \quad \text{for } 0 < r \leq y, \quad (36)$$

where

$$\pi_n \equiv \sum_{r=0}^y \eta_n^r \Phi_n^r, \quad (37)$$

and

$$\sum_{r=0}^y \eta_n^r = 1. \quad (38)$$

The dynamical system simplifies since the equations for u^r ($0 < r \leq y$) and Φ^r ($0 \leq r \leq y$) are decoupled, and η^0 can be eliminated using the constraint (38). We thus obtain

$$\eta_{n+1}^r = \eta_n^{r+1} \quad \text{for } 0 < r < y, \quad (39)$$

$$\eta_{n+1}^y = u_n^0 \left(\frac{1-s}{1-s^n} \right) \left(1 - \sum_{r=1}^y \eta_n^r \right), \quad (40)$$

$$u_{n+1}^0 = s u_n^0 + \left[1 - u_n^0 \left(\frac{1-s}{1-s^n} \right) \right] q(\pi_n) + u_n^0 \left(\frac{1-s}{1-s^n} \right) [1 - \theta(\pi_n - \pi_c)], \quad (41)$$

where

$$\pi_n \equiv F(\eta_n^1, \dots, \eta_n^y, u_n^0) \equiv u_n^0 \left(\frac{1-s}{1-s^n} \right) + \left[1 - u_n^0 \left(\frac{1-s}{1-s^n} \right) \right] \sum_{r=1}^y \eta_n^r. \quad (42)$$

We write the state of the system as $(\eta_n^1, \dots, \eta_n^y, u_n^0)$; the domain of the dynamical system is $\mathcal{D} = [0, 1]^y \times [0, 1/(1-s))$.

Fixed point analysis. Due to discontinuity at $\pi = \pi_c$, we distinguish two complementary domains: $\mathcal{D}_1 = \{(\eta^1, \dots, \eta^y, u^0) | F(\eta^1, \dots, \eta^y, u^0) \geq \pi_c\}$ and $\mathcal{D}_2 = \{(\eta^1, \dots, \eta^y, u^0) | F(\eta^1, \dots, \eta^y, u^0) < \pi_c\}$.

Case 1: \mathcal{D}_1 . In the limit $n \rightarrow \infty$, Eqs. (39)–(41) become the following dynamical system;

$$\eta_{n+1}^r = \eta_n^{r+1} \quad \text{for } 0 < r < y, \quad (43)$$

$$\eta_{n+1}^y = u_n^0 (1-s) \left(1 - \sum_{r=1}^y \eta_n^r \right), \quad (44)$$

$$u_{n+1}^0 = s u_n^0, \quad (45)$$

which has no attractors in \mathcal{D}_1 . However, if we extend the domain to \mathcal{D} , then the system has a fixed point in \mathcal{D}_2 at $(0, \dots, 0)$. This fixed point is an attractor since the Jacobian of the dynamical system evaluated at $(0, \dots, 0)$ has one eigenvalue equal to s ($0 \leq s < 1$) and y eigenvalues equal to 0. The attractor is global since u^0 is decreasing, corresponds to the situation where no individual vaccinates, and fully characterizes the dynamics in \mathcal{D}_1 : orbits in \mathcal{D}_1 will be attracted to $(0, \dots, 0)$ until they land in \mathcal{D}_2 . In \mathcal{D}_2 , the orbit is iterated with a different smooth map.

Case 2: \mathcal{D}_2 . We now obtain the following dynamical system:

$$\eta_{n+1}^r = \eta_n^{r+1} \quad \text{for } 0 < r < y, \quad (46)$$

$$\eta_{n+1}^y = u_n^0 (1-s) \left(1 - \sum_{r=1}^y \eta_n^r \right), \quad (47)$$

$$u_{n+1}^0 = u_n^0 + [1 - u_n^0 (1-s)] q(\pi_n), \quad (48)$$

that has no fixed points in \mathcal{D}_2 . However, if we extend the domain to \mathcal{D} , then the system has two fixed points. The first fixed point is

$$[(y+1)^{-1}, (y+1)^{-1}, \dots, (y+1)^{-1}, (1-s)^{-1}] \quad (49)$$

and corresponds to the situation where everybody vaccinates (i.e., $\pi = 1$). It can be shown straightforwardly that $1 + q(0) \times (1-s)(\pi_c^{-1} - 1)$ and 1 are eigenvalues of the Jacobian of the system at this fixed point; thus, the fixed point is unstable. The second fixed point of the extended system is

$$\rho^* = \{\pi_c / (y+1), \pi_c / (y+1), \dots, \pi_c / (y+1), \pi_c / (1-s) / [1 + y(1 - \pi_c)]\} \quad (50)$$

corresponding to $\pi = \pi_c$. The fixed point may be interpreted as follows. The population of N individuals is divided into two groups. The individuals in the first group never vaccinate

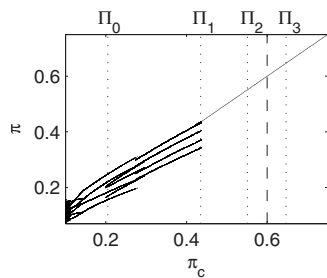


FIG. 7. Bifurcation diagram of the dynamical system given by Eqs. (39)–(41) versus π_c [$y=1$, $s=0.7$ and $q(0)=0.8$]. The dotted lines, from left to right, indicate the approximated position of $\Pi_0 \approx 0.205$ ($k=2$), $\Pi_1 \approx 0.435$ ($k=5$), $\Pi_2 \approx 0.551$ ($k=5$), and $\Pi_3 \approx 0.647$ ($k=6$). The dashed line marks our chosen value of $\pi_c = 0.6$. In this case, incentive 2 with $y=1$ or $y=2$ does not induce a qualitative change in the dynamics of the coverage, while for $y=3$ such a change occurs through a border-collision bifurcation.

while those in the second group always vaccinate. The number of individuals that vaccinate keep the vaccination coverage π at π_c every year and always feel motivated to get vaccinated. This implies that the η^r ($0 < r \leq y$) values of ρ^* are all the same. $\eta^0 = 1 - y\pi_c/(y+1)$ includes both individuals that never vaccinate and individuals that just finished a vaccination program and will participate in a new one. The individuals who do not vaccinate do not get infected because $\pi = \pi_c$ and always benefit from herd immunity. The characteristic equation at ρ^* is analytically intractable. However, it can be established numerically that ρ^* is a potential attractor of the map defined for \mathcal{D}_2 when the domain is extended to \mathcal{D} . For the dynamical system describing the basic model with superimposed public health incentive 2 [Eqs. (39)–(41)], ρ^* may be attracting from \mathcal{D}_2 since ρ^* is on $\partial\mathcal{D}_2$ and the basin of ρ^* intersects \mathcal{D}_2 ; ρ^* is asymptotically approached, yet never reached.

Bifurcation diagram. At high values of π_c , the orbits are attracted to ρ^* which yields $\pi = \pi_c$. With decreasing π_c , a border collision bifurcation occurs. At a particular value of π_c , a critical periodic orbit is created in phase space which with further decreasing π_c turns into a periodic attractor (see Fig. 7). Denoting the k th iterate of the $(y+1)$ -dimensional map given by Eqs. (39)–(41) by $M_y^{[k]}$, the equation of the critical orbit is

$$M_y^{[k]}(\hat{\rho}; \pi_c) = \hat{\rho}. \quad (51)$$

Equation (51) may be understood as follows. For a hypersurface in the parameter space, a point $\hat{\rho} \in \partial\mathcal{D}_2$ (having $\pi = \pi_c$) is reached in a finite number of iterates; $\hat{\rho}$ is first iterated with the smooth map that applies in \mathcal{D}_1 and the orbit lands in \mathcal{D}_2 , then the orbit is evolved with the smooth map that applies in \mathcal{D}_2 for $(k-1)$ iterates until $\hat{\rho}$ is reached again. Equation (51) and $F(\hat{\rho}) = \pi_c$ can be simultaneously solved for $\hat{\rho}$ and Π_y , the value of π_c where the critical orbit is created. For values of π_c in the neighborhood of Π_y ($\pi_c < \Pi_y$), the critical periodic orbit turns into a periodic attractor. It is important to note that, except at $\pi_c = \Pi_y$, the periodic attractor is bounded away from $\partial\mathcal{D}_2$, and is robust to arbitrarily small noise. As an

illustration, we choose $y=1$, $s=0.7$, $\pi_c=0.6$, and $q(0)=0.8$. Figure 7 presents the bifurcation diagram of the map with these parameters and varying π_c . We numerically solve the equations for the critical orbit with $k=5$ and obtain the threshold value of π_c , $\Pi_1 \approx 0.435$ in agreement with Fig. 7 [30]. Figure 7 also shows the positions of Π_0 , Π_2 , and Π_3 relative to the bifurcation diagram of M_1 . We note that Π_y increases with y increasing from 0 to 3 and even further (results not shown).

3. Biological implications of the bifurcation structure

These results provide insights in the potential impact of public health incentives. In particular, the study of these codimension one border collision bifurcations is critical for understanding the effect of public health incentive 2. In practice, it would be unlikely that π_c and/or s are parameters that can be easily changed since the critical vaccination coverage π_c is strongly determined by the transmissibility and virulence of the viral strain, and the memory parameter s is a feature of the individuals. On the other hand, the number of prepaid vaccinations y would be a parameter of the incentive 2 that is easy to change. Since Π_y increases with y , for given s , π_c , and $q(0)$, there exists a threshold value of y (which we denote Y) such that the orbits of the dynamical system with parameters $y=Y-1$, s , π_c , and $q(0)$ are attracted to the corresponding ρ^* (i.e., to $\pi = \pi_c$). Such orbits are very sensitive to noise (i.e., display severe epidemics), while orbits of the dynamical system with parameters $y=Y$, s , π_c , and $q(0)$ go to a period k attractor which is robust to noise (severe epidemics might be prevented). For example, for $s=0.7$, $\pi_c=0.6$, and $q(0)=0.8$, we obtain $Y=3$ and $k=6$; see Fig. 6(b). This phenomenon is very important since it establishes a threshold value for the number of prepaid vaccinations such that incentive 2 makes a qualitative difference in the dynamics of the mean-field coverage. In this case, public health incentive 2 may be very effective in ameliorating influenza epidemics; see Fig. 6(b). For discussion of other possible public health benefits of incentive 2 see Ref. [1].

IV. DISCUSSION AND CONCLUSIONS

In the United States, influenza vaccination is voluntary and the demand for influenza vaccines (70 to 75 million vaccine doses per season) is generally met. The vaccine is very effective in offering protection [31]. However, due to evolution of the virus and waning immunity, the vaccine is good only for one influenza season. In recent years, the vaccination coverage has steadily increased, reaching values between 20% to 25% [32,33]. Although these values have increased they suggest that the current vaccination programs are not very effective. Therefore, every year, up to 25% of the United States population is infected with influenza [10] causing 36 000 deaths [33,34]. One of the national health objectives of the United States is to further increase the vaccination coverage [32,33] which currently is below the Healthy People 2010 objective [35]. Therefore, it is important to understand the vaccination dynamics and to identify incentives that could be used to increase the vaccination cov-

erage and to control influenza epidemics. Our mean-field analyses sheds considerable light on how to increase the yearly influenza vaccination coverage.

Our previous analyses based on simulations [1], have shown that even without the introduction of pandemic strains severe influenza epidemics cannot be prevented unless incentive-based vaccination programs are offered. During the years where epidemics are not prevented, the vaccination coverage tends to increase: individuals that did vaccinate continue to do so in successive years while those that remained susceptible might have become infected and consequently begin to vaccinate. However, once the coverage has reached the critical level and an epidemic is prevented, many vaccinating individuals decide to try to take advantage of herd immunity and avoid infection without getting vaccinated. This results in the reoccurrence of a severe influenza epidemic and the cycle repeats. To understand these dynamics we have analyzed our model by formulating mean-field equations. However, we have found that mean-field dynamics were able to reproduce the simulations at biologically plausible parameter values only when first order effects of the fluctuations were included. Therefore, our analysis shows that, for realistic parametrization, the coverage dynamics of our model is driven by stochasticity. Interestingly, mean-field analysis also shows that for low enough critical vaccination coverages the dynamics undergoes a border-collision bifurcation. At this point, the coverage follows a periodic orbit and the dynamics becomes robust to fluctuations. However, we note that the transition between fluctuation-sensitive and fluctuation-robust dynamics occurs at very small critical vaccination coverages that are biologically implausible for influenza.

We have also studied the potential impact of two public health incentives on our model. The first incentive, which leaves the vaccination decision to the head of the family, reveals similar coverage dynamics as that found for the basic model for the same individual-level and epidemiological parameters. However, compared to the basic model the magnitude of the fluctuations in the coverage dynamics are larger. The reason is that the number of independent decision makers is reduced since family members follow the vaccination decision of the head of family. Larger fluctuations increase the frequency at which the coverage exceeds the critical vaccination coverage and triggers a severe epidemic. Thus, severe epidemics occur more frequently.

The second public health incentive that we have studied is based on prepayment of vaccination. In this case, mean-field analysis of our model explains why this incentive can be used to prevent severe influenza epidemics. Compared to the basic model, the border-collision bifurcation occurs at a larger critical coverage, that increases with the duration of

the prepaid period. When the bifurcation occurs the coverage dynamics fundamentally change from fluctuation sensitive to fluctuation robust. For a value of the critical coverage in agreement with the literature [11–14], we found that an incentive requiring prepayment for 3 years of vaccination would induce a border-collision bifurcation. The resulting fluctuation-robust dynamics yields a vaccination coverage that remains close to the critical level. Thus, this incentive could be used to alleviate the severity of yearly influenza epidemics. At the individual level, these results can be understood from the close relationship between the length of the vaccination program and the time scale of the memory parameter (s ; $s=0.7$ determines a half-life of 1.9 years). Every year a fraction of the population leave the vaccination program and will consider whether or not they should renew their enrollment. Individuals who decide not to renew their participation in the program could become infected, and therefore may reenroll the following year. This produces a high turnover in the number of participants that enroll in the vaccination program every year. Since the time scale of the program is comparable to that of the memory parameter, when they next leave the program, those individuals that were previously infected will benefit from their past influenza experiences before enrollment to decide whether or not they should further reenroll.

By formulating a model that combines human cognition and vaccination behavior with influenza epidemiology and conducting a mean-field analysis we have obtained results that may be useful in guiding public health policy in the event of an influenza pandemic. Our findings show that severe influenza epidemics cannot be prevented unless vaccination programs offer incentives. We found that a public health intervention program that focuses on vaccinating families is likely to increase the frequency of severe epidemics. However, this frequency could be reduced if programs provide, as an incentive to vaccinate, several years of free vaccines to individuals who pay for 1 year of vaccination. Notably, our analysis shows that there exists a threshold for the number of years of vaccination that should be offered. At or above this threshold severe epidemics would be prevented. Therefore, our analysis provides a practical method for identifying how many years of free vaccination the incentive should provide in order to successfully ameliorate influenza epidemics.

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- [1] R. Vardavas, R. Breban, and S. Blower, *PLOS Comput. Biol.* **3**, e85 (2006).
- [2] W. B. Arthur, *Am. Econ. Rev.* **84**, 406 (1994).
- [3] D. Challet, M. Marsili, and Y.-C. Zhang, *Minority Games* (Nova Science Publishers, New York, 2004).
- [4] E. Moro, in *Advances in Condensed Matter and Statistical Physics*, edited by E. Korutcheva and R. Cuerno (Nova, 2004), p. 1.
- [5] D. Challet and Y. C. Zhang, *Physica A* **246**, 407 (1997).
- [6] D. Challet, A. Chessa, M. Marsili, and Y. C. Zhang, *Quant. Finance* **1**, 168 (2001).
- [7] P. Y. Geoffard and T. Philipson, *Am. Econ. Rev.* **87**, 222 (1997).
- [8] C. T. Bauch, A. P. Galvani, and D. J. Earn, *Proc. Natl. Acad. Sci. U.S.A.* **100**, 10564 (2003).
- [9] C. T. Bauch and D. J. Earn, *Proc. Natl. Acad. Sci. U.S.A.* **101**, 13391 (2004).
- [10] T. D. Szucs and D. Muller, *Vaccine* **23**, 5055 (2005).
- [11] N. M. Ferguson, D. A. Cummings, S. Cauchemez, C. Fraser, S. Riley, A. Meeyai, S. Iamsirithaworn, and D. S. Burke, *Nature (London)* **437**, 209 (2005).
- [12] B. S. Cooper, I. M. Longini, W. J. Edmunds, and N. J. Gay, *PLoS Med.* **3**, e212 (2006).
- [13] M. E. Halloran and I. M. Longini, *Science* **311**, 615 (2006).
- [14] C. E. Mills, J. M. Robins, and M. Lipsitch, *Nature (London)* **432**, 904 (2004).
- [15] We do not include the option of treatment against influenza. However, the effects of treatment can be implicitly included in our model by decreasing the effective critical vaccination coverage π_c .
- [16] Our model is designed to describe the case of large N . We do not explicitly model disease transmission as we simply assign a probability of getting infected. However, the results that we present here would not disagree with more elaborate models including disease transmission. In the cases when disease transmission chains scale sublinearly with N (i.e., $p < \pi_c$), we say that epidemics are prevented. When there is the possibility of transmission chains that scale linearly with N (i.e., $p \geq \pi_c$), we say that epidemics will occur. As N increases, the epidemic to nonepidemic transition becomes sharper.
- [17] H. E. Nusse, E. Ott, and J. A. Yorke, *Phys. Rev. E* **49**, 1073 (1994).
- [18] M. di Bernardo, C. J. Budd, and A. R. Champneys, *Phys. Rev. Lett.* **86**, 2553 (2001).
- [19] M. Dutta, H. E. Nusse, E. Ott, J. A. Yorke, and G. Yuan, *Phys. Rev. Lett.* **83**, 4281 (1999).
- [20] J. Laugesen and E. Mosekilde, *Comput. Oper. Res.* **33**, 464 (2006).
- [21] H. E. Nusse and J. A. Yorke, *Physica D* **57**, 39 (1992).
- [22] M. di Bernardo, M. I. Feigin, S. J. Hogan, and M. E. Homer, *Chaos, Solitons Fractals* **10**, 1881 (1999).
- [23] M. di Bernardo, C. J. Budd, and A. R. Champneys, *Physica D* **160**, 222 (2001).
- [24] As the period two orbit is created, its basin jumps from $\{\pi_c, s\pi_c\}$ at $\pi_c = \Pi_0$ to $[0, s(\pi_c - \delta_\epsilon)] \cup [(\pi_c + \delta_\epsilon), 1]$ at $\pi_c = \Pi_0 - \epsilon$, where ϵ and δ_ϵ are strictly positive and arbitrarily small.
- [25] It can be argued that the distribution of w 's over the population approaches $(1 - \pi_c)\delta(w) + \pi_c\delta(1 - w)$. See [1] for discussion of numerical results.
- [26] S. Kraut and U. Feudel, *Phys. Rev. E* **66**, 015207(R) (2002).
- [27] P. Reimann, *J. Stat. Phys.* **82**, 1467 (1996).
- [28] J. C. Sommerer, E. Ott, and C. Grebogi, *Phys. Rev. A* **43**, 1754 (1991).
- [29] H. McCallum, N. Barlow, and J. Hone, *Trends Ecol. Evol.* **16**, 295 (2001).
- [30] Furthermore, for $k=4$, we obtain the threshold value of π_c to be approximately 0.419 in agreement with the bifurcation structure in Fig. 7.
- [31] W. E. Beyer, I. A. de Bruijn, A. M. Palache, R. G. Westendorp, and A. D. Osterhaus, *Arch. Intern. Med.* **159**, 182 (1999).
- [32] S. A. Harper, K. Fukuda, N. J. Cox, and C. B. Bridges, *MMWR Morb Mortal Wkly Rep.* **52**, 1 (2003).
- [33] CDC, *Influenza (flu)*, <http://www.cdc.gov/flu/>
- [34] W. W. Thompson, D. K. Shay, E. Weintraub, L. Brammer, N. Cox, L. J. Anderson, and K. Fukuda, *J. Am. Med. Assoc.* **289**, 179 (2003).
- [35] S. A. Harper, K. Fukuda, T. M. Uyeki, N. J. Cox, and C. B. Bridges, *MMWR Morb Mortal Wkly Rep.* **54**, 1 (2005).