How hard it is scoring a bullseye: Public policies and compliance hesitancy in a heterogeneous population

Giorgio Rampa[*] & Margherita Saraceno[♣]

Abstract

The present dynamic Bayes-conjectural model deals with individual compliance and the effectiveness of policies aimed at reducing welfare losses associated to compliance hesitancy. The model fits with various problems when compliance hesitancy contributes to greater welfare losses, including higher average severity of vaccinable diseases associated to insufficient immunization coverage, too severe average consequences for insurable risks associated to insufficient insurance coverage, excessive tax evasion related to low tax compliance, disproportionate pollution associated with poor compliance with individual environmental practices, etc. The model focuses on how people decide to comply with given *good practices/rules* aimed at reducing welfare losses based on their conjectures, the available information, and their private costs. At each date, people decide and then update their conjectures based on Public Authority's communications about the average welfare loss they could suffer, that in turn depends on the *overall* rate of compliance with the good practices/rules. The resulting conjectural equilibrium compliance rate is unique, however, is compatible with infinitely many different equilibrium subpopulations' priors. These determine the dynamical features of the equilibrium itself. The equilibrium welfare loss is then compared with a policy target-welfare loss to discuss the impact of various policies including nudging and subsidies, information campaigns, mandatory compliance, and investments in new technologies able to reduce welfare losses, either directly or indirectly by making compliance more effective. We conclude that, though the Public Authority can implement effective policies, a specific policy goal cannot be *hit with precision*. In fact, policy-paths could approach non-equilibrium positions surrounding a target equilibrium or could even be "captured" by the basin of attraction of another equilibrium.

JEL classifications: D83, K32, I12, I18.

Keywords: Compliance, Conjectural Equilibria, Dynamical features, Policy effectiveness.

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[The whole paper – that is complete– is going to be rewritten in general terms without recurring to the specific case of vaccination hesitancy]

1. Introduction

The recent outbreaks of vaccine-preventable illnesses - including the COVI19-pandemic, monkeypox as emerged in Europe in 2022, the reemergence of polio cases in 2022 (potentially eradicated in 1979), recurrent Ebola pandemics in African countries (just to mention the most known recent cases)- remind that immunization policies and related compliance attitudes are indeed a present problem. In 2015, the World Health Organization (WHO hereafter) started considering *vaccine hesitancy* (the delay in acceptance or refusal of safe vaccines despite their availability) as an increasingly relevant issue.[1] Actually, even for well-known and established immunization programs, including those for protecting children and elders, compliance rates are very heterogenous depending on the disease, country, target population, and even the flavor of the month. In many European countries where the immunization coverage was high in the past, many parents and people started to not fully complying with immunization programs (Tafuri et al. 2014, Larson et al. 2018). Finally, in 2019 the WHO has declared vaccine hesitancy one of ten threats to global health.[2]

The reasons why people decide to get vaccinated, or to vaccinate their children, or not, are complex. A considerable public health literature tried to identify various relevant determinants (Geoffard and Philipson 1997, Dubé et al. 2014, MacDonald et al. 2015). Economic studies of vaccination behavior formulated static game theory models showing that self-interest would lead to suboptimal vaccination coverage while subsidies and educational policies would reduce suboptimality (Bauch and Earn. 2004, Galvani et al. 2007, Shim et al. 2009 and 2012a). Classical theoretical analyses of vaccinating behavior typically predict a single stable equilibrium for any given choice of parameter values. In their systematic literature review, Chang et al. (2020) show that the Nash-equilibrium classical approach still continue to retain its prominence, though network-based models are attracting research interest (Fukuda, 2015, Bhattacharyya et al. 2019).

Recent contributions investigated behavioral aspects (Siram et al. 2022), shedding light on the role of beliefs and cognitive biases (Chen and Stevens 2017, Becchetti et al. 2021), information, fake news, social media (Jolley and Douglas 2014, Aquino et al. 2017, Dhaliwal et al. 2020), (dis)trust in institutions (Yaqub et al. 2014), altruism, herding and social norms (Shim et al. 2012, Agranov et al. 2021). In addition, policies regulating specific immunization programs – including mandatory immunization (Gulano et al. 2019), vaccine passports (de Figueiredo et al. 2021), moral suasion and information campaigns (Honkanen 1996, Bigaard and Franceschi 2021) and nudging (Reñosa eta al. 2021) – might affect individual behavior significantly (Vrdelja, 2020, Becchetti et al. 2021, Odone et al. 2021). However, clearcut evidence has not always emerged in this sense (Vaz 2020, Charrier et al. 2022, Gravagna et al. 2020).

The present model frames the problem of individual vaccination compliance and related optimal policies in a dynamic Bayesian-learning model with heterogeneous and uninformed agents. Some previous contributions, too, provided dynamic models. Francis (1997) provided a first model with homogeneous decision-makers (both respect to beliefs and costs) and a deterministic epidemic function, finally showing that the optimal-time-of-vaccination

[1] https://www.who.int/news/item/18-08-2015-vaccine-hesitancy-a-growing-challenge-for-immunization-programmes
[2] See https://www.who.int/news/item/12-09-2019-vaccination-european-commission-and-world-health-organization-join-forces-to-promote-the-benefits-of-vaccines

problem must be framed dynamically. Bauch (2005) explained oscillations in the coverage levels by providing a game-theoretic dynamic model. In that model with herding, players must estimate their probability of infection and their decisions are based on disease prevalence and perceived risks. Bhattacharyya and Bauch (2010) prove the relevance of both feedback mechanisms and feed-forward mechanism. Coelho and Codeço (2009) acknowledge that dynamic subjective beliefs about the safety of a given vaccine affect adherence to vaccination programs. In their model, the update process is based on a logarithmically pooling process.

Differently from previous contributions, in the present dynamical model with learning, decision-makers belong to two different subpopulations (difference concerns beliefs and costs to get vaccinated); on each vaccination time they decide to get vaccinated (or not) by comparing their private costs and the expected severity of the disease that they conjecture based on the available information and their subjective beliefs. At the end of each time, decision-makers update their beliefs based on two common pieces of information communicated by the Public Health Authority (PHA, hereafter): the current share of vaccinated people and the current average severity of the disease. The latter is a random function of the vaccine coverage, but the true relation between the severity of the disease and each level of coverage remains unknown. We study both the dynamics of vaccination decisions over timeand the possible conjectural equilibria, i.e., those situations when immunization behavior stops adjusting time by time because the learning process comes to an end, given that conjectures locally guess rightly the true coverage-severity relation.

Results show that that there is a unique conjectural equilibrium vaccination coverage: this, however, is compatible with a two-dimension manifold (roughly, a doubly-infinite set) of conjectural equilibrium priors on the part of the two subpopulations. The exact equilibrium priors determine the dynamical features of the equilibrium itself: its (in)stability, convergence time, presence of oscillations.

Finally, given the socially optimal disease severity level depending on social costs and benefits of severity reduction (Hethcote and Waltman 1973; Fine and Clarkson 1986, Szucs 2000, Donadel et al. 2021), we study how the conjectural equilibrium levels of severity and vaccination coverage might be corrected by using different approaches in order to achieve the optimal disease severity. Thanks to the model, joint with simulations, we assess investments in better vaccines or in new treatments, nudging and subsidies, moral suasion/information campaigns and mandatory vaccinations with respect to their suitability in promoting socially optimal level of disease. Each policy has a different impact both in terms of marginal effectiveness and time/dynamics to achieve the policy goal (time of convergence/oscillation period/stability).

2. The Model

2.1. Hypotheses

Assumption 1 (Timing).

Time is discrete. At the beginning of date 0, a vaccinable disease outbreak emerges. At each date $t \geq 0$ people must decide to get vaccinated or not. In fact, immunization is temporary, and at the beginning of each date each

individual must decide once again. This assumption could be relaxed in future research. Individuals decide to get vaccinated by comparing the expected consequences (severity) of the disease and their own cost of being vaccinated.

Assumption 2 (True relation between average severity and immunization coverage)

Define μ_t as the average severity of the disease (that is, the average "cost" of its consequences on the population of infected subjects), as can be observed at date *t*. We assume that there exists a *true* random relationship between μ_t and the immunization coverage π_t such that $\mu_t \approx N(\alpha - \beta\pi_t; 1)$, $\alpha, \beta \in (0,1]$. The parameters α and β are unknown to individuals. Randomness is due to different factors: individual genetic predisposition, exposure, precautions, etc. The normality assumption seems to be quite reasonable; the linearity of the mean and the unitary variance are chosen for simplicity.

The parameter α measures the severity of the disease with a null vaccination coverage. We are assuming that vaccines are, at worse, completely ineffective (β approaching 0), but not an aggravating factor.

Assumption 3 (Information)

When the outbreak emerges at the beginning of $t = 0$, the PHA communicates the fact to the population, clarifying that the vaccination can help to reduce the severity. At the end of each date *t*, the PHA observes μ_t and π_t and communicates them to the population.

Assumption 4 (Subpopulations)

The overall population is composed of two components characterized by specific features (described in the next Assumptions 5 and 6), subpopulation *A* and subpopulation *B*. The weight of sub-population *A* is γ and the weight of sub-population *B* is $1-\gamma$.

Assumption 5 (Individual costs)

In case of vaccination, individuals bear an individual "cost" to be vaccinated. This does not only depend on the possible economic cost of immunization, but may also depend on individual risk aversion, fear of side effects of vaccination, non-monetary costs, etc. This individual characteristic is a random variable distributed among each population according to a Uniform Distribution of support $[0, \theta_i]$, $i = A, B, 1 \leq \theta_A \leq \theta_B$.

Assumption 6 (Conjectures)

At $t = 0$, when the outbreak emerges, given that the true relationship between μ_t and π_t is unknown, people get an idea on that relation. In particular, they correctly assume that the severity of the disease is a random variable $N(\mu_t^e = \alpha - \beta\pi_t, 1)$. However, given that the couple of *structural* parameters (α, β) are unknown, individuals have a subjective prior on those parameters. We assume that all individuals within one of the two subpopulations share the same prior on them, and priors differ between the two subpopulations. In particular, the prior on (α, β)

for subpopulation *i* is a normal bivariate, and the mean and precision parameters of this subjective distribution at date 0 are, respectively, the following vector $\mathbf{z}_{i,0}$ and symmetric "precision"[3] matrix $\mathbf{H}_{i,0}$:

$$\mathbf{z}_{i,0} = \begin{bmatrix} \alpha_{i,0} \\ \beta_{i,0} \end{bmatrix}, \quad \mathbf{H}_{i,0} = \begin{bmatrix} \eta_{i,\alpha,0} & \eta_{i,\alpha\beta,0} \\ \eta_{i,\alpha\beta,0} & \eta_{i,\beta,0} \end{bmatrix}, i = A, B.$$

$\alpha_{i,0}$ and $\beta_{i,0}$ are the initial parameters conjectured by subpopulation *i* on α and β, respectively; $\eta_{i,\alpha,0}$, and $\eta_{i,\beta,0}$ are positive, whereas we assume $\eta_{i,\alpha\beta,0} = 0$ because people have no reason to hypothesize any specific initial value for co-variances.

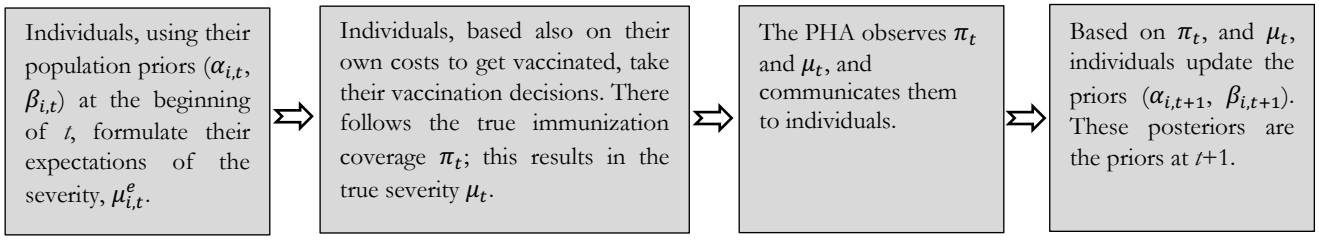
Henceforth, we use the term "*hyperparameters*" to indicate the quantities $\alpha_{i,t}$, $\beta_{i,t}$ and $\eta_{i,\alpha(\beta),t}$, as opposed to the "*structural parameters*" α and β. In particular, $\alpha_{i,t}$, $\beta_{i,t}$ are the *mean* hyperparameters, while $\eta_{i,\alpha(\beta),t}$ are the *precision* hyperparameters.

Since at the beginning of date *t,* individuals do not know the actual π_t when formulating their expectation μ_t^e, they provisionally assume that the immunization coverage remains constant from *t*-1 to *t*. Therefore, at each date they expect $\mu_{i,t}^e = \alpha_{i,t} - \beta_{i,t}\pi_{t-1}$.

Assumption 7 (Bayesian learning: Timing)

At the end of each date *t*, once the immunization decisions have been taken, people learn μ_t and π_t from the PHA. At that point, each subpopulation formulates a posterior along a Bayesian learning process, by updating the prior based on μ_t and π_t. The posterior at date *t* becomes the prior for date *t*+1: see Figure 1.

FIGURE 1- Timing of the model on date *t*



2.2. Decisions and learning at *t*

The individuals of each subpopulation decide to get vaccinated at the beginning of each date *t* if and only if the expected severity of the disease μ_t^e is greater than the cost of being vaccinated, that is, if $\alpha_{i,t} - \beta_{i,t}\pi_{t-1} > \theta_i$. Given Assumption 5, the share of each subpopulation who decides to get immunized is[4]:

$$\pi_{i,t} = \frac{\alpha_{i,t} - \beta_{i,t}\pi_{t-1}}{\theta_i}, \; i=A, B \qquad (1)$$

[3] *Precision (or robustness)* of the subjective prior is a term of Bayesian statistics; it is related to the inverse of the variance and indeed the matrices $\mathbf{H}_{i,0}$ are the inverse of the variance-covariance matrices conjectured by the populations.

[4] Although decisions are based on expected values, recall that θ_i with *i =A, B*, embodies subpopulations' risk aversion as stated in Assumption 5.

The higher the maximum subjective cost of each subpopulation θ_i, the lower the subpopulation propensity to get vaccinated for any given perceived severity of the disease.

The overall immunization coverage at date *t* is:

$$\pi_t = \frac{\gamma}{\theta_A}\left(\alpha_{A,t} - \beta_{A,t}\pi_{t-1}\right) + \frac{1-\gamma}{\theta_B}\left(\alpha_{B,t} - \beta_{B,t}\pi_{t-1}\right) \tag{2}$$

Note that at $t = 0$ we have $\pi_0 = \frac{\gamma}{\theta_A}\alpha_{A,0} + \frac{1-\gamma}{\theta_B}\alpha_{B,0}$, since at the outbreak of the disease nobody is yet vaccinated.

Given the immunization coverage π_t and its *true* relation with the severity, the observed mean severity at *t* is:

$$\mu_t = \alpha - \beta\left[\frac{\gamma}{\theta_A}\left(\alpha_{A,t} - \beta_{A,t}\pi_{t-1}\right) + \frac{1-\gamma}{\theta_B}\left(\alpha_{B,t} - \beta_{B,t}\pi_{t-1}\right)\right] \tag{3}$$

At $t = 0$ we have $\mu_0 = \alpha - \beta\left(\frac{\gamma}{\theta_A}\alpha_{A,0} + \frac{1-\gamma}{\theta_B}\alpha_{B,0}\right)$.

By inspecting (2) and (3), we conclude the following:

RESULT 1 (a) *The overall immunization coverage at a given date* $t \geq 0$ *depends on both structural parameters and mean hyper-parameters. In particular, the immunization coverage at a given date t positively depends on the subpopulation hyperparameters* $\alpha_{i,t}, i = A, B$ *(the expected severities under zero immunization, as conjectured by both subpopulations), and negatively depends on hyperparameters* $\beta_{i,t}, i = A, B$ *(the effectiveness of vaccine conjectured by both subpopulations). The weight of the subpopulations' hyperparameters in determining the immunization rate depends on the relative weight of the two subpopulations.*

(b) *The maximum individual cost of vaccination* θ_i *negatively affects the subpopulation and the overall coverage.*

Now, once vaccination decisions are taken and π_t and μ_t are observed and then communicated by the PHA, the two subpopulations update their priors, finally formulating their posteriors along the Bayesian lines defined in Assumptions 6 and 7.

In particular, define the vector $\mathbf{x'}_t = [1 \quad -\pi_t]$, that is, the vector of the "regressors" of the equation $\mu^e_{i,t} = \alpha_{i,t} - \beta_{i,t}\pi_t$ that people would conjecture after being informed of π_t, given their previous prior. Under our assumptions, the updated hyper-parameters are as follows (see De Groot 1970, chapter 11):

$$\mathbf{z}_{i,t+1} = \left[\mathbf{H}_{i,t} + \mathbf{x}_t\mathbf{x'}_t\right]^{-1}\left[\mathbf{H}_{i,t}\mathbf{z}_{i,t} + \mathbf{x}_t\mu_t\right] \qquad \text{and} \qquad \mathbf{H}_{i,t+1} = \mathbf{H}_{i,t} + \mathbf{x}_t\mathbf{x}'_t,\ i = I, H \tag{4a}$$

After some simple passages, the first expression becomes:

$$\mathbf{z}_{i,t+1} = \mathbf{z}_{i,t} + \left[\mathbf{H}_{i,t} + \mathbf{x}_t\mathbf{x'}_t\right]^{-1}\left[\mathbf{x}_t\left(\mu_t - \mathbf{x'}_t\mathbf{z}_{i,t}\right)\right] \tag{4b}$$

The final parenthesis contains the *forecasting error* for subpopulation *i,* $e_{i,t} \equiv \left(\mu_t - \mathbf{x'}_t\mathbf{z}_{i,t}\right)$, given that μ_t is the *true* average severity at the end of time *t*, communicated by the PHA together with the actual π_t, while $\mathbf{x'}_t\mathbf{z}_{i,t}$ is the one that would be computed by population *i* on the basis of its prior and of the actual π_t.

Looking at (4a and 4b), we deduce the following:

RESULT 2 *The updated mean hyperparameter vector* $\mathbf{z}_{i,t+1}$ *is equal to the previous one, plus the term* $\left[\mathbf{x}_t\left(\mu_t - \mathbf{x'}_t\mathbf{z}_{i,t}\right)\right]$*, containing the forecast error* $e_{i,t}$*, "deflated" by the inverse of the precision matrix. The precision matrix* $\mathbf{H}_{i,t}$ *"grows" in time (in the sense of the positive-definite-matrix ordering), so its inverse "decreases" in time.*

Observe that $\mathbf{z}_{i,t+1} = \mathbf{z}_{i,t}$ *if and only if* the individuals of subpopulation *i* learns from the PHA that $\mu_t = \mathbf{x'}_t\mathbf{z}_{i,t}$, that is, if the forecast $e_{i,t}$ error is null. On the other side, because the precision matrix grows over time, the strength of the correction mechanism reduces in time. Therefore, the updating process becomes increasingly slower.

Now, by exploiting expression (3) and the definition of the forecasting errors we derive some properties of the forecasting errors, that are so important for the recursive adjustment of conjectures. Observe that we measure the errors as true severity *minus* expected severity. Using (3) and (4b), the forecasting error of each population can be written as follows:

$$e_{i,t} = \alpha - \beta\left[\frac{\gamma}{\theta_A}\left(\alpha_{A,t} - \beta_{A,t}\pi_{t-1}\right) + \frac{1-\gamma}{\theta_B}\left(\alpha_{B,t} - \beta_{B,t}\pi_{t-1}\right)\right] - \alpha_{i,t} + \beta_{i,t}\pi_t \qquad (5)$$

It is then easy to verify that the derivatives of the errors with respect to the four mean hyperparameters at *t* are:

$$\frac{\partial e_{A,t}}{\partial \alpha_{A,t}} = -1 - \frac{\beta\gamma}{\theta_A}; \qquad \frac{\partial e_{A,t}}{\partial \beta_{A,t}} = \frac{\beta\gamma}{\theta_A}\pi_{t-1} + \pi_t; \qquad \frac{\partial e_{A,t}}{\partial \alpha_{B,t}} = -\beta\frac{1-\gamma}{\theta_B}; \quad \frac{\partial e_{A,t}}{\partial \beta_{B,t}} = \beta\frac{1-\gamma}{\theta_B}\pi_{t-1} \qquad (6a)$$

$$\frac{\partial e_{B,t}}{\partial \alpha_{B,t}} = -1 - \frac{\beta(1-\gamma)}{\theta_B}; \quad \frac{\partial e_{B,t}}{\partial \beta_{B,t}} = \frac{\beta(1-\gamma)}{\theta_B}\pi_{t-1} + \pi_t; \quad \frac{\partial e_{B,t}}{\partial \alpha_{A,t}} = -\beta\frac{\gamma}{\theta_A}; \qquad \frac{\partial e_{B,t}}{\partial \beta_{A,t}} = \beta\frac{\gamma}{\theta_A}\pi_{t-1} \qquad (6b)$$

These derivatives depend only on the structural parameter of the model (given the existing vaccination coverage). That is, they depend on the true parameters of the relationship between the vaccination coverage and the severity of the disease, on the cost distributions within the two subpopulations, and on the population shares. The derivative of $i's$ error with respect to $j's$ hyperparameters is affected of course by population $j's$ share ($i, j = A, B$). In particular, the forecasting error of each subpopulation depends negatively on the intercept of both populations' conjectures, and positively on the conjectured slopes, since both a higher α_i and a lower β_i increase the expected severity. In addition, the positive or negative effects on the errors become stronger (in absolute value) if the cost distributions in the two populations decrease: this in reasonable, since a lower θ_i accentuates the oscillations of the vaccination coverage of the corresponding subpopulation; this, in turn, strengthens the oscillations of the true average severity, thus reinforcing the movements of the forecasting errors. Finally, the positive effect of the β's on the errors is obviously accentuated at higher vaccination coverages.

Coupling the above arguments with Result 2 leads us to the following

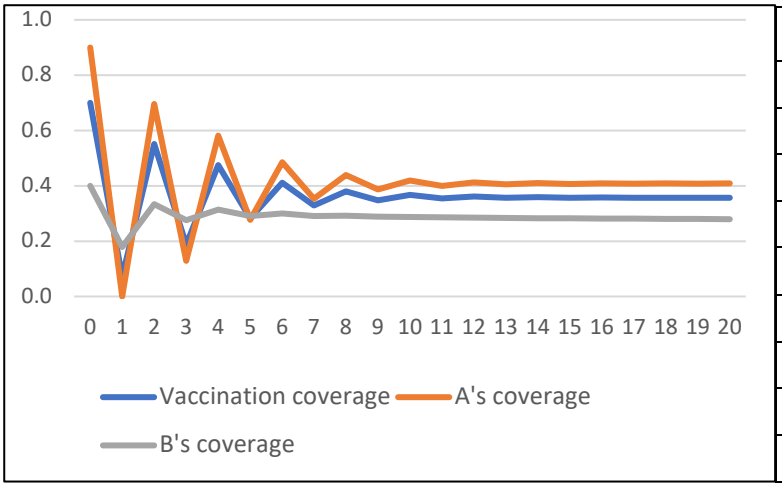
RESULT 3 *The updating process, while abating in time due to the increasing precisions of conjectures, is driven significantly by the populations' forecasting errors. These errors, in turn, are obviously induced by the conjectured hyperparameters; however, the marginal relationships between errors and hyperparameters depend only on the structural parameters. Said differently, the recursive dynamical interplay between conjectures, coverage decisions, forecasting errors and updating is strongly shaped by the structural parameters of the model.*

Some general implications can be derived from Results 1-3. In particular, Result (1a) sheds light on the (positive and negative) *external effects* that are related to vaccination decisions: when a subpopulation is reluctant to get vaccinated the whole population suffers from a lower coverage level and a higher severity (negative externality), while when a subpopulation is prompt to get vaccinated the whole population benefits from a higher coverage and a lower severity (positive externality).

Furthermore, by observing the properties described in Results 1-3, an *overshooting* effect can be detected: at the first dates, when adjustments are large since the learning has a relevant impact, the vaccination coverage temporarily overreacts to changes in observed levels of severity/coverage. Indeed, from expression (4b) we see that $\mathbf{z}_{i,t+1} - \mathbf{z}_{i,t}$ moves in the same direction as $\mu_t - \mathbf{x}'_t\mathbf{z}_{i,t}$, meaning that a population's hyperparameters and hence its vaccination coverage –given expression (1)– negatively depend on their past values. This aspect is indeed common to most "adaptive" expectations models.

Figure 2 illustrates a possible overshooting effect for a given set of parameters. The overshooting effect is greater at the initial dates, and it gets smaller in time due to the slowing down of the adjustment process.

FIGURE 2 - Vaccination coverage dynamics: an example.



Structural parameters			
α	0.8		
β	1.1		
γ	0.6		
θ_A	1	θ_B	1.5
Hyperparameters			
$\alpha_{A,0}$	0.9	$\alpha_{B,0}$	0.6
$\beta_{A,0}$	1.2	$\beta_{B,0}$	0.2
$\eta_{A,\alpha,0}$	1	$\eta_{B,\alpha,0}$	2
$\eta_{A,\beta,0}$	1	$\eta_{B,\beta,0}$	2

The simulation is carried out for the parameters above.

In Appendix 1 we graphically illustrate some simulations in order to deepen how different hyperparameters affect subpopulations' vaccination decision and the overall dynamics of vaccination coverage. Although many different scenarios could be compared, the main implication derived from the proposed simulations is that hyperparameters significantly affect the dynamic towards some stable coverage rates. Indeed, they determine both the *short-run* populations' behavior -together with the related vaccination coverage rates- and the time required for those variables to converge to a stable position. Simulations also show that the dynamic is characterized by (*a*) overshooting effects associated to the adaptive expectations, (*b*) external effects related to the heterogeneity of subpopulations, and (*c*) a herd effect induced by learning.

2.3. Conjectural equilibria

Now, we introduce the concept of conjectural equilibria in order to study whether and how the updating process can reach a position of rest, so that the vaccination coverage rates stabilize to certain levels.

We define a *conjectural equilibrium* (*CE*, hereafter) as a configuration of subpopulations' hyperparameter such that, if the *learning dynamical system* is set in one of these positions, it stays there except when a further shock perturbs the system. In such situations, people (of both subpopulations) have no reason to modify their beliefs and the related vaccination decisions.[5]

As observed in Section 2.2, vaccination coverage depends on the hyperparameters of subpopulations' conjectures. Hence, if these parameters remain unchanged from date t to date t+1, both the vaccination coverage and the disease severity remain unchanged. On the other side, if the observed severity remains unchanged, the populations' vaccination decisions (consequently, the vaccination coverage) remain unchanged. This means that a *stationary state of the dynamical learning process has been reached*, and no further change will take place.

Now, we can state precisely the conditions of a conjectural equilibrium. Since the *CE* definition implies that equilibrium variables remain constant in time, we omit the time subscripts. As it is clear from the definition above and from equations (2) and (4b), these conditions require that (*i*) the observation of the true μ on the part of people of both subpopulations confirms their expectations, and (*ii*) the actual vaccination coverage π is the weighed sum of the populations' decided coverages. Hence, the *CE* condition can be represented by the following system:

$$\begin{cases} \alpha_A - \beta_A \pi = \alpha - \beta\pi & (7) \\ \alpha_B - \beta_B \pi = \alpha - \beta\pi & (8) \\ \pi = \beta\left[\frac{\gamma}{\theta_A}(\alpha_A - \beta_A\pi) + \frac{1-\gamma}{\theta_B}(\alpha_B - \beta_B\pi)\right] & (9) \end{cases}$$

By replacing (7) and (8) in (9), we obtain the *CE* vaccination coverage:

$$\pi_{CE} = \frac{\left(\frac{\gamma}{\theta_A} + \frac{1-\gamma}{\theta_B}\right)\alpha}{1 + \left(\frac{\gamma}{\theta_A} + \frac{1-\gamma}{\theta_B}\right)\beta} \tag{10}$$

This corresponds to the following *CE* average severity:

$$\mu_{CE} = \alpha - \beta \frac{\left(\frac{\gamma}{\theta_A} + \frac{1-\gamma}{\theta_B}\right)}{1 + \left(\frac{\gamma}{\theta_A} + \frac{1-\gamma}{\theta_B}\right)\beta}\alpha \tag{11}$$

[5] The notion of "conjectural equilibrium" was first introduced by Hahn (1977). Fudenberg-Levine (1993) and Dekel-Fudenberg-Levine (2004) reworded it in terms of *self-confirming equilibrium*.

The above passages enable us to state the following:

RESULT 4 *The precise location of* π_{CE} *and* μ_{CE} *in the final stationary state does not depend on the subpopulations' conjectures (priors). There is a unique equilibrium vaccination coverage-severity couple depending only the structural parameters.*

In spite of this uniqueness result, things are quite different as regards the configuration of the *CE conjectures* of the two subpopulations. Indeed, by replacing (10) in conditions (7) and (8), we are able to deduce that the *CE* mean hyperparameters of each subpopulation are characterized by the following relations:

$$\beta_{CE,i} = \frac{\alpha_{CE,i} - \alpha}{\pi_{CE}} + \beta \qquad i = A, B \tag{12}$$

Hence, we have:

RESULT 5 *There exist a doubly-infinite set of quadruplets of hyperparameters that are compatible with CE. More technically, we have a two-dimensional CE manifold in the four-dimensional space of the subpopulations' mean hyperparameters.*

This infinity is easily interpreted from the geometrical point of view. For each subpopulation, it consists of all passible *intersections* between the true line, $\mu = \alpha - \beta\pi$, and the conjectured one, $\mu = \alpha_{CE,i} - \beta_{CE,i}\pi$: given any freely chosen α_i, the hyperparameter β_i must satisfy (12), or vice versa.

The infinity of possible *CE* situations implies that the two subpopulations end up, in equilibrium, with different interpretations of the steady state: even if they correctly guess the *local* relationship between vaccination coverage and disease severity, they envisage different relationships between the two variables. It is even possible that one subpopulation conjectures a positive relationship, meaning that they believe the vaccination is dangerous for health. Said differently: given an exogenous change in the overall vaccination coverage, the two subpopulations predict different (possibly, quite different) changes in the average severity of the disease. This observation will turn out to be important when discussing (in Section 3) the effects of possible public policies, interpreted as exogenous shocks with respect to a pre-existing conjectural equilibrium.

2.4. Dynamic stability of *CE*s

In general, it is highly improbable that the populations' initial priors obey the *CE* conditions (7-9) and people typically need to update them due to their forecasting errors, so inducing a dynamical interplay among the relevant variables. This leads us to ask whether, starting from a non-equilibrium position, the system will converge towards an equilibrium or, otherwise, it will diverge from it, making that *CE* non-interesting from the practical point of view. This is a *dynamic stability problem*.

In order to tackle this problem, observe that the dynamical system whose stability properties we wish to examine is defined by equations (4b) and (2), coupled with the formulation (5) of the forecasting errors: therefore, five

variables are involved. Defining the vector $\mathbf{y}_t \equiv (\alpha_{A,t}, \beta_{A,t}, \alpha_{B,t}, \beta_{B,t}, \pi_t)$, we have the following formal definition of our discrete-time dynamical system:

$$\mathbf{y}_t = F(\mathbf{y}_{t-1}) \tag{13}$$

The system of equations (13), besides being five-dimensional, is *non-linear*; hence, analyzing its global properties is quite difficult. Therefore, we propose to study the *local* stability of *CE*s. As it is well known[6], this requires to compute the Jacobian matrix of system (13) evaluated at a *CE*, call it $\mathbf{J} = [J_{i,j}] \;\; i,j = 1,\dots,5$, and to study the eigenvalues of this matrix.[7] The characteristics of the eigenvalues determine how the variables move after a tiny displacement from the *CE* at which the Jacobian and its eigenvalues are evaluated (Appendix 2 provides a brief guide about that).

To start with, we need to stress a caveat. We know from expression (4b), and related comments, that the adjustment process of conjectures/decisions/outcomes slows down in time, due to the increasing subjective precision hyperparameters of people's conjectures: this implies that the initial forces at work after a displacement from a *CE* tend to weaken in time. Our strategy, then, is to evaluate the local dynamical properties of a *CE* keeping precisions *constant* at their initial values: in other terms, we evaluate the *potential* trajectories of variables in the very first periods after a shock, well knowing that these trajectories will tend to relax in the subsequent periods.

Now, we are able to prove the following result (Proof in Appendix 2, where some symbols are defined):

RESULT 6

(*a*) *The Jacobian of system* (13)*, computed at a CE, is similar to the following matrix; hence, the two matrices have the same eigenvalues:*

$$\begin{bmatrix} 1 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 \\ J_{3,1} & J_{3,2} & -\delta_A(t)\eta_{A,\beta}\left(\frac{\theta_A+\beta\gamma}{\theta_A}\right)+1+\delta_A(t)\eta_{A,\alpha}\left(\frac{\theta_A+\beta\gamma}{\theta_A}\right)\pi^2 & -\delta_A(t)\eta_{A,\beta}\beta\frac{1-\gamma}{\theta_B}+\delta_A(t)\eta_{A,\alpha}\beta\frac{1-\gamma}{\theta_B}\pi^2 & \delta_A(t)\eta_{A,\alpha}\beta\left(\frac{\gamma}{\theta_A}\beta_A+\frac{1-\gamma}{\theta_B}\beta_B+\beta_A\right)\pi \\ J_{4,1} & J_{4,2} & -\delta_B(t)\eta_{B,\alpha}\frac{\eta_{A,\beta}}{\eta_{A,\alpha}}\beta\frac{\gamma}{\theta_A}+\delta_B(t)\eta_{B,\alpha}\beta\frac{\gamma}{\theta_A}\pi^2 & -\delta_B(t)\eta_{B,\alpha}\frac{\eta_{A,\beta}}{\eta_{A,\alpha}}\left(\frac{\theta_B+\beta(1-\gamma)}{\theta_B}\right)+1+\delta_B(t)\eta_{B,\alpha}\left(\frac{\theta_B+\beta(1-\gamma)}{\theta_B}\right)\pi^2 & \delta_B(t)\eta_{B,\alpha}\left(\frac{\gamma}{\theta_A}\beta_A+\frac{1-\gamma}{\theta_B}\beta_B+\beta_B\right)\pi \\ J_{5,1} & J_{5,2} & \frac{\eta_{A,\beta}}{\eta_{A,\alpha}\pi}\frac{\gamma}{\theta_A}\Sigma_{j=1}^4 J_{j,1}-\frac{\gamma}{\theta_A}\pi\Sigma_{j=1}^4 J_{j,2} & \frac{\eta_{A,\beta}}{\eta_{A,\alpha}\pi}\frac{1-\gamma}{\theta_B}\Sigma_{j=1}^4 J_{j,3}-\frac{1-\gamma}{\theta_B}\pi\Sigma_{j=1}^4 J_{j,4} & \left(\beta_B\frac{1-\gamma}{\theta_B}+\beta_A\frac{\gamma}{\theta_A}\right) \end{bmatrix}$$

(*b*) *The above matrix is clearly decomposable into the "north-west"* 2x2 *block and the "south-east"* 3x3 *block. Therefore, the eigenvalues of the matrix are the two ones of the north-west block, the* 2x2 *identity matrix, plus the three ones of the south-east block. This explains why the six elements of the south-west block are not reported explicitly, being irrelevant for our purposes.*

(*c*) *It follows that two eigenvalues of the Jacobian matrix of system* (13) *are equal to one, and the remaining three eigenvalues are those of the south-east block of the above matrix.*

A first comment is in order about Result 6. The presence of two unitary eigenvalues means that, if the system is slightly displaced from a *CE* in certain particular directions[8], the variables will *not* converge to (nor diverge from) the previous equilibrium: they will simply remain in the new position. This is nothing else than the implication of

[6] See, e.g., Gandolfo (1980).

[7] The Jacobian matrix contains the partial derivatives of each variable at date *t* with respect to all variables at date *t*–1. In our case, it is a 5x5 matrix.

[8] See footnote 9.

the presence of a double *infinity* of *CE*s: if variables are moved from a *CE* to another *CE*, there will not be any further change. In other terms, *CE*s are neither completely stable nor completely unstable.[9]

There exists a further implication of the infinity of *CE*s. If stationary states were *isolated*, each of them would be surrounded by a full set of points such that any trajectory starting from these points will converge to, or diverge from, the stationary state (the so-called "basin of attraction or repulsion" of the stationary state). In the presence of a *continuum* of stationary states, instead, each of them is endowed with its own basin of attraction/repulsion[10]. It follows that, if the system is displaced from a CE in a *generic* direction (that is, neither exactly along the *CE* manifold, nor exactly along the eigenvector associated with an eigenvalue)[11], even if we are in a zone of stable *CE*s the variables will converge towards *a different CE*.

Now, in order to ascertain whether a *CE*, if perturbed, will display stability or instability, we need to evaluate the remaining three eigenvalues of the Jacobian matrix. A general result for the analytical form of these eigenvalues is very difficult, and its interpretation would even more difficult, given the complexity of the terms appearing in the matrix of Result 6*a*. Therefore, in Appendix 3 we offer a series of *numerical computations* for different relevant parameters, reporting the periods of oscillation and the stability features associated with the resulting eigenvalues.

Despite the difficulty of finding a general analytical result for the last three eigenvalues of the Jacobian matrix of system (13), we can nevertheless put forward some hints. A glance at the matrix reported in Result (6*a*), coupled with the Jacobian matrix $\mathbf{J_4}$ written and Appendix 2, suggests that: (*i*) and increase in the equilibrium level of π (necessarily accompanied by a decrease in the equilibrium level of μ) *tends to increase the absolute value* of the elements of the matrix appearing in Result 6*a*; (*ii*) the same tendency is induced by an increase in β, that, however, depresses π_{CE}, as shown by expression (10); (*iii*) a similar effect is produced by increases in the initial precision hyperparameters relating to the basic severity, $\eta_{i,\alpha}$, or by reductions in those relating to the vaccine efficacy, $\eta_{i,\beta}$; (*iv*) supposing, as we will do in Section 2.6, that population *A* is more confident in the efficacy of the vaccine ($\beta_A > \beta_B$), and that it bears a lower cost to be vaccinated ($\theta_A < \theta_B$), an increase of *A*'s weigh γ tends, again, to increase the absolute value of the elements of the matrix of Result (6*a*). These changes, then, tend to push in the direction of an increase in the absolute value of the last three eigenvalues, which implies a *slower convergence* (or an *emphasized divergence*).

We can add a single more precise result related to the last argument. Looking at expression (*A*3) of Appendix 2, we can notice the any change/intervention that, starting from an existing *CE*, reduces the true severity μ causes a *negative* forecasting error on the part of subpopulations. Therefore, the learning mechanism (*A*3) forces the two subpopulations to *reduce* both α_i and β_i: this gives rise to a *greater difference in steepness* between the conjectured and the true relation π - μ (when the latter is greater than the former, that is, in "normal" cases: see Tables 1-4 of

[9] This is a case of "Lyapunov stability".
[10] This means that there exists a continuum of basins of attraction/repulsion, one for each of the *CE*s.
[11] It should be noticed that each of these five particular directions of perturbation (the *CE* manifold, and the four eigenvector directions) is very "tiny" in the space of the five state variables: technically, each of them has "zero measure" in the five-dimensional space. Instead, the set of generic directions (that is, all combinations of those five particular directions) has probability one in that space.

Appendix 3): hence, there results in lower stability or greater instability, as usual in many adaptive expectations models. This suggests us to put forward the following

RESULT 7

Every change/intervention pushing the true severity μ *down reduces stability or increase instability*

Finally, a decrease of the θ_is generates, of course, a *higher* variability in the vaccination decisions, expressed by the shares $\pi_{i,t}$ of vaccinated people (see expression 1); this, again, reduces stability or increases instability.

All the above observations might be useful in Section 3.2, where we discuss the dynamical effects of policies.

Concerning the numerical computations reported in Appendix 3, they allow us to put forward some general conclusions about how changes in the parameters affect the *CE* location and the out-of-equilibrium dynamics. Briefly, (a) *increases in* α tend reduce time of convergence (TOC, hereafter)/increase time of divergence (negative TOC) while both π_{CE} and μ_{CE} increase, (b) *increases in* β tend to reduce both stability and π_{CE} and μ_{CE}, (c) when θ_β increases, time of convergence change slightly while, π_{CE} goes down and μ_{CE} rises, (d) *increases in* γ reduce stability and, as expected, π_{CE} rises while μ_{CE} goes down, (e) *increases in both* $\alpha_{A,CE}$ *and* $\alpha_{B,CE}$ (implying higher $\beta_{A,CE}$ $/\beta_{B,CE}$) generally tend to destabilize the *CE*s, both in terms of oscillation period and of convergence/divergence. Concluding, when structural parameters and/or *CE* conjectured hyperparameters change, not only the system moves towards CEs that are characterized by different stability properties. In particular, some changes might lead to longer convergence time/ shorter divergence time. As we discuss in the next section, policymakers intervening on parameters must be aware of that.

3. Social optimum and policies

3.1. Actual *CE* coverage vs social optimum.

The average "cost" of disease on the infected subjects μ_t can be considered as the main public health policy target-variable. Clearly, a trade-off exists between social benefits of reducing μ_t (milder consequences for individuals, lower impact on hospital, health care systems and social security systems, etc.) and social costs to achieve this reduction goal (health assistance and social security costs to support infected people etc.). Pandemics clearly showed the reach of such trade-offs.
Now, it looks sound assuming the following:

Assumption 8 (Social welfare)

Define $B(\mu)$ as the social benefit associated with the reduction of μ (with omit time subscript because not relevant here) , with $B' = \frac{\partial B}{\partial \mu} < 0, \frac{\partial^2 B}{\partial \mu^2} < 0$. Define then $C(\mu)$ as the social cost associated to the reduction of μ, with $C' = \frac{\partial C}{\partial \mu} < 0, \frac{\partial^2 C}{\partial \mu^2} > 0$. In order to further develop the present analysis, we specifically assume the following functional forms of social benefits and costs:

$$B(\mu) = (1-\mu)^b, 0 < b < 1 \qquad\qquad C(\mu) = (1-\mu)^c, c > 1$$

where parameters b and c capture marginal benefits and costs, respectively, of μ reductions.

The PHA is now able to derive the socially optimal level of μ^* by trading off social benefits and costs of reducing it. The optimal μ^*, in turn, corresponds to an optimal vaccination coverage $\pi^* = \frac{\alpha-\mu^*}{\beta}$.

RESULT 7 (a) *The socially optimal severity μ^* must satisfy the usual first-order condition $B'(\mu^*) = C'(\mu^*), 0 < \mu^* < 1$. In particular, given the assumed functional forms, the socially optimal severity is:*

$$\mu^* = 1 - \left(\frac{b}{c}\right)^{\frac{1}{c-b}} \in (0,1) \qquad (11)$$

It is decreasing in b and increasing in c.

(b) *Given μ^*, the socially optimal level of vaccination coverage (as derived from the model) is $\pi^* = \frac{\alpha - 1 - \left(\frac{b}{c}\right)^{\frac{1}{c-b}}}{\beta}$ that depends not only on marginal benefits and costs of severity reduction (positively and negatively, respectively.), but also on structural parameters α and β (positively and negatively, respectively).*

Clearly, the socially optimal disease severity may differ from the existing *CE* severity. The PHA interested in aligning the μ_{CE} to μ^* must be aware that only shocks affecting the structural parameters can be effective to achieve the purpose. In fact, μ_{CE} can be changed only through policies and interventions that are able to affect the structural parameters, even if the PHA might not, and in general does not, know these parameters precisely. Moreover, we know that each specific policy (i.e., a change aimed at modifying the structural parameters) will have a different impact in terms of both marginal effectiveness and the time dynamics needed to achieve the policy goal (time of convergence/period/stability).

In the next subsection various policies are discussed. Their implementation typically entails specific fixed costs that, thought socially relevant, do not determine the optimal level of severity at the margin. E.g., the costs of developing effective treatments that are able to reduce the severity do not depend on severity reduction and can be simply added as an additional fixed cost to $C(\mu)$.

3.2. Policies and their dynamical effects

Now we are ready to characterize different policies that can be used to align the *CE* level of disease severity to the socially optimal severity. In fact, as shown by our computations (reported in Appendix 3) reasonable sets of parameters are typically associated with relatively high *CE* severity and too low *CE* vaccination coverage with respect to hypothetical optimal values. This calls for policy interventions. Of course, actual public interventions can, and in general do, involve a mix of policies; however, we will examine each of them separately, in order to understand its potential and limits.

A general observation is in order before going on. In what follows we will also mention the *stability* effects of some policies. As we saw in previous section, some parameter changes (representing indeed those policies) can reduce

the stability, or even induce instability, of the associated *CE*s. Recall, however, that our numerical experiments were implemented by *fixing the precision hyperparameters*, so that we can appreciate the characteristics of the dynamical paths in the very first periods after a displacement from a *CE*. On the other side, we know from Result 3 and from Figures 2 and A1-A4 that the actual increase of precisions dampens the oscillations after some periods. This implies that those paths that are unstable in the initial periods will *not* diverge indefinitely: they could approach some non-equilibrium position surrounding a *CE*, or could even be "captured" by the basin of attraction of a stable *CE*.

***Improvements in treatment effectiveness* (*reducing* α)**

Resources can be invested in researching, developing and/or making available treatments that are able to reduce the impact of the disease on individuals (es. antibiotics/antiviral/medical treatments). As usual, the suitability of this policy is strictly related to costs and effectiveness of research and development of drugs and treatments. In fact, improvements in treatment effectiveness result in a diminished α. When $\mu^* < \mu_{CE}$ (and, obviously, $\pi^* > \pi_{CE}$), developing treatments can reduce μ_{CE} favoring its realignment to the socially optimal level of severity. In fact, since μ^* does not depend on α and μ_{CE} is a decreasing function of it, the strategy might be effective. Reductions of α decrease also both the CE vaccination coverage and the optimal vaccination coverage. This sheds light on the fact that treatment effectiveness and vaccination coverage are *substitutes*. However, given that $\frac{d\pi^*}{d\alpha} = \frac{1}{\beta} > \frac{d\pi_{CE}}{d\alpha} = \frac{\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)}{1+\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\beta}$, the optimal coverage decreases faster than the *CE* coverage, finally favoring their alignment.

Concerning the dynamical effects of this policy, as observed in Section 2.4 (and Appendix 3), reductions of α increases the *time of convergence* to the new *CE*, or even results in an unstable *CE*, thus implying a slower approach to, or rapid initial divergence from, the target levels.

Note that when $\mu^* > \mu_{CE}$, resources eventually devoted to maintaining α at its structural level could be destinated, at least partially, to combating other diseases.

Now, consider a baseline scenario characterized by the *structural* parameters $\alpha = 0.7, \beta = 0.8, \gamma = 0.5, \theta_A = 1, \theta_B = 2$ (the same used in Appendix 3 for numerical computations) According to the model (expressions 10-11) *CE* levels of severity and coverage are $\mu_{CE} = 0.437$ and $\pi_{CE} = 0.328$, respectively. Let's now assume that the optimal level of severity is $\mu^* = 0.1$. Focusing of α, the PHA should work on developing (or paying for) a *terrific* therapy able to bring down α from 0.7 to 0.16 (a reduction of more than 400%), implying $\mu_{CE|\alpha=0.16} = \mu^*$. Note that, without opting for a policy-mix, the extraordinary therapy might imply a low vaccination coverage ($\pi_{CE|\alpha=0.16} = 0.075$). The substitution effect between therapies and vaccination coverage force to a significant decrease of α in absence of other combined policies.

By using the computation program applied to obtain numerical computations and assuming "middle" values $\alpha_{A,CE} = 0.7$ and $\alpha_{B,CE} = 0.5$, we observe that moving α from 0.7 to 0.16 dramatically changes the stability properties of *CE*s: starting from a convergence time of 12.9 passing through higher and higher TOCs, we reach even a negative TOC (-1.7) implying divergence in the first phases of policy intervention.

Concluding, the required therapy must be extremely effective and safe, usually requiring many years of research and trials. This policy might be a long-term and very expensive one, besides requiring very long time or even the impossibility to reach immediately the exact policy goal.

Improvements in vaccine effectiveness (increasing β)

Resources can be also invested in researching, developing and/or making available (more) effective vaccines. Once again, the suitability of this policy is related to costs and effectiveness of the process. In fact, improvements in vaccine effectiveness result in an augmented β. When $\mu^* < \mu_{CE}$ (and, obviously, $\pi^* > \pi_{CE}$), developing better (or new) vaccines reduces μ_{CE} ($\frac{d\mu_{CE}}{d\beta} = -\frac{\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\alpha}{1+\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\beta} + \frac{\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\alpha}{\frac{1}{\beta}+\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\beta} < 0$) and favors its realignment to the socially optimal level of severity. Intuitively, given that $\frac{d\pi^*}{d\beta} = -\frac{\alpha-1-\left(\frac{b}{c}\right)^{\frac{1}{c-b}}}{\beta^2} > 0$ while $\frac{d\pi_{CE}}{d\beta} = -\frac{\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\alpha}{1+\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\beta^2} < 0$, increases of β further decrease *CE* vaccination coverage while increasing the optimal coverage. This shows that vaccine effectiveness and vaccination coverage are substitutes; nonetheless, increasing vaccine effectiveness might be a good policy to reduce the disease severity.

Concerning the dynamical effects of this policy, as observed in Section 2.4 (and Appendix 3),, an increase in β suffers from the same limitations of a reduction of α studied above (slower convergence/faster divergence).

If we consider the usual baseline scenario ($\mu_{CE} = 0.437$ and $\pi_{CE} = 0.328$) and the hypothetical optimal level of severity $\mu^* = 0.1$, a policy exclusively based on the development of a furthermore effective vaccine is extremely difficult. Given the present parameters, parameter β should increase ten times (above the parameter limits).

Without guaranteeing a sufficiently high level of vaccination coverage, the implementation of this policy looks unaffordable and the *CE*s possibly achievable might be strongyle unstable in the first periods.

Nudging and subsidies (reducing θ_B and/or θ_A)

In order to favor vaccination compliance, resources might be invested in subsidizing and/or nudging individual decisions to get vaccinated. Public vaccination campaigns involve both subsidies and nudging, since (*a*) people (or specific categories like newborns, kids, elders, fragile people, etc.) can be vaccinated for free or for a capped price; (*b*) individuals are invited to comply with a vaccinal agenda; (*c*) vaccinations are facilitated, being provided by general practitioners/pediatricians, local vaccination centers, or even the pharmacies. In general, most of PHA actions aiming at reducing vaccination hesitancy and favoring higher vaccination coverages are based on a significant reduction of individual costs to get vaccinated. According to our model, this policy can be seen as a shrinking of the individual-cost distributions such that θ_B and/or θ_A become smaller. Once again, note that when $\mu^* < \mu_{CE}$ (and, obviously, $\pi^* > \pi_{CE}$), reducing $\theta_i, i = A, B$ optimal levels are not affected, while μ_{CE} decreases ($\frac{d\mu_{CE}}{d\theta_i} > 0$) and π_{CE} increases ($\frac{d\pi_{CE}}{d\theta_i} < 0$), finally favoring the realignment of *CE* levels to the socially optimal

values.[12] Note that green-pass (Campanozzi et al. 2022) and other immunization passports (Sharun et al. 2021) can be modelled as nudging mechanisms that, by increasing the individual cost of being not vaccinated, in fact result in lower θ_i.

Concerning the dynamical effects of subsidies/nudging, as pointed in Section 2.4 (and Appendix 3), a reduction of θ_B might imply shorter oscillation periods (the overshooting effect slows-down) and weak effects on TOCs. However, the combined effects of reductions of both θ_i must be carefully considered from a dynamical perspective, as showed in the following example.

In the usual baseline scenario, the hypothetical optimal level of severity $\mu^* = 0.1$ corresponding to a vaccination coverage $\pi^* = 0.75$ can be achieved by *significantly* shrinking the two subpopulations' intervals of individual costs to get vaccinated. As already explained, this can be done by mixing proper economic subsidies and various actions making easier or more comfortable to get vaccinated. Given the present parameters, *CE* levels of severity and coverage can be aligned to the optimal levels by reducing both parameter θ_i ten times (θ_A should decrease from 1 to 0.1 and θ_B should decrease from 2 to 0.2). Looking at the stability properties, by implementing this very strong policy, from a convergence time of 12.9 , the new *CE* is unstable, intuitively because such shrined cost intervals induce very strong oscillations in the subpopulations' decisions (negative TOC -1.2), at least in the first dates.

Concluding, this policy might be expensive but feasible and with a clear impact but at the cost of possible initial high instability, meaning long time or even the impossibility to immediately reach the exact policy goal.

Educational campaign/moral suasion (increasing γ)

A further strategy to favor vaccination compliance is promoting information or educational campaigns to encourage or persuade people to get vaccinated. These interventions are typically targeted to specific subpopulations particularly needing to benefit of a larger vaccination coverage. According to our model, we can assume that the policy is targeted to the subpopulation *B*, including hesitant individuals given their initial beliefs and individual costs. A successful policy might move some individuals from subpopulation *B* to subpopulation *A*, finally increasing γ. Changes of parameter γ do not affect the optimal levels of severity and coverage, while they modify *CE* levels. In particular when $\mu^* < \mu_{CE}$ (and, obviously, $\pi^* > \pi_{CE}$), by increasing γ μ_{CE} decreases ($\frac{d\mu_{CE}}{d\gamma} > 0$) and increases π_{CE} ($\frac{d\pi_{CE}}{d\gamma} < 0$), once again favoring the realignment of *CE* levels to the socially optimal values.[13]

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[12] $\frac{d\pi_{CE}}{d\theta_A} = -\alpha\frac{\gamma}{\theta_A^2}\frac{1}{\left(1+\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\beta\right)^2} < 0, \frac{d\pi_{CE}}{d\theta_B} = -\alpha\frac{1-\gamma}{\theta_B^2}\frac{1}{\left(1+\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\beta\right)^2} < 0, \frac{d\mu_{CE}}{d\theta_i} = -\beta\frac{d\pi_{CE}}{d\theta_i} > 0$

[13] $\frac{d\pi_{CE}}{d\gamma} = \frac{\alpha\left(\frac{1}{\theta_A}-\frac{1}{\theta_B}\right)}{\left(1+\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\beta\right)^2} > 0$ because $\theta_B > \theta_A$. $\frac{d\mu_{CE}}{d\theta_i} = -\beta\frac{d\pi_{CE}}{d\gamma} < 0$.

Even in this case, from a the dynamical perspective, as reported Section 2.4, an increase in γ brings about an initial slower convergence or faster divergence.

Although educational campaign might be ideally useful to pursuing optimality, when we consider our usual scenario and the objective of $\mu^* = 0.1$, we conclude that even persuading all the hesitant (subpopulation *B*) to adhere to beliefs and individual costs as perceived by the less hesitant subpopulation *A* (an extremely difficult goal, we guess!), the resulting *CE* severity would remain significantly higher with respect to the optimal level ($\mu_{CE|\gamma=1} = 0.389 >> 0.1$). In addition, the stability properties of the possible new *CE* worsen, since passing from a TOC=12.9 to a negative TOC = −30.1, implying slow divergence.

Mandatory vaccination

In many countries, some vaccinations are mandatory and the decision to not comply is variously sanctioned. People who do not comply with mandatory infant vaccinations, for example, can be sanctioned with monetary sanctions and children cannot attend schools. During the Covid 19 pandemic, several countries introduced mandatory vaccinations, at least for some categories of people. In the model, we assume that a fixed penalty $S > 0$ is certainly paid by those who do not comply with mandatory vaccinations. In this case, the individuals of each subpopulation decide to get vaccinated at the beginning of each date *t* iff $\alpha_{i,t} - \beta_{i,t}\pi_{t-1} + S > \theta_i$. Reconsidering expressions (1) and (2), we easily conclude that when vaccination noncompliance is punished with a sanction, coverage increases. Looking at the *CE* equilibrium, we obtain $\pi_{CE,S} = \frac{\left(\frac{\gamma}{\theta_A+S}+\frac{1-\gamma}{\theta_B+S}\right)\alpha}{1+\left(\frac{\gamma}{\theta_A+S}+\frac{1-\gamma}{\theta_B+S}\right)\beta} > \pi_{CE} = \frac{\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\alpha}{1+\left(\frac{\gamma}{\theta_A}+\frac{1-\gamma}{\theta_B}\right)\beta}$. By introducing sanction S, *CE* severity decreases consequently: $\mu_{CE,S} = \alpha - \beta\frac{\left(\frac{\gamma}{\theta_A+S}+\frac{1-\gamma}{\theta_B+S}\right)\alpha}{1+\left(\frac{\gamma}{\theta_A+S}+\frac{1-\gamma}{\theta_B+S}\right)\beta}$.

A punishment system can allow the PHA to pursue optimality at relatively low costs (costs to implement the sanction system, net of the inflicted sanctions), although, as in the case of subsidies and nudging, this might be associated to long time or even the impossibility to immediately reach the specific target level of immunization/severity.

Again, consider our baseline scenario and the usual PHA objective $\mu^* = 0.1$. By introducing a sanction $S = 0.9285$ (a value that is in reasonable if compared with individual costs to get vaccinated) the optimal levels of severity and coverage is achieved. Not surprisingly, mandatory vaccinations and related sanctions are the most common actions of PHA to pursue their policy goals. However, starting from a *CE* characterized by TOC = 12.9, the new *CE* has negative TOC (−1.0) implying, once again, a strong initial divergence. This suggest that the PHA, when intervening, must be aware that its policy goals can be effectively pursued, but at the cost of an initial complex dynamic and possibly, not perfectly achieving the specific target level of immunization/severity.

Overall, it seems clear that if the PHA is interested in controlling disease severities by achieving sufficiently high vaccination coverages, policy interventions are needed, because without them *CE* levels of coverage tends to be relatively low. Policies can be effective but can increase instability of *CE*s. The PHA must be account for that,

finally cautiously calibrating interventions. Policy mixes might be very useful to achieve policy goals because acting on various structural parameters might be more effective and less costly than promoting a unique kind of intervention. Certainly, mandatory immunization (with sanctions) appears the easiest way to promote vaccination compliance. However, nudging, subsidies joint with sanctions appear to be a very effective policy for the PHA, because substitution effects are avoided. For these reasons, countries must guarantee updated vaccinal agenda, granting both easy access to immunization and sanctions in the case of non-compliance.

According to our analysis, trying to change populations' beliefs, even if this were possible, would *not* affect the *CE* vaccination coverage/disease severity. Not only: since the PHA is not informed of the private beliefs prevailing in an existing *CE*, there would exist high *uncertainty* about the dynamical effects related to changes of these beliefs (induced, say, by informational campaigns). In addition, high $\alpha_{i,CE}$'s (necessarily coupled with high $\beta_{i,CE}$'s) work against easy convergence. Therefore, campaigns oriented towards inducing people to be more aware about severity of the disease and/or more confident with vaccine effectiveness might result in longer convergence times.

In general, our arguments suggest that the PHA should be *pragmatic* when deciding any policy objective: in fact, most policies that are potentially useful to reduce the disease severity to certain levels might not *hit the target with precision*. Besides the uncertainty due to ignorance of private beliefs, policies tend to induce lower stability or higher instability of the associated *CE*s, at least initially. Therefore, the system might eventually stop in a position that is not *precisely* the projected one.

4. Conclusions

The present dynamic Bayes-conjectural model contributes to understanding how heterogeneous and uninformed agents decide to get vaccinated based on various conjectures, the available information, and their private costs of getting vaccinated. As showed in the model, without any policy intervention, immunization coverage tends to be relatively low and the dynamics over time shows the relevance of (*a*) overshooting effects associated to the adaptive expectations, (*b*) external effects related to the heterogeneity of subpopulations, and (*c*) a herd effect induced by learning.

Looking at the *CE*s, given specific structural parameters, a unique *CE* severity/vaccination coverage is achieved. However, this is compatible with infinity of possible *CE* situations implying that the subpopulations keep envisaging different relationships between severity and vaccination coverage. This clearly implies that a PHA interested in controlling the average severity of a disease must intervene not so much on subpopulations' beliefs (the hyperparameters of the model) but on the structural elements of the relation between average severity and vaccination coverage (that is, the structural parameters of the model). Beliefs and their precisions, in fact, do not determine the *CE* severity/immunization coverage while being relevant for the *CE*'s dynamical features.

By analyzing different public health policy interventions, some remarks are possible. Although taking action on any of the structural parameters might help pursuing average severity reductions, policies aimed at developing new treatments (excluding vaccines) or at increasing the efficacy of vaccines tend to displace immunization behavior.

This means that working only on new treatments and/or more effective vaccines -both substitutes for the vaccination coverage- without simultaneously supporting the overall immunization requires very massive interventions, that are expensive and long-term because of the costs and times of R&D in this field. Balanced policy mixes, also including actions against the vaccination hesitancy, look more suitable.
Information campaigns aimed at reducing the weight of "truthers" and incorrigible skeptics, though helpful in theory, might be not only a hard task (it is not easy influencing individual costs and initial conjectures of a subpopulation) but even not very effective because the policy impact depends on the overall population features.
Nudging and subsidization, on the one hand, and mandatory vaccinations with sanctions for those who do not comply, on the other hand, represent a valid approach to reduce the average severity of a disease by favoring larger vaccination coverage.

However, by intervening on structural parameters, the PHA must account for the relevant dynamical effects of policies. Policies can effectively influence severity and immunization behavior in the wished direction, but not necessarily with immediate precision. In fact, once a policy (that is an exogenous shock moving the system from a previous *CE* towards another possible one) is implemented, the dynamical path in the first periods could approach non-equilibrium positions surrounding the target *CE* or could even be "captured" by the basin of attraction of another stable *CE*. In other words, policies on structural parameters can be used to pursue target levels of severity/immunization coverage, but reaching the exact policy goal might be like scoring a bullseye when playing blindfolded: hit the target with precision can be a long story or even impossible in few shots.

For these reasons, affordable public health policies pursuing optimality through actions on structural parameters must be in place and constantly supported, even remembering the incomparable costs of pandemics and epidemics with respect to policy costs (Nandi and Shet 2020, Ozawa et al. 2016, Silver et al. 2021, Standaert et al. 2020).

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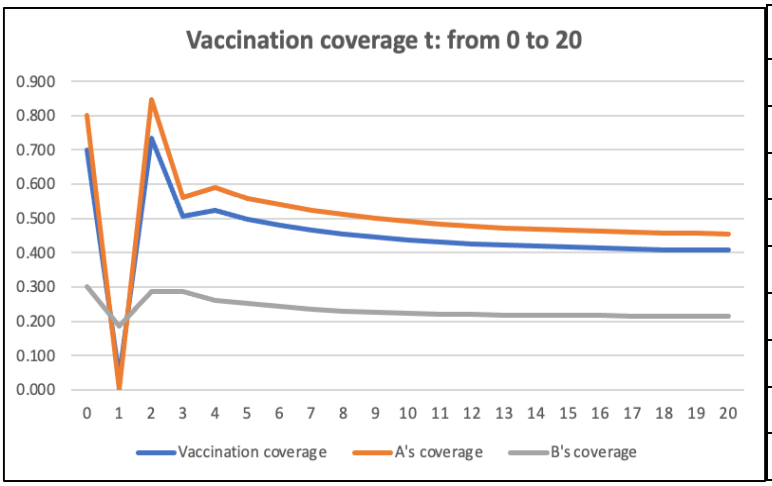
Appendix 1 - Vaccination coverage dynamics: some simulations for different scenarios

The following scenarios illustrate how different hyperparameters affect subpopulations' vaccination decision and the overall dynamics of vaccination coverage.

Baseline scenario with different precision hyperparameters: sticky convictions vs malleable beliefs

The structural parameters of the model are initially set in such a way that we illustrate the case of a serious disease (relatively high α) for which a quite effective vaccine is available (relatively high β). We consider a largely predominant subpopulation A ($\gamma \gg 0.5$) of individuals who are sufficiently open-minded about medical information, confident about vaccination and characterized by relatively low individual costs to get vaccinated. The residual subpopulation B is stickily convinced that the disease is not so severe, and vaccines are not really effective; their individual cost to get vaccinated is relatively high. See Figure A1. As expected, the coverage rates *stabilize* after some dates with overshooting effects. The overall coverage is not very high (there are no regulatory interventions at the moment) and is a weighted average of the subpopulations' coverage rates.

FIGURE A1 -Vaccination coverage dynamics: Baseline scenario with sticky hesitant individuals.

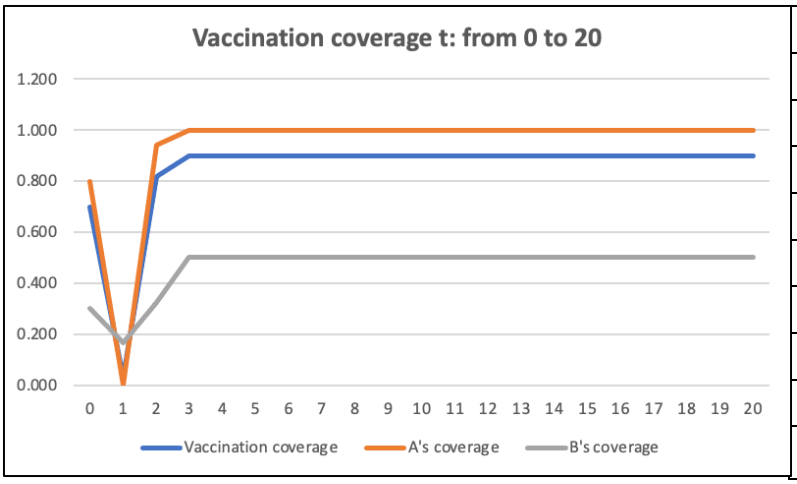


Structural parameters			
α	0.8		
β	0.9		
γ	0.8		
θ_A	1	θ_B	2
Hyperparameters			
$\alpha_{A,0}$	0.8	$\alpha_{B,0}$	0.6
$\beta_{A,0}$	1	$\beta_{B,0}$	0.2
$\eta_{A,\alpha,0}$	0.5	$\eta_{B,\alpha,0}$	1
$\eta_{A,\beta,0}$	0.5	$\eta_{B,\beta,0}$	1

If we decrease the initial precision hyperparameters, making the people more malleable and open-minded, we observe that the dynamic towards *stable* coverage rates significantly changes. Actually, in this case people learn faster, and the vaccination coverage rates stabilize in few dates without too many overshooting movements: see Figure A2.

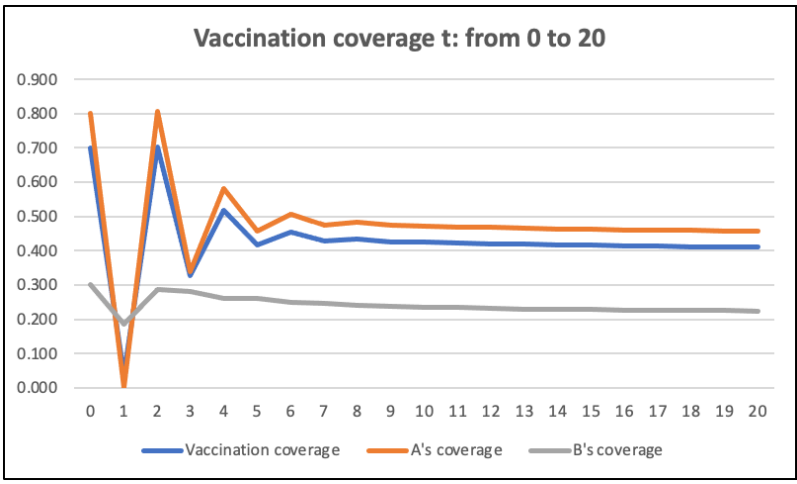
When, instead, both subpopulations are staunchly convinced about their initial beliefs, coverage rates stabilize later in time and overshooting lasts for more periods (Figure A3).

FIGURE A2: Vaccination coverage dynamic: Baseline scenario with malleable beliefs.



Structural parameters			
α	0.8		
β	0.9		
γ	0.8		
θ_A	1	θ_B	2
Hyperparameters			
$\alpha_{A,0}$	0.8	$\alpha_{B,0}$	0.6
$\beta_{A,0}$	1	$\beta_{B,0}$	0.2
$\eta_{A,\alpha,0}$	0.2	$\eta_{B,\alpha,0}$	0.2
$\eta_{A,\beta,0}$	0.2	$\eta_{B,\beta,0}$	0.2

FIGURE A3: Vaccination coverage dynamic: Baseline scenario with sticky beliefs.



Structural parameters			
α	0.8		
β	0.9		
γ	0.8		
θ_A	1	θ_B	2
Hyperparameters			
$\alpha_{A,0}$	0.8	$\alpha_{B,0}$	0.6
$\beta_{A,0}$	1	$\beta_{B,0}$	0.2
$\eta_{A,\alpha,0}$	1	$\eta_{B,\alpha,0}$	1
$\eta_{A,\beta,0}$	1	$\eta_{B,\beta,0}$	1

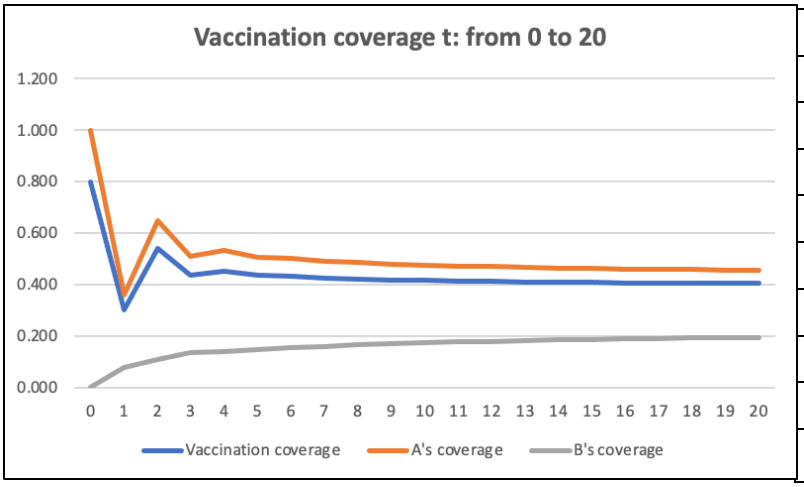
Baseline scenario with different beliefs about severity: alarmed people and truthers.

Keeping the structural parameters of the model unchanged, we now consider a largely predominant subpopulation *A* who is strongly convinced that the disease is very severe ($\eta_{A,\alpha,0}$ relatively high), while being still open-minded about vaccination ($\eta_{A,\beta,0}$ relatively low). The residual subpopulation *B* is strongly convinced ($\eta_{B,\alpha,0}$ relatively high) that the disease does not exist, or its severity is a fake news, while vaccines *could* be ($\eta_{B,\beta,0}$ relatively low) even dangerous ($\beta_{B,0} < 0$). Looking at Figure A4, we observe that, even if subpopulation *B* is initially against vaccination, its vaccination coverage rate slowly increases thanks to the learning process that is partially induced by subpopulation *A*'s behavior. We might conclude that there is some *herd effect*.

Although many different scenarios could be compared, the main implication derived from the proposed simulations is that hyperparameters significantly affect the dynamic towards some stable coverage rates. Indeed,

they determine both the *short-run* populations' behavior -together with the related vaccination coverage rates- and the time required for those variables to converge to a stable position.

FIGURE A4: Vaccination coverage dynamic: Baseline scenario with alarmed people and truthers.



Structural parameters			
α	0.8		
β	0.9		
γ	0.8		
θ_A	1	θ_B	2
Hyperparameters			
$\alpha_{A,0}$	1	$\alpha_{B,0}$	0
$\beta_{A,0}$	0.9	$\beta_{B,0}$	-0.2
$\eta_{A,\alpha,0}$	1	$\eta_{B,\alpha,0}$	1
$\eta_{A,\beta,0}$	0.5	$\eta_{B,\beta,0}$	0.5

Appendix 2 – Dynamic stability of *CE*s

Jacobian and its eigenvalues

In order to study the *local* stability of *CE*s, the Jacobian matrix of system whose stability properties we wish to examine must be computed at a *CE*. Then, the eigenvalues of this matrix must be evaluated. The characteristics of the eigenvalues determine how the variables move after a tiny displacement from the *CE* at which the Jacobian and its eigenvalues are evaluated. In particular,

(*a*) the presence of a positive eigenvalue means that there is a component of the out-of-equilibrium trajectory[14] that is *monotonic* with respect to the *CE*;

(*b*) the presence of a negative eigenvalue implies a *two-period oscillation* around the *CE*;

(*c*) the presence of a couple of complex-conjugate eigenvalues implies a *longer-period oscillation*, whose length depends on the argument of the complex eigenvalues;

(*d*) the modulus of an eigenvalue determines whether the movements are *convergent or divergent* with respect to the *CE*: a modulus lower (resp. greater) than one means convergence, or *stability* (resp. divergence, or *instability*).

Proof of Result 6(*a*)

[14] To be precise, a "component" of the trajectories is one of the different dynamical forces at work after the displacement: it can be isolated if the displacement takes place exactly along the direction of the *eigenvector* associated with the corresponding eigenvalue. In general, every *actual* trajectory is a combination of these specific components.

Rewrite (4b) of the text as follows (make vector products explicit, and use our definitions of $\mathbf{z}_{i,t}$ and $\mathbf{x}_{i,t}$):

$$\begin{bmatrix}\alpha_{i,t+1}\\ \beta_{i,t+1}\end{bmatrix} = \begin{bmatrix}\alpha_{i,t}\\ \beta_{i,t}\end{bmatrix} + \left[\mathbf{H}_{i,t+1}\right]^{-1}\begin{bmatrix}1\\ -\pi_t\end{bmatrix}\left(\mu_t - \begin{bmatrix}1 & -\pi_t\end{bmatrix}\begin{bmatrix}\alpha_{i,t}\\ \beta_{i,t}\end{bmatrix}\right), i = A, B \qquad (A1)$$

Recall that $\mathbf{H}_{i,t+1} \equiv \mathbf{H}_{i,t} + \begin{bmatrix}1 & -\pi_t\\ -\pi_t & \pi^2\end{bmatrix}$. Expression (*A*1) includes two dynamical equations, so that we have four equations for the two subpopulations; to these, we must add equation (2) of the text (shifted by one period ahead):

$$\pi_{t+1} = \frac{\gamma}{\theta_A}\left(\alpha_{A,t+1} - \beta_{A,t+1}\pi_t\right) + \frac{1-\gamma}{\theta_B}\left(\alpha_{B,t+1} - \beta_{B,t+1}\pi_t\right) \qquad (A2)$$

In order to analyze the stability of a *CE*, we suppose that our system has stayed in that *CE* for t periods after date 0. In addition, we assume that the initial precision matrices, as of date 0, are $\mathbf{H}_{i,0} = \begin{bmatrix}\eta_{i,\alpha} & 0\\ 0 & \eta_{i,\beta}\end{bmatrix}$, coherently with Assumption 6. Finally, since we are considering a *CE*, we drop the time suffix from π.

Therefore, it is not difficult to see that $\mathbf{H}_{i,t+1} = \begin{bmatrix}\eta_{i,\alpha} + t & -t\pi\\ -t\pi & \eta_{i,\beta} + t\pi^2\end{bmatrix} = t\begin{bmatrix}\frac{\eta_{i,\alpha}}{t} + 1 & -\pi\\ -\pi & \frac{\eta_{i,\beta}}{t} + \pi^2\end{bmatrix}$. As regards the inverse of this matrix, appearing in expression (*A*1), one can show that it is given by the following formula (details available on request): $\left[\mathbf{H}_{i,t+1}\right]^{-1} = \delta_i(t)\begin{bmatrix}\frac{\eta_{i,\beta}}{t} + \pi^2 & \pi\\ \pi & \frac{\eta_{i,\alpha}}{t} + 1\end{bmatrix}$, where we defined $\delta_i(t) \equiv \frac{1}{\frac{\eta_{i,\alpha}\eta_{i,\beta}}{t} + \eta_{i,\alpha}\pi^2 + \eta_{i,\beta}}$. Hence, we can finally write the term $\left[\mathbf{H}_{i,t+1}\right]^{-1}\begin{bmatrix}1\\ -\pi\end{bmatrix}$, appearing in (A1), as $\left[\mathbf{H}_{i,t+1}\right]^{-1}\begin{bmatrix}1\\ -\pi\end{bmatrix} = \delta_i(t)\begin{bmatrix}\frac{\eta_{i,\beta}}{t} + \pi^2 & \pi\\ \pi & \frac{\eta_{i,\alpha}}{t} + 1\end{bmatrix}\begin{bmatrix}1\\ -\pi\end{bmatrix} = \delta_i(t)\begin{bmatrix}\frac{\eta_{i,\beta}}{t}\\ \frac{\eta_{i,\alpha}}{t}\pi\end{bmatrix} \equiv S_{i,t}$ The final round parenthesis contained in (*A*1) was defined as the "forecasting error" in the text ($e_{i,t} \equiv \left(\mu_t - \mathbf{x'}_t\mathbf{z}_{i,t}\right)$). Then, we can write expression (*A*1) as

$$\begin{bmatrix}\alpha_{i,t+1}\\ \beta_{i,t+1}\end{bmatrix} = \begin{bmatrix}\alpha_{i,t}\\ \beta_{i,t}\end{bmatrix} + S_{i,t}e_{i,t}, i = A, B \qquad (A3)$$

Now, for the study of the local stability properties of a *CE*, we need to compute the derivatives of the five equations (*A*3) and (*A*2) with respect to the five variables of system defined in (13) of the text, and evaluate them in that *CE*.

As regards the derivatives of equations (*A*3), to be evaluated in a *CE*, recall the that $e_{i,t}$ is *equal to zero* in equilibrium, by definition. On the other hand, the derivatives of $e_{i,t}$ with respect to the conjectured mean hyperparameters were computed in expressions (6*a-b*) of the text. Using the usual rules of derivatives, we are finally able to write the following set of derivatives, organized in the 4x4 matrix $\mathbf{J_4}$ (details available on request):

$$\mathbf{J}_4 = \frac{1}{t}\begin{bmatrix}
1 - \delta_A(t)\eta_{A,\beta}\left(\frac{\theta_A + \beta\gamma}{\theta_A}\right) & \delta_A(t)\eta_{A,\beta}\left(\frac{\theta_A + \beta\gamma}{\theta_A}\right)\pi & -\delta_A(t)\eta_{A,\beta}\beta\frac{1-\gamma}{\theta_B} & \delta_A(t)\eta_{A,\beta}\beta\frac{1-\gamma}{\theta_B}\pi\\
-\delta_A(t)\eta_{A,\alpha}\pi\left(\frac{\theta_A + \beta\gamma}{\theta_A}\right) & 1 + \delta_A(t)\eta_{A,\alpha}\left(\frac{\theta_A + \beta\gamma}{\theta_A}\right)\pi^2 & -\delta_A(t)\eta_{A,\alpha}\pi\beta\frac{1-\gamma}{\theta_B} & \delta_A(t)\eta_{A,\alpha}\beta\frac{1-\gamma}{\theta_B}\pi^2\\
-\delta_B(t)\eta_{B,\beta}\beta\frac{\gamma}{\theta_A} & \delta_B(t)\eta_{B,\beta}\beta\frac{\gamma}{\theta_A}\pi & 1 - \delta_B(t)\eta_{B,\beta}\left(\frac{\theta_B + \beta(1-\gamma)}{\theta_B}\right) & \delta_B(t)\eta_{B,\beta}\left(\frac{\theta_B + \beta(1-\gamma)}{\theta_B}\right)\pi\\
-\delta_B(t)\eta_{B,\alpha}\pi\beta\frac{\gamma}{\theta_A} & \delta_B(t)\eta_{B,\alpha}\beta\frac{\gamma}{\theta_A}\pi^2 & -\delta_B(t)\eta_{B,\alpha}\pi\left(\frac{\theta_B + \beta(1-\gamma)}{\theta_B}\right) & 1 + \delta_B(t)\eta_{B,\alpha}\left(\frac{\theta_B + \beta(1-\gamma)}{\theta_B}\right)\pi^2
\end{bmatrix}$$

Now we have to "border" $\mathbf{J_4}$ with a fifth column and a fifth row, containing, respectively, the derivatives of the first four variables with respect to π_t, and the derivatives of π_{t+1} with respect to all five variables. Again, we evaluate these derivatives in a *CE*. For brevity, call $J_{j,k}, j, k = 1, \ldots, 4$, the elements of the above matrix $\mathbf{J_4}$; then, we can see that, also using expression (A2), the whole Jacobian matrix of system (13) is given by the following 5x5 matrix:

$$\mathbf{J}_5 = \begin{bmatrix} J_{1,1} & J_{1,2} & J_{1,3} & J_{1,4} & \delta_A(t)\eta_{A,\beta}\left(\beta\frac{\gamma}{\theta_A}\beta_A + \beta\frac{1-\gamma}{\theta_B}\beta_B - \beta_A\right) \\ J_{2,1} & J_{2,2} & J_{2,3} & J_{2,4} & \delta_A(t)\eta_{A,\alpha}\left(\beta\frac{\gamma}{\theta_A}\beta_A + \beta\frac{1-\gamma}{\theta_B}\beta_B - \beta_A\right)\pi \\ J_{3,1} & J_{3,2} & J_{3,3} & J_{3,4} & \delta_B(t)\eta_{B,\beta}\left(\beta\frac{\gamma}{\theta_A}\beta_A + \beta\frac{1-\gamma}{\theta_B}\beta_B - \beta_B\right) \\ J_{4,1} & J_{4,2} & J_{4,3} & J_{4,4} & \delta_B(t)\eta_{B,\alpha}\left(\beta\frac{\gamma}{\theta_A}\beta_A + \beta\frac{1-\gamma}{\theta_B}\beta_B - \beta_B\right)\pi \\ \frac{\gamma}{\theta_A}\Sigma_{j=1}^4 J_{j,1} & -\frac{\gamma}{\theta_A}\pi\Sigma_{j=1}^4 J_{j,2} & \frac{1-\gamma}{\theta_B}\Sigma_{j=1}^4 J_{j,3} & -\frac{1-\gamma}{\theta_B}\pi\Sigma_{j=1}^4 J_{j,4} & -\left(\beta_B\frac{1-\gamma}{\theta_B} + \beta_A\frac{\gamma}{\theta_A}\right) \end{bmatrix}$$

The β_A and β_B appearing in the last column are the *CE* ones, and can be computed using expression (12) in the text.

Matrix $\mathbf{J}_5$ is quite complicated. However, we can make a change that helps to simplify somehow its form. Define the following transformation matrix:

$$\mathbf{T} = \begin{bmatrix} \frac{\eta_{A,\alpha}}{\eta_{A,\beta}}\pi & -1 & 0 & 0 & 0 \\ 0 & 0 & \frac{\eta_{A,\alpha}}{\eta_{A,\beta}}\pi & -1 & 0 \\ 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix}$$

whose inverse is

$$\mathbf{T^{-1}} = \begin{bmatrix} \frac{\eta_{A,\beta}}{\eta_{A,\alpha}\pi} & 0 & \frac{\eta_{A,\beta}}{\eta_{A,\alpha}\pi} & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 \\ 0 & \frac{\eta_{A,\beta}}{\eta_{A,\alpha}\pi} & 0 & \frac{\eta_{A,\beta}}{\eta_{A,\alpha}\pi} & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix}$$

Compute now the matrix product $\mathbf{T} \cdot \mathbf{J}_5 \cdot \mathbf{T^{-1}}$, that is, a *similarity* transformation: the result is precisely the matrix written in Result 6(*a*) of the text.

Appendix 3 - Numerical computations: convergence to, and oscillations around, *CE*s

We now provide some numerical computations[15] in order to deepen both how different levels of the structural parameters and the hyperparameters of the model affect the *CE* levels of vaccination coverage and severity and, more important for our present purposes, how they determine the dynamic features of the *CE*.

The *precision* hyperparameters affect only the time required for the dampening of the updating process in the longer period (Section 2.2 and Appendix 1). Instead, the precise configuration of the *CE*s is determined by the structural parameters and the *CE mean* hyperparameters $\alpha_{CE,i}$ and $\beta_{CE,i}, i = A, B$ (Section 2.3); in addition, these same parameters determine also the movements -oscillations and convergence/divergence- that take place *just after* a tiny

[15] Computations are carried out by means of Mathematica©. Commands available upon request.

displacement from a *CE*, neglecting the dampening effect of the increasing precision hyperparameters (Section 2.4).

In what follows, in order to appreciate the role of conjectures in shaping the dynamics around a *CE*, we assume that the two subpopulations are characterized by different *CE* hyperparameters: subpopulation *A* is more worried about the basic severity of the disease (relatively high $\alpha_{CE,A}$) and is more confident in the efficacy of the vaccine (relatively high $\beta_{CE,A}$); in addition, this subpopulation is readier to learn from the PHA announcements (relatively low $\eta_{A,\alpha,0}$ and $\eta_{A,\beta,0}$, where "0" stands for the moment when the system is displaced from the *CE*). Subpopulation *B* has opposite characteristics, besides bearing a higher cost to be vaccinated ($\theta_B > \theta_A$). Wishing to neglect, at least in the first instance, the dampening effect of precisions that keep increasing after the displacement, we keep fixed the η's, and set $\eta_{A,\alpha,0} = \eta_{A,\beta,0} = 0.2$ and $\eta_{B,\alpha,0} = \eta_{B,\beta,0} = 1$ in all our computations. For the very same reason, we provisionally put aside the effect of the passing of time (which is, again, dampening): therefore, we keep *t* equal to 1, well knowing that higher *t*s slow down the pace of oscillations.

We envisage a baseline scenario characterized by the following set of *structural* parameters: $\alpha = 0.7, \beta = 0.8, \gamma = 0.5, \theta_A = 1, \theta_B = 2$. We operate then variations of these structural parameters, one at a time, around their baseline values, except for θ_A, kept fixed at 1. Tables 1-4 provide all the results.

Since any *CE* configuration depends also on the *mean* hyperparameters, each table contains different rows, corresponding to sixteen different combinations of the parameters $\alpha_{i,CE}, i = A, B$ (four cases each): again, the different $\alpha_{CE,i}$ are chosen coherently with the assumption of differing subpopulation conjectures. Recall that, once the $\alpha_{CE,i}$ hyperparameters and the structural parameters are known, the corresponding $\beta_{i,CE}, i = A, B$ can be computed using expression (10-12).

It follows that every table presents eighty different *CE*s. For each *CE* we report three results: the $\beta_{i,CE}, i = A, B$; the *oscillation period* around the *CE*; and the *time of convergence* (TOC, hereafter). By TOC we mean the time required in order that the distance from the *CE* is dampened to 5% of the initial displacement size. A *negative* TOC signals *instability*: if time "flowed backwards", the system would converge to the *CE*, while in the ordinary direction of time it diverges from it.[16] As regards the *oscillation period*, recall that a 2-period derives form a negative real eigenvalue (the "overshooting" effect discussed after Result 3), while longer oscillation periods derive from a couple of complex conjugate eigenvalues (this case implies a slower overshooting effect).

Finally, π_{CE} and μ_{CE}, as we know, do *not* depend on the conjectured hyperparameters, and can be computed using expressions (10-11). Their values are reported in the last two rows of the tables.

The results reported in Tables 1-4 allow us to put forward some general conclusions about how changes in the parameters affect the *CE* location and the out-of-equilibrium dynamics. The results are obtained looking at *partial* effects, keeping fixed the remaining parameters (we move along single rows or columns of the Tables). Observe

[16] We report the TOC associated with the eigenvalue of *maximum modulus*, i.e., the one implying the slowest convergence rate or the faster divergence rate. In the unstable case, the absolute value of the negative TOC indicates the time required in order that the amplitude diverges to twenty times (not one twentieth) the initial displacement.

that there is a prevalence -about 70%- of period-2 oscillations (the "direct" overshooting effect), while less the one third of cases display longer-period oscillations. Longer-period oscillations are associated with *higher* $\alpha_{A,CE}$ *and lower* $\alpha_{B,CE}$ hyperparameters, suggesting that somehow "divergent" conjectures tend to cause more complex dynamical interactions.

Our conclusions are divided into those depending on the *structural* parameters (S1-S4), and those depending on *hyperparameters* (H1-H2). While the former depend on exogenous factors or on public policies (see Section 3), the latter depend on the populations' beliefs at the starting date (see Section 2.5).

S1) *Increases in* α (Table 1) This implies a higher basic, zero-coverage, disease severity. If α rises, the oscillation period, when different from 2, is increased: this means that these changes tend to reduce the stability of *CE*s. On the other side, the TOC reacts in a stabilizing direction: either the positive TOCs decrease (shorter convergence time), or the absolute values of negative TOCs increase (slower divergence time). As expected, both π_{CE} and μ_{CE} increase.

S2) *Increases in* β (Table 2). This implies a higher vaccine efficacy. If β rises, the oscillation period, if different from 2, is increased: this, again, means that these changes tend to reduce stability. Similarly, the TOC reacts in a destabilizing direction, differently from α increases: the positive TOCs decrease, or the absolute values of negative TOCs increase. As expected, both π_{CE} and μ_{CE} decrease.

S3) *Increases in* θ_β (Table 3). This means a higher vaccination cost for subpopulation *B*, keeping the cost of subpopulation *A* fixed: population *B* becomes relatively less willing to get vaccinated. If θ_β rises, the oscillation period, when different from 2 increases, while the TOC change slightly. Finally, π_{CE} goes down and μ_{CE} rises: due to the lower inclination of B to get vaccinated, the overall coverage decreases, and the disease gravity increases.

S4) *Increases in* γ (Table 4). Population *A*'s share increases, i.e., the "truthers" have a lower weigh. In this case, again, the oscillation period, when different from 2, is increased, and stability is reduced (higher positive TOC and lower absolute-vale TOC). Therefore, surprisingly, it is not sufficient that a higher share of people is worried of the disease (and believe in the vaccine efficacy) in order that the system converges more rapidly to a CE: this is probably due to the fact that, in our scenarios, population *A* is readier to change their mind in the initial periods. On the other side, the effects on π_{CE} and μ_{CE} are as expected: the former rises and the latter goes down.

Concerning the impact of hyperparameters, we observe the following:

H1) *Increases in* $\alpha_{A,CE}$ (all Tables). Given expression (12), this implies a higher $\beta_{A,CE}$ (this holds in algebraic terms, if $\beta_{A,CE}$ is negative). In general, these changes tend to destabilize the CEs, both in terms of oscillation period and of convergence/divergence. Note that when $\alpha_{A,CE}$ is very low, $\beta_{A,CE}$ can be even negative, meaning that people become very worried about vaccination effects.

H2) *Increases in* $\alpha_{B,CE}$ (all Tables). The effects are of course similar to those found in point H1.

Concluding, when structural parameters and/or *CE* conjectured hyperparameters change, not only the system moves towards CEs that are characterized by different stability properties. In particular, some changes might lead to longer convergence time/ shorter divergence time. As we discuss in the next section, policymakers intervening on parameters must be aware of that.

Table 1. Dynamic stability of *CE*s for different $\alpha_{i,CE}$ values: varying α

		α =0.5				α =0.6				α =0.7				α =0.8				α =0.9			
$\alpha_{A,CE}$	$\alpha_{B,CE}$	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period
0.5	0.1	0.80	-0.91	8.4	74.9	0.44	-0.98	4.0	102.5	0.19	-1.03	4.4	2	0.00	-1.07	4.9	2	-0.15	-1.10	5.4	X
0.5	0.3	0.80	-0.05	12.9	2	0.44	-0.27	4.5	2	0.19	-0.42	4.7	2	0.00	-0.53	5.1	2	-0.15	-0.62	5.6	8.6
0.5	0.5	0.80	0.80	30.2	2	0.44	0.44	5.7	2	0.19	0.19	4.8	2	0.00	0.00	5.2	2	-0.15	-0.15	5.6	5.8
0.5	0.7	0.80	1.65	-80.9	2	0.44	1.56	7.9	2	0.19	0.80	4.8	2	0.00	0.53	5.2	3.3	-0.15	0.33	5.7	4.6
0.7	0.1	1.65	-0.91	-18.8	58.2	1.16	-0.98	22.6	62.0	0.80	-1.03	6.8	68.0	0.53	-1.07	4.8	79.9	0.33	-1.10	5.0	121.9
0.7	0.3	1.65	-0.05	-11.5	2	1.16	-0.27	90.3	2	0.80	-0.42	8.9	2	0.53	-0.53	5.0	2	0.33	-0.62	5.5	2
0.7	0.5	1.65	0.80	-8.3	2	1.16	0.44	-44.1	2	0.80	0.19	12.9	2	0.53	0.00	5.3	2	0.33	-0.15	5.6	2
0.7	0.7	1.65	1.65	-6.5	2	1.16	1.56	-17.7	2	0.80	0.80	24.1	2	0.53	0.53	6.7	2	0.33	0.33	5.6	2
0.9	0.1	2.51	-0.91	-6.1	56.7	1.87	-0.98	-12.8	60.7	1.41	-1.03	303.6	65.5	1.07	-1.07	11.4	71.2	0.80	-1.10	5.6	78.4
0.9	0.3	2.51	-0.05	-5.3	126.7	1.87	-0.27	-9.4	109.9	1.41	-0.42	-40.6	113.0	1.07	-0.53	17.2	130.9	0.80	-0.62	6.7	190.9
0.9	0.5	2.51	0.80	-4.6	2	1.87	0.44	-7.5	2	1.41	0.19	-19.0	2	1.07	0.00	34.9	2	0.80	-0.15	8.6	2
0.9	0.7	2.51	1.65	-4.1	2	1.87	1.56	-6.3	2	1.41	0.80	-12.5	2	1.07	0.53	-1879.0	2	0.80	0.33	12.0	2
1.1	0.1	3.36	-0.91	-4.1	57.2	2.58	-0.98	-6.0	62.6	2.02	-1.03	-10.7	69.2	1.60	-1.07	-45.0	76.9	1.27	-1.10	19.7	85.4
1.1	0.3	3.36	-0.05	-3.7	74.3	2.58	-0.27	-5.3	74.3	2.02	-0.42	-8.5	78.0	1.60	-0.53	-21.5	84.3	1.27	-0.62	40.4	93.2
1.1	0.5	3.36	0.80	-3.4	2	2.58	0.44	-4.7	2	2.02	0.19	-7.1	2	1.60	0.00	-14.2	2	1.27	-0.15	-1207.1	433.8
1.1	0.7	3.36	1.65	-3.2	2	2.58	1.56	-4.2	2	2.02	0.80	-6.1	2	1.60	0.53	-10.6	2	1.27	0.33	-38.6	2
		π_{CE}=0.234				**π_{CE} =0.281**				**π_{CE} =0.328**				**π_{CE} = 0.375**				**π_{CE} =0.422**			
		μ_{CE}=0.312				**μ_{CE} =0.375**				**μ_{CE} =0.437**				**μ_{CE} = 0.500**				**μ_{CE} =0.562**			

$\beta = 0.8, \gamma = 0.5, \theta_A = 1,\ \theta_B = 2, \eta_{A,j,0} = 0.2, \eta_{B,j,0} = 1$

Table 2. Dynamic stability of *CE*s for different $\alpha_{i,CE}$ values: varying β

		β =0.6				β =0.7				β =0.8				β =0.9				β = 1			
$\alpha_{A,CE}$	$\alpha_{B,CE}$	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period
0.5	0.1	0.05	-1.06	3.9	42.7	0.12	-1.04	4.0	58.4	0.19	-1.03	4.4	2	0.26	-1.01	4.9	2	0.33	-1.00	5.2	2
0.5	0.3	0.05	-0.05	4.8	2	0.12	-0.46	4.7	2	0.19	-0.42	4.7	2	0.26	-0.37	4.8	2	0.33	-0.33	4.7	2
0.5	0.5	0.05	0.05	5.2	2	0.12	0.12	5.0	2	0.19	0.19	4.8	2	0.26	0.26	4.7	2	0.33	0.33	4.6	2
0.5	0.7	0.05	0.60	5.4	2	0.12	0.70	5.1	2	0.19	0.80	4.9	2	0.26	0.90	4.6	2	0.33	1.00	4.5	2
0.7	0.1	0.60	-1.06	5.1	33.9	0.70	-1.04	5.0	41.3	0.80	-1.03	6.8	68.0	0.90	-1.01	9.4	2	1.00	-1.00	13.8	2
0.7	0.3	0.60	-0.05	4.7	48.1	0.70	-0.46	6.4	74.5	0.80	-0.42	8.9	2	0.90	-0.37	12.7	2	1.00	-0.33	19.5	2
0.7	0.5	0.60	0.05	6.3	2	0.70	0.12	8.9	2	0.80	0.19	12.9	2	0.90	0.26	20.3	2	1.00	0.33	36.2	2
0.7	0.7	0.60	0.60	9.3	2	0.70	0.70	14.1	2	0.80	0.80	24.1	2	0.90	0.90	53.7	2	1.00	1.00	602.4	2
0.9	0.1	1.15	-1.06	13.5	38.3	1.28	-1.04	28.3	44.4	1.41	-1.03	303.6	65.5	1.54	-1.01	-42.4	2	1.67	-1.00	-21.7	2
0.9	0.3	1.15	-0.05	26.6	43.82	1.28	-0.46	271.3	51.5	1.41	-0.42	-40.6	113.0	1.54	-0.37	-20.7	2	1.67	-0.33	-14.7	2
0.9	0.5	1.15	0.05	345.3	60.0	1.28	0.12	-36.7	96.4	1.41	0.19	-19.1	2	1.54	0.26	-13.6	2	1.67	0.33	-11.1	2
0.9	0.7	1.15	0.60	-33.2	2	1.28	0.70	-17.4	2	1.41	0.80	-12.5	2	1.54	0.90	-10.1	2	1.67	1.00	-8.8	2
1.1	0.1	1.70	-1.06	-25.8	44.4	1.86	-1.04	-14.7	49.3	2.02	-1.03	-10.7	69.2	2.17	-1.01	-8.7	2	2.33	-1.00	-7.5	2
1.1	0.3	1.70	-0.05	-15.0	47.3	1.86	-0.46	-10.7	51.3	2.02	-0.42	-8.5	78.0	2.17	-0.37	-7.3	2	2.33	-0.33	-6.5	2
1.1	0.5	1.70	0.05	-10.7	54.8	1.86	0.12	-8.4	61.9	2.02	0.19	-7.1	2	2.17	0.26	-6.3	2	2.33	0.33	-5.7	2
1.1	0.7	1.70	0.60	-8.3	76.1	1.86	0.70	-7.0	128.0	2.02	0.80	-6.1	2	2.17	0.90	-5.5	2	2.33	1.00	-5.1	2
		$\boldsymbol{\pi_{CE}}$**=0.362**				$\boldsymbol{\pi_{CE}}$ **=0.344**				$\boldsymbol{\pi_{CE}}$ **=0.328**				$\boldsymbol{\pi_{CE}}$ **= 0.313**				$\boldsymbol{\pi_{CE}}$ **=0.300**			
		$\boldsymbol{\mu_{CE}}$**=0.482**				$\boldsymbol{\mu_{CE}}$ **=0.459**				$\boldsymbol{\mu_{CE}}$ **=0.437**				$\boldsymbol{\mu_{CE}}$ **= 0.418**				$\boldsymbol{\mu_{CE}}$ **=0.400**			
$\alpha = 0.7, \gamma = 0.5, \theta_A = 1,\ \theta_B = 2, \eta_{A,j,0} = 0.2, \eta_{B,j,0} = 1$																					

Table 3. Dynamic stability of *CE*s for different $\alpha_{i,CE}$ values: varying θ_B

		$\theta_B = 1$				θ_B =1.5				θ_B =2				θ_B =2.5				$\theta_B = 3$			
$\alpha_{A,CE}$	$\alpha_{B,CE}$	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period
0.5	0.1	0.28	-0.74	5,3	19.8	0.23	-0.91	4.5	31.8	0.19	-1.03	4.4	2	0.16	-1.11	4.9	2	0.14	-1.17	5.1	2
0.5	0.3	0.28	-0.23	3.6	47.6	0.23	-0.34	4.4	2	0.19	-0.42	4.7	2	0.16	-0.47	4.9	2	0.14	-0.51	4.9	2
0.5	0.5	0.28	0.28	4.9	2	0.23	0.23	4.8	2	0.19	0.19	4.8	2	0.16	0.16	4.8	2	0.14	0.14	4.8	2
0.5	0.7	0.28	0.80	5.2	2	0.23	0.80	5.0	2	0.19	0.80	4.9	2	0.16	0.80	4.8	2	0.14	0.80	4.8	2
0.7	0.1	0.80	-0.74	5.4	19.0	0.80	-0.91	6.0	31.6	0.80	-1.03	6.8	68.0	0.80	-1.11	7.4	2	0.80	-1.17	8.0	2
0.7	0.3	0.80	-0.23	7.1	24.1	0.80	-0.34	8.1	54.7	0.80	-0.42	8.9	2	0.80	-0.47	9.4	2	0.80	-0.51	9.9	2
0.7	0.5	0.80	0.28	12.8	2	0.80	0.23	12.9	2	0.80	0.19	12.9	2	0.80	0.16	13.0	2	0.80	0.14	13.1	2
0.7	0.7	0.80	0.80	71.7	2	0.80	0.80	31.7	2	0.80	0.80	24.1	2	0.80	0.80	21.0	2	0.80	0.80	19.4	2
0.9	0.1	1.31	-0.74	14.6	18.5	1.37	-0.91	37.2	32.0	1.41	-1.03	303.6	65.5	1.43	-1.11	-83.1	2	1.46	-1.17	-44.8	2
0.9	0.3	1.31	-0.23	56.8	21.2	1.37	-0.34	-107.6	39.3	1.41	-0.42	-40.6	113.0	1.43	-0.47	-28.7	2	1.46	-0.51	-23.8	2
0.9	0.5	1.31	0.28	-29.4	46.4	1.37	0.23	-22.0	2	1.41	0.19	-19.1	2	1.43	0.16	-17.4	2	1.46	0.14	-16.3	2
0.9	0.7	1.31	0.80	-11.8	2	1.37	0.80	-12.3	2	1.41	0.80	-12.5	2	1.43	0.80	-12.5	2	1.46	0.80	-12.5	2
1.1	0.1	1.83	-0.74	-41.7	18.2	1.94	-0.91	-14.6	32.5	2.02	-1.03	-10.7	69.2	2.07	-1.11	-9.2	2	2.11	-1.17	-8.3	2
1.1	0.3	1.83	-0.23	-14.5	20.0	1.94	-0.34	-10.1	36.2	2.02	-0.42	-8.5	78.0	2.07	-0.47	-7.8	2	2.11	-0.51	-7.3	2
1.1	0.5	1.83	0.28	-8.9	27.6	1.94	0.23	-7.7	55.5	2.02	0.19	-7.1	2	2.07	0.16	-6.8	2	2.11	0.14	-6.5	2
1.1	0.7	1.83	0.80	-6.4	2	1.94	0.80	-6.3	2	2.02	0.80	-6.1	2	2.07	0.80	-6.0	2	2.11	0.80	-5.9	2
		π_{CE}=0.389				**π_{CE} =0.350**				**π_{CE} =0.328**				**π_{CE} =0.314**				**π_{CE} =0.304**			
		μ_{CE}=0.389				**μ_{CE} =0.420**				**μ_{CE} =0.437**				**μ_{CE} =0.448**				**μ_{CE} =0.456**			

$\alpha = 0.7, \beta = 0.8, \gamma = 0.5\ \theta_A = 1, \eta_{A,j,0} = 0.2, \eta_{B,j,0} = 1$

Table 4. Dynamic stability of *CE*s for different $\alpha_{i,CE}$ values: varying γ

		γ =0.2				γ =0.4				γ =0.5				γ =0.6				γ =0.8			
$\alpha_{A,CE}$	$\alpha_{B,CE}$	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period	$\beta_{A,CE}$	$\beta_{B,CE}$	TOC	Period
0.5	0.1	0.09	-1.31	5.9	10.6	0.16	-1.11	4.4	20.4	0.19	-1.03	4.4	2	0.21	-0.96	5.9	2	0.25	-0.84	6.5	2
0.5	0.3	0.09	-0.61	3.5	12.6	0.16	-0.47	3.6	2	0.19	-0.42	4.7	2	0.21	-0.37	5.3	2	0.25	-0.29	5.9	2
0.5	0.5	0.09	0.09	4.0	2	0.16	0.16	4.6	2	0.19	0.19	4.8	2	0.21	0.21	5.1	2	0.25	0.25	5.5	2
0.5	0.7	0.09	0.80	4.9	2	0.16	0.80	4.9	2	0.19	0.80	4.9	2	0.21	0.80	4.9	2	0.25	0.80	5.2	2
0.7	0.1	0.80	-1.31	6.0	11.5	0.80	-1.11	4.9	22.2	0.80	-1.03	6.8	68.0	0.80	-0.96	12.3	2	0.80	-0.84	744.7	2
0.7	0.3	0.80	-0.61	4.2	13.6	0.80	-0.47	5.4	28.7	0.80	-0.42	8.9	2	0.80	-0.37	16.2	2	0.80	-0.29	-319.7	2
0.7	0.5	0.80	0.09	3.5	26.7	0.80	0.16	8.0	2	0.80	0.19	12.9	2	0.80	0.21	24.2	2	0.80	0.25	-128.3	2
0.7	0.7	0.80	0.80	8.0	2	0.80	0.80	15.2	2	0.80	0.80	24.1	2	0.80	0.80	49.2	2	0.80	0.80	-79.0	2
0.9	0.1	1.50	-1.31	6.1	12.2	1.43	-1.11	11.9	23.5	1.41	-1.03	303.6	65.5	1.38	-0.96	-18.6	2	1.34	-0.84	-7.9	2
0.9	0.3	1.50	-0.61	4.7	14.0	1.43	-0.47	26.1	26.7	1.41	-0.42	-40.6	113.0	1.38	-0.37	-14.1	2	1.34	-0.29	-7.5	2
0.9	0.5	1.50	0.09	6.8	19.5	1.43	0.16	-160.1	39.1	1.41	0.19	-19.1	2	1.38	0.21	-11.3	2	1.34	0.25	-7.3	2
0.9	0.7	1.50	0.80	26.3	127.0	1.43	0.80	-20.5	2	1.41	0.80	-12.5	2	1.38	0.80	-9.5	2	1.34	0.80	-7.0	2
1.1	0.1	2.21	-1.31	6.2	12.7	2.07	-1.11	-51.9	24.5	2.02	-1.03	-10.7	69.3	1.97	-0.96	-6.8	2	1.89	-0.84	-4.6	2
1.1	0.3	2.21	-0.61	6.0	14.2	2.07	-0.47	-17.6	26.5	2.02	-0.42	-8.6	78.1	1.97	-0.37	-6.2	2	1.89	-0.29	-4.5	2
1.1	0.5	2.21	0.09	16.3	17.8	2.07	0.16	-10.7	32.1	2.02	0.19	-7.1	2	1.97	0.21	-5.7	2	1.89	0.25	-4.4	2
1.1	0.7	2.21	0.80	-40.2	28.5	2.07	0.80	-7.8	57.5	2.02	0.80	-6.1	2	1.97	0.80	-5.2	2	1.89	0.80	-4.3	2
		π_{CE}=0.284				**π_{CE} =0.3141**				**π_{CE} =0.328**				**π_{CE} =0.341**				**π_{CE} =0.366**			
		μ_{CE}=0.473				**μ_{CE} =0.448**				**μ_{CE} =0.437**				**μ_{CE} =0.427**				**μ_{CE} =0.407**			

$\alpha = 0.7, \beta = 0.8, \theta_A = 1,\ \theta_B = 2, \eta_{A,j,0} = 0.2, \eta_{B,j,0} = 1$