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Mean Field Game modeling of epidemic propagation

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Abstract

We consider the propagation of epidemics for which individuals have control on some parameters like their vaccination rate or social interactions. We present general properties of discrete Mean Field Games which are used to describe the behavior of individuals. Then we focus on two models deriving from the standard compartmental SIR model used in epidemiology and discuss their Mean Field Game version. The first one is a SIR model with a vaccination rate. Individuals have to choose their vaccination rate depending of the epidemic situation in order to minimize a certain cost. If individuals choose to be vaccinate too early, then the epidemic does not take off, but the cost will be high because too many people are uselessly vaccinated. On the other hand, if they do not vaccinate themselves, the epidemic grows and the cost due to risk of infection will be high too. A Mean Field Nash equilibrium at the population level is formed, and we compute this equilibrium numerically. The second one is a SIR model with a structure of social contacts : there are some settings where individuals have contacts and could be contaminated. Individuals are grouped by age and they control their rate of contacts in each setting in order to minimize a certain cost. We implement the Mean Field Game paradigm on this model and we find numerically the Nash equilibrium. Furthermore, we develop a genetic algorithm which provides an alternative route to the optimization process when the usual approach, through the Bellman equation, is not practicable.

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1 Introduction

Since many years and in particular since the beginning of the COVID-19 pandemic, the question of modeling as precisely as possible epidemics propagation is central. The most famous model used since decades is the compartmental SIR (susceptible - infected - recovery) model. Here, we make an homogeneous assumption at the society level, that is we say that all individuals have the same probability of contact with any other individual. The model is described by the following set of equations :

$$\begin{cases} \dot{S} = -\hat{\beta} S(t) I(t) \\ \dot{I} = (\hat{\beta} S(t) - \hat{\gamma}) I(t) \ , \\ \dot{R} = \hat{\gamma} I(t) \end{cases} \tag{1.1}$$

where S, I and R are respectively the proportion of susceptible, infected and recovery people in our society. $\hat{\beta}$ is the transmission rate of the disease considered and $\hat{\gamma}$ the recovery rate. The SIR model is very simple and has a lot of variations to gain in precision. The most common ones are the SIRD model (D for deceased, [11]), SIRV (V for vaccination, [12]), MSIR (M for maternally derived immunity, [8], [9]), or SIRC (C for carrier but asymptomatic, [10]) and SEIR models (E for exposed class, [4]) but there are a lot of other variations, see [8] and [9] for a more detailed literature on the subject of compartmental models.

In a lot of models, the two parameters $\hat{\beta}$ and $\hat{\gamma}$ are fixed but it is well known that at least the transmission rate $\hat{\beta}$ change a lot with time. Indeed, there are a fix part in $\hat{\beta}$ intrinsic to the disease and a changing part which depends of human interactions (which evolve with time, see for example the different lockdown periods in almost all countries in the world during the covid-19 pandemic). Thus, we can assume a time dependence $\hat{\beta}(t)$. The variations of $\hat{\beta}$ and thus of human interactions (frequency and number of contacts) are unknown a priori. This parameter is extrinsic to the model, we need some guess about it, but this guess has to be time dependent which makes extremely difficult to extract $\hat{\beta}(t)$ from experimental data as it involves the evolution of humans behaviors at society level will evolve in the future. As an example, we can see that the Institut-Pasteur in France, responsible for advising policy decisions during the COVID-19 epidemic crisis make a lot of predictions with several possible evolution for the reproduction number R_0 and the vaccination rate because they do not model the behavior of people (see their work in [13]). Instead of this, they take an empirical approach like in [14] to estimate the impact of restrictive measures on the transmission rate $\hat{\beta}$ and use it afterwards in their predictions.

We propose here to take another approach, and to turn extrinsic functional parameters of the model into intrinsic ones (e.g $\hat{\beta}$ with the simple SIR but there may be other parameters for variations of the SIR). That is we want to develop a theoretical apparatus providing a prediction for these functional parameters. In the aim of modeling humans behavior, we will use the "Mean Field Game" paradigm. Introduced by Lasry and Lions in [15], [16] and [17] and independently by M.Huang, R.P. Malhamé and P. E. Caines [18]. MFG focus on the derivation of a Nash equilibrium within a population containing a larger number of individuals. The classical way to write down the dynamic of these games is through a (coupled) system of Kolmogorov and Bellamn equations (see [19] or [1] for a complete mathematical description). They are also used by theoretical physicists, for example in the case of crowd dynamics (see [20] for an introduction to the continuous games with a physicist point of view).

Our final goal is to develop refined SIR models with a description of the society at a mesoscopic level, that is a level where there are heterogeneous groups but still far from the individual description (to make a homogeneous assumption at this level rather than at society level). Then, the idea is to obtain the functional parameters by a MFG approach. In order to cope with increase complexity our models, we will need generic methods to solve our Mean Field Games because the associated Bellman equations with the refined models might be very complicated to solve and even to derive. This is why we will develop genetic algorithms which can find numerically the solution of the Mean Field Game without resorting to the Bellman equation.

The question of control of epidemics is often modeled through combination of isolation and vaccination strategies. That is the number of contacts of individuals and the vaccination are the main levers to impact epidemic evolution. Some work has already been done from a global point of view, for example [21] and [22] but they do not explore the individual behavior. Some other papers tries to evaluate the best individual response without the Mean Field Game paradigm ([23], [24]).

In our work, we keep the two main approaches to control epidemics, that are isolation and vaccination

strategies and we will study them within MFG. This report is organize as follow : after an introduction to discrete Mean Field Games which relies on the paper [1], we study a first paper of Turinici [2] which uses Mean Field Games in order to determine a vaccination campaign. We present the main ideas, the physical interpretations of the results of [2] and we implement the numerical part of the paper (which is mainly focus on mathematical properties). Then, we introduce genetic algorithms with a description based mainly on the book on evolutionary computing [3] and we apply it in order to recover the results of the paper [2] without the Bellman derivation. In a fourth part, we introduce a SIR model with a structure of social contacts proposed by [4] and [25] to get a more detailed description of the society, at a mesoscopic scale. We include the Mean Field Game paradigm on this scheme following the spirit the paper of Turinici [5] who did it for a classical SIR model. We make numerical simulations in order to understand the behavior of our model and we find the Nash equilibrium with two different methods (inspired by [6] and [7]). Finally, we conclude our work by proposing some possible enhancement to our model and some openings to other models.

2 Discrete Mean Field Games

In this section, we provide a general description of discrete Mean Field Games, based mainly on the paper [1]. Reader can refer to this paper for a rigorous mathematical approach. Symbol $\equiv$ in an equation means "by definition" of the left or right hand side in the following.

2.1 General framework of discrete Mean Field Games

Mean Field Games (MFG) are part of game theory in mathematics. We consider a game during a certain (continuous) time T with a large number of players (or agents) N. At a certain time t, each player is completely described by his state which could be continuous (for instance in the case of crowd dynamics, with his position $\vec{x}$, see [20]) or discrete (e.g in a case of a limited number of choices for the players, i). We will deal only with discrete games in the following. We have a finite number of states $a = 1, ..., d$ and we denote by $\theta^a(t) = \frac{k_a}{N}$ the proportion of players in the state a at time t (there is k_a players in a here) with : $\sum_a \theta^a(t) = 1$. In our SIR model above, the N players are the individuals of our population, T the characteristic time at the end of the epidemics and the discrete states are the different compartments : susceptible, infected and recovery. The associated proportions are respectively S, I and R (with $S + I + R = 1$). In the following, we will also denote by S, I and R the different compartments if there is no ambiguity. The players have control over some parameters $\alpha(t)$ in order to modify their own state, but only at a probabilistic level. Indeed, α takes the form of a transition rate between states (Markov process). In the example above, the control parameter could be the transmission rate $\hat{\beta}(t)$ (as it is done in [5]) and thus susceptible individuals (at t) control their probability to be infected at $t+dt$ which is $\hat{\beta}(t) \cdot I(t) \cdot dt$ but have not directly control over their next state.

We consider that all players are equivalent (symmetric assumption), therefore the proportion of players in each state θ^i is sufficient to describe the game and we can consider a reference player to study it. But this problem is still very hard to solve because we have N players in interaction (many body game theory). In order to turn the problem into a solvable one, we can make the following mean field approximation : since the number of players is very large, we consider that players are only sensitive to $\bar{\theta}^i(t) = \langle \theta^i(t) \rangle$ (stochastic average), that is we consider that N is sufficiently large to neglect fluctuations around $\bar{\theta}^i(t)$. Now we can deal with $\bar{\theta}^i(t)$ which is a deterministic quantity. Actually, this mean field approximation was already done in the SIR model (1.1) ; we wrote indeed S, I and R as deterministic quantities, with a well known variation at each step. In reality, there are stochastic fluctuations in $\sqrt{N}$ for these quantities but we wrote directly $\bar{S}, \bar{I}, \bar{R}$.

Since all players are equivalent, we denote by $\beta(t)$ the strategy of all other players (we call strategy a certain choice of control parameters during the game), $\beta(t)$ is a matrix of transition rates. We stress that β is a strategy and not the transmission rate $\hat{\beta}$ of the SIR model. The problem is said to be consistent (this will be referred to below as a "Nash equilibrium") if the best strategy α^* for the reference player is $\beta : \alpha^*(\beta) = \beta$ because by symmetry this strategy β will be the best for each player. This is an equilibrium point in the sense that nobody has an interest in changing his strategy if everybody else keep the strategy β (which is the general definition of a (symmetric) Nash equilibrium).

Since the problem is described by a Markov process with a continuous time, we naturally obtain the dynamical equation of θ^i, also known as the Kolmogorov [1] equation (we write $\theta^i(t)$ instead of $\bar{\theta}^i(t)$ for

1. In reference to continuous games where we deal with the original Kolmogorov equation

simplicity)

$$\frac{d\theta^i(t)}{dt} = \sum_j \theta^j(t)\beta_{ji}(t) \ , \tag{2.1}$$

with $\theta(0) = \theta_0$. $\beta_{ij} \geq 0$ is the transition rate from i to j and θ is the vector of all the θ^i. Note that for the SIR model, (2.1) correspond to (1.1) and we can directly read on it what are the rates $\beta_{ij}(t)$ in this case (it could be that only some rates $\beta_{ij}(t)$ are controlled by players). Remark : by convention, $\beta_{ii}(t) \equiv -\sum_{j\neq i}\beta_{ij}(t)$ (that is the rate to stay in i is the opposite of the total rate to leave i).

During the game, each player pay a certain instantaneous cost c which depends of his own state but also on the state of all other players. For a given player, the total cost incurred is the integral over time of his instantaneous cost (plus eventually a final cost) for a certain strategy α. Denoting by i_t the state of our reference player at t and assuming that he knows $\theta(s)$ for all $s \geq t$, the total cost paid between t and T has the following general form starting with $i_t = i$ and with θ given (this is also the case in the following equations) :

$$u^i_\theta(t,\alpha) = \mathbb{E}^\alpha_{i_t=i}\left[\int_t^T c(i_s,\theta(s),\alpha(s))\mathrm{ds} + \Psi^{i_T}(\theta(T))\right] \ . \tag{2.2}$$

The expectation here is on i_s which is the only undetermined quantity (the subscript α is here to say : "expectation for a given strategy α"). The goal for each player is to minimize their own total cost. In the SIR model, a strategy is what is chosen for $\hat{\beta}(t)$ at each t. We can imagine a cost where we pay a fix price r_I at t if we are infected by the disease at t and a continuous price at each time t if we choose to reduce our social contacts $\hat{\beta}(t)$. Thus, the cost depends of the risk of infection and therefore of the situation of all other individuals (how many infected are there ?). Naturally, all individuals want to minimize their total cost during the epidemic. To minimize this function $u^i_\theta(t,\alpha))$, each player choose the best strategy α^* to obtain the value function : the minimum price (on average) paid by a player between t and the end of the game T :

$$u^i_\theta(t) = \min_\alpha \left(u^i_\theta(t,\alpha)\right) = u^i_\theta(t,\alpha^*) \ . \tag{2.3}$$

The goal of the problem is to find $\alpha^*(t)$ (i.e the best strategy of players) and $\theta(t)$ (the evolution of proportion of players in each state).

2.2 The Hamilton-Jacobi Bellman equation

In order to get the best strategy α^* we derive "physically" the equation followed by the value function $u^i_\theta(t)$ (see [1] for a rigorous derivation). By definition of the Markov process, we have (at first order with $dt \longrightarrow 0$) :

$$P(i_{t+dt} = j | i_t = i) = \alpha_j(t)\cdot dt \quad (j \neq i) \tag{2.4}$$

$$P(i_{t+dt} = i | i_t = i) = 1 - \sum_{j\neq i}\alpha_j(t)\cdot dt \ . \tag{2.5}$$

We use an argument "à la Bellman" , writing $u^i_\theta(t)$ in terms of $u^i_\theta(t+dt)$ (see (2.2) and (2.3) for the definitions)

$$u^i_\theta(t) = \min_{\alpha(t)\in\mathbb{R}^d} \mathbb{E}_{i_t=i}\left[u^{i_{t+dt}}_\theta(t+dt) + c(i,\theta(t),\alpha(t))\cdot dt\right] \ . \tag{2.6}$$

Then, if we write explicitly $\mathbb{E}$ with the probabilities of the Markov process :

$$u^i_\theta(t) = \min_{\alpha(t)\in\mathbb{R}^d}\left[\sum_{j\neq i}\alpha_j(t)\cdot dt\cdot u^j_\theta(t+dt) + \left(1 - \sum_{j\neq i}\alpha_j(t)\cdot dt\right)\cdot u^i_\theta(t+dt) + c(i,\theta(t),\alpha(t))\cdot dt\right], \tag{2.7}$$

where the first term corresponds to $i_{t+dt} = j$ for some j and the second term to $i_{t+dt} = i$. Then, since $u^i_\theta(t+dt)$ is independent of $\alpha(t)$, this gives the Hamilton-Jacobi Bellman equation :

$$-\frac{du^i_\theta}{dt} = \min_{\alpha(t)\in\mathbb{R}^d}\left[c(i,\theta(t),\alpha(t)) + \alpha(t)\cdot\Delta_i u_\theta(t)\right] \ , \tag{2.8}$$

where we define the difference operator on i as : $\Delta_i u \equiv (u^1 - u^i, ..., u^j - u^i, ..., u^d - u^i)$ whith $u \in \mathbb{R}^d$. Moreover,

$$\alpha^* \equiv \underset{\alpha(t)\in\mathbb{R}^d}{\mathrm{argmin}}\left[c(i,\theta,\alpha(t)) + \alpha(t)\cdot\Delta_i u_\theta(t)\right] \tag{2.9}$$

is the optimal Markovian control. Remark : below the Hamilton-Jacobi-Bellman [2] equation is sometimes called (for simplicity) the Bellman equation.

2.3 Mean field Nash equilibrium

We obtain a Nash equilibrium when the best strategy of the reference player is β itself, that is at each time s, for all j, i

$$\beta_{ji}(s) = \alpha_i^*(\Delta_j u(s), \theta(s), j) \ . \tag{2.10}$$

The Nash equilibrium is therefore characterized by the system of Kolmogorov and Hamilton-Jacobi-Bellman equations (replacing β_{ji} by (2.10))

$$\begin{cases} \dfrac{d\theta^i}{dt} = \displaystyle\sum_j \theta^j \alpha_i^*(\Delta_j u, \theta, j) \\ -\dfrac{du^i}{dt} = \displaystyle\min_{\alpha(t)\in\mathbb{R}^d} \left[c(i, \theta(t), \alpha(t)) + \alpha(t) \cdot \Delta_i u_\theta(t)\right] \ , \end{cases} \tag{2.11}$$

together with the initial-terminal conditions

$$\theta(0) = \theta_0 \qquad u^i(T) = \Psi^{i_T}(\theta(T)) \ . \tag{2.12}$$

This form the initial-terminal value problem (ITVP) for the Mean Field Game. The solution of this problem is the solution of the MFG. Remark : in general, we have some final cost $\Psi^{i_T}(\theta(T))$ but in the case of epidemic propagation we will take $\Psi^{i_T}(\theta(T)) = 0$ in the following. Under some monotonicity assumptions on the cost, authors of [1] shows that there exists a unique solution (θ,u) to ((2.11) - (2.12)), that is there is a unique Nash equilibrium.

2.4 Numerical methods to find the Nash equilibrium

We describe here two numerical methods we have implemented to find the Nash equilibrium.

1^{st} method : Inductive sequence

The Nash equilibrium correspond to a fixed point of the function $f_{\theta_0} : \beta \longrightarrow \alpha^*$ (β global strategy, α^* the best individual strategy, and θ_0 the initial conditions). The idea is to use an inductive sequence $U_{n+1} = f_{\theta_0}(U_n)$ to reach this point.

To compute $f_{\theta_0}(U_n)$, we apply the following general iterative scheme, with $U_n = \beta_n$ a global strategy (and our inductive sequence)

$$\beta_n \underset{Kolmogorov}{\longrightarrow} \theta(\beta_n) \underset{Bellman}{\longrightarrow} \tilde{f}_\theta(\beta_n) = \alpha_n^* \underset{Symmetric}{\longrightarrow} \beta_{n+1} = \alpha_n^* = f_{\theta_0}(\beta_n) \ . \tag{2.13}$$

On the first step, we start from a global strategy β_n and we compute numerically $\theta(\beta_n)$ using (2.1). Then, we solve numerically or analytically backwards in time the Bellman equation (2.8) to find α_n^* the best individual strategy in response to β_n (this correspond to $\tilde{f}$). Finally by a symmetric assumption on players, each player choose the same best strategy, therefore the new global strategy is simply $\beta_{n+1} = \alpha_n^*$. We continue this scheme until the convergence of (β_n). If this sequence converges, we reach indeed a fixed point : $f_{\theta_0}(\beta) = \beta$.

Picard-Banach fixed point theorem provides us with sufficient conditions for convergence : it states that every contractive mapping on a complete metric space (the metric is denoted d) has a unique fixed point (an application is said to be contractive on E if there exists $0 < k < 1$ such that : $\forall (x, y) \in E$, $d(f(x), f(y)) \leq k \cdot d(x, y)$). The theorem gives also that every inductive sequence of the above form converges to the fixed point geometrically : $d(U_n, l) \leq k^n \cdot d(U_0, l)$ (see [6]). Advantages of this method are that the convergence is fast (geometrical) and sure (we reach the Nash equilibrium certainly). But in order to use it, we have to be able to compute $f_{\theta_0}(\beta)$ analytically or numerically. Furthermore, if f_{θ_0} is not contracting, (U_n) will not always converge.

If (β_n) does not converges, one possibility is to rather use $\beta_{n+1} = a \cdot f_{\theta_0}(\beta_n) + (1 - a) \cdot \beta_n$ with $a \in [0, 1]$ which is a more robust sequence convergence cannot be guaranteed here either, even with small values of a.

2. This equation can be seen as a discrete version of a Hamilton-Jacobi equation

In fact such a sequence converges whenever the original sequence (β_n) does, and works better for f decreasing with a large negative slope, because we can avoid oscillations of the original sequence.

2^{nd} method : Gradient descent
The second method is to use a kind of gradient descent on the first variable of the cost (ie the individual strategy). This is an equilibrium flow descent with an explicit Euler discretization (explained in [5] and defined rigorously in [7]). Starting from a certain strategy α_0, we have :

$$\alpha_{n+1} = \alpha_n - h \cdot \nabla_1 u(\alpha_{n+1}, \alpha_n) \ . \tag{2.14}$$

The first variable of the cost u is for the individual strategy (here α_{n+1}) and the second variable is for the global strategy $\beta = \alpha_n$. h is the step of the gradient descent. If this sequence converges until a certain α, then $\beta = \alpha$ (we can said that it is a Nash "candidate" because individual and global strategies are the same) and $\nabla_1 u(\alpha, \alpha) = 0$, we have reached at least a local minima at least. The authors of [5] use this method successfully to find the Nash equilibrium of their model. The issue of this method is that we are not certain to reach a Nash equilibrium, because a Nash equilibrium correspond to a "Nash candidate" which is a global minimum of u with respect to the first variable α (that is the translation of "the best individual strategy in response to the global strategy $\beta = \alpha$ is α itself").

3 SIR model with a vaccination campaign [Following [2]]

3.1 Theoretical framework

We present here a first application of Mean Field Games to epidemic propagation made by Turnici in [2] with 4 states : the Susceptible - Infected - Recovery - Vaccinated model. We add a global vaccination rate $u(t)$ to the SIR model which was presented in section 1 :

$$\begin{cases} \dot{S} = -\hat{\beta} S(t) I(t) - u(t) \\ \dot{I} = (\hat{\beta} S(t) - \hat{\gamma}) I(t) \\ \dot{R} = \hat{\gamma} \cdot I(t) \quad , \\ \dot{V} = u(t) \end{cases} \tag{3.1}$$

with the initial conditions : $S(0) = S_0$, $I(0) = I_0$, $R(0) = 0$, $V(0) = 0$. S, I, R, V are respectively the proportion of susceptible people, infected people, recovery people and vaccinated people. We define ψ_I the probability that an individual which is not vaccinated is infected before t, which thus fulfill

$$\dot{\psi_I} = (1 - \psi_I(t)) \hat{\beta} I(t) \ . \tag{3.2}$$

$\hat{\beta}$ is the transmission rate of the disease and $\hat{\gamma}$ the recovery rate as in (1.1), which one are taken fixed here. We assume $u(t) \leq u_{max}$ which is the maximal vaccination rate of the population (because of a maximal capacity of vaccination) ; u is such that $u(t) = 0$ if $S(t) = 0$. In [2], the authors derive some mathematical properties of such a model (like the existence and uniqueness of a Nash equilibrium). We propose here to recap their main results and add some physical interpretations.

The author's idea is to allow people to choose their own vaccination rate $\lambda(t)$, this is the control parameter. Then, $u(t)$ will be simply the sum of individual choices, $u(t) = S(t) \cdot \lambda(t)$ (only susceptible could be vaccinated). Thus, this model correspond to a discrete Mean Field Game (where S, I, R and V are the possible states). We define ψ_v the probability that an individual which is not infected is vaccinated before t,

$$\dot{\psi_v} = (1 - \psi_v) \lambda(t) \ . \tag{3.3}$$

We suppose that all individuals are equivalent, in the sense that they all search to minimize the individual cost function.

One way to build this cost is the following : between t and $t + dt$, we have a probability $(1 - \psi_I(t))(1 - \psi_v(t)) \hat{\beta} I(t) dt$ to be infected (we must be susceptible at t) and in that case we pay the price of infection $r_I e^{-Dt}$. On the other hand, we have a probability $(1 - \psi_I(t))(1 - \psi_v(t)) \lambda(t) dt$ to be vaccinated (we also have to be susceptible at t) and we pay then $r_v e^{-Dt}$. The factor e^{-Dt} is included for each cost because one prefers to be infected (or suffer of undesirable effects) late rather than immediately. D is called the discount factor

which is supposed to be constant. r_I is the cost of the infection (quite intuitive) and r_v is the cost of the vaccination (in money or undesirable effects), both are supposed to be constant (they can evolve in reality). Thus we obtain a total cost function J :

$$J \equiv \int_0^\infty (1-\psi_I(t))(1-\psi_v(t)) \cdot (\hat{\beta} I(t) r_I e^{-Dt} + \lambda(t) r_v e^{-Dt}) dt \ . \tag{3.4}$$

We can check that we recover the formula of the paper [2] by identifying $\dot{\Phi}_I$ in the last formula and integrating by parts :

$$J(\phi_v, u) = r_I \Phi_I(\infty) + \int_0^\infty \left[r_I(\Phi_I(t) - \Phi_I(\infty)) + r_v e^{-Dt}(1-\psi_I(t)) \right] d\psi_v(t) \ , \tag{3.5}$$

where $\Phi_I(t)$ is defined as

$$\Phi_I(t) \equiv \int_0^t e^{-Ds} \dot{\psi_I}(s) ds = \int_0^t e^{-Ds}(1-\psi_I(s)) \hat{\beta} I(s) ds \ , \tag{3.6}$$

which correspond to the probability to be infected before t weighted by the discount factor.

This form of the cost can be interpreted by considering each term separately : first, without vaccination, we can say that an individual pay a price $r_I e^{-Dt}$ when he is infected. But the time of infection is uncertain, so we take the mean (over time of infection) of the price paid to construct the cost function :

$$J_I = \int_0^\infty r_I \cdot e^{-Ds} \dot{\psi_I}(s) ds = r_I \Phi_I(\infty) \ , \tag{3.7}$$

where the last term $\dot{\psi_I}(s)ds$ correspond to the probability to be infected between s and $s+ds$. Considering now the impact of vaccination, the positive effect is that after a vaccination at time t, one will pay only $r_I \Phi_I(t)$ instead of $r_I \Phi_I(\infty)$ because the probability of infection vanishes after t. Taking the average over the time of vaccination, this gives a positive impact (so a negative contribution to the cost) :

$$J_{VI} = \int_0^\infty (r_I \Phi_I(t) - r_I \Phi_I(\infty)) d\psi_v(t) \ < 0 \ , \tag{3.8}$$

where $d\psi_v(t)$ is indeed the probability of vaccination at time t for people who were not infected before t (because the cost is only reduced for these people).

Finally, we add the cost of vaccination (undesirable effect). One pay a price $r_v e^{-Dt}$ for a vaccination at t and the probability of vaccination at t is here $(1-\psi_I(t)) \cdot (1-\psi_v(t)) \cdot \lambda(t) \cdot dt = (1-\psi_I(t)) \cdot d\psi_v(t)$ because people who have already been infected will be never vaccinated. Thus the cost of the vaccination is :

$$J_V = \int_0^\infty r_v e^{-Dt} (1-\psi_I(t)) d\psi_v(t) \ , \tag{3.9}$$

And we obtain indeed : $J \equiv J_I + J_{VI} + J_V$.

Let us denote by $g(t)$ the integrand of (3.5) :

$$g(t) = r_I(\Phi_I(t) - \Phi_I(\infty)) + r_v e^{-Dt}(1-\psi_I(t)) \ , \tag{3.10}$$

with its derivative :

$$\dot{g}(t) = e^{-Dt}(1-\psi_I(t)) \cdot [(r_I - r_v)\hat{\beta} I(t) - r_v D] \ . \tag{3.11}$$

Considering the beginning of optimization at t, the minimization of the cost function J correspond to the minimization of V :

$$V(t) \equiv \inf_\lambda \left[\int_t^\infty g(\tau) d\psi_v(\tau) \right] \ . \tag{3.12}$$

Physically, this means that in order to minimize V, people will wait for $g(t)$ to become negative to get vaccinated ($\lambda(t) > 0$). This is natural because $g(t) \leq 0$ correspond to the fact that the vaccination represent a gain (in statistical average) for people who are still susceptible. We can briefly analyse the behavior of g : since $I(0)$ is small (beginning of the epidemic), $\dot{g}(0) < 0$ and g reaches an extrema when $I(t) = \frac{r_v D}{(r_I - r_v)\cdot\hat{\beta}} \equiv I_{thr}$ (above this infection threshold there is a vaccination campaign). If $I_{max} \geq I_{thr}$ (I_{max} is the maximum of I during the epidemic), g decreases until $I(t) = I_{thr}$ (I increases with the beginning of the epidemic), increases until $I(t) = I_{thr}$ for the second time (passing by $I(t) = I_{max}$ at the inflection point of g) and decreases finally

(end of the epidemic, $I(t) \longrightarrow 0$). We can remark that in all cases, $g(t) \underset{t \longrightarrow \infty}{\longrightarrow} 0$. If $I_{max} \leq I_{thr}$, g decreases and is always positive, there is no vaccination campaign. (NB : we consider only the relevant case where $g(0) \geq 0$)

We now show how that V obeys the following Hamilton-Jacobi-Bellman equation (HJB), while $S(t) > 0$:

$$-\left[\dot{V} + \min_{\lambda(t) \in [0, \frac{u_{max}}{S(t)}]} \left[(g(t) - V(t)) \cdot \lambda(t)\right]\right] = 0 \ . \tag{3.13}$$

We propose an argument "à la Bellman" to derive the above formula (the reasoning is essentially the same as in section 2.2). If we denote by i_t the state of an individual i at t, then (3.12) correspond to the value function of a Mean Field Game describe by two states : the susceptible (which therefore can be vaccinated) and the vaccinated. Indeed, if $g(t)$ is the cost of the vaccination, λ our control parameter, then the argument of inf in (3.12) is the total cost of such a game with two states. The cost function $C(t)$ is here dependent of the state at time $t+dt$. If we are susceptible at t and vaccinated at $t+dt$, then we pay $g(t)$ at time t. If we are still susceptible at $t+dt$ we pay nothing at t (nothing happened). $i_t = S$ if i is susceptible at t and $i_t = V$ if i is vaccinated.

We have a Markov process, so $P(i_{t+dt} = V | i_t = S) = \lambda(t)dt$ and $P(i_{t+dt} = S | i_t = S) = 1 - \lambda(t)dt$. Furthermore, if i is vaccinated at $t + dt$, then the value function $V(t + dt) = 0$ since $\lambda(t^{'}) = 0$ for $t^{'} \geq t$ (the value function is unchanged if we are still susceptible, because nothing happened in the interval). Thus, considering only the relevant case where $V(t) \neq 0$ (that is $i_t = S$), we obtain

$$V(t) = \min_{\lambda(t)} \left[\mathbb{E}_{i_t = S}\left[V_{i_{t+dt}} + C(t, i_{t+dt})\right]\right] \ , \tag{3.14}$$

which gives, writing explicitly $\mathbb{E}$ and using the expression for the Markov process

$$V(t) = \min_{\lambda(t)} \left[(1 - \lambda(t)dt) \cdot V(t + dt) + \lambda(t)dt \cdot g(t)\right] \ . \tag{3.15}$$

At first order, $\lambda(t) \cdot dt \cdot V(t + dt) = \lambda(t) \cdot dt \cdot V(t)$ and since $V(t + dt)$ is independent of $\lambda(t)$

$$-\frac{dV}{dt} = \min_{\lambda(t)} \left[(g(t) - V(t)) \cdot \lambda(t)\right] \ . \tag{3.16}$$

An equilibrium is realized when the global vaccination strategy $u(t)$ matches with the individual choices $\lambda(t)$. Since all individuals are equivalents, this requires : $u(t) = S(t)\lambda(t)$. It is a consistent equation translating the Nash equilibrium. Indeed, if the best individual strategy in response to u is λ such that $u = S \cdot \lambda$, we are at equilibrium.

The authors of [2] shows under monotonicity assumptions that (3.13) has a unique solution V. Then, they try a solution of the form $\lambda(t) = \frac{u_{max}}{S(t)}$ for $t \in [t_1^*, t_2^*]$ (and $\lambda(t) = 0$ otherwise) where $t_1^* < t_2^*$ such that $g(t_1^*) = V(t_1^*)$ and $g(t_2^*) = V(t_2^*) = 0$. This is a particular solution of (3.13) and thus the solution because of the uniqueness. There is no analytical expression of the times (t_1^*, t_2^*), they must be estimate numerically.

They finally find a Nash equilibrium solution of the form $u(t) = u_{max}$ when $t \in [t_1^*, t_2^*]$ and $u(t) = 0$ otherwise. Thus, all the dynamics depends on (t_1^*, t_2^*), we can therefore reduce our search space of different strategies (individual and global strategies) to a space of two dimensions to find the Nash equilibrium easily.

3.2 Numerical approach

We propose to illustrate (and check) numerically the results of the paper [2]. We use the inductive sequence defined in section 2.4 with $a = 0.1$ to find the Nash equilibrium. That is we implement the scheme (2.13) with $u_n(t)$ the sequence of global vaccination rates :

$$u_n(t) \underset{Kolmogorov}{\longrightarrow} I(u_n) \underset{Bellman}{\longrightarrow} f_{I(U_n)}(u_n) = \lambda_n \underset{Symmetric}{\longrightarrow} u_{n+1}(t) = a \cdot S(t) \cdot \lambda_n(t) + (1-a) \cdot u_n(t) \ . \tag{3.17}$$

Once we reach the convergence of u_n, we can plot the time evolution (free scale, we will say "weeks" in the following for concreteness) of all compartments of our system. We observe in fig 1 that the vaccination

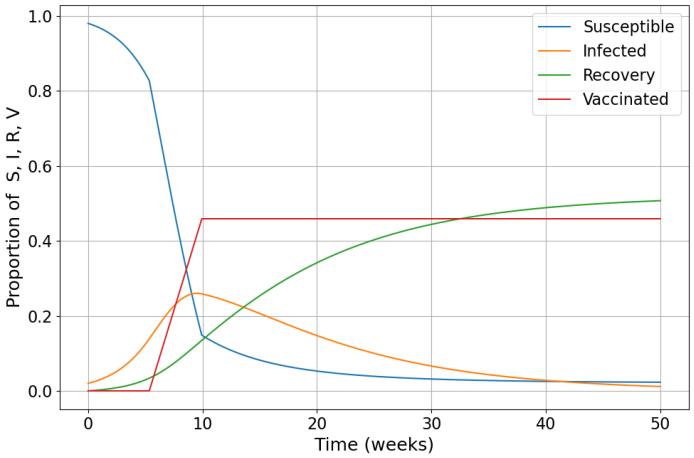

FIGURE 1 – Nash equilibrium of the SIR model with a vaccination campaign. Susceptible are (blue), Infected (orange), Vaccinated (red) and Recovery (green).
Parameters : $[S_0 = 0.98,\ I_0 = 0.02,\ r_v = 0.2,\ r_I = 0.4,\ D = 0.1,\ \hat{\beta} = 0.5,\ \hat{\gamma} = 0.1,\ u_{max} = 0.1\ ]$

campaign appears between approximately 5 and 10 weeks. The vaccination slows down quickly the peak of infected people and the total quantity of infected people is much lower than in the case without vaccination (this quantity correspond to the proportion of recovery people at the end of the epidemic). To be convince, we can compare with the classical SIR model without vaccination (and otherwise the same parameters) shown on fig 2. We can see that the proportion of people "saved" by vaccination is almost one half of the total po-

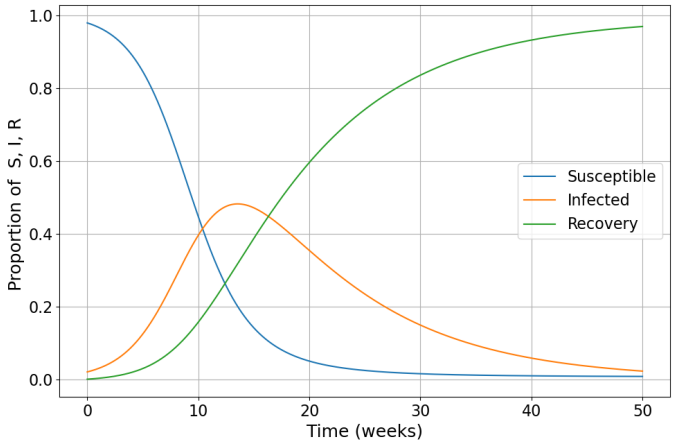

FIGURE 2 – Typical evolution of the SIR model without vaccination. Susceptible are (blue), Infected (orange), and recovery (green). Parameters : $[S_0 = 0.98,\ I_0 = 0.02,\ \hat{\beta} = 0.5,\ \hat{\gamma} = 0.1\ ]$

pulation, which is more than the total number of vaccinated people. This illustrates that vaccination protect globally the population as it allows to terminate quickly the epidemic. Naturally before vaccination, the two figures are the same.

Further insights can be obtained by considering the value function $V(t)$ and the vaccination cost $g(t)$ on fig 3, focusing in particular on the vaccination campaign. The global behavior that was expected in the discussion after (3.11) is recovered. Vaccination campaign occurs between t_1^* and t_2^*. The vaccination campaign begins a little bit before the first extrema of g, that is when $I(t)$ increases quickly and is just below I_{thr} (which is the value of I where $\dot{g}(t) = 0$). People see the fast evolution of $I(t)$ and consider that it is time to be vaccinated (because the risk of infection is high). At t_2^*, we are just after the inflection point of g, which

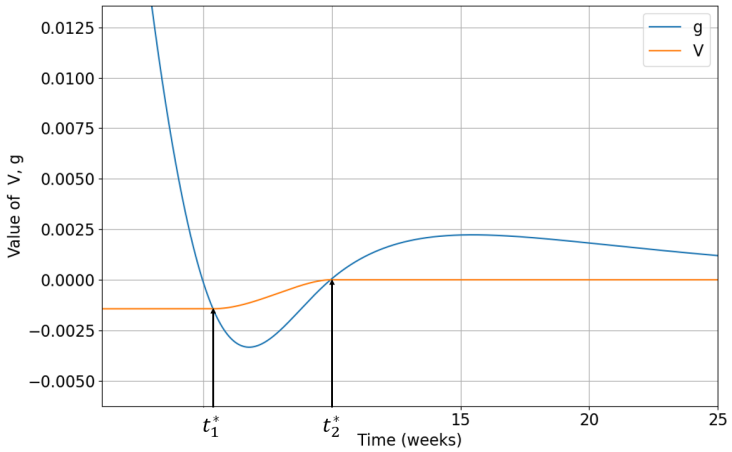

FIGURE 3 – Evolution of the vaccination cost g (blue) and the value function V (orange) around the vaccination campaign with the same parameters as fig 1

correspond to $I = I_{max}$ (see fig 1). Therefore, people understand that now the epidemic decreases, the risk of being infected is lower and they can stop the vaccination.

On the other hand if the epidemic is not virulent enough, there is no vaccination campaign. This is illustrated in fig 4 in which $\hat{\beta}$ has been changed from 0.5 to 0.2 (we use a longer time scale because the epidemic dynamics is slower). Now since the epidemic does not take off ($I_{max} < I_{thr} = 0.5$), the best strategy

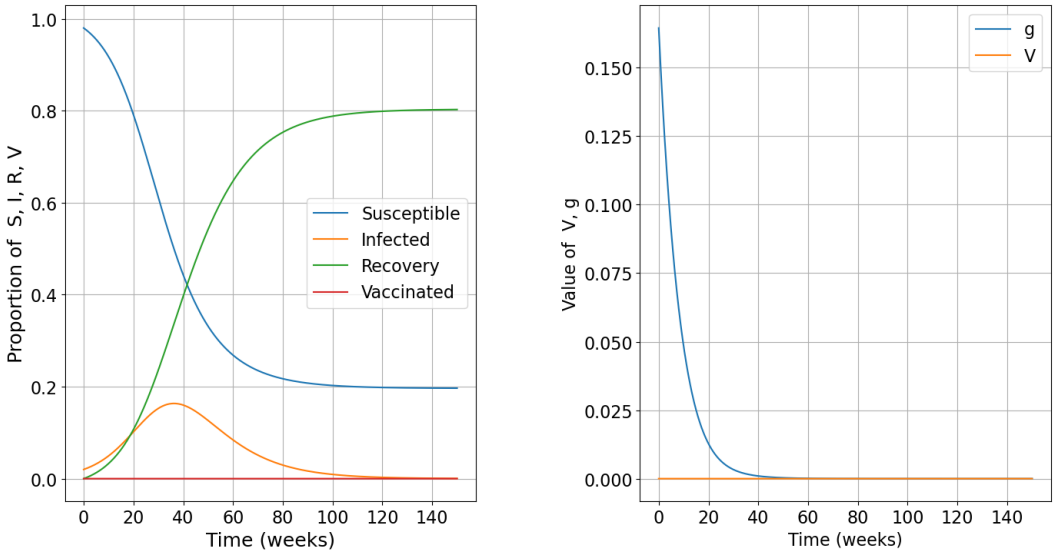

FIGURE 4 – Left : Nash equilibrium of the SIR model without vaccination campaign (same color code as figure 1). Right : evolution of g (blue) and the value function V (orange).
Parameters : [$S_0 = 0.98$, $I_0 = 0.02$, $r_v = 0.2$, $r_I = 0.4$, $D = 0.1$, $\hat{\beta} = 0.2$, $\hat{\gamma} = 0.1$, $u_{max} = 0.1$]

is to take $\lambda(t) = 0 \ \forall t$. We do not have any vaccination here : $V(t) = 0$. Furthermore, we can check that $g(t) \geq 0 \ \forall t$, that is the vaccination represent an absolute cost for individuals at each time.

4 Genetic algorithms

4.1 Theoretical framework

We introduce a particular type of algorithm that are the genetic algorithms (GA) summarizing here the chapter of [3] on this subject. GA are a way to solve an optimization problem, that is to find $\vec{x}_{opt}$ such that $Q(\vec{x}_{opt}) = \max_{\vec{x}}(Q(\vec{x}))$ for a certain function Q which is call the quality or the "fitness" function. This could be very useful for Mean Field Games where the Bellman equation cannot be derived, that is Mean Field Games where the "Bellman" step in the general scheme presented in (2.13) cannot be realized. Therefore we will have to find the best individual response to a global strategy numerically without the Bellamn formalism. We solve directly the problem from the cost function (Q is thus the cost function up to a minus sign). The general idea is to test some individuals strategies, compare the (individual) cost between them and select the best ones in order to converge to the best individual strategy by imitating the natural selection.

Genetic algorithms (and more generally evolutionary computing) are based on the natural selection principle. They follow the following scheme :

— We define our quality function $Q(\vec{x})$ that we want maximize.

— We define our search space. That is the space of parameters that we want to optimize. It could be directly the space where $\vec{x}$ lives but it could be also a representation of $\vec{x}$.

— We start with a certain (random) distribution of individuals (that are "candidates" $\vec{x}$ to maximize our quality function Q)

— We make a "natural" selection of individuals : those with the best quality function are more adapted to their environment and have more chance to survive. Individuals who survive are called "parents".

— We create several offspring from parents. We make a mix in the characteristics of two (or more) random parents to create a new individual. Furthermore, to get diversity and explore our space, we add mutation : we add a little random value at one or more parameters of each offspring.

— Then we have a new generation of individuals and we can iterate the procedure in order to find $\vec{x}_{opt}$.

This type of algorithm is general. We have to choose the number of selected individuals (i.e the number of parents at each generation), the number of offspring, the number of initial candidates. We also have to define the offspring procedure (the way that we mix the characteristics of parents) and the mutation operator (what characteristics do we change, and how many). All these quantities have an impact on the efficiency of the algorithm. We have to adapt it to each problem. As an example, if the search space is very large, we should take a large number of offspring compare to the number of parents to explore the space (we generally work with a fix number of individuals at each generation for convenience, but it is not necessary).

The algorithm terminate after a fix number of iterations (generations) or when we are near enough from the optimum of Q (if we know it). We can also imagine other process to end up the algorithm.

4.2 Application to the SIR model with vaccination

As a first application, we propose to recover the results of [2] with GA. Authors of [2] derive and solve the Bellman equation for V in order to find the best individual vaccination strategy λ^*. This is possible because the cost function J (see (3.5)) is clear enough. But we can imagine situations where the cost function is very complex and it is not possible to derive a Bellman equation. Thus, we want realize $f_I : u \longrightarrow \lambda^*$ with a genetic algorithm instead of use the Bellman paradigm.

Thus, we start from J, u (see section 3.1) and we want to recover λ^* (which is of the form $\lambda^* = \frac{u_{max}}{S(t)}$ on $[t_1^*, t_2^*]$ and 0 otherwise).

We describe the general scheme applied to our problem :

— Our quality function is $Q(\lambda) = -J(\lambda, u)$. We want maximize it for a certain global strategy u.

— Our search space is : $\mathbb{S} = \{0 \leq \lambda(t) \leq \frac{u_{max}}{S(t)}\ ,\ 0 \leq t \leq T\}$ which correspond to the space of all possible individual strategies. After discretization of time, we obtain a huge space with np finite dimensions (np is the number of discretization points)

— Our initial candidates are taken randomly. We take 200 candidates per generation.
— As a first guess, we select simply the candidates with the best quality function. We choose 10 to 50 parents. (and so 190 to 150 offspring).
— Offspring procedure : we choose two parents A and B randomly and for each t, $\lambda(t)_{off} = \frac{\lambda_A(t)+\lambda_B(t)}{2} + \frac{u_{max}}{S(t)} \cdot random(-1,1)$. We define like this the offspring procedure and the mutation operator.
— We end up the process after a certain number of iterations (ie a certain number of generations)

The problem is that $\mathbb{S}$ is huge because we have np dimensions ($\simeq 4000$). Thus, when we take initial candidates $\lambda(t)$ randomly, the quality function $Q(\lambda)$ is very bad (worse than $\lambda(t) = 0\ \forall t$) and grows very slowly at each generation. Actually, the algorithm explore a huge part of $\mathbb{S}$ which is flat and we are far from the peak of Q because $\lambda(t)$ has to be ordered for that.

To solve this issue, we propose to first take $\lambda(t) = C = cste$ on $[0, T]$ and make an optimization on this C. When the best C is find, we divide the interval into two smaller intervals $[0, \frac{T}{2}]$ and $[\frac{T}{2}, T]$ and we take $\lambda(t) = cste$ on each one. For the first offspring generation with "two values", we make mutations on each interval to get new candidates with different values on $[0, \frac{T}{2}]$ and on $[\frac{T}{2}, T]$. This method works because the search space is considerably diminished (only one dimension for the first step, then two, etc). When the algorithm converges with two intervals, we cut each interval in two equals parts and we repeat the procedure. We define a threshold in order to know if we reached the maximum of Q with a certain number of intervals (that is we go further in number of intervals when the quality function do not evolve during few generations).

We test our algorithm with the same parameters as above ($\hat{\beta} = 0.5$) and since we do not know anything about the best vaccination campaign, we start with a global vaccination rate $u(t) = 0\ \forall t$. Numerical and analytical results of section 3 allow to show that $\lambda^* = \frac{u_{max}}{S(t)}$ for $t \in [t_1^*, t_2^*]$ and 0 otherwise with $t_1^* = 4.11$ weeks and $t_2^* = 21.89$ weeks (we take the same parameters as the ones of 1). With the genetic algorithm, we found the following results.

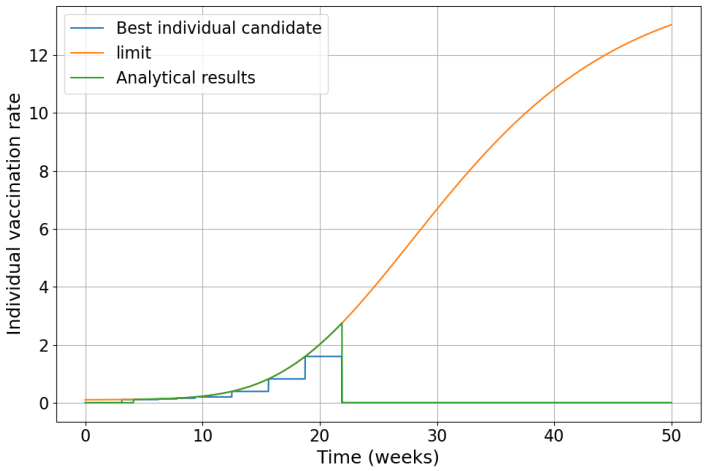

FIGURE 5 – Results of the genetic algorithm after 50 generations (16 intervals). The limit in orange is $\lambda_{max}(t) = \frac{u_{max}}{S(t)}$. In green are the analytical expected results and in blue the best candidate found by the GA

We see on fig 5 that the global behavior is recover by the algorithm : the vaccination occurs between the right bounds (the first bound is not perfectly reach here) and the right intensity (we do not reach the limit at each time because of the interval structure). An issue here was that there is a "plateau" in $\mathbb{S}$ because $g(t) \longrightarrow 0$ quickly after the epidemic peak, so the algorithm found that there is almost no difference between the above results and the same with a huge vaccination campaign at the end of the epidemic (this does not change Q).

In order to avoid this, we had a new offspring process to our algorithm : a "cloning" process. That is we clone many times the best candidate of a given generation. Then we associated with each interval some clones

(5 in our algorithm) and on this interval only we change the value of λ randomly (local mutation) for each clone. We end up with many new offspring. We make a first selection among this clones to choose the best ones. The cloning process allow us to explore this flat part of $\mathbb{S}$ and then we just have new candidates for the next generation in competition with the "natural" offspring. In order to accelerate the process, we add also a manual clone which is the one that maximize Q when we change only the value of λ on one interval (this allows to cut the vaccination campaign at the end of epidemic quickly). These clones are also in competition with other clones to be the offspring of a generation. At the end, we take 20 parents, 60 natural offspring and 120 clones (which are selected among all clones), and so 200 candidates per generation.

We can go further and wait instead of few minutes (50 generations) some more time until 200 generations. This is illustrated in fig 6 and fig 7.

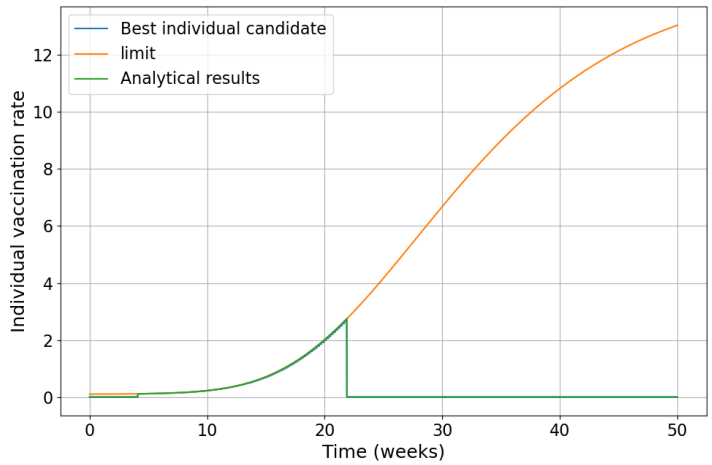

FIGURE 6 – Results of the genetic algorithm after 200 generations (512 intervals). The limit in orange is $\lambda_{max}(t) = \frac{u_{max}}{S(t)}$. In green are the analytical expected result and in blue the best candidate found by the GA

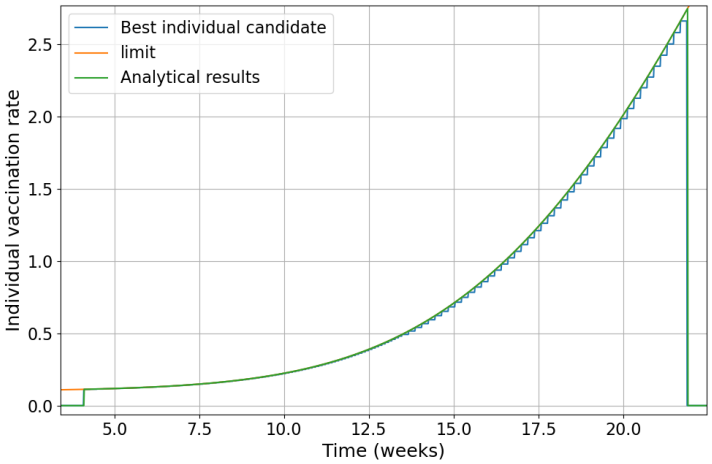

FIGURE 7 – Focus on the vaccination campaign with the same color code as fig 6

When we focus on the vaccination campaign on fig 7, we can see that we recover perfectly the expected results. That is our algorithm is able to compute $f_I(u) = \lambda^*$ and thus to make the "Bellman arrow" of (2.13). Then, we can use the inductive sequence (2.13) to find the Nash equilibrium of our problem without the Bellman equation. This result for this example confirms that in principle, we will be able to use genetic algorithms to simulate our models if the Bellman paradigm is not usable.

5 SIR model with a social structure

5.1 Theoretical framework

Now we want to develop Mean Field Game approach on models which present a mesoscopic description of society. A first model that we will consider is a SIR model with a structure of social contacts defined in [4]. We start with a a general description of this model before include Mean Field Games. The idea is to class each individual by his age class i and define some settings where there are contacts between individuals : the schools, the community, the workplaces and the households. Let introduce M_{ij}^k the average frequency of contact between an individual of age class i and someone of age class j in the setting k. Each individual of age class i are equivalent in the sense that they have the same frequency of contacts with other individuals (we take the average over individuals at this level). The effective number of contacts with individuals of age class j is given when we multiply M_{ij}^k by the contact rate $N_i^k(t)$ for individuals of age class i in the setting k. That is $N_i^k(t)$ is only a number of contact for a certain time but we say nothing here about the nature of contacts (the detailed about who are our contacts are in M_{ij}^k). For example, a child at school will have lot of contacts per days with other children but almost no contacts with adults (the teachers here) or old people.

In the paper [4], authors infers the matrix M_{ij}^k from the demographic structure. That is they use the composition in number and age of individuals with (at least one) contacts in each school, workplace, household. Then they defined a frequency of contacts for each individual of age class i with individuals of age class j in a certain household or in a certain school (if this individual has contacts with this school or this household). They construct the matrix M_{ij}^k by taking the average over individuals of age class i taking into account the fact that some individuals have no contacts with some settings (for example adult people do not go to school, unless they are teachers). This construction could be very useful to define and construct properly a "virtual society".

Now we can write down the SIR equations, indexing by i the susceptible/ infected/ recovery people of age class i. We denote by q the probability of transmission (of the virus) per effective contact (between a susceptible and an infected). We do not have any vaccination in this model. The SIR equations are (with n age classes) :

$$\begin{cases} \dot{S}_i = -\left[\displaystyle\sum_{j=1}^{n}\sum_{k} q \cdot N_i^k(t) \cdot M_{ij}^k \cdot I_j(t)\right] \cdot S_i(t) \\ \\ \dot{I}_i = \left[\displaystyle\sum_{j=1}^{n}\sum_{k} q \cdot N_i^k(t) \cdot M_{ij}^k \cdot I_j(t)\right] \cdot S_i - \hat{\gamma} I_i(t) \ , \\ \\ \dot{R}_i = \hat{\gamma} \cdot I_i(t) \end{cases} \tag{5.1}$$

Indeed, the probability for someone of age class i to be infected between t and $t + dt$ (if this person is still susceptible at t) is

$$\left[\sum_{j=1}^{n}\sum_{k} q \cdot N_i^k(t) \cdot M_{ij}^k \cdot I_j(t)\right] \cdot dt \ \equiv \ \lambda_i(t) \cdot dt \ , \tag{5.2}$$

for each setting k, for each age class j, our individual of age class i has an average number of effective contacts $N_i^k(t) \times M_{ij}^k$ during dt with someone of age class j in the setting k. For each contact, there is a probability I_j that the contact person is infected and if this person is infected, there is a probability q that there is a transmission of the disease during the contact. Finally, we naturally sum over all age class of the contacts and all settings. Therefore, we obtain (5.2) and the SIR equations (5.1) follow. λ_i is called the "force" of infection for an individual of age class i.

Notice that here we take for simplicity a recovery ratio $\hat{\gamma}$ independent of i and we consider only 3 compartments (S,I,R). We could include more, but beyond an increase of the number of equations, this would not change the structure of the problem, and the following analysis is extendable to these more refined models. Note also that we assume the average frequency of contacts for individuals M_{ij}^k constant, which could be not true in reality.

5.2 Mean Field Game approach

We propose to derive a Mean Field Game version of this model. We consider n types of agent. An agent of type i is an individual of age class i in our society. The control parameters of the agents (of type i) are the contact rates of individuals of age class i in the setting k $N_i^k(t)$. Thus, we have a Mean Field Game with $3n$ states $(S_1, S_2, ..., S_n, I_1..., R_1, ...)$ but each agent of type i can only be in S_i, I_i or R_i.

The idea is that if the epidemic is virulent, agents will reduce their contact rates $N_i^k(t)$ during this period to avoid infection but this reduction has a certain cost $f\left(\{N_i^k\}\right)$. We assume this cost to be decreasing, with a second derivative positive. Indeed, this means that the larger the effort made to reduce N_i^k, the higher the increases of the price to be paid. For the cost of infection due to the epidemic, we take a simple form $r_I(i)$ when an individual of age class i is infected. This cost increases with i, modeling that we suffer more from infection when we are older.

Introducing $\phi_I^i(t)$ the probability for an individual of age class i to be infected before t, an infection for this individual happens between t and $t+dt$ with a probability $(1-\phi_I^i(t)) \cdot \lambda_i(t) \cdot dt$. From an egoistic point of view, the total cost associated with the reduction of contacts is zero when we are infected, because the risk of a new infection is zero also. Thus, the total cost function for an individual of age class i (i is fixed in the following), starting the optimization at t for a certain individual strategy $\{N_i^k\}$ (and a certain global strategy which is implicit in the notation) is

$$U^i(\{N_i^k\}, t) \equiv \int_t^T \left[\lambda_i(s) \cdot r_I(i) + f\left(\{N_i^k(s)\}\right)\right] \cdot (1-\phi_I^i(s)) \cdot ds \,. \tag{5.3}$$

Therefore the value function is :

$$U^i(t) = \min_{\{N_i^k\}} \left[U^i\left(\{N_i^k\}, t\right)\right]. \tag{5.4}$$

Now, we are able to derive the Hamilton Jacobi Bellman equation follows by U^i. We denote by St_{i_t} the state of a reference agent of age class i at t. The Markov process of this Mean Field Game is :

$$\begin{cases} P(St_{i_{t+dt}} = I_i | St_{i_t} = S_i) = \lambda_i(t)dt \\ P(St_{i_{t+dt}} = S_i | St_{i_t} = S_i) = 1 - \lambda_i(t)dt \\ P(St_{i_{t+dt}} = R_i | St_{i_t} = I_i) = \hat{\gamma}dt \end{cases} . \tag{5.5}$$

We use a Bellman argument to find the evolution of U^i backwards in time (we consider as in previous cases that the state at t is S_i otherwise $U^i(t) = 0$) :

$$U^i(t)|_{St_{i_t}=S_i} = \min_{\{N_i^k(t)\}} \mathbb{E}_{St_{i_t}=S_i}\left[U^i(t+dt)|_{St_{i_{t+dt}}} + C^i(t)|_{St_{i_{t+dt}}}\right] . \tag{5.6}$$

From the Markov process (5.5) and the definition of U^i we have :

$U^i(t+dt) = 0$ if $St_{i_{t+dt}} = I_i$ and is unchanged if $St_{i_{t+dt}} = S_i$.

$C^i(t) = f\left(\{N_i^k(t)\}\right) \cdot dt + r_I(i)$ if $St_{i_{t+dt}} = I_i$ and $C^i(t) = f\left(\{N_i^k(t)\}\right) \cdot dt$ if $St_{i_{t+dt}} = S_i$.

Writing explicitly the expectation in (5.6)

$$U^i(t) = \min_{\{N_i^k(t)\}} \left[\lambda_i(t)dt\left(r_I(i) + f\left(\{N_i^k(t)\}\right)dt\right) + (1-\lambda_i(t)dt)\left(U^i(t+dt) + f\left(\{N_i^k(t)\}\right)dt\right)\right]; \tag{5.7}$$

this gives the Hamilton-Jacobi-Bellman equation of our Mean Field Game

$$-\left[\frac{dU^i(t)}{dt} + \min_{\{N_i^k(t)\}}\left[\lambda_i(t)\left(r_I(i) - U^i(t)\right) + f\left(\{N_i^k(t)\}\right)\right]\right] = 0 \,. \tag{5.8}$$

Where $\lambda_i(t)$ depends on $N_i^k(t)$ (see its definition (5.2)). For an epidemic evolution given by the SIR system (5.1), we can compute the behavior of our reference agent of age class i. This agent will take an optimal strategy N_i^{k*} at each time t

$$\{N_i^{k*}(t)\} = \underset{\{N_i^k(t)\}}{\operatorname{argmin}}\left[\lambda_i(t)\left(r_I(i) - U^i(t)\right) + f\left(\{N_i^k(t)\}\right)\right] . \tag{5.9}$$

5.3 Practical implementation

We propose a form for f inspired from [5] where the cost of the contact rate ($\hat{\beta}$ in the paper) is of the form

$$c(\hat{\beta}) = \frac{\hat{\beta}_0}{\hat{\beta}} - 1 \quad (\hat{\beta} \in [\hat{\beta}_{min}, \hat{\beta}_0]) \,, \tag{5.10}$$

expressing the discrepancy between the strategy chosen, $\hat{\beta}$, and the strategy without efforts $\hat{\beta}_0$. There is no cost associated with the "effortless" strategy $\hat{\beta}_0$, and the farther is $\hat{\beta}$ from $\hat{\beta}_0$, the higher the price to be paid.

In the same spirit, we propose here a cost of the form :

$$c(k, N_i^k) = \left(\frac{N_{i0}^k}{N_i^k}\right)^{\mu_k} - 1 \,, \tag{5.11}$$

which has the same qualitative properties as (5.10), but here μ_k models the "attachment" to the setting k. It is for example easier to reduce once contacts at work rather than inside once family. Notice that this cost function is clearly decreasing, with a positive second derivative. We furthermore impose bounds of N_i^k : a minimum of contact rate N_{imin}^k (denoted after simply N_{im}^k) and a maximum N_{i0}^k which is the contact rate without efforts. Then, the total cost of contact reduction is :

$$f\left(\{N_i^k(t)\}\right) = \sum_k c(k, N_i^k(t)) \,. \tag{5.12}$$

Notice that we take a form of the cost independent of the age class i (for simplicity and because we assume that contact reduction is painful regardless of age).

If we use our expression of f (5.12), we can derive the expression of argmin in (5.9) noticing that

$$\left[\lambda_i(t)\left(r_I(i) - U^i(t)\right) + f\left(\{N_i^k(t)\}\right)\right] = \left[\sum_k \left(q \sum_j M_{ij}^k I_j(t) N_i^k(t)(r_I(i) - U^i(t)) + \left(\frac{N_{i0}^k}{N_i^k}\right)^{\mu_k} - 1\right)\right] \,. \tag{5.13}$$

Then, since each N_i^k appears in only one term, we can minimize independently each term of the sum. We can see that this formula diverges asymptotically, when N_i^k tends to 0 (f diverges) or when N_i^k diverges (λ_i diverges). Furthermore, we can check by elementary computations that this quantity decreases until a unique minimum and increases afterwards.

The minimum is the minimum of a standard function with one variable (see (5.13))

$$\underset{\{N_i^k\} \in [N_{im}^k, N_{i0}^k]}{\operatorname{argmin}} \left(\sum_k [...]\right) = \sum_k \underset{N_i^k \in [N_{im}^k, N_{i0}^k]}{\operatorname{argmin}} [...] \,, \tag{5.14}$$

and by taking the derivative equals to zero, we find that

$$N_i^{k*} = \left(\frac{\mu_k (N_{i0}^k)^{\mu_k}}{q \sum_j M_{ij}^k I_j \cdot (r_I(i) - U^i(t))}\right)^{\frac{1}{\mu_k + 1}} \,. \tag{5.15}$$

This formula correspond indeed to the true optimal N_i^{k*} if we are inside the bounds $[N_{im}^k, N_{i0}^k]$. If we are above N_{i0}^k, the optimal N_i^{k*} is $N_i^{k*} = N_{i0}^k$ and if we are below N_{im}^k, the optimal N_i^{k*} is $N_i^{k*} = N_{im}^k$.

We can understand the behavior of the value function U^i from (5.8) : as $t \longrightarrow \infty$, $U^i(\infty) = 0$ because the total cost is zero if we begin the optimization at the end of epidemic. When we go backwards in time, $r_I(i) - U^i(t) \geq 0$ and thus the minimum in (5.8) is positive implying :

$$-\frac{dU^i}{dt} \geq 0 \,. \tag{5.16}$$

If at a time t $U^i(t)$ reaches $r_I(i)$, then $\frac{dU^i}{dt} = 0$ (because the optimal N_i^k is N_{i0}^k and $U^i(t)$ becomes constant). Thus, (5.16) is true for all t and therefore U^i decreases. Remark : by analyzing directly (5.8), $U^i(t)$ tends to $r_I(i)$ from below and in this limit, $N_i^{k*} = N_{i0}^k$.

5.4 Numerical simulations

Our numerical simulations are made under the following assumptions.

— We take only 3 age classes (youth, adults, retired), corresponding respectively to the i^{th} column or row in our contact matrices or directly to $i = 1, 2, 3$.

— $r_I(i) = r_I \times 10^i$ (to simulate the impact of infection when we are older). We use $r_I = 1$ (unless other indication) to get a competition between the infection cost and the cost due to contact reduction.

— We take 4 settings as in [4] : schools, workplaces, community and households.

— Our matrices M_{ij}^k are inspired from the data analysis of [4] to obtain a qualitative comprehension of agent's behavior. But we do not made any fit to get these matrices.

$$M^S = \begin{pmatrix} 0.95 & 0.05 & 0 \\ 0.005 & 0.005 & 0 \\ 0 & 0 & 0 \end{pmatrix} \; ; \; M^W = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$M^C = \begin{pmatrix} 0.25 & 0.5 & 0.25 \\ 0.25 & 0.5 & 0.25 \\ 0.25 & 0.5 & 0.25 \end{pmatrix} \; ; \; M^H = \begin{pmatrix} 0.3 & 0.5 & 0.2 \\ 0.5 & 0.3 & 0.2 \\ 0.3 & 0.2 & 0.5 \end{pmatrix}$$

— We choose N_{i0}^k in the same way from [4]. We write it as a matrix for concision (first row for schools, second for workplaces, third for community, fourth for households) :

$$N_{(ki)}^0 = \begin{pmatrix} 7.5 & 2.5 & 0 \\ 0 & 5 & 0 \\ 2.5 & 2.5 & 2.5 \\ 2.5 & 2.5 & 2.5 \end{pmatrix}$$

We set $N_{im}^k = \frac{N_{i0}^k}{A_k}$. That is the minimum contact rate is independent of i. We take $A_S = 3$ (schools), $A_C = A_W = 5$ (community and workplace), $A_H = 2$ (Household) to induce the fact that we cannot reduce as much our contacts in our household as inside schools or at work.

— To induce the fact that the reduction of contacts is harder inside households and easier in community, we take : $\mu_S = \mu_W = 2, \mu_H = 3, \mu_C = 1$

— We take 25% of "young" and "retired" in our society and 50% of adults.

— We take a probability of transmission by real effective contact $q = 0.2$ and we take the recovery rate $\hat{\gamma} = 0.1$. Then, we work with fix initial conditions : $(S_i(0), I_i(0)) = ([0.98, 0.98, 0.98], [0.02, 0.02, 0.02])$.

Since all these quantities are typical, observed behaviors are a priori general. This was actually check by running many simulations with different parameters.

As in previous MFG, we use the inductive sequence procedure of section 2.4, with the following scheme :

$$\{\bar{N}_i^k\}_n \underset{Kolmogorov}{\longrightarrow} I(\{\bar{N}_i^k\}_n) \underset{Bellman}{\longrightarrow} f(\{\bar{N}_i^k\}_n) = \{N_i^{k*}\}_n \underset{Symmetric}{\longrightarrow} \{\bar{N}_i^k\}_{n+1} = \{N_i^{k*}\}_n \; . \quad (5.17)$$

Notice that here we were be able to find the optimal individual response N_i^{k*} through an analytical minimization of the Bellman equation (5.8), namely (5.15) where there is an implicit dependence in the global strategy $\{\bar{N}_i^k\}$ in $I_j(t)$. We follow the above scheme until $N_i^{k*}(t) = \bar{N}_i^k(t) \; \forall \, t, k, i$, obtaining in this way the Nash equilibrium. This Nash equilibrium is actually reached quite quickly (few iterations) with this model, because the cost function is sufficiently flat.

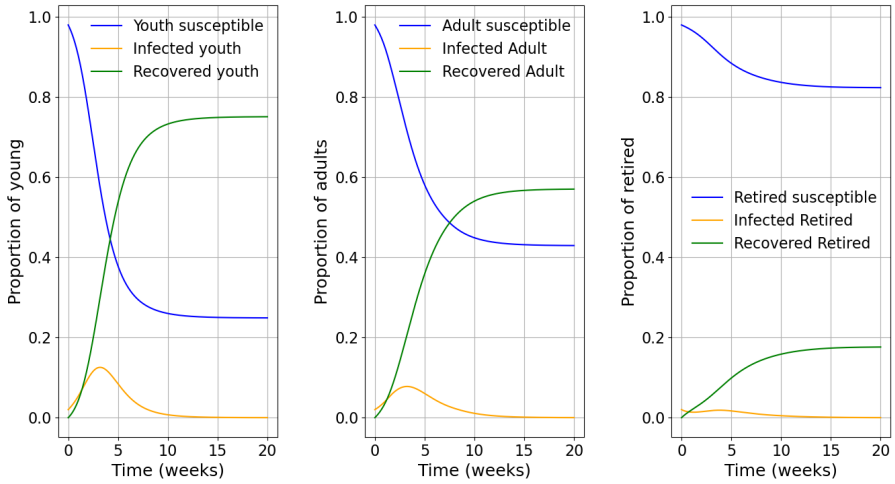

FIGURE 8 – Evolution of epidemic by age group. From left to right : young, adult and retired epidemic. Recovered are in green, Infected in orange, Susceptible in blue

We can see on fig 8 that epidemic peaks are around 3 and 4 weeks, but the precise time of the peak is different for each classes because of the interactions and different behaviors. We observe that the number of infected retired is very low compare to the one of young people, which is due to the fact that being infected is more dangerous for retired people than for adults and young. We can also observe that there is a little epidemic peak for the retired despite their efforts from the beginning to reduce epidemic, which is due to interactions with other classes.

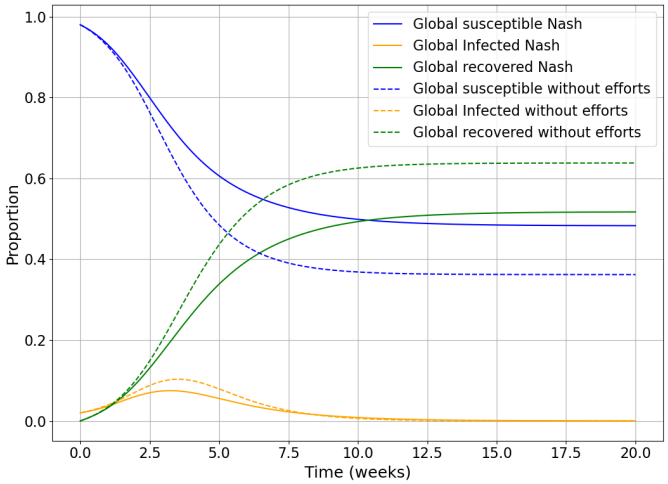

FIGURE 9 – Evolution of global epidemic. Comparison between the Nash equilibrium strategy and the strategy without efforts (dot lines). Recovered are in green, Infected in orange and Susceptible in blue

Fig 9 illustrates the effect of contact reduction compared with the scenario where no change in the control rates are made. The peak of epidemic comes earlier and is less important with the Nash equilibrium. The importance of contact reduction is significantly affected by r_I which determines how much efforts the individuals are ready to make to reduce their contacts (with a small r_I we observe a little reduction and with a very high r_I the reduction is maximum). We can check it by plotting contact rates.

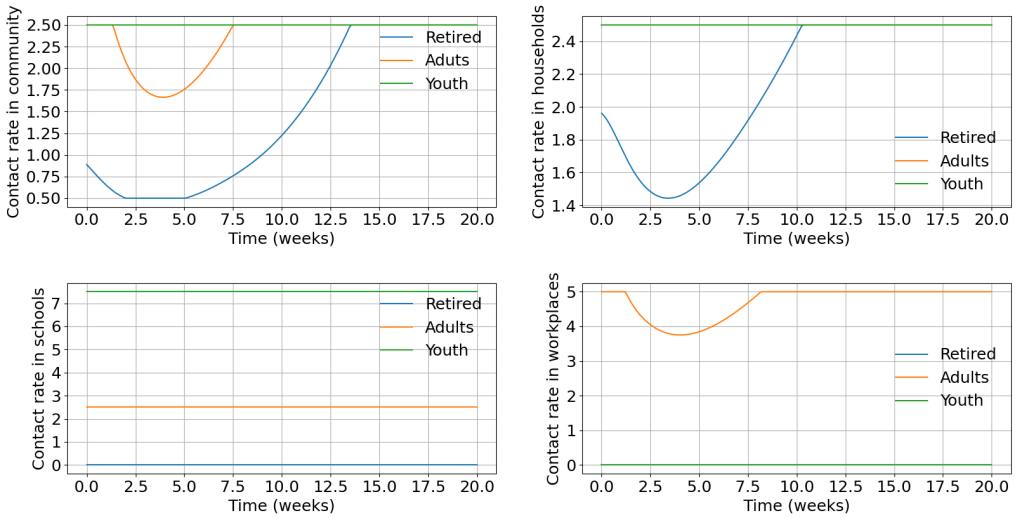

FIGURE 10 – Evolution of contact rates. Retired people are in blue, adults in orange and young in green

Fig 10 shows the different behaviors of agents : retired people reduce indeed their contacts because they have more risk. This reduction is very significant in community (they reaches N_{min} for a certain period of time) because it is easy for them to reduce their contacts, but in households, we see that the period of reduction is less important. The situation for adults is intermediate : they reduce their contacts in community and in workplaces during the epidemic peak, but they do not change anything in households because the peak is not high enough. For young people, the risk with such epidemic is low and therefore, they do not change their behavior. Notice that in schools, adults do not change their contacts because the probability for an adult to be in contact with young at school is very low.

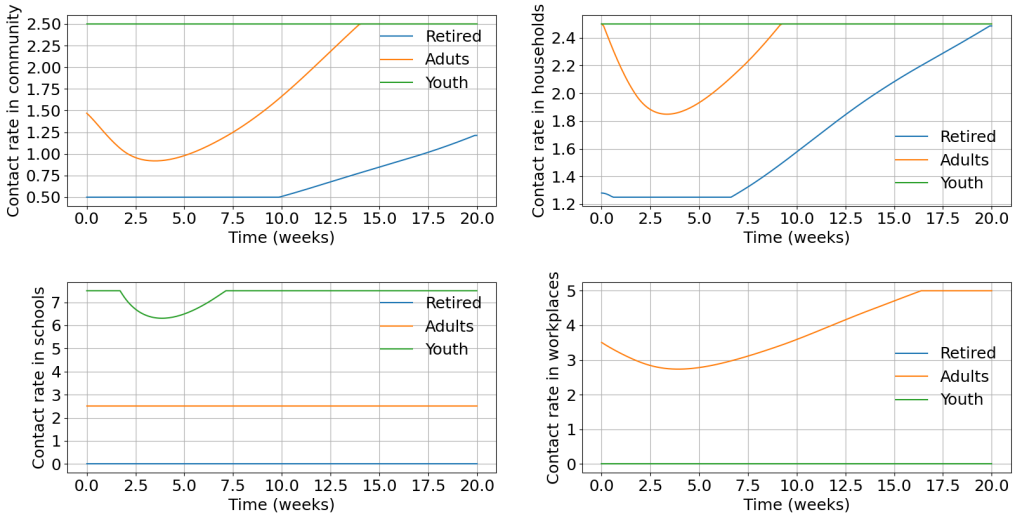

FIGURE 11 – Evolution of contact rates. Retired are in blue, adults in orange and young in green. We take here $r_I = 5$ (instead of $r_I = 1$ above)

As we expected, we see on fig 11 that when the cost due to infection is higher, the contact reduction is much more drastic. Young start to reduce their contacts in schools. Furthermore, to avoid an epidemic rebound the growth of contacts after the epidemic peak is very low.

5.5 Other method to reach the Nash equilibrium

The inductive sequence method used until here works well because functions are contracting (see section 2.4). But sometimes it is not the case and this method does not converge. This is the case in particular in our model if we want to implement global constraints, such as the ones that would be implied by a lockdown (for example, $N_i^k \in [N_{i,m}^k, N_{i0}^k]$ if $I < I_{sat}$ and $N_i^k = N_{i,m}^k$ if $i > I_{sat}$).

The impact on the cost function is that a small change of the global strategy $\bar{N}_i^k$ change I and therefore the bounds of the "lockdown", implying that the associated cost paid by individuals change a lot. Our cost function is not contracting anymore and this is why the inductive sequence does not converge.

Instead of this, we can use the second method presented in section 2.4 : the gradient descent. This method find a priori a local minima (with respect to the individual strategy) among the Nash candidates. But we can test if we reach the same equilibrium than the first method (when the latest converges).

In order to implement it numerically, we have to compute the Gateau derivative of the cost (see [31] for a rigorous definition and [5] for a similar application to what we do here). This cost is (5.3) at $t = 0$. Note that for clarity and to avoid heavy notations, we denote after $\{\bar{N}_i^k\}$ by $\bar{N}$, $\{N_i^k\}$ by N, $N(t)$ by N_t and $\Phi_I^i(t)$ by $\Phi_i^{N,\bar{N}}(t)$:

$$\mathcal{C}^i(N, \bar{N}) = \int_0^T [\lambda_i^{N,\bar{N}}(t) \cdot r_I(i) + f(N_t)] \cdot (1 - \phi_i^{N,\bar{N}}(t)) \cdot dt \ . \tag{5.18}$$

With the Markov process (5.5) we know that

$$\phi_i^{N,\bar{N}}(t) = 1 - e^{-\int_0^t \lambda_i^{N,\bar{N}}(s) \cdot ds} \ , \tag{5.19}$$

by integrating over the first term of $\mathcal{C}$ we obtain (we recognize $\dot{\phi}$)

$$\mathcal{C}^i(N, \bar{N}) = \int_0^T f(N_t)(1 - \phi_i^{N,\bar{N}}(t))dt + r_I(i)\phi_i^{N,\bar{N}}(T) \ . \tag{5.20}$$

Now we can compute the Gateau derivative $D_h\mathcal{C}$ of $\mathcal{C}$ with respect to the first variable in the direction h :

$$D_h\mathcal{C}^i(N, \bar{N}) \equiv \lim_{\epsilon \longrightarrow 0} \frac{1}{\epsilon}(\mathcal{C}^i(N + h\epsilon, \bar{N}) \ - \ \mathcal{C}^i(N, \bar{N})) \tag{5.21}$$

$$= r_I(i) D_h\phi_i^{N,\bar{N}}(T) + \int_0^T \left[D_h f(N_t)(1 - \phi_i^{N,\bar{N}}(t)) - f(N_t) D_h\phi_i^{N,\bar{N}}(t) \right] dt \ , \tag{5.22}$$

and the two Gateau derivatives appearing in (5.22)

$$D_h\phi_i^{N,\bar{N}}(t) \equiv \lim_{\epsilon \longrightarrow 0} \frac{1}{\epsilon}(\phi_i^{N+h\epsilon,\bar{N}}(t) \ - \ \phi_i^{N,\bar{N}}(t)) = (1 - \phi_i^{N,\bar{N}}(t)) \cdot \int_0^t h(s) \frac{d\lambda_i^{N,\bar{N}}(s)}{dN} ds \tag{5.23}$$

$$D_h f(N_t) \equiv \lim_{\epsilon \longrightarrow 0} \frac{1}{\epsilon}(f(N_t + h_t\epsilon) \ - \ f(N_t)) = f^{'}(N_t) h_t \ . \tag{5.24}$$

We therefore obtain by replacing in (5.22) :

$$D_h\mathcal{C}^i(N, \bar{N}) = \langle \, h, \, \nabla_1\mathcal{C}^i(N, \bar{N}) \, \rangle_T \ , \tag{5.25}$$

where

$$\nabla_1\mathcal{C}^i(N, \bar{N})(.) = \frac{d\lambda_i^{N,\bar{N}}(.)}{dN} \left(r_I(1 - \phi_i^{N,\bar{N}}(T)) - L_.(N, \bar{N}) \right) + (1 - \phi_i^{N,\bar{N}}(.)) f^{'}(N_.) \ , \tag{5.26}$$

with

$$L_t(N, \bar{N}) \equiv \int_t^T f(N_s)(1 - \phi_i^{N,\bar{N}}(s))ds \ . \tag{5.27}$$

Equation (5.26) is used for numerical simulation of the gradient descent. Writing (2.14) explicitly for the cost :

$$N^{n+1} = N^n - h \cdot \nabla_1\mathcal{C}(N^{n+1}, N^n) \ . \tag{5.28}$$

We use $\nabla_1 \mathcal{C}(N^n, N^n)$ numerically (we take the first order and it was sufficient). h is the step of the gradient descent here and N means as previously $\{N_i^k\}$.

Now we can check on fig 12 the convergence of the gradient descent method until the Nash equilibrium find with the inductive sequence method :

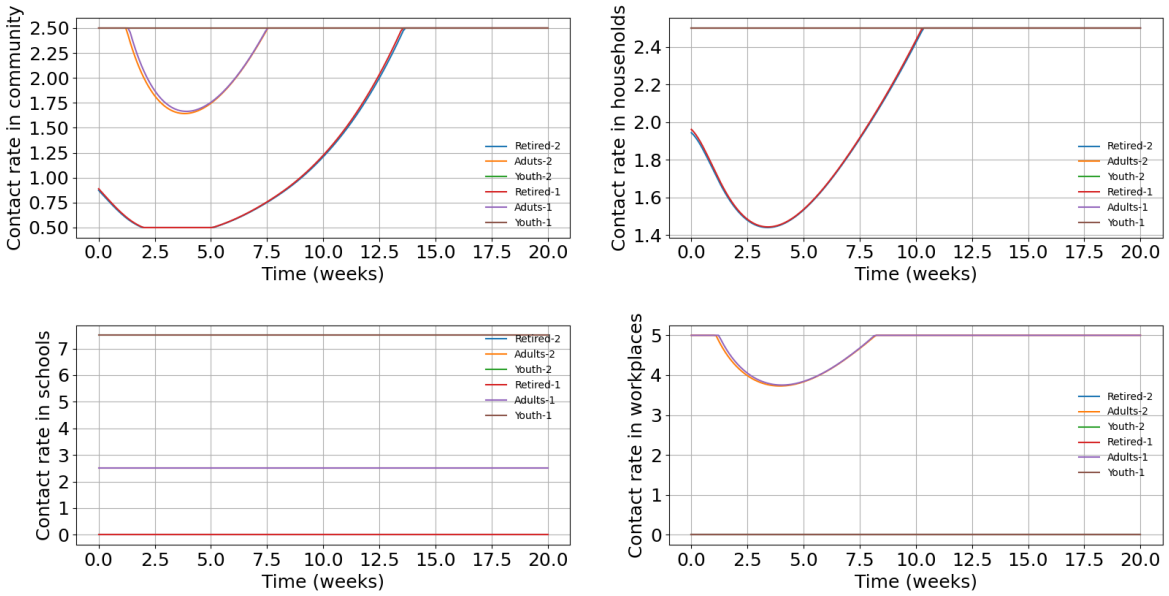

FIGURE 12 – Convergence of two methods with the same parameters as fig 10. Method indicating by -1 : Inductive sequence. Method indicating by -2 : gradient descent

We clearly see on fig 12 a great convergence of the gradient descent method towards the Nash equilibrium find by the inductive sequence method. This second method could be useful to treat the cases where the first method does not converge (it is a slower method but more robust to the form of the application). Even if we cannot guarantee that the minima obtain in this way is a true minima (i.e a true Nash equilibrium) and not just a local minima, this can be checked afterwards : by inserting this obtained results as an entry of the iterative scheme (5.17) and check that this is indeed a fixed point. If this turns out not to be the case, we can think of an hybrid algorithm (with a mix of the two above methods) to converge effectively towards the true Nash equilibrium.

6 Conclusion

In our work, we develop a SIR model with a social structure where the contact rates between individuals are intrinsic instead of extrinsic and treated with the Mean Field Game paradigm. We find the Nash equilibrium using two different approaches, inductive sequence and gradient descent which allows us to address models for which the inductive sequence method does not works. We develop also a genetic algorithm which realize the optimization of the problem and allow us to solve numerically a Mean Field Game when the Bellman equation cannot be derived.

We have developed some tools which can be used to further develop this line of research. Such extension of our work could be, in the sort term :

- Add global constraints ([21], [22])
- Study other kinds of mesoscopic description

On the longer term :

- Add a spatio-temporal dynamics to our models ([26], [27], [28])
- Calibrate the cost functions (i.e comparison with empirical data and discussion with other researchers)

We can finally think about other models with other approaches to describe heterogeneous interactions in the society. An example are the networks based models ([29], [30]), we can try to homogeneous them at an appropriate level to model globally the spread of epidemics and use Mean Field Games on it.

Finally, I would like to thanks sincerely Denis Ullmo for his great support and all the LPTMS team for their warm welcome to the Laboratory.

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