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On the modeling of epidemics under the influence of risk perception

S. De Lillo
Department of Mathematics and Computer Science, University of Perugia, via Vanvitelli 1, 06123 Perugia, Italy
Istituto Nazionale di Fisica Nucleare, Sezione di Perugia, Italy
silvana.delillo@pg.infn.it

G. Fioriti*
Department of Mathematics and Computer Science Ulisse Dini, University of Florence, viale Morgagni 67/A, 50134 Florence, Italy
Istituto Nazionale di Fisica Nucleare, Sezione di Perugia, Italy
fioriti@math.unifi.it

M. L. Prioriello
Department of Mathematics and Computer Science, Eindhoven University of Technology, P. O. Box 513, 5600 MB Eindhoven, The Netherlands
and
Department of Mathematics, Physics and Computer Science, University of Modena and Reggio Emilia, via G. Campi 213/b, 41125 Modena, Italy
marialuisa.prioriello@unimore.it

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An epidemic spreading model is presented in the framework of the kinetic theory of active particles. The model is characterized by the influence of risk perception which can reduce the diffusion of infection. The evolution of the system is modeled through nonlinear interactions, whose output is described by stochastic games. The results of numerical simulations are discussed for different initial conditions.

Keywords Kinetic theory; active particles; risk perception; epidemics.

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1. Introduction

Modeling the epidemics of infectious diseases motivated a very extended literature, starting from the early studies of Kermack and McKendrick. In Ref. 26, the authors

*Corresponding author.
present an interesting survey of mathematical models and analytical results, to be compared with laboratory data in order to understand epidemiological trends and to control the spread of infection and disease within human communities. On the other hand, in Ref. 13 the author analyzes and classifies epidemic models according to their mathematical structure. Two main classes are identified: one of them related to order preserving dynamical systems, the other one related to Lyapunov methods. The mathematical models discussed in Refs. 13 and 26 are deterministic; however, as pointed out also in Ref. 13, spontaneous stochastic fluctuations have to be taken into account in order to get a more realistic model, able to fit experimental data. Indeed, more recently several studies were devoted to the development of stochastic epidemic models, mainly in the framework of random networks.1,3,4,11,19,23,25,31 In this respect, an extensive review concerning the spread of infectious diseases is presented in Ref. 18, where the authors focus on the interplay between network theory and epidemiology. Important aspects determining the diffusion of global epidemics, appear to be influence of large scale properties on the airline transportation network15 and also the spatial structure of the population, as pointed out in Ref. 16. We also observe that modifications of the global spread of epidemics account for the influence of resource14,9 and spatial10 constraints. Moreover, importance of developing an effective immunization approach in order to restrain an epidemic spreading on networks is pointed out in Ref. 29. On the other hand in recent years, a mathematical approach has been developed to describe complex systems belonging to the domain of life sciences. Description of such systems requires the use of appropriate techniques and mathematical methods that differ substantially from those used for the description of the inert matter. The mathematical formalism is called kinetic theory of active particles (KTAP theory). In such formalism the complex system is characterized by a large number of interacting entities named “active particles”. This means that the physical state of the particles is described not only by geometric and mechanical variables but also by a new variable named “activity”. This variable characterizes the type of strategy and the type of interactions that the particles of the complex system are able to develop.

The KTAP theory allowed the derivation of various models of practical interest in life science such as the description of crowds,30 the formulation of models of social and immune competition,5,22,7,8 the modeling of vehicular traffic flow6 and of the spread of epidemics contrasted by immune defense.21 It is the aim of this paper to propose some new ideas developed in the context of the model.21 In particular, we focalize our attention on two fundamental issues:

- **Nonlinear interactions:** Recent studies20,17 have introduced new concepts concerning nonlinear additivity of interactions. In this paper, the evolution of the system is ruled by nonlinear interactions between the active particles. The outcome of such interactions is described by stochastic games.
Risk perception: It is assumed in this model that susceptible individuals may be aware of the risk to contract the infection. According to the level of awareness they can take the necessary precautions.

The approach refers to the kinetic theory of active particles, reviewed in Ref. 17, which leads to equations suitable to describe the time and space dynamics of appropriate probability distributions over the micro-scale state of a large system of interacting entities. The derivation of the said equations is based on suitable developments of the methods of the mathematical kinetic theory, while interactions are modeled by theoretical tools of evolutionary game theory.

2. Mathematical Structure

Let us consider a large system of many interacting entities, called active particles, grouped into several different functional subsystems. Within the same subsystem, each individual is characterized by a microscopic state called activity, with a different meaning in each functional subsystem.

The number of particles in the whole system is assumed to be constant.

The evolution of the system is determined by interactions between pairs belonging to the same subsystem or to different ones.

The system consists of four subsystems, also called populations, labeled by the index $i \in \{1, \ldots, 4\}$:

- $i = 1$: doctors;
- $i = 2$: susceptible individuals;
- $i = 3$: individuals affected by the disease;
- $i = 4$: healed individuals (these individuals cannot be infected again).

The activity is a discrete scalar variable $u \in [0, 1]$ describing, for each $i$th population, the main properties of its respective individuals. In particular, it represents the four distinct subsystems:

- $i = 1$: the ability and the experience of doctors to treat the disease;
- $i = 2$: the susceptibility (to contract the infection);
- $i = 3$: the progression of the pathological state;
- $i = 4$: getting back in shape.

Remark 2.1. We assume that in the disease under consideration the severity of the pathological state is highest in the first stage of the disease. Specifically, for the third population $u = 0$ and $u = 1$ correspond, respectively, to the highest and to the lowest levels of severity of the pathological state.

We assume that the infectivity is constant, i.e. it is the same for all individuals of the third population.
In the following, we assume that the activity of individuals is heterogeneously distributed in each functional subsystem and we introduce the set \( I_u = \{ u_1 < \cdots < u_m \} \).

The overall state is described by the probability distributions:

\[
f_i(t, u = u_r) : [0, T] \rightarrow \mathbb{R}^+, \quad i \in 1, \ldots, 4, \quad r = 1, \ldots, m.
\]  

(2.1)

The interaction terms are defined as follows:

- \( \eta_{hk}^{pq} = \eta_{hk}[\bar{f}](u_p, u_q) \) is the encounter rate between the active particle of the \( h \)th functional subsystem with state \( u_p \) and the active particle of the \( k \)th functional subsystem with state \( u_q \), where \( h, k \in \{ 1, \ldots, 4 \} \) and \( p, q \in \{ 1, \ldots, m \} \);

- \( B_{hk}^i(r) = B_{hk}^i[\bar{f}](u_p \rightarrow u_r | u_p, u_q) \) is the probability that an active particle of the \( h \)th subsystem, with state \( u_p \), ends up into the \( i \)th subsystem with state \( u_r \), after interacting with the active particle of the \( k \)th subsystem, with state \( u_q \).

Then, for \( i = 1, \ldots, 4 \) and for \( r = 1, \ldots, m \), the balance equation is given by:

\[
d\frac{df_{ir}(t)}{dt} = Q_{ir}[\bar{f}](t) = \sum_{h,k=1}^{4} \sum_{p,q=1}^{m} \eta_{hk}[\bar{f}](u_p, u_q) B_{hk}^i[\bar{f}](u_p \rightarrow u_r | u_p, u_q) f_{hp}(t) f_{kq}(t) 
- f_{ir}(t) \sum_{k=1}^{4} \sum_{q=1}^{m} \eta_{hk}[\bar{f}](u_r, u_q) f_{kq}(t),
\]  

(2.2)

where \( \bar{f} \) denotes the set of all \( f_{ir} \) components of the probability density.

3. Qualitative Analysis

In this section, the initial value (I.V.) problem for Eq. (2.2) is formulated. We take into account the general case of \( n \) populations, \( n \geq 4 \). For the time evolution of the distribution functions \( f_{ir}(t), i \in \{ 1, \ldots, n \} \), we write:

\[
\begin{cases}
\frac{d}{dt} f_{ir}(t) = Q_{ir}[\bar{f}](t), & i = 1, \ldots, n, \quad r = 1, \ldots, m, \\
n_{ir}(0) = f_i(0, u_r),
\end{cases}
\]  

(3.1)

where, due to (2.2)

\[
\frac{d}{dt} f_{ir}(t) = Q_{ir}[\bar{f}](t) = \sum_{h,k=1}^{n} \sum_{p,q=1}^{m} \eta_{hk}[\bar{f}](u_p, u_q) B_{hk}^i[\bar{f}](u_p \rightarrow u_r | u_p, u_q) f_{hp}(t) f_{kq}(t) 
- f_{ir}(t) \sum_{k=1}^{n} \sum_{q=1}^{m} \eta_{hk}[\bar{f}](u_r, u_q) f_{kq}(t). 
\]  

(3.2)

We introduce the space:

\[
X = \{ f_i : [0, T] \rightarrow \mathbb{R}, \ f_i \in C^1([0, T]), \ i = 1, \ldots, n, \ T > 0 \}
\]  

(3.3)
characterized with the norm:

$$\|f(t)\|_X = \sum_{r=1}^{m} |f_r(t)|.$$

Moreover, we define the Banach space $X = X^n = \{ f = (f_1, \ldots, f_n), f_i \in C^1([0,T]), T > 0 \}$ with the corresponding norm:

$$\|f(t)\|_X = \sum_{i=1}^{n} |f_i(t)|_X,$$

and introduce:

$$X_+ = \{ f \in X | f_i \geq 0, i = 1, \ldots, n \}.$$

The following theorem states a result of local existence and uniqueness for the solution of the I.V. problem (3.1). We omit the proof for brevity.

**Theorem 1.** Consider the I.V. problem (3.1) with $f_0 = \{ f_1(0,u), \ldots, f_n(0,u) \} \in X_+$. Assume that

$$\eta_{hk}^{pq} \geq 0, \quad R_{hk}^i(r) \geq 0, \quad \sum_{i=1}^{m} \sum_{r=1}^{m} R_{hk}^i[f](u_p \rightarrow u_r|u_p,u_q) = 1, \quad \forall f,$$

hold together with the following hypotheses:

- The encounter rate $\eta_{hk}^{pq}$ satisfies the condition:

  $$\sum_{r=1}^{m} \eta_{hk}[f](u_p,u_q) \leq C, \quad \forall h,k = 1, \ldots, n \forall p,q \in \{1, \ldots, m\} \text{ and } \forall f \in X$$

  with $C$ a positive constant;
- $\forall f, g \in X$ the transition probability density $R_{hk}^i(r)$ and the encounter rate $\eta_{hk}^{pq}$ are Lipschitz continuous in $X$, that is, $\forall p,q \in \{1, \ldots, m\}$ it results

  $$\sum_{h,k,i=1}^{n} \sum_{r=1}^{m} (|R_{hk}^i[f](u_p \rightarrow u_r|u_p,u_q)| + |R_{hk}^i[g](u_p \rightarrow u_r|u_p,u_q)|) \leq L_1 \|fg\|_X,$$

  $$\sum_{h,k=1}^{n} \sum_{r=1}^{m} \left| \eta_{hk}[f](u_p,u_q) - \eta_{hk}[g](u_p,u_q) \right| \leq L_2 \|f-g\|_X,$$

  with $L_1, L_2$ positive constants.

Then, there exist $T > 0$ and a unique solution $f(t)$ in $X$ for the I.V. problem (3.1) on the time interval $[0,T]$. Moreover $f(t) \in X_+, t \in [0,T]$.

**Proof.** We start observing that, since the interactions are assumed number conservative, see (3.7), it results that:

$$\frac{d}{dt} \sum_{i=1}^{n} \sum_{r=1}^{m} f_{ir}(t) = 0,$$
which implies:
\[ \|f(t)\|_X = \|f(0)\|_X, \quad \text{for any } t \geq 0. \] (3.9)

Therefore, the solution of (3.1), if it exists, remains bounded in \( X \) for any time \( t \geq 0 \).

The latter observation assures that the operator \( Q_i[f](t) \) in the right-hand side of (3.1) is a closed map in \( X \).

Let us now prove that \( Q_i[f](t) \) is Lipschitz continuous in \( X \), i.e. given \( \|f\|_X \) and \( \|g\|_X \leq M \) it follows that:
\[ \|Q_i[f](t) - Q_i[g](t)\|_X \leq L\|f - g\|_X, \] (3.10)
with \( L \) a positive constant depending on \( M \). Indeed, when (3.2) is used together with (3.4) and (3.5), for the right-hand side of (3.10) we can write (see Ref. 12):
\[
\sum_{i=1}^{n} \sum_{r=1}^{m} \left[ \sum_{k,l=1}^{n} \sum_{p,q=1}^{m} \eta_{hk}\mathcal{B}_l^i(u_p, u_q)\mathcal{B}_k^i[u_p \to u_r, u_q]f_{hp}(t)f_{hq}(t) \\
- f_{ir} \sum_{k=1}^{n} \sum_{q=1}^{m} \eta_{kq}[u_r, u_q]f_{kq}(t) \\
- \left[ \sum_{k,l=1}^{n} \sum_{p,q=1}^{m} \eta_{lk}\mathcal{B}_l^i(u_p, u_q)\mathcal{B}_k^i[u_p \to u_r, u_q]g_{hp}(t)g_{kq}(t) \\
- g_{ir} \sum_{k=1}^{n} \sum_{q=1}^{m} \eta_{kq}[u_r, u_q]g_{kq}(t) \right] \right] \leq 2m^2n^3CM\|f - g\|_X + m^2n^3M^2CL_1\|f \\
- g\|_X + n^2m^2M^2L_2\|f - g\|_X + 2n^2mCM\|f - g\|_X \]
\[ + nmM^2L_2\|f - g\|_X \leq L\|f - g\|_X. \] (3.11)

that proves (3.10). Then, there follows the existence of a unique solution \( f(t) \) in \( X \) of (3.1) local in time. Next, the non-negativity of the solution, in its domain of existence, is easily obtained along the same lines of the proof reported in Ref. 17 observing that the components \( f_{ir}(t) \) of the solution satisfy the condition:
\[ f_{ir}(t) \geq 0 \quad \forall i = 1, \ldots, n \quad \text{and} \quad \forall j = 1, \ldots, m, \] (3.12)
when \( f(0) \in X_+ \). We omit the proof here for brevity.

Moreover, when (3.12) is used together with (3.9), we obtain that the solution to (3.1) is uniformly bounded on any compact time interval \([0,T], T > 0\). This latter observation leads immediately to the following result of global existence of the solution in \( X_+ \).

**Theorem 2.** Consider the I.V. problem (3.1) under the assumptions of Theorem 1. Then the solution \( f(t) \) exists for any finite time \( t \geq 0 \).
4. Modeling and Simulations

In order to model the encounter rates we introduce a distance between the probability densities:

\[ d(f_h, f_k)(t) = \sum_{r=1}^{m} \sum_{r'=1}^{m} |f_{hr}(t) - f_{khr'}(t)|, \quad h, k \in \{1, \ldots, 4\}. \quad (4.1) \]

The interaction rates are modeled according to:

\[
\begin{align*}
\eta_{22}^{pq} & = \eta_{14}^{pq} = \eta_{24}^{pq} = \eta_{42}^{pq} = \eta_{44}^{pq} = \alpha_1, \\
\eta_{11}^{pq} & = \alpha_2, \\
\eta_{12}^{pq} & = e^{-\frac{1}{2(uq)}} \\
\eta_{21}^{pq} & = e^{-\frac{1}{2(uq)}} \\
\eta_{13}^{pq} & = e^{-\frac{1}{(uq)^2}} \\
\eta_{31}^{pq} & = e^{-\frac{1}{(uq)^2}} \\
\eta_{23}^{pq} & = e^{-\beta(1+uq)(1+d(f_2,f_3))}, \\
\eta_{32}^{pq} & = e^{-\beta(1+uq)(1+d(f_2,f_3))}, \\
\eta_{33}^{pq} & = \eta_{34}^{pq} = \eta_{43}^{pq} = \alpha_4,
\end{align*}
\]

where \(\alpha_1, \alpha_2, \alpha_3, \alpha_4\) are positive constants, and \(0 < \beta < 1\) denotes the risk perception.

The above choice for the interaction rates indicates that the interaction rate \(\eta_{12}^{pq}\) doctor/susceptible increases when the value of activity \(u_q\) decreases: indeed people at a low level of susceptibility are more induced to get immunized. On the other hand, the risk perception induces susceptible individuals to stay away from infected ones, which explains the interaction rates in (4.8) and (4.9) that are exponentially decreasing as the distance between the distributions is increasing. Finally, the encounter rates in (4.6) and (4.7), corresponding to the interactions doctor/infected, tend to increase in the first stage of the illness, when the doctors are more invoked to prescribe the cure. In all other cases they are assumed to be constant.

4.1. Tables of games

Interactions modeled by the terms \(B_{hk}(r)\), are called stochastic games since the microscopic state of the active particles is known in probability and the output is identified by a probability density.

In order to describe the tables of games we need to introduce the following parameters:

- **Doctors ability**: \(0 \leq \delta \leq 1\)
- **Intensity of the vaccine reaction**: \(0 \leq \gamma \leq 1\)
- **Infectivity**: \(0 \leq \chi \leq 1\)
Moreover, we consider the first-order moment for \( i = 1, \ldots, 4 \) which we identify with the mean value:

\[
\mathbb{E}_i^1[f_i](t) = \sum_{r=1}^{m} u_r f_{ir}(t),
\]

(4.11)

and

\[
B_p^i(\mathbb{E}_i^1[f_i]) = \varepsilon_i |\mathbb{E}_i^1[f_i] - u_p|,
\]

(4.12)

which is proportional to the distance between the activity of the interacting particle \( p \) and the mean value \( \mathbb{E}_i^1[f_i] \), with \( p = 1, \ldots, m \) and \( 0 < \varepsilon_i \leq 1 \).

For sake of brevity, in the following we only report the most relevant tables of games.

4.1.1. Tables of games for \( \mathcal{B}_{2k}^i(r) \), for \( k = 1, 3 \). (susceptible individuals)

- \( \mathcal{B}_{21}^i(r) = \mathcal{B}_{21}^i[f](u_q - u_p | u_p, u_q) \).

We consider the table referring to interactions between a susceptible and a doctor.

As an example we here show only the case when the susceptibility is above the mean value. The derivation of the expressions reported below is based on empirical assumptions.

In detail: for \( p < m \), we assume that a susceptible individual interacting with a doctor has a probability density \( B_{21}^i \) to make a transition into the healed population, which increases as the vaccine reaction \( \gamma \) increases. For the same individual instead, the probability density \( B_{21}^i \) to remain in the same population, decreases as \( \gamma \) increases.

In the case \( p = m \) (last class of activity) the behavior of \( B_{21}^i \) does not change, while there is a probability density \( B_{21}^3 \) to make a transition into the infected population. Such probability density decreases as the vaccine reaction increases.

We therefore write:

\[
\begin{align*}
p < m & \quad \begin{cases} 
\mathcal{B}_{21}^i(r = m) = 1 - \left(1 - \tanh\left(\frac{1}{1-\gamma}\right)\right)B_p^i \\
\mathcal{B}_{21}^i(r = p + 1) = \left(1 - \tanh\left(\frac{1}{1-\gamma}\right)\right)B_p^i \\
\mathcal{B}_{21}^i(r \neq p + 1) = 0 \\
\mathcal{B}_{21}^i(r \neq m) = 0
\end{cases} \\
p = m & \quad \begin{cases} 
\mathcal{B}_{21}^i(r = m) = 1 - \left(1 - \tanh\left(\frac{1}{1-\gamma}\right)\right)B_p^i \\
\mathcal{B}_{21}^i(r = 1) = \left(1 - \tanh\left(\frac{1}{1-\gamma}\right)\right)B_p^i \\
\mathcal{B}_{21}^i(r \neq m) = 0 \\
\mathcal{B}_{21}^i(r \neq 1) = 0
\end{cases}
\end{align*}
\]
From the above table we see that when the susceptibility is greater than the mean value, then \( u_p \rightarrow u_{p+1} \) in the case of a small vaccine reaction \( \gamma \); when \( \gamma \) increases instead, the susceptible individual will be driven toward the healed population. In the special case \( p = m \) for small \( \gamma \), the individual will be driven to the first stadium of the third population (illness).

- \( B_{23}^i(r) = B_{23}^i[u_f(u_p \rightarrow u_r, u_q)] \).

We are considering here the interaction between a susceptible individual and an infected one. In this case the parameter playing a fundamental role is the infectivity \( \chi \).

On the basis of empirical considerations we assume that the probability density of susceptible individual \( B_{23}^i \) to remain in the same population with a bigger (the same) susceptibility, increases (decreases) as \( \chi \) increases.

On the other hand, when \( p = m \) the susceptible individual has a probability density \( B_{23}^3 \) to make a transition in the first class of the infected population. Such probability density is an increasing function of \( \chi \).

We therefore write:

\[
\begin{align*}
\begin{cases}
\quad & p < m \\
\quad & p = m
\end{cases}
\end{align*}
\]

\[
\begin{align*}
    u_p \geq E_2^1[f_2] \quad & \begin{cases}
    B_{23}^2(r = p - 1) = 0 \\
    B_{23}^2(r = p) = 1 - e^{\chi - 1} \\
    B_{23}^2(r = p + 1) = e^{\chi - 1} \\
    B_{23}^2(r \neq p - 1, p, p + 1) = 0 \\
    B_{23}^2(r = m) = 1 - e^{\chi - 1} \\
    B_{23}^2(r \neq m) = 0 \\
    B_{23}^2(r \neq 1) = 0
    \end{cases} \\
    u_p < E_2^1[f_2] \quad & \begin{cases}
    B_{23}^2(r = p - 1) = 0 \\
    B_{23}^2(r = p) = 1 - e^{(1 - B_{23}^2)x - 1} \\
    B_{23}^2(r = p + 1) = e^{(1 - B_{23}^2)x - 1} \\
    B_{23}^2(r \neq p - 1, p, p + 1) = 0
    \end{cases}
\end{align*}
\]

As specified before, the above table refers to the interactions susceptible/affected. When the susceptibility is above the mean value, the probability transition \( u_p \rightarrow u_{p+1} \) increases as the infectivity \( \chi \) increases. For \( p = m \) the transition refers to the first stadium of the third population.

When \( u_p \) is above the mean value, the transition \( u_p \rightarrow u_{p+1} \) is again driven by the infectivity \( \chi \) but now there is a dumping effect due to the distance from the mean value.

4.1.2. Tables of games for \( B_{3k}^i(r) \), for \( k = 1, \ldots, 4 \). (individuals affected by the disease)

The following tables are derived on the basis of empirical considerations along the same lines of the one reported in (see 4.1.1).
In particular, in the table reported below, we consider the interactions between infected individuals \( (k = 3) \) and individuals belonging to the susceptible \( (k = 2) \), infected \( (k = 3) \) and removed \( (k = 4) \) populations.

- \( \mathcal{B}^{ij}_{3k}(r) = \mathcal{B}^{ij}_{3k}[f](u_p \rightarrow u_q | u_p, u_q) \), for \( k = 2, 3, 4 \).

\[
\begin{align*}
\mathcal{B}^{3k}_{3k}(r) & = 0 \\
\mathcal{B}^{3k}_{3k}(r) & = 1 - B^p_k \\
\mathcal{B}^{3k}_{3k}(r + 1) & = B^p_k \\
\mathcal{B}^{3k}_{3k}(r - 1) & = 1 \\
\mathcal{B}^{3k}_{3k}(r) & = 0 \\
\mathcal{B}^{3k}_{3k}(r) & = 0 \\
\mathcal{B}^{3k}_{3k}(r) & = 0 \\
\mathcal{B}^{3k}_{3k}(r) & = 0 \\
\end{align*}
\]

When we consider individuals affected by the disease (above table) we observe that when the level of illness is below the mean value, the activity \( u_p \) does not change. In all other cases the probability transition \( u_p \rightarrow u_{p+1} \) increases as the distance \( B^p_k \) from the mean value increases. We point out that in the special case \( p = m \), the individuals end up in the healed individuals population.

Next, we report the table referring to the interactions between infected individuals \( (k = 3) \) and doctors \( (k = 1) \).

- \( \mathcal{B}^{ij}_{31}(r) = \mathcal{B}^{ij}_{31}[f](u_p \rightarrow u_q | u_p, u_q) \).

\[
\begin{align*}
\mathcal{B}^{31}_{31}(r) & = 0 \\
\mathcal{B}^{31}_{31}(r) & = 0 \\
\mathcal{B}^{31}_{31}(r) & = 1 - B^p_1 \\
\mathcal{B}^{31}_{31}(r) & = 1 - B^p_1 \left(1 - B^p_3 \right) \\
\mathcal{B}^{31}_{31}(r) & = 0 \\
\mathcal{B}^{31}_{31}(r) & = 0 \\
\mathcal{B}^{31}_{31}(r) & = 0 \\
\mathcal{B}^{31}_{31}(r) & = 0 \\
\end{align*}
\]
In the first part of the above table is described the case when the degree of illness is above the mean value. Then the output of the interaction depends both on the doctors’ ability and on the distance $B^3_{q3}$ of the activity $u_p$ from the mean value. The illness will tend to evolve toward the healed state as rapidly as $B^3_{q3}$ increases and the distance $B^3_{q1}$ decreases. In the second case, when $u_p$ is below the mean value, we have the opposite behavior.

5. Numerical Simulations

The results of numerical simulations are shown in Figs. 1–4, where initial configurations for the populations distribution among the different activity classes ($m=6$) are shown vs final and also intermediate (Fig. 1) configurations. In our simulations we used realistic initial values of the parameters, coming from the CIRI database (Interuniversity Center Flu Research).

In Figs. 1–4 we represent the different populations from top to bottom, reporting on the left the initial values and on the right the final (asymptotic) values or intermediate values. The $\alpha_j$ parameters concerning the encounter rates (4.2)–(4.10) are fixed as $\alpha_1 = 0.9$, $\alpha_2 = 2.5$, $\alpha_3 = 0.5$, $\alpha_4 = 0.7$. Moreover, in Figs. 1–3, we keep fixed the intensity of vaccine reaction $\gamma = 0.94$, the infectivity $\chi = 0.9$ and the doctors’ ability $\delta = 0.9$; we wish to observe how the evolution of the system changes by changing the risk perception parameter $\beta$.

In Figs. 1 and 2 we put $\beta = 0.23$. The initial distribution for doctors, susceptible individuals and infective individuals is chosen to be uniform. In Fig. 1, we look at the configuration taken at an intermediate time, while in Fig. 2, we look at the configuration taken at the final (asymptotic) time. In Fig. 1, we can follow the evolution of the epidemics, and see how the doctors progressively migrate to the last class, while the susceptible individuals became part infected and part susceptible to the last stage. Also, the infected people start to migrate toward the removed population.

In Fig. 2, as expected, all the doctors migrate eventually in the last class. They learn from experience by treating many different cases and become expert both in prevention and in the handling of the epidemic. When we look at the second
Fig. 1. Initial (left) and intermediate (right) configurations for the populations distribution among the different activity classes. Here, the parameters are fixed as $\alpha_1 = 0.9$, $\alpha_2 = 2.5$, $\alpha_3 = 0.5$, $\alpha_4 = 0.7$, $\gamma = 0.94$, $\delta = 0.9$, $\chi = 0.9$, $\beta = 0.23$.

Fig. 2. Initial (left) and asymptotic (right) configurations for the populations distribution among the different activity classes. Here, the parameters are fixed as $\alpha_1 = 0.9$, $\alpha_2 = 2.5$, $\alpha_3 = 0.5$, $\alpha_4 = 0.7$, $\gamma = 0.94$, $\delta = 0.9$, $\chi = 0.9$, $\beta = 0.23$. 
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Fig. 3. Initial (left) and asymptotic (right) configurations for the populations distribution among the different activity classes. Here, the parameters are fixed as $\alpha_1 = 0.9$, $\alpha_2 = 2.5$, $\alpha_3 = 0.5$, $\alpha_4 = 0.7$, $\gamma = 0.94$, $\delta = 0.9$, $\chi = 0.9$, $\beta = 0.34$.

Fig. 4. Initial (left) and asymptotic (right) configurations for the populations distribution among the different activity classes. Here, the parameters are fixed as $\alpha_1 = 0.9$, $\alpha_2 = 2.5$, $\alpha_3 = 0.5$, $\alpha_4 = 0.7$, $\gamma = 0.94$, $\delta = 0.9$, $\chi = 0.1$, $\beta = 0.25$. 

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population, we see that there are no susceptible individuals in the corresponding final states. The same happens with the individuals affected by the disease. On the other hand, the total number of individuals of the two populations, susceptible + infective, make a migration in the last class of the removed population which constitute their final (asymptotic state). In other words, the risk perception is not big enough to prevent susceptible individuals from exposing themselves to the infection. It appears that all of them contract the disease, and then, as time goes on they end up in the removed population.

The situation is instead different in Fig. 3 where the risk perception parameter $\beta$ is higher ($\beta = 0.34$). The first population (doctors) has the same behavior as in Fig. 2. Also the third population (infected individuals) has the same behavior as in Fig. 2: all the individuals eventually end up in the last class of the removed population. More interesting is the evolution of the second population: looking carefully at the final values for the different classes of the susceptible population, it appears that there are two migration phenomena. First of all, there are individuals with a low level of susceptibility which get immunized (vaccinate) and migrate into the removed population; then there are those who migrate to a different class (higher level of susceptibility) and either stay there or contract the disease and then, as time goes on, end up in the removed population. In our last figure we take now the risk perception at a lower level, $\beta = 0.25$, with a low level of infectivity $\chi = 0.1$ and a high reaction to the vaccine $\gamma = 0.94$. We start with uniform initial distribution for the different populations. When we look at the final (asymptotic) configuration we find a situation which is almost identical to the one corresponding to high risk perception $\beta = 0.34$, with high infectivity $\chi = 0.9$ and the same reaction to the vaccine $\gamma = 0.94$, shown in Fig. 3. In other words, from the comparison of Figs. 3 and 4, it appears that a variation in $\chi$ (infectivity) of order $8 \cdot 10^{-1}$ has the same effect as a variation in $\beta$ (risk perception) of order $9 \cdot 10^{-2}$.

6. Conclusions

In this paper, we introduced a mathematical model suitable to describe the onset and the evolution of epidemics. The model is characterized by three fundamental parameters: the risk perception, the infectivity and the vaccine reaction.

Our results show how such parameters influence both the onset and the evolution of the infective disease. In particular, as expected, the epidemic spread can be controlled by increasing the risk perception. At the same time we observe that an increase of the infectivity induces and promotes the diffusion of the infection.

The approach presented here can be further developed in order to include some important aspects. In particular:

- we believe that the mathematical structure should be generalized to open systems, incorporating birth and death processes;
a space dependence of the model could be possibly derived through an asymptotic analysis of the microscopic model described by the kinetic theory approach. This would open the way to the description of propagation phenomena with finite velocity and also to relate the spread of epidemics to space dynamics of individuals affected by the pathology.

References

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