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A kinetic framework under the action of an external force field: Analysis and application in epidemiology

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ABSTRACT

The tools of kinetic theory allow to describe the dynamics and evolution of a system composed of stochastically interacting particles. The interaction is modeled by means of two classes of parameters, i.e. interaction rates and transition probabilities. Therefore, a system of nonlinear ordinary differential equations is derived. Nevertheless, in general, this structure does not consider the action of an external environment. This paper aims at providing a new kinetic model where an external action occurs. Specifically, this action over the system is modeled by introducing an external force field. Then, a new kinetic model is derived, and some analytical results towards the solution of the related Cauchy problem are provided, in the conservative case: existence, uniqueness, positivity and boundedness. Finally, an application in the contest of mathematical epidemiology is given; the new kinetic framework is characterized for three classical compartmental models: SIR, SEIIR and SEIIRS. Stability results and numerical simulations, in agreement with classical theory, confirm the adherence to reality of this new model.

1. Introduction

In the last decades the interest towards interacting systems has been growing in the scientific community, due to the studies regarding complex systems [1]. This interest is referred to systems composed of *agents*, also called *particles*, such that their interactions follow stochastic rules. In particular, each interaction is binary and is modeled by some probabilities. Therefore the evolution of such systems is defined by these stochastic interactions.

An interacting system is studied at different scales. At *microscopic scale*, the interest is focused on each particle, by gaining the related evolution equation; then the final dynamics is the sum of all these equations. At *macroscopic scale*, the overall state of the system is considered, regarding it as a unique entity. Nevertheless, there is an intermediate level that allows to study statistically the evolution of the system, by introducing a *distribution function* over the system. This is the *mesoscopic scale*. At this level, the microscopic state of the systems is defined by some variables, on whom the distribution function is defined. The macroscopic state is describe by some moments of the distribution function itself.

Among others, *Kinetic Theory* is widely used for the study of a system at a mesoscopic level, [2–6]. In particular, the microscopic level is described by classical *mechanical variables*, i.e. *space* and *velocity*, whereas there is another real variable, generally called *activity*, whose particular meaning depends on the application taken into account. This real variable attains its value in a continuous or discrete subset of $\mathbb{R}$. Moreover, the overall system is divided into *functional subsystems* [7], such that particles belonging to the same functional subsystem share the same *strategy*. On each functional subsystem, a distribution function is introduced. Then the evolution of the ith functional subsystem is described by a suitable integro-differential equation, partial differential equation or ordinary differential equation, in dependence of the shape of microscopic variables. One of the most successful aspect of kinetic theory is the huge versatility of its models and equations. Indeed, it has been applied to biology [8–12], economy [13–15], opinion dynamic [16–18], vehicular traffic [19,20], psychology [21,22] and so on.

In general, kinetic models are *closed*, that is the dynamics is modeled only by binary and stochastic interactions among the particles. And so, the action of an external force field is neglected. However, some applications require these kinds of interactions, otherwise kinetic equations would be not so realistic. For example, in a ecological model, the action of external environment is fundamental for the evolution of the overall system, e.g. rainfall, drought, water levels, climate change.

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Nevertheless, the introduction of an external force field in the system of kinetic equations causes some problems. Among others, form an analytical viewpoint, this could make the system *nonconservative*, i.e. the total density is not conserved during the evolution, and so blow-up phenomena of solutions may appear, [23]. In order to avoid such critical situations, when external actions are considered, thermostatted kinetic models have been recently introduced in [24–29].

Firstly, in this paper, a system *homogeneous* with respect to the mechanical variables is considered, where the microscopic state is described by the only activity variable. This variable acquires its values in a discrete real subset. Nevertheless, the evolution depends not only on the binary conservative interactions among the particles, but also on the action of an *external force field* $\mathbf{F}[\mathbf{f}](t)$. In this paper there is not the most general external force field, but it has a particular shape. Indeed, each component of $\mathbf{F}[\mathbf{f}](t)$ depends on the density of each functional subsystem, with some coefficients. But some assumptions on these coefficients ensure that the total density of the system is conserved, and then any thermostat is required.

The main novelty of this paper is the introduction of a new discrete kinetic framework under the action of an external force field, which has a specific analytical shape. This is not only a theoretical interest towards kinetic modeling, but it is relevant for the applications, as widely demonstrated in what follows. Moreover, the action of an external environment on a stochastically interacting system, with its consequences, has to depend on the current state of the overall system, with its components. This motivates the particular analytical shape for the external force field. Accordingly, this choice ensures some analytical results towards existence, uniqueness, positivity and boundedness of the solution, globally in time. The latter property is possible thanks to conservative assumptions on the external force field. At the best of our knowledge, this represents a first attempt in the modeling of a kinetic framework under the action of a specific external force field, whose components depend on the current state of the system. Furthermore, the generality of this scheme may represent a new possible approach for nonconservative interacting systems.

It is worth stressing that the novelty of the introduction of an eternal force field in a kinetic framework goes beyond the theoretical viewpoint. Indeed, a specific application is presented, in the contest of mathematical epidemiology (see [30,31] and references therein). In the last years, this research area has been growing, with several new results, [32–38]. Kinetic theory framework has just been applied in mathematical epidemiology (see [39–43] and references therein), also thanks to what happened with the recent COVID-19 pandemic. Nevertheless, the new kinetic framework, here presented, allows to model recovery process and reinfections, if they occur, as consequences of external factors, regardless of binary and stochastic interactions. This is a realistic assumption. Then, the new kinetic model proposed in this paper is applied with respect to three different compartmental models: SIR, SEIIR and SEIIRS. This application aims at proving how well this new kinetic model models fits with *classical* schemes, also in terms of some stability results, which are not so common in kinetic theory. However, analytical and numerical results gained for these models furnish some interesting novelties and perspectives, among the others, in terms of bifurcations and statistical viewpoint.

After this brief introduction, the paper is organized in four more sections. Section 2 presents some tools of kinetic theory, regarding models homogeneous with respect space and velocity, with discrete activity variable. In Section 3, the new model with external force field is presented, with some analytical results. The application to mathematical epidemiology is shown in Section 4, where some stability results are proved. Finally, Section 5 discusses final remarks and future research perspective.

2. The kinetic framework

Let consider an *interacting system* composed of *particles* (or *agents*), that have *stochastic interactions*. Specifically, the system is divided into $n \in \mathbb{N}$ *functional subsystems*. The *microscopic state* of the system is described by a *discrete variable* u that acquires values in a discrete real subset, i.e.

$$u \in \mathcal{I} = \{u_1, u_2, \dots, u_n\} \subseteq \mathbb{R}.$$

The *distribution function* of the ith functional subsystem is

$$f_i(t) : [0, T] \to \mathbb{R}^+,$$

and gives the number of particles at time $t > 0$ in the microscopic state u_i. Whereas $\mathbf{f}(t) = (f_1(t), f_2(t), \dots, f_n(t))$ is the *vector distribution function* of the overall system.

The *macroscopic state* of the system is derived by the introduction of the *pth-order moment*, for $p \in \mathbb{N}$, related to the distribution function $\mathbf{f}$. Specifically, it defines

$$\mathbb{E}_p[\mathbf{f}](t) := \sum_{i=1}^{n} u_i^p \, f_i(t).$$

By acquiring a physical viewpoint, the zeroth-order moment, the first-order moment and the second-order moment correspond to density, linear momentum and global activation energy, respectively.

The stochastic microscopic dynamics of the system is defined by some suitable quantities that model the interactions between pairs of particles. Specifically:

- The *interaction rate* η_{hk}, for $h, k \in \{1, 2, \dots, n\}$, gives the number of encounters between particles of the hth function subsystem and particles of the kth functional subsystem.
- The *transition probability* B^i_{hk}, for $i, h, k \in \{1, 2, \dots, n\}$, gives the probability that a particle of the hth functional subsystem falls into the ith functional subsystem, after interacting with a particle of the kth functional subsystem. Since B^i_{hk} is a probability, hereafter it is assumed that

$$\sum_{i=1}^{n} B^i_{hk} = 1, \qquad \forall h, k \in \{1, 2, \dots, n\}.$$

Bearing all above in mind, the evolution of the ith functional subsystem, for $i \in \{1, 2, \dots, n\}$, is described by the following system of kinetic equations:

$$\begin{aligned} \frac{df_i}{dt} &= G_i[\mathbf{f}](t) - L_i[\mathbf{f}](t) \\ &= \sum_{h,k=1}^{n} \eta_{hk} B^i_{hk} f_h(t) f_k(t) - f_i(t) \sum_{k=1}^{n} \eta_{ik} f_k(t). \end{aligned} \tag{1}$$

It is a system of nonlinear ordinary differential equations with quadratic nonlinearity. Specifically:

- $G_i[\mathbf{f}](t) := \sum_{h,k=1}^{n} \eta_{hk} B^i_{hk} f_h(t) f_k(t)$ is the *gain term operator* that gives the number of particles that fall into the ith functional subsystem, after interacting with a particle of the kth functional subsystem.
- $L_i[\mathbf{f}](t) := f_i(t) \sum_{k=1}^{n} \eta_{ik} f_k(t)$ is the *loss term operator* that gives the number of particles that leave the ith functional subsystem due to interactions with other particles.

Then the operator

$$J_i[\mathbf{f}](t) := G_i[\mathbf{f}](t) - L_i[\mathbf{f}](t)$$

gives the *net flux* of particles related to the ith functional subsystem, for $i \in \{1, 2, \dots, n\}$. The framework (1) is *conservative* since B^i_{hk} is a probability, for all $h, k \in \{1, 2, \dots, n\}$, and there is not any *nonconservative*

term. Therefore, the density, i.e. the 0th-order moment, of the system is conserved. Moreover, we require the solution $\mathbf{f}(t)$, if it exists, to satisfy

$$\mathbb{E}_0[\mathbf{f}](t) = \sum_{i=1}^{n} f_i(t) = 1, \qquad \forall t > 0.$$

Then $\mathbf{f}(t)$ is a probability distribution, whereas this is not true in a general framework, where $\mathbf{f}(t)$ is only a distribution over the system.

3. A kinetic model with an external force term

The conservative kinetic framework (1) is useful to describe the evolution of systems where interactions among particles are the predominant elements of the dynamics. Nevertheless, this framework does not consider external actions. However, in some applications these external interactions may not be neglected, if a realistic description of the dynamics is required.

In order to consider such external actions, an *external force term* is introduced in the conservative kinetic framework (1). Specifically, the *external force field* is modeled by a function

$$\mathbf{F}[\mathbf{f}](t) : [0, T] \to \mathbb{R}^n, \qquad t > 0,$$

where

$$\mathbf{F}[\mathbf{f}](t) = \left(F_1(t), F_2(t), \dots, F_n(t)\right).$$

Then, the kinetic equation for the evolution of the ith function subsystem now is

$$\frac{df_i}{dt} = \sum_{h,k=1}^{n} \eta_{hk} B_{hk}^i f_h(t) f_k(t) - f_i(t) \sum_{k=1}^{n} \eta_{ik} f_k(t) + F_i(t). \tag{2}$$

The structure and analytical properties of the system (2) depend on the particular shape of the external force field $\mathbf{F}[\mathbf{f}](t)$, which is related to the particular application taken into consideration. This paper aims at deriving and studying kinetic equations for an external force field with the following analytical shape:

$$\mathbf{F}[\mathbf{f}](t) = \left(\sum_{k=1}^{n} \Gamma_1^k(t) f_k(t), \sum_{k=1}^{n} \Gamma_2^k(t) f_k(t), \dots, \sum_{k=1}^{n} \Gamma_n^k(t) f_k(t) \right), \tag{3}$$

where $\Gamma_i^k(t)$ is a continuous real-valued function, for all $i, k \in \{1, 2, \dots, n\}$. Then, the kinetic equation that models the evolution of the ith functional subsystem writes

$$\frac{df_i}{dt} = \sum_{h,k=1}^{n} \eta_{hk} B_{hk}^i f_h(t) f_k(t) - f_i(t) \sum_{k=1}^{n} \eta_{ik} f_k(t) + \sum_{k=1}^{n} \Gamma_i^k(t) f_k(t). \tag{4}$$

Roughly speaking, the analytical shape (3) of the external force field, i.e. $F_i(t) = \sum_{k=1}^{n} \Gamma_i^k(t) f_k(t)$, for $i \in \{1, 2, \dots, n\}$, may have the following physical interpretation. For $i \in \{1, 2, \dots, n\}$, $F_i(t)$ gives the number of particles that enter or leave the ith functional subsystem due to external actions that depend on functional subsystems and related densities. Indeed, the external action $\Gamma_i^k(t) f_k(t)$ on the ith functional subsystem, for $i \in \{1, 2, \dots, n\}$, related to the kth functional subsystem, for $k \in \{1, 2, \dots, n\}$, is modeled by the time-dependent function $\Gamma_i^k(t)$, whereas the dependence of this action with respect to the density of th kth functional subsystem is defined by the presence of the distribution function $f_k(t)$ itself. Therefore, the external force field (3) is a *non-direct* interaction among the ith and the kth functional subsystem, since these interactions do not depend on the stochastic interactions among the particles, that are modeled by parameters η_{hk} and B_{hk}^i, for $i, h, k \in \{1, 2, \dots, n\}$.

Given a *suitable initial data* $\mathbf{f}^0 = \left(f_1^0, f_2^0, \dots, f_n^0\right) \in \mathbf{R}^n$, the *Cauchy problem* related to the new kinetic framework (4) writes

$$\begin{cases} \dfrac{df_i}{dt} = J_i[\mathbf{f}](t) + \sum_{k=1}^{n} \Gamma_i^k(t) f_k(t), \quad t > 0 \\ \mathbf{f}(0) = \mathbf{f}^0. \end{cases} \tag{5}$$

In general, the action of an external force field makes the system *nonconservative*, that is the case of kinetic model (4), presented in this paper. Indeed, the overall density of the system may change during the evolution, then the solution $\mathbf{f}(t)$ of the Cauchy problem (5), if exists, may not be a probability. Moreover, the nonconservative structure does not ensure, in general, other analytical properties of solution: uniqueness, boundedness and positivity.

In order to preserve the analytical properties of conservative case (1), the following assumptions towards the external force field (3) are assumed:

A1 The functions $\Gamma_i^k(t)$ are time-independent, i.e., for all $i, k \in \{1, 2, \dots, n\}$

$$\frac{d\,\Gamma_i^k(t)}{dt} = 0, \qquad t \geq 0.$$

A2 For all $i \in \{1, 2, \dots, n\}$,

$$\Gamma_i^k = \begin{cases} \geq 0, & k \neq i \\ \\ \leq 0, & k = i. \end{cases}$$

A3 The coefficients Γ_i^k are such that, for all $k \in \{1, 2, \dots, n\}$,

$$\sum_{i=1}^{n} \Gamma_i^k = 0.$$

The assumption **A2** may have the following physical interpretation. For $i \in \{1, 2, \dots, n\}$, the ith functional subsystem can only acquire particles due to the external actions related to the other functional subsystem, i.e. $\Gamma_i^k f_k(t)$, for $k \neq i$. Whereas, it may only loose particles due to the part of external force field related to the ith functional subsystem itself, i.e. $\Gamma_i^i f_i(t)$. Roughly speaking, the external actions $\Gamma_i^k f_k(t)$, for $k \neq i$, moves particles out of the ith functional subsystems, whereas $\Gamma_i^i f_i(t)$ is the only part of $F_i(t)$ that furnishes new particles.

The assumption **A3** ensures the conservative structure of the system. Indeed,

$$\frac{d}{dt} \sum_{i=1}^{n} f_i(t) = \sum_{i=1}^{n} \left(\eta_{hk} B_{hk}^i f_h(t) f_k(t) - f_i(t) \sum_{k=1}^{n} \eta_{ik} f_k(t) \right) + \sum_{i=1}^{n} \sum_{k=1}^{n} \Gamma_i^k f_k(t) = 0.$$

Then, the solution $\mathbf{f}(t)$ of the Cauchy problem (5), if exists, remains a probability as well as for the conservative model (1).

Bearing all above in mind, the following analytical result holds true.

Theorem 1.

Let consider the Cauchy problem (5), such that

$$\sum_{i=1}^{n} B_{hk}^i = 1, \qquad \forall h, k \in \{1, 2, \dots, n\}.$$

*Let assume that assumptions **A1**-**A2**-**A3** hold true. Moreover, there exist two positive constants η, Γ such that:*

- $\eta_{hk} \leq \eta$, *for all* $h, k \in \{1, 2, \dots, n\}$.
- $\Gamma_i^k \leq \Gamma$, *for all* $i, k \in \{1, 2, \dots, n\}$.

Furthermore, the initial data $\mathbf{f}^0 \in \mathbb{R}^n$ is such that $\sum_{i=1}^{n} f_i^0 = 1$. Then, there exists a unique, positive and bounded solution $\mathbf{f}(t) \in (C^\infty([0, +\infty[))^n$ of the related Cauchy problem, such that

$$\mathbb{E}_0[\mathbf{f}](t) = \sum_{i=1}^{n} f_i(t) = 1, \qquad \forall t \geq 0.$$

Proof. The Eq. (4) rewrites, for $i \in \{1, 2, \dots, n\}$,

$$\frac{df_i}{dt} = P_i[\mathbf{f}](t),$$

where

$$P_i[\mathbf{f}](t) := \sum_{h,k=1}^{n} \eta_{hk} B^i_{hk} f_h(t) f_k(t) - f_i(t) \sum_{k=1}^{n} \eta_{ik} f_k(t) + \sum_{k=1}^{n} \Gamma^k_i f_k(t)$$

is an operator on the set of C^∞ functions. Let, now, $\mathbf{f}, \mathbf{g} \in (C^\infty([0,T]))^n$, for $T > 0$, such that they are positive and satisfy $\sum_{h=1}^n f_h(t) = \sum_{h=1}^n g_h(t) = 1$. Straightforward computations show that

$$\begin{aligned} \left|P_i[\mathbf{f}](t) - P_i[\mathbf{g}](t)\right| &= \left| \sum_{h,k=1}^{n} \eta_{hk} B^i_{hk} f_h(t) f_k(t) - f_i(t) \sum_{k=1}^{n} \eta_{ik} f_k(t) + \sum_{k=1}^{n} \Gamma^k_i f_k(t) \right. \\ &\quad \left. - \sum_{h,k=1}^{n} \eta_{hk} B^i_{hk} g_h(t) g_k(t) + g_i(t) \sum_{k=1}^{n} \eta_{ik} g_k(t) - \sum_{k=1}^{n} \Gamma^k_i g_k(t) \right|, \end{aligned}$$

from which

$$\begin{aligned} \left|P_i[\mathbf{f}](t) - P_i[\mathbf{g}](t)\right| \leq & \left| \sum_{h,k=1}^{n} \eta_{hk} B^i_{hk} \left(f_h(t) f_k(t) - g_h(t) g_k(t) \right) \right| + \\ & \left| \sum_{k=1}^{n} \eta_{ik} \left(g_i(t) g_k(t) - f_i(t) f_k(t) \right) \right| \\ & + \left| \sum_{k=1}^{n} \Gamma^k_i \left(f_k(t) - g_k(t) \right) \right|. \end{aligned} \tag{6}$$

By using the assumptions of boundedness, the (6) rewrites

$$\begin{aligned} \left|P_i[\mathbf{f}](t) - P_i[\mathbf{g}](t)\right| \leq & \eta \left| \sum_{h,k=1}^{n} \left(f_h(t) f_k(t) - g_h(t) g_k(t) \right) \right| \\ & + \eta \left| \sum_{k=1}^{n} \left(g_i(t) g_k(t) - f_i(t) f_k(t) \right) \right| \\ & + \Gamma \left| \sum_{k=1}^{n} \left(f_k(t) - g_k(t) \right) \right|. \end{aligned} \tag{7}$$

Since

$$\begin{aligned} f_h(t) f_k(t) - g_h(t) g_k(t) &= f_h(t) f_k(t) - f_h(t) g_k(t) + f_h(t) g_k(t) - g_h(t) g_k(t) \\ &= f_h(t) \left(f_k(t) - g_k(t) \right) + g_k(t) \left(f_h(t) - g_h(t) \right), \end{aligned}$$

the (7) writes

$$\begin{aligned} \left|P_i[\mathbf{f}](t) - P_i[\mathbf{g}](t)\right| \leq & \eta \sum_{h=1}^{n} f_h(t) \sum_{k=1}^{n} \left| f_k(t) - g_k(t) \right| + \eta \sum_{k=1}^{n} g_k(t) \sum_{h=1}^{n} \left| f_h(t) - g_h(t) \right| \\ & + \eta f_i(t) \sum_{k=1}^{n} \left| f_k(t) - g_k(t) \right| + \eta \sum_{k=1}^{n} g_k(t) \left| f_i(t) - g_i(t) \right| \\ & + \Gamma \sum_{k=1}^{n} \left| f_k(t) - g_k(t) \right| \leq 4\eta \|\mathbf{f}(t) - \mathbf{g}(t)\|_{l^1} + \Gamma \|\mathbf{f}(t) - \mathbf{g}(t)\|_{l^1} \\ & \leq (4\eta + \Gamma) \|\mathbf{f}(t) - \mathbf{g}(t)\|_{l^1}. \end{aligned} \tag{8}$$

Passing to the sup in the (8) on $[0, T]$, for $T > 0$, it follows

$$\|P_i[\mathbf{f}](t) - P_i[\mathbf{g}](t)\|_{C^\infty([0,T[)} \leq C \|\mathbf{f}(t) - \mathbf{g}(t)\|_{C^\infty([0,T[)}, \tag{9}$$

where C is a positive constant that depends on the parameters of the system, i.e. η and Γ among others. Then, there exists a unique local solution of the Cauchy problem (5).

In order to prove the positivity of the solution $\mathbf{f}(t)$, the (4) has to be rewritten in the following form, for $i \in \{1, 2, \dots, n\}$,

$$\frac{df_i}{dt} + f_i(t) A_i[\mathbf{f}](t) = B_i[\mathbf{f}](t), \tag{10}$$

where

$$A_i[\mathbf{f}](t) = \sum_{k=1}^{n} \left(\eta_{ik} + \Gamma^k_i \right) f_k(t)$$

$$B_i[\mathbf{f}](t) = \sum_{h,k=1}^{n} \eta_{hk} B^i_{hk} f_h(t) f_k(t) + \Gamma^i_i f_i(t).$$

Let now

$$\gamma_i(t) = \int_0^t A_i[\mathbf{f}](\tau)\, d\tau.$$

Then, straightforward computations show from the (10) that, for $i \in \{1, 2, \dots, n\}$,

$$f_i(t) = f_i^0 e^{-\gamma_i(t)} + \int_0^t e^{\gamma_i(\tau) - \gamma_i(t)} B_i[\mathbf{f}](\tau)\, d\tau. \tag{11}$$

Bearing the expression (11) in mind, the positivity of function $\mathbf{f}(t)$ is ensured by the positivity of exponential function and assumption **A2**.

Finally, let consider the integral form of Eq. (4). Then, by integrating in $[0, t]$, one has

$$\begin{aligned} f_i(t) = f_i^0 + & \int_0^t \left(\eta_{hk} B^i_{hk} f_h(\tau) f_k(\tau) - f_i(\tau) \sum_{k=1}^{n} \eta_{ik} f_k(\tau) \right) d\tau \\ & + \int_0^t \sum_{k=1}^{n} \Gamma^k_i f_k(\tau)\, d\tau. \end{aligned} \tag{12}$$

By summing the (12) on $i = 1, 2, \dots, n$ and bearing assumption**A3** in mind, the following is gained

$$\begin{aligned} \sum_{i=1}^{n} f_i(t) &= \sum_{i=1}^{n} f_i^0 + \int_0^t \sum_{i=1}^{n} \left(\eta_{hk} B^i_{hk} f_h(\tau) f_k(\tau) - f_i(\tau) \sum_{k=1}^{n} \eta_{ik} f_k(\tau) \right) d\tau \\ &\quad + \int_0^t \sum_{i,k=1}^{n} \Gamma^k_i f_k(\tau)\, d\tau \\ &= 1. \end{aligned} \tag{13}$$

Then, the boundedness of the solution is proved. Moreover, the relation (13) ensures that

$$\mathbb{E}_0[\mathbf{f}](t) = \sum_{i=1}^{n} f_i(t) = 1, \qquad \forall t \geq 0.$$

Since $f_i(t) < +\infty$, for all $i \in \{1, 2, \dots, n\}$ and for all $t > 0$, then the global existence and uniqueness of a positive solution is gained. This concludes the proof. □

Remark 1. Theorem 1 ensures that the new kinetic framework with external force field (4), under the assumptions **A1**-**A3**, is conservative. In particular, the assumption **A3** is sufficient condition for having a conservative framework. It is worth stressing that, since $\mathbb{E}_0[\mathbf{f}](t) = 1$, for all $t \geq 0$, the solution $\mathbf{f}(t)$ can be regarded as a probability.

4. An application to epidemiology

This Section aims at applying the new kinetic framework with external force field (4), under the assumptions **A1**-**A3**, for a disease spreading. The overall population is divided into compartments. Then the system (4) writes

$$\frac{df_i}{dt} = \sum_{h,k=1}^{n} \eta_{hk} B^i_{hk} f_h(t) f_k(t) - f_i(t) \sum_{k=1}^{n} \eta_{ik} f_k(t) + \sum_{\substack{k=1 \\ k \neq i}}^{n} \Gamma^k_i f_k(t) + \Gamma^i_i f_i(t). \tag{14}$$

In particular, each functional subsystem represents a specific compartment. Moreover, the parameters of (14) acquire the following meaning:

- The interaction rate η_{hk}, for $h, k \in \{1, 2, \dots, n\}$, represents the number of encounter between agents of the hth compartment and agents of the kth compartment.
- The transition probability B^i_{hk}, for $i, h, k \in \{1, 2, \dots, n\}$, represents the probability that an agent of the hth compartment moves to the ith one after an interaction with an agent of the kth compartment.
- The coefficient Γ^k_i, for $i \in \{1, 2, \dots, n\}$ and $i \neq k$, models the action of the external force field related to the kth functional subsystem, and in dependence of its density $f_k(t)$, that moves particles inside the ith functional subsystem. Otherwise, the coefficient Γ^i_i refers to the action of the external force field which causes the loss of particles, in dependence of the density $f_i(t)$ of the ith functional

subsystem itself. According to the assumption **A2**: $\Gamma_i^k \geq 0$, for $i \neq k$, whereas $\Gamma_i^i \leq 0$.

Given a suitable initial data $\mathbf{f}^0 = \left(f_1^0, f_2^0, \dots, f_n^0\right)$, the Cauchy problem related to the system (14) is obtained. The existence, uniqueness, positivity and boundness of solution $\mathbf{f}(t) = \left(f_1(t), f_2(t), \dots, f_n(t)\right)$ are ensured by previous Theorem 1. Hereafter, it is assumed that

$$\sum_{i=1}^{n} f_i^0 = 1,$$

then the solution $\mathbf{f}(t)$ is a probability, i.e.

$$\sum_{i=1}^{n} f_i(t) = 1, \qquad \forall t \geq 0.$$

Moreover, $f_i(t)$ can be seen as the fraction of individuals of the i functional subsystem, for $i \in \{1, 2, \dots, \}$.

In the next Subsections, three different compartmental models for disease spreading are described by using the new kinetic framework with external force field (14): SIR, SEIIR, SEIIRS. The related Cauchy problems are derived. For each model, the basic reproduction number $\mathcal{R}_0$ is obtained by employing the method of next generation matrix [44], a first stability analysis is presented by studying equilibria, and some numerical simulations are performed by using MATLAB routines for ODE.

4.1. SIR

Firstly, SIR model is considered. The overall population is divided into three functional subsystems according to the three compartments of this model: *susceptible*, *infectious* and *recovered*. Then, three distribution functions over the related functional subsystems are introduced:

- $f_1(t)$ gives the number of susceptible individuals at time $t > 0$;
- $f_2(t)$ gives the number of infectious individuals at time $t > 0$;
- $f_3(t)$ gives the number of recovered individuals at time $t > 0$.

Bearing the assumptions of Theorem 1 in mind, it is assumed that $\sum_{i=1}^{3} f_i(t) = 1$, for all $t \geq 0$, that is the distribution function $f_i(t)$, for $i \in \{1, 2, 3\}$, refers to the fraction of individuals of the ith functional subsystem.

According to the previous analytical considerations and some usual assumptions of compartmental models, the parameters of the system (14) have some specific properties:

- An individual does not change functional subsystem after an interaction with another individual of the same functional subsystem, that is
$$B_{ii}^k = \begin{cases} 1, & k = i \\ 0, & k \neq i. \end{cases}$$
- The transitions from the infectious group to the recovered one are modeled by an external force field (3). Specifically, the only non-zero coefficients Γ_i^2 are: Γ_2^2 and Γ_3^2. Due to the assumption **A3**, one has
$$\Gamma_2^2 + \Gamma_3^2 = 0,$$
and then
$$\Gamma_3^2 = -\Gamma_2^2.$$
Since $\Gamma_2^2 < 0$, then $\Gamma_3^2 > 0$. If a parameter $\Gamma \geq 0$ is introduced, then
$$\Gamma_3^2 = \Gamma$$
$$\Gamma_2^2 = -\Gamma.$$

Bearing all above in mind, the kinetic framework with external force field (14) for the SIR model writes

$$\begin{cases} \dfrac{df_1}{dt} = -\eta_{12} B_{12}^2 f_1(t) f_2(t) \\ \dfrac{df_2}{dt} = \eta_{12} B_{12}^2 f_1(t) f_2(t) - \Gamma f_2(t) \\ \dfrac{df_3}{dt} = \Gamma f_2(t). \end{cases} \tag{15}$$

In particular, B_{12}^2 is the transition probability that a susceptible individual gets infected after the interaction with an infectious one. This is the only transition probability B_{hk}^i that appears explicitly in the system. Roughly speaking, this quantity is a kinetic version of the transmission rate of epidemiological models.

It is worth stressing that, given a suitable initial data $\mathbf{f}^0 = \left(f_1^0, f_2^0, f_3^0\right)$, the Cauchy problem related to the model (15) admits a unique positive solution $\mathbf{f}(t)$ such that

$$\mathbb{E}_0[\mathbf{f}](t) = 1, \qquad t > 0.$$

Generally, in an SIR model, it is assumed that $f_3^0 = 0$, i.e. the number of recovered individuals is zero at initial time $t = 0$.

In order to compute the basic reproduction number $\mathcal{R}_0$ for this model, the next generation matrix method is applied. Let consider the equation for the infectious compartment, i.e. the second equation of the system (15)

$$\frac{df_2}{dt} = \eta_{12} B_{12}^2 f_1(t) f_2(t) - \Gamma f_2(t).$$

This equation rewrites

$$\frac{df_2}{dt} = f_2(t)\left(\eta_{12} B_{12}^2 f_1(t) - \Gamma\right),$$

and, at the initial time, one has

$$\frac{df_2}{dt} = f_2^0\left(\eta_{12} B_{12}^2 f_1^0 - \Gamma\right). \tag{16}$$

Then, the *Transmission matrix* T (matrix of second infections) and the *Transition matrix* Ξ (matrix of change of compartments) are obtained by separating the events leading to new infections from all other events of the disease spreading dynamics (for details, see [44] and references therein). Since $f_1^0 \simeq 1$, the matrix T and the matrix Ξ for this kinetic model read

$$T = \left[\eta_{12} B_{12}^2\right]$$
$$\Xi = [-\Gamma].$$

Accordingly, the following matrix is derived

$$K_L = -T \cdot \Xi^{-1} = \left[\frac{\eta_{12} B_{12}^2}{\Gamma}\right]. \tag{17}$$

The eigenvalues of the previous matrix K_L (17) allow to derive the basic reproduction number $\mathcal{R}_0$ of this SIR model. In particular, $\mathcal{R}_0$ is the spectral radius of matrix K_L, i.e.

$$\mathcal{R}_0 = \rho(K_L) := \max_i(|\lambda_i|).$$

Finally, the basic reproduction number $\mathcal{R}_0$ for the model (15) is

$$\mathcal{R}_0 = \frac{\eta_{12} B_{12}^2}{\Gamma}. \tag{18}$$

Since

$$\frac{df_2}{dt} = \Gamma f_2(t)\left(\mathcal{R}_0 f_1(t) - 1\right),$$

then the value of the basic reproduction number $\mathcal{R}_0$ is fundamental for the outbreak of a disease spreading event. In particular, if $\mathcal{R}_0 < 1$, then $f_2(t)$ is decreasing and the outbreak does not occur. Otherwise, for $\mathcal{R}_0 > 1$, the disease event starts. The case $\mathcal{R}_0 = 1$ is critical.

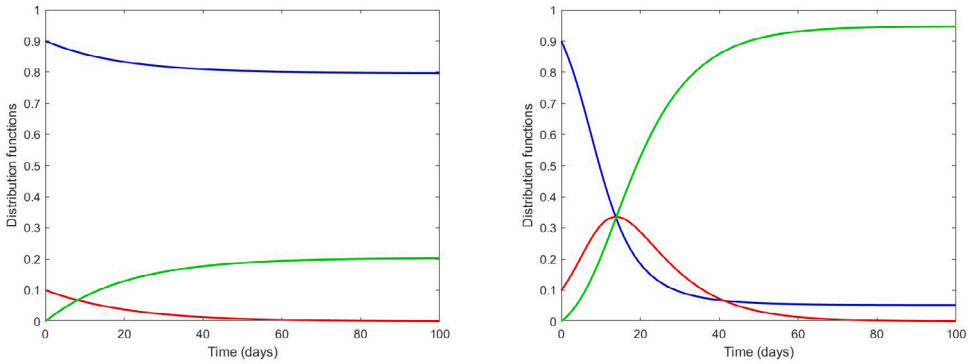

Fig. 1. Time evolution of the three distribution functions $f_1(t)$ (blue line), $f_2(t)$ (red line) and $f_3(t)$ (green line), with $\beta_{12}^2 = 0.3$, $\Gamma = 0.1$ and initial condition $\mathbf{f}^0 = (0.9, 0.1, 0)$ in two different scenario for SIR model: (a) for $\mathcal{R}_0 < 1 (\eta_{12} = 0.2)$, where the DFE is $(0.7966, 0, 0.2034)$; (b) for $\mathcal{R}_0 > 1 (\eta_{12} = 1)$, where the DFE is $(0.0524, 0.0005, 0.9471)$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Therefore, the kinetic model (15) is in accordance with the known results of classical epidemiological models.

4.1.1. Equilibrium points

Let consider the region

$$\Omega = \left\{(f_1, f_2, f_3) \in \mathbb{R}^3 : f_1, f_2, f_3 \geq 0, f_1 + f_2 + f_3 = 1\right\},$$

which is positively invariant. The equilibrium points of the system (15), in the region Ω, are obtained by equating to zero the right hand side of the system (15). From the second equation, one gains $f_2 = 0$. Then, the equilibrium points are

$$(p, 0, 1-p), \quad \text{with } p \in (0, 1).$$

Since the compartment of infectious individuals is empty, this point is called *disease free equilibrium* point, also known as *DFE*. In particular, $1-p$ is the fraction of recovered individuals, that is individuals infected during the disease spreading. Therefore, p is the final size of susceptible individuals (see Fig. 1).

Remark 2. The condition $f_2 = 0$ gives infinity equilibrium points, i.e. $(p, 0, 1-p)$.

4.2. SEIIR

In the previous SIR model, there is not an exposure period to the infection, i.e. an infected individual is immediately infectious. However, this may not be a realistic assumption in some situations. Therefore, a fourth compartment, with the related functional subsystem, is considered: the exposed individuals. An exposed individual is an individual who is infected but not still infectious. Then, the overall population is now divided into four compartments: susceptible, exposed, infectious and recovered. In order to derive a more realistic model, we further divide the group of infectious individuals, since an infectious individual may be asymptomatic or symptomatic. Finally, there are five functional subsystems, with the related distribution functions. Specifically:

- $f_1(t)$ gives the fraction of susceptible individuals at time $t > 0$;
- $f_2(t)$ gives the fraction of exposed individuals at time $t > 0$;
- $f_3(t)$ gives the fraction of asymptomatic infectious individuals at time $t > 0$;
- $f_4(t)$ gives the fraction of symptomatic infectious individuals at time $t > 0$;
- $f_5(t)$ gives the fraction of recovered individuals at time $t > 0$.

This is called SEIIR scheme. Following the previous arguments of the SIR scheme, this Subsection aims at modeling the disease spreading of this compartmental model by using the new kinetic framework with external force field (14).

Since the system is conservative, one has

$$\mathbb{E}_0[\mathbf{f}](t) = f_1(t) + f_2(t) + f_3(t) + f_4(t) + f_5(t) = 1, \qquad \forall t > 0.$$

Keeping all above in mind, the kinetic scheme related to the SEIIR model is gained by assigning specific values to the parameters of framework (14): interaction rates η_{hk}, for $h, k \in \{1, 2, 3, 4, 5\}$, the transition probabilities B_{hk}^i, for $i, h, k \in \{1, 2, 3, 4, 5\}$, and coefficients Γ_i^k, for $i, k \in \{1, 2, 3, 4, 5\}$. Therefore, the following assumptions are taken into consideration:

- An individual does not change functional subsystem after an interaction with another individual of the same functional subsystem, that is

$$B_{ii}^k = \begin{cases} 1, & k = i \\ 0, & k \neq i. \end{cases}$$

- Only infectious individuals can infect susceptible ones, whereas exposed ones cannot infect. Moreover, a susceptible individual passes, with some probability, into the exposed group after an interaction with an infectious individual, symptomatic or asymptomatic. Then the following transition probabilities model the disease spreading dynamics:
  - B_{13}^2, which is the transition probability that a susceptible individual passes into the exposed group after the interaction with an asymptomatic infectious individual;
  - B_{14}^2, which is the transition probability that a susceptible individual passes into the exposed group after the interaction with a symptomatic infectious individual.

  Moreover, the related interaction rates η_{13} and η_{14} are assigned.
- Exposed individuals pass into the asymptomatic or symptomatic infectious group, and infectious individuals into the recovered one. These dynamics are modeled by an external force field (3). For the model of the current Subsection, the only non-zero coefficients Γ_i^k are:
  1. $\Gamma_2^2, \Gamma_3^2, \Gamma_4^2$, which are related to the exposed individuals that become infectious (asymptomatic or symptomatic);
  2. Γ_3^3, Γ_3^5, which are related to the asymptomatic infectious individuals that recover;
  3. Γ_4^4, Γ_5^4, which are related to the symptomatic infectious individuals that recover.

Since the assumption **A3** holds true, one has

$$\begin{aligned} \Gamma_2^2 + \Gamma_3^2 + \Gamma_4^2 &= 0, \\ \Gamma_3^3 + \Gamma_5^3 &= 0, \\ \Gamma_4^4 + \Gamma_5^4 &= 0. \end{aligned}$$

and then

$$\begin{aligned} \Gamma_3^2 + \Gamma_4^2 &= -\Gamma_2^2, \\ \Gamma_5^3 &= -\Gamma_3^3, \\ \Gamma_5^4 &= -\Gamma_4^4. \end{aligned}$$

Since $\Gamma_2^2, \Gamma_3^3, \Gamma_4^4 < 0$, then $\Gamma_3^2, \Gamma_4^2, \Gamma_5^3, \Gamma_5^4 > 0$, according to the previous analytical considerations. Let $\Gamma, \Gamma_A, \Gamma_S, \gamma_A, \gamma_S \geq 0$. Then

$$\begin{aligned} &\Gamma_3^2 = \Gamma_A, \quad \Gamma_4^2 = \Gamma_S \quad \Gamma_2^2 = -(\Gamma_A + \Gamma_S) = -\Gamma, \\ &\Gamma_5^3 = \gamma_A, \quad \Gamma_3^3 = -\gamma_A, \\ &\Gamma_5^4 = \gamma_S, \quad \Gamma_4^4 = -\gamma_S. \end{aligned} \tag{19}$$

Then, the kinetic framework with external force field (14) for the SEIIR model writes

$$\begin{cases} \dfrac{df_1}{dt} = -\eta_{13} B_{13}^2 f_1(t) f_3(t) - \eta_{14} B_{14}^2 f_1(t) f_4(t) \\ \dfrac{df_2}{dt} = \eta_{13} B_{13}^2 f_1(t) f_3(t) + \eta_{14} B_{14}^2 f_1(t) f_4(t) - \Gamma f_2(t) \\ \dfrac{df_3}{dt} = \Gamma_A f_2(t) - \gamma_A f_3(t) \\ \dfrac{df_4}{dt} = \Gamma_S f_2(t) - \gamma_S f_4(t) \\ \dfrac{df_5}{dt} = \gamma_A f_3(t) + \gamma_S f_4(t). \end{cases} \tag{20}$$

The assumptions of Theorem 1 are satisfied. If the initial data $\mathbf{f}^0 = (f_1^0, f_2^0, f_3^0, f_4^0, f_5^0)$ is such that $\sum_{i=1}^n f_i^0 = 1$, then there exists a unique, positive and bounded solution $\mathbf{f}(t)$ for the system (20), such that

$$\sum_{i=1}^{5} f_i(t) = 1, \qquad t > 0.$$

The next generation matrix method allows to compute the basic reproduction number $\mathcal{R}_0$ for the SEIIR framework (20). Firstly, the following system is considered

$$\begin{aligned} \frac{df_2}{dt} &= \eta_{13} B_{13}^2 f_1(t) f_3(t) + \eta_{14} B_{14}^2 f_1(t) f_4(t) - \Gamma f_2(t) \\ \frac{df_3}{dt} &= \Gamma_A f_2(t) - \gamma_A f_3(t) \\ \frac{df_4}{dt} &= \Gamma_S f_2(t) - \gamma_S f_4(t). \end{aligned} \tag{21}$$

The system (21) is obtained by considering only infected individuals (i.e. exposed, asymptomatic, symptomatic). Indeed, the production of new infections and changes of compartments for infected individuals are considered for applying the next generation matrix method. Following the same considerations of SIR model, the Transmission matrix T (i.e. that one of second infections) and the Transition matrix Ξ (i.e. that one of changes of compartments) are obtained by separating the events leading to new infections from all other events of the disease spreading dynamics. Specifically, each element T_{ij} is the rate at which an infected individual, $j \in \{2,3,4\}$, leads to new infections in the infected state $i \in \{2,3,4\}$. Therefore, the matrix T and the matrix Ξ write

$$\begin{aligned} T &= \begin{bmatrix} 0 & \eta_{13} B_{13}^2 f_1^0 & \eta_{14} B_{14}^2 f_1^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \\ \Xi &= \begin{bmatrix} -\Gamma & 0 & 0 \\ \Gamma_A & -\gamma_A & 0 \\ \Gamma_S & 0 & -\gamma_S \end{bmatrix}. \end{aligned} \tag{22}$$

Moreover, the inverse matrix of Ξ reads

$$\Xi^{-1} = \begin{bmatrix} \dfrac{-1}{(\Gamma_A + \Gamma_S)} & 0 & 0 \\ \dfrac{-\Gamma_A}{\gamma_A(\Gamma_A + \Gamma_S)} & \dfrac{-1}{\gamma_A} & 0 \\ \dfrac{-\Gamma_S}{\gamma_S(\Gamma_A + \Gamma_S)} & 0 & \dfrac{-1}{\gamma_S} \end{bmatrix}. \tag{23}$$

Keeping the shape of matrices T (22) and Ξ^{-1} (23) in mind, the final matrix K_L is

$$K_L = -T \cdot \Xi^{-1} = \begin{bmatrix} \dfrac{\eta_{13} B_{13}^2 \Gamma_A}{\gamma_A(\Gamma_A + \Gamma_S)} + \dfrac{\eta_{14} B_{14}^2 \Gamma_S}{\gamma_S(\Gamma_A + \Gamma_S)} & \dfrac{\eta_{13} B_{13}^2}{\gamma_A} & \dfrac{\eta_{14} B_{14}^2}{\gamma_S} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}. \tag{24}$$

In order to computer the eigenvalues of the previous matrix K_L (24), one has

$$\begin{aligned} det(K_L - \lambda I) &= \begin{vmatrix} \dfrac{\eta_{13} B_{13}^2 \Gamma_A}{\gamma_A(\Gamma_A + \Gamma_S)} + \dfrac{\eta_{14} B_{14}^2 \Gamma_S}{\gamma_S(\Gamma_A + \Gamma_S)} - \lambda & \dfrac{\eta_{13} B_{13}^2}{\gamma_A} & \dfrac{\eta_{14} B_{14}^2}{\gamma_S} \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix} \\ &= \lambda^2 \left(\frac{\eta_{13} B_{13}^2 \Gamma_A}{\gamma_A(\Gamma_A + \Gamma_S)} + \frac{\eta_{14} B_{14}^2 \Gamma_S}{\gamma_S(\Gamma_A + \Gamma_S)} - \lambda \right). \end{aligned} \tag{25}$$

Straightforward computations on (25) show

$$\lambda_{1,2} = 0, \qquad \lambda_3 = \frac{\eta_{13} B_{13}^2 \Gamma_A}{\gamma_A(\Gamma_A + \Gamma_S)} + \frac{\eta_{14} B_{14}^2 \Gamma_S}{\gamma_S(\Gamma_A + \Gamma_S)}.$$

Then, the basic reproduction number $\mathcal{R}_0$ of the SEIIR scheme (20), modeled by using the new kinetic framework with external force field (14), is gained by computing spectral radius of the matrix K_L, that is

$$\mathcal{R}_0 = \rho(K_L) := \max_i(|\lambda_i|).$$

Finally, some algebraic computation show that

$$\mathcal{R}_0 = \frac{\eta_{13} B_{13}^2 \Gamma_A}{\gamma_A(\Gamma_A + \Gamma_S)} + \frac{\eta_{14} B_{14}^2 \Gamma_S}{\gamma_S(\Gamma_A + \Gamma_S)}. \tag{26}$$

The (26) gives the basic reproduction number $\mathcal{R}_0$ for the SEIIR framework (20). In particular, it is the sum of two term, both related to interactions among susceptible individuals and infectious ones (asymptomatic or symptomatic). The former is related to the contribution of asymptomatic individuals, i.e. agents of the 3rd functional subsystem. The latter is the contribution of symptomatic individuals, i.e. agents of the 4th functional subsystem.

4.2.1. *Equilibrium points*

Let consider the region

$$\Omega = \left\{ (f_1, f_2, f_3, f_4, f_5) \in \mathbb{R}^5 : \ f_1, \ f_2, \ f_3, \ f_4, \ f_5 \geq 0, \ f_1 + f_2 + f_3 + f_4 + f_5 = 1 \right\},$$

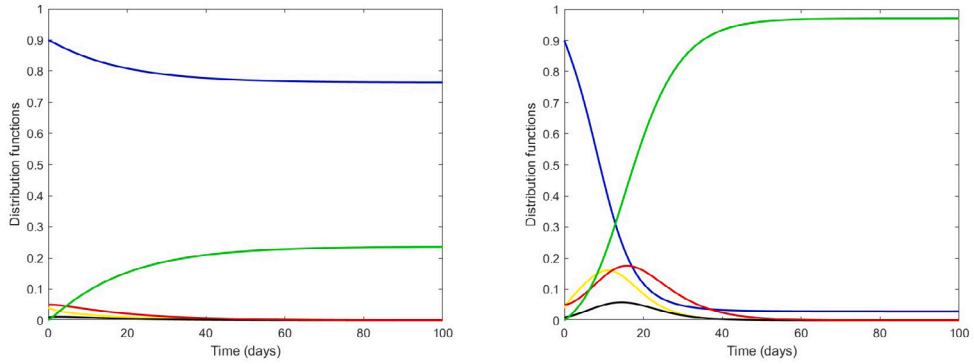

Fig. 2. Time evolution of the five distribution functions $f_1(t)$ (blue line), $f_2(t)$ (yellow line), $f_3(t)$ (red line), $f_4(t)$ (black line) and $f_5(t)$ (green line), with $\beta^2_{13} = 0.3$, $\beta^2_{14} = 0.7$, $\Gamma_A = 0.1$, $\Gamma_S = 0.2$, $\gamma_A = 0.25$, $\gamma_S = 0.15$ and initial condition $\mathbf{f}^0 = (0.9, 0.04, 0.01, 0.05, 0)$ in two different scenario for SEIIR model: (a) For $\mathcal{R}_0 < 1(\eta_{13} = 0.1, \eta_{14} = 0.2)$, where the DFE is $(0.7631, 0, 0, 0, 0.2370)$; (b) For $\mathcal{R}_0 > 1(\eta_{13} = 1, \eta_{14} = 1)$, where the DFE is $(0.0287, 0, 0, 0, 0.9713)$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

that is positively invariant. Therefore, by standard procedure (see [45] and references therein), one derive that, starting from and initial data $\mathbf{f}^0 \in \Omega$, the solution $\mathbf{f}(t)$ still remains in Ω, for all $t > 0$, by regarding the analytical shape of equations of the system (20).

The equilibrium points of the system (20) are obtained by equating to zero the right hand side of the system (20). The *DFE* (disease free equilibrium) is gained, and reads

$$(p, 0, 0, 0, 1-p) \quad \text{with } p \in (0, 1),$$

where $1-p$ is the fraction of recovered individuals, that is individual infected during the disease spreading, and p is the final size of susceptible individuals (see Fig. 2).

Remark 3. The condition $f_2 = 0, f_3 = 0, f_4 = 0$ gives infinity equilibrium points, i.e. points of the form $(p, 0, 0, 0, 1-p)$.

Remark 4. As well as for the SIR scheme, the results gained for the SEIIR framework (20), modeled by the new kinetic framework (14), are in agreement with the ones of the classical epidemiological models.

4.3. SEIIRS

Let now consider an SEIIR model where reinfections occur. We call this model SEIIRS. The related kinetic scheme is gained by considering the model of the previous Subsection with the following further assumption:

- Reinfection process is also modeled by the external force field (3). Specifically, the coefficients Γ_1^5 and Γ_5^5 describe the recovered individuals that loose their immunity and return into the susceptible group. Due to the assumption **A3**, one has

  $$\Gamma_1^5 + \Gamma_5^5 = 0,$$

  and then

  $$\Gamma_1^5 = -\Gamma_5^5.$$

  Therefore, if $\nu > 0$, let

  $$\Gamma_1^5 = \nu$$
  $$\Gamma_5^5 = -\nu.$$

Then, the kinetic framework with external force field (14) for the SEIIRS model writes

$$\begin{cases} \dfrac{df_1}{dt} = -\eta_{13} B^2_{13} f_1(t) f_3(t) - \eta_{14} B^2_{14} f_1(t) f_4(t) + \nu f_5(t) \\ \dfrac{df_2}{dt} = \eta_{13} B^2_{13} f_1(t) f_3(t) + \eta_{14} B^2_{14} f_1(t) f_4(t) - \Gamma f_2(t) \\ \dfrac{df_3}{dt} = \Gamma_A f_2(t) - \gamma_A f_3(t) \\ \dfrac{df_4}{dt} = \Gamma_S f_2(t) - \gamma_S f_4(t) \\ \dfrac{df_5}{dt} = \gamma_A f_3(t) + \gamma_S f_4(t) - \nu f_5(t). \end{cases} \tag{27}$$

The assumptions of Theorem 1 are satisfied. Let $\mathbf{f}^0 = (f_1^0, f_2^0, f_3^0, f_4^0, f_5^0)$ be a suitable initial data, such that $\sum_{i=1}^5 f_i^0 = 1$, then there exists a unique, positive and bounded solution $\mathbf{f}(t)$ for the system (20), such that

$$\sum_{i=1}^{5} f_i(t) = 1, \qquad t > 0.$$

The next generation matrix method shows that the basic reproduction number $\mathcal{R}_0$ for the current SEIIRS framework (27) is equal to the $\mathcal{R}_0$ (26) of SEIIR model. Indeed,

$$\mathcal{R}_0 = \frac{\eta_{13} B^2_{13} \Gamma_A}{\gamma_A(\Gamma_A + \Gamma_S)} + \frac{\eta_{14} B^2_{14} \Gamma_S}{\gamma_S(\Gamma_A + \Gamma_S)} = \frac{\eta_{13} B^2_{13} \gamma_S \Gamma_A + \eta_{14} B^2_{14} \gamma_A \Gamma_S}{\gamma_A \gamma_S (\Gamma_A + \Gamma_S)}, \tag{28}$$

4.3.1. Equilibrium points

Let consider the region

$$\Omega = \left\{(f_1, f_2, f_3, f_4, f_5) \in \mathbb{R}^5 : f_1, f_2, f_3, f_4, f_5 \geq 0, f_1 + f_2 + f_3 + f_4 + f_5 = 1\right\}.$$

that is positively invariant, for the same reasons of the previous case.

The equilibrium points of the system (27) are obtained by equating to zero the right hand side of the system (27). Specifically, the *DFE* (disease free equilibrium) reads

$$(1, 0, 0, 0, 0).$$

Moreover, the Jacobian matrix of the system (27) writes

$$J = \begin{bmatrix} -\eta_{13}B_{13}^2 f_3(t) - \eta_{14}B_{14}^2 f_4(t) & 0 & -\eta_{13}B_{13}^2 f_1(t) & -\eta_{14}B_{14}^2 f_1(t) & \nu \\ \eta_{13}B_{13}^2 f_3(t) + \eta_{14}B_{14}^2 f_4(t) & -(\Gamma_A + \Gamma_S) & \eta_{13}B_{13}^2 f_1(t) & \eta_{14}B_{14}^2 f_1(t) & \nu \\ 0 & \Gamma_A & -\gamma_A & 0 & 0 \\ 0 & \Gamma_S & 0 & -\gamma_S & 0 \\ 0 & 0 & \gamma_A & \gamma_S & -\nu \end{bmatrix}, \tag{29}$$

where $J = J(f_1, f_2, f_3, f_4, f_5)$. Towards the stability of the DFE, the following theorem holds true.

Theorem 2. *Let consider the system (27). If $\mathcal{R}_0 < 1$, the DFE is globally asymptotically stable. If $\mathcal{R}_0 > 1$, the DFE is unstable.*

Proof. Let $\mathcal{R}_0 < 1$. We apply the method developed in [46]. Let

$$\mathbf{x} = \left(f_1(t),\, f_5(t)\right)$$
$$\mathbf{I} = \left(f_2(t),\, f_3(t),\, f_4(t)\right).$$

The system (27) is divided into two parts, i.e.

$$\frac{d\mathbf{x}}{dt} = F(\mathbf{x}, \mathbf{I}), \tag{30}$$
$$\frac{d\mathbf{I}}{dt} = G(\mathbf{x}, \mathbf{I}), \qquad G(\mathbf{x}, \mathbf{0}) = \mathbf{0}. \tag{31}$$

Specifically,

$$F_1(\mathbf{x}, \mathbf{I}) = -\eta_{13}B_{13}^2 f_1(t) f_3(t) - \eta_{14}B_{14}^2 f_1(t) f_4(t) + \nu f_5(t)$$
$$F_2(\mathbf{x}, \mathbf{I}) = \gamma_A f_3(t) + \gamma_S f_4(t) - \nu f_5(t),$$

and

$$G_1(\mathbf{x}, \mathbf{I}) = \eta_{13}B_{13}^2 f_1(t) f_3(t) + \eta_{14}B_{14}^2 f_1(t) f_4(t) - \Gamma f_2(t)$$
$$G_2(\mathbf{x}, \mathbf{I}) = \Gamma_A f_2(t) - \gamma_A f_3(t)$$
$$G_3(\mathbf{x}, \mathbf{I}) = \Gamma_S f_2(t) - \gamma_S f_4(t).$$

Let $\mathbf{U}_0 = (\mathbf{x}^*, \mathbf{0})$ be the DFE of the system, where $\mathbf{x}^* = (1, 0)$. In order to apply the method of [46], the following two assumptions need to be satisfied:

H1 For $\dfrac{d\mathbf{x}}{dt} = F(\mathbf{x}, \mathbf{0})$, $\mathbf{x}^*$ is globally asymptotically stable;

H2 $G(\mathbf{x}, \mathbf{I}) = A\mathbf{I} - \hat{G}(\mathbf{x}, \mathbf{I})$, where $\hat{G}(\mathbf{x}, \mathbf{I}) \geq 0$ for $(\mathbf{x}, \mathbf{I}) \in \Omega$, and $A = D_I G(\mathbf{x}^*, \mathbf{0})$ is an M-matrix.

Now, since $\mathbf{F}(\mathbf{x}, \mathbf{0}) = \left(-\eta_{13}B_{13}^2 f_1(t) f_3(t) - \eta_{14}B_{14}^2 f_1(t) f_4(t) + \nu f_5(t),\ \gamma_A f_3(t) + \gamma_S f_4(t) - \nu f_5(t)\right)$, the system $\dfrac{d\mathbf{x}}{dt} = F(\mathbf{x}, \mathbf{0})$ rewrites

$$\begin{cases} \dfrac{df_1}{dt} = \nu f_5(t) \\ \dfrac{df_5}{dt} = -\nu f_5(t). \end{cases}$$

Then, the point $\mathbf{x}^* = (1, 0)$ is globally asymptotically stable for this system, and the assumption **H1** is satisfied.

Bearing the Jacobian matrix (29) in mind, one has

$$A = D_I G(\mathbf{x}^*, \mathbf{0}) = \begin{bmatrix} -\Gamma & \eta_{13}B_{13}^2 & \eta_{14}B_{14}^2 \\ \Gamma_A & -\gamma_A & 0 \\ \Gamma_S & 0 & -\gamma_S \end{bmatrix}. \tag{32}$$

Then,

$$A\mathbf{I} = \begin{bmatrix} -\Gamma f_2(t) + \eta_{13}B_{13}^2 f_3(t) + \eta_{14}B_{14}^2 f_2(t) \\ \Gamma_A f_2(t) - \gamma_A f_3(t) \\ \Gamma_S f_2(t) - \gamma_s f_4(t) \end{bmatrix}.$$

Since $G(\mathbf{x}, \mathbf{I}) = A\mathbf{I} - \hat{G}(\mathbf{x}, \mathbf{I})$, then $\hat{G}(\mathbf{x}, \mathbf{I}) = A\mathbf{I} - G(\mathbf{x}, \mathbf{I})$. Therefore,

$$\hat{G}(\mathbf{x}, \mathbf{I}) = \begin{bmatrix} \left(1 - f_1(t)\right)\left(\eta_{13}B_{13}^2 f_3(t) + \eta_{14}B_{14}^2 f_4(t)\right) \\ 0 \\ 0 \end{bmatrix}.$$

Trivially, $\hat{G}(\mathbf{x}, \mathbf{I}) \geq \mathbf{0}$, for $(\mathbf{x}, \mathbf{I}) \in \Omega$, as $f_1(t) \leq 1$. Then

$$G(\mathbf{x}, \mathbf{I}) = A\mathbf{I} - \hat{G}(\mathbf{x}, \mathbf{I}).$$

The matrix A is trivially an M-matrix, that is the off-diagonal elements are non-negative. The condition **H2** is satisfied too. The global asymptotic stability for $\mathcal{R}_0 < 1$ is thus gained.

Let now $\mathcal{R}_0 > 1$. The Jacobian matrix (29) at DFE reads

$$J(1, 0, 0, 0, 0) = \begin{bmatrix} 0 & 0 & -\eta_{13}B_{13}^2 & -\eta_{14}B_{14}^2 & \nu \\ 0 & -(\Gamma_A + \Gamma_S) & \eta_{13}B_{13}^2 & \eta_{14}B_{14}^2 & \nu \\ 0 & \Gamma_A & -\gamma_A & 0 & 0 \\ 0 & \Gamma_S & 0 & -\gamma_S & 0 \\ 0 & 0 & \gamma_A & \gamma_S & -\nu \end{bmatrix}. \tag{33}$$

If we prove that, at least, one of the eigenvalues of the matrix (29) has positive real part, then the proof is concluded. In order to get this aim, the characteristic polynomial of the matrix, that is (29) is considered

$$p(\lambda) = -\lambda(-\lambda - \nu)\, g(\lambda), \tag{34}$$

where

$$g(\lambda) = a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0. \tag{35}$$

Specifically, the coefficients of $g(\lambda)$, after straightforward computations, write

$$\begin{aligned} a_3 &= -1, \\ a_2 &= -(\gamma_A + \gamma_S + \Gamma_A + \Gamma_S), \\ a_1 &= \eta_{13}B_{13}^2\Gamma_A + \eta_{14}B_{14}^2\Gamma_S - \gamma_A(\Gamma_A + \Gamma_S) - \gamma_S(\Gamma_A + \Gamma_S) - \gamma_A\gamma_S, \\ a_0 &= \eta_{13}B_{13}^2\gamma_S\Gamma_A + \eta_{14}B_{14}^2\gamma_A\Gamma_S - \gamma_A\gamma_S(\Gamma_A + \Gamma_S) \\ &= (\mathcal{R}_0 - 1)\gamma_A\gamma_S(\Gamma_A + \Gamma_S). \end{aligned} \tag{36}$$

Now, the Routh–Hurwitz criterion [47,48] is applied. Indeed, some computations prove that there is a change of sign in the coefficients of the first column of Routh's matrix. Trivially one has that $a_3, a_2 < 0$. Since $\mathcal{R}_0 > 1$, then $a_0 > 0$. Now, one has

$$b_2 = -\frac{\begin{vmatrix} a_3 & a_1 \\ a_2 & a_0 \end{vmatrix}}{a_2} = \frac{a_0 + a_1 a_2}{a_2}$$
$$b_1 = -\frac{\begin{vmatrix} a_3 & 0 \\ a_2 & 0 \end{vmatrix}}{a_2} = 0$$
$$c_1 = -\frac{\begin{vmatrix} a_2 & a_0 \\ b_2 & 0 \end{vmatrix}}{b_2} = a_0.$$

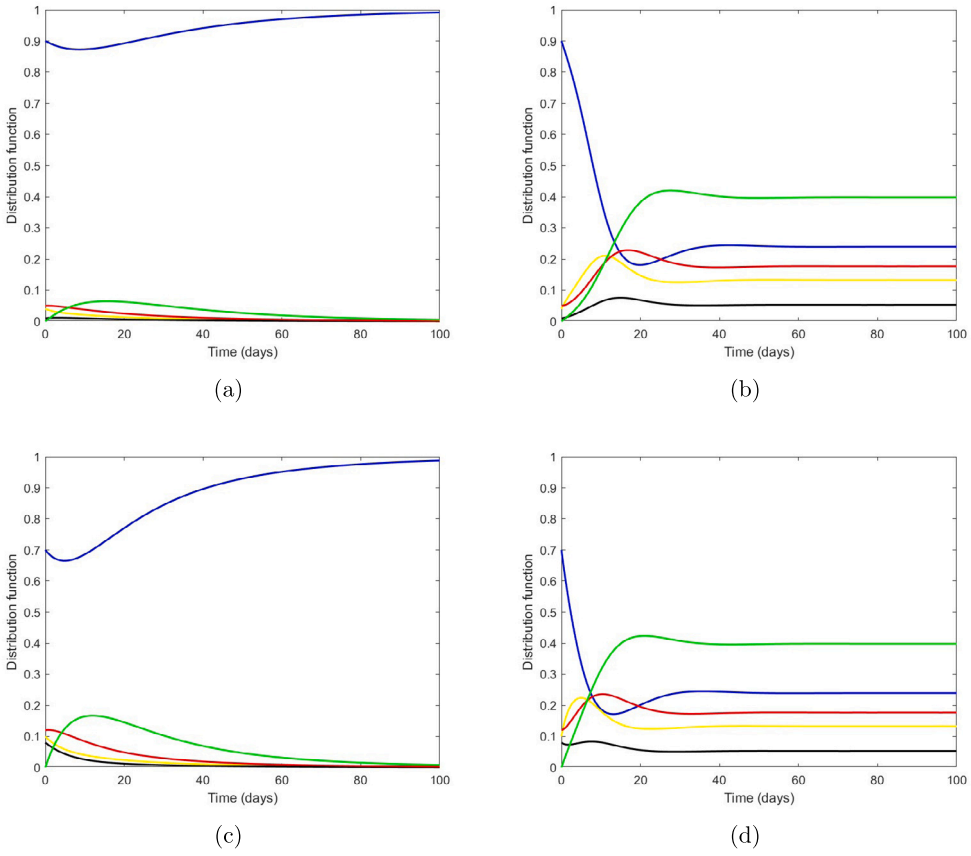

Fig. 3. Time evolution of the five distribution functions $f_1(t)$ (blue line), $f_2(t)$ (yellow line), $f_3(t)$ (red line), $f_4(t)$ (black line) and $f_5(t)$ (green line) for SEIIRS model with parameters $\beta_{13}^2 = 0.8$, $\beta_{14}^2 = 0.7$, $\Gamma_A = 0.1$, $\Gamma_S = 0.2$, $\gamma_A = 0.25$, $\gamma_S = 0.15$, $\nu = 0.1$. Two scenarios are considered for the initial condition: (a)–(b) $\mathbf{f}^0 = (0.9, 0.04, 0.01, 0.05, 0)$; (c)–(d) $\mathbf{f}^0 = (0.7, 0.1, 0.08, 0.12, 0)$. Specifically, the left panels refer to the case $\mathcal{R}_0 < 1 (\eta_{13} = 0.1, \eta_{14} = 0.2)$, whereas the right panels to the case $\mathcal{R}_0 > 1 (\eta_{13} = 1, \eta_{14} = 1)$. The endemic equilibrium point E^* is: $(f_1^*, f_2^*, f_3^*, f_4^*, f_5^*) = (0.2394, 0.1327, 0.0531, 0.1769, 0.3980)$ for case (b), and $(f_1^*, f_2^*, f_3^*, f_4^*, f_5^*) = (0.2394, 0.1326, 0.0531, 0.1769, 0.3980)$ for case (d). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

The first column of the Routh matrix is the vector

$$\mathbf{v} = \begin{bmatrix} a_3 \\ a_2 \\ b_2 \\ c_1 \end{bmatrix},$$

with

$$a_3 < 0$$
$$a_2 < 0$$
$$b_2 = \frac{a_0 + a_1 a_2}{a_2}$$
$$c_1 > 0.$$

Since a_2 and c_1 have opposite sign, this ensures that a change of sign occurs, regardless of value of the coefficient b_2. Then, there exists at least one eigenvalue with positive real part. This ensures that the DFE is unstable for $\mathcal{R}_0 > 1$. □

Since reinfections occur in this framework, then *endemic equilibrium* points may appear. Therefore, the following result holds true (see Fig. 3).

Theorem 3. *Let consider the framework (27). If $\mathcal{R}_0 > 1$, the system has an endemic equilibrium point. Otherwise, the DFE is the unique equilibrium point.*

Proof. The equilibrium points of system are obtained by equating to zero the right hand side of the system (27). Beyond the DFE, the further equilibrium point E^* is obtained, that is

$$E^* = (f_1^*, f_2^*, f_3^*, f_4^*, f_5^*), \tag{37}$$

where

$$f_1^* = \frac{\gamma_A \gamma_S \left(\Gamma_A + \Gamma_S\right)}{\eta_{13}\beta_{13}^2 \Gamma_A \gamma_S + \eta_{14}\beta_{14}^2 \gamma_A \Gamma_S} = \frac{1}{\mathcal{R}_0}$$
$$f_2^* = \frac{\gamma_A}{\Gamma_A} f_3^*$$
$$f_4^* = \frac{\gamma_A \Gamma_S}{\gamma_S \Gamma_A} f_3^*$$
$$f_5^* = \frac{\gamma_A(\Gamma_A + \Gamma_S)}{\nu \Gamma_A} f_3^*.$$

Since $f_1^* + f_2^* + f_3^* + f_4^* + f_5^* = 1$, then

$$f_3^* = \frac{\nu \gamma_S \Gamma_A}{\nu \gamma_S \Gamma_A + \gamma_A \left[\nu \Gamma_S + \gamma_S(\nu + \Gamma_A + \Gamma_S)\right]} \left(1 - \frac{1}{\mathcal{R}_0}\right).$$

Finally, some straightforward computations show

$$E^* = \left(\frac{1}{\mathcal{R}_0}, \frac{\nu \gamma_A \gamma_S}{W}\left(1 - \frac{1}{\mathcal{R}_0}\right), \frac{\nu \gamma_S \Gamma_A}{W}\left(1 - \frac{1}{\mathcal{R}_0}\right), \right.$$
$$\left. \frac{\nu \gamma_A \Gamma_S}{W}\left(1 - \frac{1}{\mathcal{R}_0}\right), \frac{\gamma_A \Gamma_S (\Gamma_A + \Gamma_S)}{W}\left(1 - \frac{1}{\mathcal{R}_0}\right)\right),$$

where

$$W = \nu \gamma_S \Gamma_A + \gamma_A \left[\nu \Gamma_S + \gamma_S(\nu + \Gamma_A + \Gamma_S)\right].$$

Nevertheless, if $\mathcal{R}_0 < 1$, then $1 - \frac{1}{\mathcal{R}_0} < 0$, and the point E^* has not an epidemiological meaning, that is no endemic equilibrium point occurs.

Moreover, for $\mathcal{R}_0 = 1$, the point E^* coincides with the DFE. Finally, for $\mathcal{R}_0 > 1$, the equilibrium point E^* has sense and it does not coincide with the DFE. This concludes the proof. □

5. Conclusion and perspectives

A realistic description of a stochastically interacting systems needs to consider the effect of external actions. Indeed, in several context the evolution is not only the consequence of binary internal stochastic interactions, but also of an external action on the overall system, or related to each functional subsystems.

The main novelty of the current paper is the derivation of the framework (14), that models the evolution of a kinetic system under the action of an external force field $\mathbf{F}[\mathbf{f}](t)$, with a specific analytical shape, that is $F_i[\mathbf{f}](t) = \sum_{k=1}^{n} \Gamma_i^k(t) f_k(t)$, for $i \in \{1, 2, \dots, n\}$. Specifically, each component of the external force field depends on two quantities: the densities of functional subsystems and on the coefficients $\Gamma_i^k(t)$. Roughly speaking, the former ensures that the action of the external force fields depends on the current state of each functional subsystem, whereas, the latter is the part of action regardless of the current state. Moreover, in the conservative case, some analytical results are gained, such that existence, uniqueness, positivity and boundedness of solution are ensured globally in time.

This new model is applied in the contest of mathematical epidemiology, by improving three typical compartmental schemes: SIR, SEIIR and SEIIRS. Here, among the others, the external force field models recovery process and reinfections. Firstly, some analytical results have been gained towards equilibria and stability, and these are in agreement with the classical ones of dynamical systems, present in literature. Analogously, some numerical simulations are provided in order to show the long-time behavior of solutions, still in agreement with classical theory. Nevertheless, this application highlights some interesting things. For instance, the expression of the basic reproduction number $\mathcal{R}_0$ (i.e. (18), (26) and (28)) depends, among the others, on the parameters that model binary and stochastic interactions. Therefore, this may be seen as a statistical description of $\mathcal{R}_0$, and it may be useful from an application viewpoint. Moreover, the derivation of stability results is of great interest in the contest of kinetic theory.

Bearing all above in mind, the analytical and numerical results of this paper, as well as in the mathematical epidemiology contest, confirm the generality and versatility of the new framework (4). Therefore, this paper may be seen as a general scheme in order to derive nonconservative kinetic frameworks, under the action of external force fields.

However, the current work represents a first step for the development of kinetic models under the action of an external force field. Firstly, the analysis towards time-dependent coefficients $\Gamma_i^k(t)$ is a future research perspective. From an application viewpoint, this is important since several models require parameters that are not constant in time. In particular, this may lead to nonautonomous nonconservative models, which are more realistic for some applications. Moreover, the epidemiological models, in particular SEIIRS (27), highlight the impact of this framework for stability studies. Indeed, according to the classical theory, a kinetic analysis towards the value $\mathcal{R}_0 = 1$ is required, since this value could represent a bifurcation case. Finally, the analysis of kinetic models with general shape of external force field $\mathbf{F}[\mathbf{f}](t)$ is another research perspective in order to have a wider field of applicability. Specifically, it is of prominent interest the research of analytical assumptions on the external force field and interaction parameters that ensure the global existence of a unique, positive and bounded solution of the related Cauchy problem.

CRediT authorship contribution statement

Marco Menale: Conceptualization, Methodology, Formal analysis, Writing – original draft, Writing – review & editing, Supervision. **Carmelo Filippo Munafò:** Conceptualization, Methodology, Formal analysis, Software, Validation, Investigation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article

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References

[1] Bar-Yam Y, McKay SR, Christian W. Dynamics of complex systems (Studies in nonlinearity). Comput Phys 1998;12(4):335–6.
[2] Arlotti L, Bellomo N, Lachowicz M. Kinetic equations modelling population dynamics. Transport Theory Statist Phys 2000;29(1–2):125–39.
[3] Bellomo N, Carbonaro B. On the complexity of multiple interactions with additional reasonings about Kate, Jules and Jim. Math Comput Modelling 2008;47(1–2):168–77.
[4] Bellomo N, Carbonaro B, Gramani L. On the kinetic and stochastic games theory for active particles: Some reasonings on open large living systems. Math Comput Model 2008;48(7–8):1047–54.
[5] Bertotti ML, Delitala M. From discrete kinetic and stochastic game theory to modelling complex systems in applied sciences. Math Models Methods Appl Sci 2004;14(07):1061–84.
[6] Tosin A. From generalized kinetic theory to discrete velocity modeling of vehicular traffic. A stochastic game approach. Appl Math Lett 2009;22(7):1122–5.
[7] Bianca C. Modeling complex systems by functional subsystems representation and thermostatted-KTAP methods. Appl Math Inf Sci 2012;6(3):495–9.
[8] Bellouquid A, Delitala M. Mathematical methods and tools of kinetic theory towards modelling complex biological systems. Math Models Methods Appl Sci 2005;15(11):1639–66.
[9] Conte M, Loy N. Multi-cue kinetic model with non-local sensing for cell migration on a fiber network with chemotaxis. Bull Math Biol 2022;84(3):1–46.
[10] De Lillo S, Delitala M, Salvatori M. Modelling epidemics and virus mutations by methods of the mathematical kinetic theory for active particles. Math Models Methods Appl Sci 2009;19(supp01):1405–25.
[11] Harrison LG. Kinetic theory of living pattern. Endeavour 1994;18(4):130–6.
[12] Loy N, Preziosi L. Kinetic models with non-local sensing determining cell polarization and speed according to independent cues. J Math Biol 2020;80(1):373–421.
[13] Bernardi E, Pareschi L, Toscani G, Zanella M. Effects of vaccination efficacy on wealth distribution in kinetic epidemic models. Entropy 2022;24(2):216.
[14] Letizia Bertotti M, Modanese G. Economic inequality and mobility in kinetic models for social sciences. Eur Phys J Spec Top 2016;225(10):1945–58.
[15] Bertotti ML, Modanese G. Microscopic models for welfare measures addressing a reduction of economic inequality. Complexity 2016;21(6):89–98.
[16] Bertotti ML, Delitala M. On a discrete generalized kinetic approach for modelling persuader’s influence in opinion formation processes. Math Comput Model 2008;48(7–8):1107–21.
[17] Crokidakis N, Blanco VH, Anteneodo C. Impact of contrarians and intransigents in a kinetic model of opinion dynamics. Phys Rev E 2014;89(1):013310.
[18] Sen P. Phase transitions in a two-parameter model of opinion dynamics with random kinetic exchanges. Phys Rev E 2011;83(1):016108.
[19] Bellouquid A, De Angelis E, Fermo L. Towards the modeling of vehicular traffic as a complex system: A kinetic theory approach. Math Models Methods Appl Sci 2012;22(supp01):1140003.
[20] Tosin A, Zanella M. Kinetic-controlled hydrodynamics for traffic models with driver-assist vehicles. Multiscale Model Simul 2019;17(2):716–49.
[21] Carbonaro B, Serra N. Towards mathematical models in psychology: A stochastic description of human feelings. Math Models Methods Appl Sci 2002;12(10):1453–90.

[22] Carbonaro B, Giordano C. A second step towards a stochastic mathematical description of human feelings. Math Comput Modelling 2005;41(4–5):587–614.
[23] Arlotti L, Bellomo N, Latrach K. From the jager and segel model to kinetic population dynamics nonlinear evolution problems and applications. Math Comput Modelling 1999;30(1–2):15–40.
[24] Bianca C. Thermostatted kinetic equations as models for complex systems in physics and life sciences. Phys Life Rev 2012;9(4):359–99.
[25] Bianca C, Riposo J. Mimic therapeutic actions against keloid by thermostatted kinetic theory methods. Eur Phys J Plus 2015;130(8):1–14.
[26] Bianca C, Brézin L. Modeling the antigen recognition by B-cell and T-cell receptors through thermostatted kinetic theory methods. Int J Biomath 2017;10(05):1750072.
[27] Bianca C, Mogno C. Qualitative analysis of a discrete thermostatted kinetic framework modeling complex adaptive systems. Commun Nonlinear Sci Numer Simul 2018;54:221–32.
[28] Bianca C, Menale M. Existence and uniqueness of nonequilibrium stationary solutions in discrete thermostatted models. Commun Nonlinear Sci Numer Simul 2019;73:25–34.
[29] Bianca C, Carbonaro B, Menale M. On the Cauchy problem of vectorial thermostatted kinetic frameworks. Symmetry 2020;12(4):517.
[30] Brauer F, Castillo-Chávez C, Brauer F, Castillo-Chávez C. Basic ideas of mathematical epidemiology. Math Models Popul Biol Epidemiol 2001;275–337.
[31] Diekmann O, Heesterbeek JAP. Mathematical epidemiology of infectious diseases: model building, analysis and interpretation. Vol. 5, John Wiley & Sons; 2000.
[32] Bauch C, d'Onofrio A, Manfredi P. Behavioral epidemiology of infectious diseases: an overview. In: Modeling the interplay between human behavior and the spread of infectious diseases. Springer; 2013, p. 1–19.
[33] Buonomo B, Lacitignola D, Vargas-De-León C. Qualitative analysis and optimal control of an epidemic model with vaccination and treatment. Math Comput Simulation 2014;100:88–102.
[34] Buonomo B, Della Marca R, d'Onofrio A, Groppi M. A behavioural modelling approach to assess the impact of COVID-19 vaccine hesitancy. J Theoret Biol 2022;534:110973.
[35] d'Onofrio A, Manfredi P. The interplay between voluntary vaccination and reduction of risky behavior: a general behavior-implicit SIR model for vaccine preventable infections. In: Current trends in dynamical systems in biology and natural sciences. Springer; 2020, p. 185–203.
[36] Della Marca R, d'Onofrio A. Volatile opinions and optimal control of vaccine awareness campaigns: Chaotic behaviour of the forward-backward sweep algorithm vs. heuristic direct optimization. Commun Nonlinear Sci Numer Simul 2021;98:105768.
[37] Della Marca R, Machado Ramos MdP, Ribeiro C, Soares AJ. Mathematical modelling of oscillating patterns for chronic autoimmune diseases. Math Methods Appl Sci 2022.
[38] Wang Z, Bauch CT, Bhattacharyya S, d'Onofrio A, Manfredi P, Perc M, et al. Statistical physics of vaccination. Phys Rep 2016;664:1–113.
[39] Albi G, Bertaglia G, Boscheri W, Dimarco G, Pareschi L, Toscani G, et al. Kinetic modelling of epidemic dynamics: social contacts, control with uncertain data, and multiscale spatial dynamics. In: Predicting pandemics in a globally connected world, volume 1: toward a multiscale, multidisciplinary framework through modeling and simulation. Springer; 2022, p. 43–108.
[40] Bertaglia G, Pareschi L. Hyperbolic models for the spread of epidemics on networks: kinetic description and numerical methods. ESAIM Math Model Numer Anal 2021;55(2):381–407.
[41] Corlan AD, Ross J. Kinetics methods for clinical epidemiology problems. Proc Natl Acad Sci 2015;112(46):14150–5.
[42] Della Marca R, Loy N, Menale M. Intransigent vs. volatile opinions in a kinetic epidemic model with imitation game dynamics. Mathematical Medicine and Biology: A Journal of the IMA 2023;40(2):111–40.
[43] Della Marca R, Loy N, Tosin A. An SIR model with viral load-dependent transmission. J Math Biol 2023;86(4):61.
[44] Diekmann O, Heesterbeek J, Roberts MG. The construction of next-generation matrices for compartmental epidemic models. J R Soc Interface 2010;7(47):873–85.
[45] Sharma S, Samanta G. Analysis of a drinking epidemic model. Int J Dyn Control 2015;3(3):288–305.
[46] Castillo-Chavez C. On the computation of r. and its role on global stability Carlos Castillo-Chavez*, Zhilan Feng, and Wenzhang Huang. In: Mathematical approaches for emerging and reemerging infectious diseases: an introduction. Vol. 1, Springer Science & Business Media; 2002, p. 229.
[47] Routh EJ. A treatise on the stability of a given state of motion, particularly steady motion: being the essay to which the adams prize was adjudged in 1877, in the University of Cambridge. Macmillan and Company; 1877.
[48] Hurwitz A. Ueber die Bedingungen, unter welchen eine Gleichung nur Wurzeln mit negativen reellen Theilen besitzt. Math Ann 1895;46(2):273–84.