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Recent Advances in Biological, Epidemiological, and Social Dynamics
Special Session B26

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Innovative mathematical models for biological, medical, epidemiological, and social dynamics are now more crucial than ever. In recent years, such models have found wide application in, for example, the study of infectious diseases (COVID-19 in particular), the behavior of large crowds, online dissemination of misinformation, and the development of new immunotherapies. This Special Session aims to collect the most recent efforts of the mathematical community to produce reliable models spanning a wide range of topics relevant to the biological and social sciences. We will explore diverse classes of models: including, but not limited to, models based on ordinary and partial differential equations, networks, kinetic theory, and agent-based approaches, as well as modern data-driven techniques. Aspects relating to phenomenological modeling, mathematical analysis, model calibration and validation, as well as applications, will be considered. The ultimate goal of the Special Session is to foster the exchange of ideas among researchers with diverse mathematical expertise, working in a broad range of application areas. The Special Session also promotes the activities of the UMI Group Modellistica Socio-Epidemiologica (*Socio-Epidemiological Modeling Group*).

Schedule and Abstracts

July 25, 2024

1. Session 1: Epidemiology, 11:30–13:00

11:30–12:00 Epidemic models incorporating the role of individuals' viral load.

Rossella Della Marca (SISSA, ITALY)
Nadia Loy (Politecnico di Torino, ITALY)
Andrea Tosin (Politecnico di Torino, ITALY)

Abstract. In classical epidemic models, a neglected aspect is the heterogeneity of disease transmission and progression linked to the viral load of each infectious individual. Here, we attempt to investigate the interplay between the evolution of individuals' viral load and the epidemic dynamics from a theoretical point of view. In the framework of multi–agent systems [3], we propose a particle stochastic model describing the infection transmission through interactions among agents and the individual physiological course of the disease. Agents have a double microscopic state: a discrete label, that denotes the epidemiological compartment to which they belong, and a microscopic trait, representing a normalized measure of their viral load. Specifically, we consider Susceptible–Infected–Removed–like dynamics where the disease transmission rate [2] or the isolation rate [1] of infectious individuals may depend on their viral load. We derive kinetic evolution equations for the distribution functions of the viral load of the individuals in each compartment, whence, via suitable upscaling procedures, we obtain a macroscopic model for the densities and viral load momentum. We perform then a qualitative analysis of the ensuing macroscopic model, and we present numerical tests in the case of both constant and viral load–dependent model parameters.

References

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12:00–12:30 Recent advances on behavioural integral epidemic models with information index.

Bruno Buonomo (University of Naples Federico II, ITALY)

Abstract. The information index has been introduced and developed by P. Manfredi, A. d'Onofrio and coauthors in a series of papers starting from 2007 [1,4,5]. It is a distributed delay which describes the opinion-driven human behavioural changes [6,7]. The information index can be considered as an extension of the concept of prevalence-dependent contact rate due to V. Capasso in the seventies [3]. In this talk, we discuss integral models describing the epidemic propagation of an infectious disease. The models are behavioural in the sense that the constitutive law for the Force of Infection includes an information index. We show some results obtained via qualitative analysis and numerical simulations. In particular, we show that when the memory of the past values of the infection is exponentially fading, the stability of an endemic state is guaranteed. Through numerical simulations, we show that self-sustained oscillations may arise when the memory is more focused in the disease's past history [2]. This research is in collaboration with E. Messina, C. Panico (University of Naples Federico II) and A. Vecchio (IAC-CNR).

References

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[6] Manfredi P., d'Onofrio A. (eds). *Modeling the interplay between human behavior and the spread of infectious diseases.* New York: Springer; 2013

[7] Wang Z. et al., *Statistical physics of vaccination.* Physics Reports;664 (2016)

12:30–13:00 Advanced compartmental models for pandemic management.

Benedetto Piccoli (Rutgers University - Camden, USA)

Abstract. The COVID-19 pandemic highlighted the need to quickly respond, via public policy, to the onset of an infectious disease breakout. Deciding the type and level of interventions a population must consider to mitigate risk and keep the disease under control could mean saving thousands of lives. Here we provide a framework for capturing population heterogeneity whose consideration may be crucial when developing a mitigation strategy based on non-pharmaceutical interventions. Precisely, we use age-stratified data to segment our population into groups with unique interactions. Our model will be fit to census data for the state of New Jersey (but could be easily adapted to other states and countries), thus we consider 7 different groups corresponding to age brackets of census data denoted by a subscript, e.g. S_j, $j = 1, \dots, 7$. The interactions among different age groups will be encoded by a matrix $L = (l_{k,j})$, where $l_{k,j}$ quantifies the

interactions that group k is having with group j. Let us denote by $N_j = S_j + E_j + I_j + R_j$ the total population in the j-th age group (which is constant), and set $\widetilde{N}_j = \sum_{k=1}^{h} l_{k,j}\ N_k$, which represents the total number of individuals the j-th susceptibles are interacting with. Then we set:

$$\begin{cases} \dot{S}_j = -u\ \dfrac{\beta S_j}{\widetilde{N}_j}\ \displaystyle\sum_{k=1}^{h} l_{k,j}\, I_k \\ \dot{E}_j = u\ \dfrac{\beta S_j}{\widetilde{N}_j}\ \displaystyle\sum_{k=1}^{h} l_{k,j}\, I_k - \delta E_j \\ \dot{I}_j = \delta E_j - \gamma I_j \\ \dot{R}_j = \gamma I_j \end{cases} \tag{1}$$

for $j = 1, \dots, h$, where $1 - u$ is the lockdown rate, β the infection rate, L the interaction matrix, δ the latent period, and γ the recovery rate. We sub-divide the interaction matrix $l = (l_{k,j})$ into a well-known socially driven subset of uniquely interacting groups. We define a set of l^i to be n interaction matrices where $l^i_{k,j}$ encodes some portion of the interaction between groups k and j due to the i-th subset of interactions. It holds that $\sum_{i=1}^{n} l^i_{k,j} = l_{k,j}$ for every k, j and thus $l = \sum_{i=1}^{n} l^i$. Each new interaction matrix brings the ability to encode the regulation of that subgroup of interactions, and we will denote by u_i the "amount of allowed interactions" such that the severity of lockdown for each group of interactions is $1 - u_i$. Therefore our final model reads

$$\begin{cases} \dot{S}_j = -\beta \dfrac{S_j}{\widetilde{N}_j} \left(u_1 \displaystyle\sum_{k=1}^{h} l^1_{k,j}\ I_k + u_2 \sum_{k=1}^{h} l^2_{k,j}\ I_k + \ldots + u_n \sum_{k=1}^{h} l^n_{k,j}\ I_k \right) \\ \dot{E}_j = \beta \dfrac{S_j}{\widetilde{N}_j} \left(u_1 \displaystyle\sum_{k=1}^{h} l^1_{k,j}\ I_k + u_2 \sum_{k=1}^{h} l^2_{k,j}\ I_k + \ldots + u_n \sum_{k=1}^{h} l^n_{k,j}\ I_k \right) - \delta E_j \\ \dot{I}_j = \delta E_j - \gamma I_j \\ \dot{R}_j = \gamma I_j \end{cases} \tag{2}$$

with $j = 1, \dots, h$ and other parameters set as in (1).

Break, 13:00-15:00

2. Session 2: Biology, 15:00-16:30

15:00–15:30 Balance Laws in Biological Models.

Rinaldo M. Colombo (University of Brescia, ITALY)
Mauro Garavello (University of Brescia, ITALY)
Francesca Marcellini (University of Brescia, ITALY)
Elena Rossi (University of Modena and Reggio Emilia, ITALY)

Abstract. Balance laws, i.e., partial differential equations of the form $\partial_t u + \nabla \cdot f(t, x, u) = g(t, x, u)$, appear in a variety of models devoted to epidemiology, structured population dynamics, cell growth, cluster formation and predator - prey or pursuit - evasion games. They may contain local and non local terms, be set in unbounded or bounded domains with different boundary conditions, and be coupled with equations of other types.

The present talk is devoted to analytic results establishing the well posedness of models based on balance laws, with numerical integrations showing the qualitative behavior of solutions in specific cases. A class of predator - prey models motivated by pest control and some structured epidemiological models will be considered in detail.

15:30–16:00 Kinetic formulation of cross-diffusion models for predator–prey dynamics.

Andrea Bondesan (University of Parma, ITALY)
Marizia Bisi (University of Parma, ITALY)
Maria Groppi (University of Parma, ITALY)
Cinzia Soresina (University of Trento, ITALY)

Abstract. We introduce a mesoscopic framework for the modeling of predator–prey dynamics in a two-dimensional space, where the different species evolve via both intraspecific and interspecific interactions. We will show how to combine in a suitable way diffusion- and fast-reaction-type limits to formally derive, at a macroscopic level, reaction–cross-diffusion systems involving a Beddington–DeAngelis-like functional response.

16:00–16:30 Modeling some aspects of stem cell therapy in cardiac tissue regeneration.

Alberto Maria Bersani (Sapienza University of Rome, ITALY)

Abstract. An acute myocardial infarction (MI) is followed by a multiphase reparative response, in which, in particular, the damaged tissue is replaced with a fibrotic scar composed of cardiac fibroblasts and collagens. This eventually leads to impaired cardiac function, resulting in adverse consequences such as cardiac atrophy and arrhythmogenicity. In the meantime, the damaged tissue tries to recover and the immune system responds by emitting damage signals so that new cells come to the aid. These signals are the chemokines.

Several studies show that stem cell therapies can help tissue repair, after an acute myocardial infarction and are used as an alternative to traditional pharmacological management of myocardial infarction. Stem cells have the capacity to regenerate cardiac tissue and improve the function of the damaged heart. Thus clearly the impact of the chemokines in the attraction of these cells toward the damaged area of the myocardium becomes very important. However several biological aspects are not yet completely understood and are still under investigation.

For example, it was not yet completely clarified if the decline in cardiac fibrosis was due to replacement of dead cardiomyocytes by the stem cells (they can divide and become specialized cells, such as cardiomyocytes), or because of the direct effects of paracrine factors released from the transplanted stem cells on the extracellular matrix and on other cells and molecules participating to the tissue repair [1].

In this talk we present two mathematical models describing two different effects of stem cells on the damaged cardiac tissue. On the one hand we generalize a recent model [2], which describes the rebuilding of the cardiac tissue by means of the differentiation of the stem cells, which replace the dead cardiomyocytes. In the new model the chemotactic effect of the chemokines on the stem cells is taken into consideration. On the other hand we generalize a recent mathematical model [3], based on a system of PDEs, which describes the processes happening in the damaged area and bringing to the formation of fibrotic scars. In our new model we take into consideration the paracrine effects of the stem cells, in order to reduce the fibrotic scar and restore the functionality of the heart. For the paracrine effetcs, we follow in particular the paper [4], which proposes a mathematical model, based on ODEs, related to the stem cell therapy after myocardial infarction.

References

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Break, 16:30-17:00

3. Session 3: Social dynamics, 17:00-18:00

17:00–17:30 On online-offline models of spread of tension and social outbursts.
Nancy Rodriguez (University of Colorado - Boulder, USA)

Abstract. Social collective behavior, like boycotts or protests, is a global phenomenon. Lately, the online spread of information and social tensions has altered the dynamics of such events. In this talk, we will introduce models that link online and offline dynamics, exploring spreading rates of these activities. Additionally, we will delve into how the online network structure impacts offline activity dynamics.

17:30–18:00 Condensation effects in social dynamics.
Mattia Zanella (University of Pavia, ITALY)

Abstract. In this talk, we discuss a class of models to understand the impact of population size on opinion formation dynamics, a phenomenon usually related to group conformity. To this end, we introduce a new kinetic model in which the interaction frequency is weighted by the kinetic density. In the quasi-invariant regime, this model reduces to a Kaniadakis-Quarati-type equation with quadratic drift, originally introduced for the dynamics of bosons in a spatially homogeneous setting. From the obtained PDE for the evolution of the opinion density, we determine the regime of parameters for which a critical mass exists and triggers blow-up of the solution. Therefore, the model is capable of describing strong conformity phenomena in cases where the total density of individuals holding a given opinion exceeds a fixed critical size. In the final part, several numerical experiments demonstrate the features of the introduced class of models and the related consensus effects.

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