BOOK OF ABSTRACTS

SS1 - Recent trends in biomathematics: modelling patterns and collective dynamics from micro to macro

2nd Joint Meeting Brazil-Italy in Mathematics

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SS32

Turing Instability and Pattern Formation in a Spatial HIV Model

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We consider a three-species reaction–diffusion model for HIV intrahost dynamics, incorporating logistic growth of healthy target cells, diffusion of all populations, and a chemotactic drift of target cells driven by cytokine signalling. The model is designed to describe the interplay between viral spread, cell proliferation, and spatial migration mechanisms that may lead to heterogeneous infection patterns.

We analyse the stability of the disease-free equilibrium and endemic equilibrium and derive threshold conditions for the onset of Turing instability of the endemic state. The results show that chemotaxis may destabilize spatially homogeneous equilibria and trigger the emergence of infection hotspots, while the inclusion of logistic proliferation lowers the critical threshold for instability with respect to previous models. To investigate the post-bifurcation regime, the linear analysis is complemented by a weakly nonlinear study and numerical simulations, providing information on the amplitude, shape, and spatial organization of the emerging patterns.

These results contribute to the mathematical understanding of pattern formation in HIV infection dynamics and highlight the role of chemotaxis and proliferation in the onset of spatially structured viral distributions.

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Multi-scale modeling of action potential dynamics in neural networks

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Large-scale dynamics in neural networks emerge from the complex interplay between microscopic single-neuron behaviors and structural network heterogeneity. Individual neurons continuously evolve according to their intrinsic biophysical properties, while interacting through complex synaptic networks. Understanding how these mechanisms integrate across scales is essential to explain the emergence of coordinated, population-level behavior, as well as abnormal synchronization patterns which are often a hallmark of neurological disorders.

In this talk, we introduce a multi-scale kinetic framework capable of reproducing action potential dynamics within a neural network by accounting for heterogeneity in both neuron-to-neuron connections and large-scale brain structure. Specifically, two levels of description are coupled: within a single brain area, pairwise neuron interactions for the exchange of membrane potential are statistically described; among different areas, a graph description of the brain network topology is embedded. Through appropriate scaling arguments, closed systems of macroscopic equations are formally derived and the equilibria of both kinetic and macroscopic settings are determined. Finally, numerical simulations on simplified graph topologies are performed to investigate how network architecture and structural heterogeneities influence membrane potential propagation and the emergence of synchronization regimes [1,2].

Joint work with M. Groppi and M. Bisi (UniPR), A. Tosin (PoliTO)

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Statistical Physics of Active Particles: From Clustering to Survival in Toxic Landscapes

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Active matter is a topic in statistical physics that studies self-propelled entities, such as microorganisms and artificial particles, which consume energy to move persistently, giving rise to a wide range of emergent behaviors. After a brief introduction to active matter and our previous work on clustering driven by persistent motion, I will discuss chemotactic active particles in a toxic chemical field. The particles adjust their tumbling rate according to the local toxin gradient, while the toxin continuously accumulates within them and is simultaneously removed by detoxification. Combining numerical simulations and Fokker-Planck equations, we uncover a non-monotonic dependence of survival probability on toxin diffusivity: intermediate diffusivities maximize lethality, whereas above a critical value the toxin profile becomes sufficiently shallow for detoxification to dominate. A minimal theoretical framework captures this transition and its underlying mechanism.

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Exploring immune pathways and remyelination in Multiple Sclerosis

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In this talk, I shall review a class of recently developed mathematical models aimed at capturing the primary mechanisms that drive the initial stages of Multiple Sclerosis (MS). A fundamental debate persists in the immunological community concerning the triggers of the immune cascade that initiate MS pathology. The first scenario involves activation of local microglia, recruitment of systemic immune responses, and oligodendrocyte apoptosis [1, 3, 4], whereas the second scenario investigates cytokine-mediated modulation of macrophage activation [5]. We proposed the following reaction-diffusion-chemotaxis model for $m(t,x)$, the density of activated macrophages, $c(t,x)$ the density of cytokines, and $d(t,x)$ the density of oligodendrocytes [2]:

$$
\begin{cases}
\partial_t m = \Delta m - \nabla \cdot (\chi(m)\nabla c) + \dfrac{c}{\xi + c}\, m(1-m), & (x,t) \in \Omega_T \\
\partial_t c = \dfrac{1}{\tau}\left(\epsilon \Delta c + \delta d - c + \beta m\right), & (x,t) \in \Omega_T \\
\partial_t d = rF(m)\, m\,(1-d)\,, & (x,t) \in \Omega_T
\end{cases} \tag{1}
$$

with $\chi(m) = \chi \frac{m}{1+m}$, $F(m) = \frac{m}{1+m}$, $\Omega_T = (0,T) \times \Omega$, where Ω is a bounded domain in $\mathbb{R}^n$ $(n = 1, 2)$ with smooth boundary $\partial\Omega$ on which no-flux boundary conditions are imposed. Upon varying the parameter ξ that rules the effect of cytokines on the macrophage activation rate, the model describes the two different scenarios.

Employing both analytical and numerical methods, we demonstrate that the model supports the emergence of various demyelination patterns. Utilizing biologically realistic parameters from existing literature, we show that the asymptotic solutions of the model system (1) replicate pathological features closely matching MRI findings.

Bifurcation analysis reveals substantial differences in pathological outcomes between the two activation scenarios. The *innate immunity* scenario produces highly aggressive pathology, characterized by intense focal inflammation and rapid progression. In contrast, the *cytokine-mediated* scenario results in less aggressive pathology, exhibiting lower inflammation levels and significantly slower disease progression.

In the last part of the talk, we will discuss ongoing extensions of the model that integrate endogenous regenerative processes driven by acute inflammation stages, aimed at promoting remyelination – a regenerative effort robustly documented during early disease phases but frequently impaired in chronic MS lesions.

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Crowding indexes as a bridge between individual behaviour and population dynamics in spatially-structured populations

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Classical theoretical ecology relies on mean-field, well-mixed assumptions that treat populations as spatially homogeneous. Yet empirical data consistently shows that most animals exhibit range residency: they confine their movements to a limited region of the available habitat and use it non-uniformly, putting into question how well mean-field assumptions hold in animal populations. Thus, bridging individual behaviour and population-level dynamics in spatially-structured populations requires new theoretical tools.

In this talk, I will introduce the crowding index for motile organisms, a dimensionless parameter that encapsulates all ecologically-relevant spatial information for population dynamics models into a single quantity that can be directly estimated from animal tracking data. The crowding index emerges naturally from a spatial moment dynamics framework [1] and is the central quantity driving the Range-Resident (RR) logistic model [2], the first analytically tractable population dynamics model incorporating range residency. I will show how the crowding index recovers demographic information using spatial quantities in the system and how carrying capacity has a non-monotonic relationship with animals' home range sizes - a pattern that has no counterpart in traditional models. Finally, I will discuss how this coarse-graining approach generalizes beyond the RR-Logistic, enabling the upscaling of complex individual behaviors to population- and community-level patterns across a variety of ecological systems.

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Growth and efficiency principles of bacterial metabolism

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Bacterial metabolism is a complex non-equilibrium process shaped by resource allocation and environmental constraints. In this talk, I will present recent data and modeling suggesting that two key features organize bacterial responses to perturbation: growth rate and carbon efficiency. Using antibiotic and energetic perturbations as a case study, I will discuss how such perturbations can alter bacterial metabolism by shifting growth rate, carbon efficiency, or both. More broadly, this work motivates finite-resource descriptions of bacterial fitness, in which the relative importance of speed and efficiency is set by ecological context.

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Modelling Cell Reorientation under Stretch

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The active response of cells to mechanical cues due to their interaction with the environment has been of increasing interest, since it is involved in many physiological phenomena, pathologies, and in tissue engineering. In particular, several experiments have shown that, if a substrate with overlying cells is cyclically stretched, they will reorient to reach a well-defined angle between their major axis and the main stretching direction. The aim of this talk will be to investigate the interplay between mechanics and cell organization. It will be shown that cells organise their internal structure to minimize an elastic energy that then drives this reorientation process. Viscoelastic effects will then be included to explain the dependence of the appearance of the phenomenon as a function of oscillation frequency. Finally, randomness is taken into account and discussed on the basis of a Fokker-Plack equation.

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Flow spatial structure determines pattern instabilities in nonlocal models of population dynamics

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A wide range of spatial scales, from cellular assemblies to entire landscapes, exhibit self-organized population patterns arising from nonlinear interactions among organisms and between organisms and their environment [1-3]. These spatial structures occur in both marine and terrestrial ecosystems and strongly influence ecological processes, including population growth, species coexistence, and extinction. We investigate how environmental flows influence spatial pattern formation and population dynamics using two nonlocal models of population dynamics coupled to two stationary flow fields. Combining numerical simulations with analytical approximations, we show that the spatial structure of the flow velocity field governs the onset of pattern-forming instabilities. In particular, we demonstrate that while shear flows aligned with the intrinsic pattern geometry leave the instability threshold essentially unchanged, vortex-like flows delay the onset of pattern formation relative to the no-flow case. Our results highlight the importance of incorporating realistic environmental transport processes into models of self-organized ecological pattern formation.

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The stochastic origins of costly sexual signals

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Sexual selection provides one of the most striking examples of pattern formation in biological populations. Yet the persistence of costly male ornaments, such as the peacock's tail, remains a long-standing evolutionary puzzle. Classical explanations are typically based on either Fisher's runaway process or the handicap principle, yet a unified mathematical framework connecting these mechanisms is still lacking.

In this work, we introduce a stochastic individual-based model for the coevolution of a female mating preference and a costly male trait in finite populations. The model combines assortative mating, trait costs, and demographic stochasticity within a Moran-type evolutionary process.

Using analytical calculations and Monte Carlo simulations, we show that stochasticity fundamentally changes the role of trait cost. Rather than acting primarily as a signal of quality, cost delays trait fixation, maintains polymorphism, and creates a temporal window that allows correlations between preference and trait to emerge. As a consequence, costly traits can increase the probability of joint fixation of trait and preference, an effect absent in deterministic models.

Our results provide a bridge between Fisher's runaway mechanism and the handicap hypothesis, highlighting how population-level evolutionary outcomes emerge from stochastic interactions at the individual level. More broadly, the study illustrates the importance of finite-size effects and fixation dynamics in the evolution of complex biological systems.

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