

# Mathematical Biology: Applications and Analysis

June 19th — June 23rd, 2023

**Abstract.** Systems of interacting particles play an important role in the mathematical modelling of biomedical phenomena. Typically, these systems are encountered across scales reaching from intracellular processes to the growth of large cell aggregates and tissues, and comprise numbers of agents that are often intractable to simulate numerically. This calls for techniques to coarse-grain and derive effective models that, systematically, establish a bridge between different scales. Concurrently, certain model properties such as phase segregation, pattern formation, or self-organisation are expected to be observed across scales, posing a particular challenge in the derivation of effective models.

## Invited Speakers:

**Sebastian Aland**

*(TU Bergakademie Freiberg  
& HTW Dresden)*

**Luca Alasio**

*(Sorbonne Université)*

**Joshua Bull**

*(University of Oxford)*

**Noemi David**

*(Université de Lyon 1)*

**Tomasz Dębiec**

*(University of Warsaw)*

**Antonio Esposito**

*(University of Oxford)*

**Gissell Estrada-Rodriguez**

*(University of Oxford  
& Universitat Politècnica de Catalunya)*

**Benjamin Friedrich**

*(TU Dresden)*

**Pierre Haas**

*(MPI of Molecular Cell Biology and Genetics)*

**Lea Happel**

*(TU Dresden)*

**Laura Kanzler**

*(Université Paris-Dauphine)*

**Tommaso Lorenzi**

*(Politecnico di Torino)*

**Duncan Martinson**

*(University of Oxford)*

**Franziska Matthäus**

*(Goethe University Frankfurt)*

**Jan-Frederik Pietschmann**

*(University of Augsburg)*

**Marco Salvalaglio**

*(TU Dresden)*

**Simon Syga**

*(TU Dresden)*

**Bao Quốc Tăng**

*(Universität Graz)*

**Chandrasekhar Venkataraman**

*(University of Sussex)*

**Chiara Villa**

*(Sorbonne Université)*

**Axel Voigt**

*(TU Dresden)*

**Havva Yoldaş**

*(TU Delft)*

## Organisers:

**Julia I. M. Hauser**

**Markus Schmidtchen**  
*(TU Dresden)*



# Timetable

## Monday, 19th of June, C207

9:30–9:45	<b>Opening</b>	
09:45–10:30	<b>Chiara Villa</b>	Modelling the evolutionary dynamics of nutrient-deprived cancer cells using structured PDEs: analysis, numerics and validation
10:30–11:00	<b>Coffee Break</b>	
11:00–11:45	<b>Noemi David</b>	Singular limits arising in mechanical models of tumor growth
11:45–12:30	<b>Joshua Bull</b>	Mathematical Methods for Spatial Analysis of Medical Imaging
12:30–14:00	<b>Lunch Break</b>	
14:00–14:45	<b>Bao Quốc Tãng</b>	An aggregation model of cockroaches with fast-or-slow motion dichotomy
14:45–15:30	<b>Pierre Haas</b>	Impossible ecologies: interaction networks and stability of coexistence in ecological communities
15:30–16:00	<b>Coffee Break</b>	
16:00–16:45	<b>Laura Kanzler</b>	Size-spectrum Evolution in Marine Ecosystems
16:45–17:30	<b>Havva Yoldaş</b>	Run and tumble model for bacteria movement
17:45–18:45	<b>Reception in Room 250</b>	
19:30	<b>Optional joined dinner at Augustiner an der Frauenkirche</b>	

## Tuesday, 20th of June, C207

09:00–09:45	<b>Antonio Esposito</b>	A gradient flow approach to Cahn-Hilliard models
09:45–10:30	<b>Lea Happel</b>	Curvotaxis - The influence of curvature on (collective) cellular motion
10:30–11:00	<b>Coffee Break</b>	
11:00–11:45	<b>Sebastian Aland</b>	Numerical simulation of active cell surfaces - from pattern formation to cell division
11:45–12:30	<b>Jan-Frederik Pietschmann</b>	On a mathematical model for neuron growth
12:30–14:00	<b>Lunch Break</b>	
14:00–14:45	<b>Gissell Estrada-Rodriguez</b>	Robust estimation of interaction potentials in nonlinear nonlocal gradient flow equations with noisy data
14:45–15:30	<b>Tomasz Dębiec</b>	On some Hookean models of dilute polymeric fluids
15:30–16:00	<b>Coffee Break</b>	
16:00–16:45	<b>Chandrasekhar Venkataraman</b>	Multiscale modelling of cell signalling
16:45–17:30	<b>Luca Alasio</b>	Mathematical modelling of the visual cycle and related diseases
19:00	<b>Conference Dinner at PlanWirtschaft</b>	

## Wednesday, 21st of June, C207 and Room 250

09:45–10:30	<b>Axel Voigt</b>	Fluid deformable surfaces
10:30–11:00	<b>Coffee Break</b>	
11:00–11:45	<b>Simon Syga</b>	Natural selection under the go-or-grow dichotomy leads to the emergence of phenotypic and genetic heterogeneity
11:45–12:30	<b>Duncan Martinson</b>	Extracellular matrix remodelling by lead chick cranial neural crest cells is a major determinant of robust collective migration
12:30–14:00	<b>Lunch Break - Starting afterwards in Room 250</b>	
14:00–14:45	<b>Franziska Matthäus</b>	Continuous and agent-based modeling approaches capture different aspects and provide complementary insights into skin patterning
14:45–15:30	<b>Tommaso Lorenzi</b>	Spatial spread and evolutionary dynamics of heterogeneous cell populations: insights from partial integro-differential equation models
15:30–16:00	<b>Coffee Break</b>	
16:00–16:45	<b>Benjamin Friedrich</b>	A mechanism of branching morphogenesis inspired by diatom silica formation
16:45–17:30	<b>Marco Salvalaglio</b>	Pattern formation and defects in Swift-Hohenberg models
	<b>Free Evening</b>	

## Thursday, 22nd of June, Library

09:00–12:30	<b>Workshop</b>
12:30–14:00	<b>Lunch Break</b>
14:30–16:30	<b>Hike</b>
16:30–17:30	<b>Wine Tasting at Schloss Wackerbarth</b>
	<b>Free Evening</b>

## Friday, 23rd of June, Library

09:00–12:15	<b>Workshop</b>
12:15–12:30	<b>Closing</b>
12:30–14:00	<b>Lunch Break</b>
14:00–16:00	<b>Open Discussion/Departure</b>

# List of Abstracts – Talks



## Monday, 19th of June

**Chiara Villa**

*Modelling the evolutionary dynamics of nutrient-deprived cancer cells using structured PDEs: analysis, numerics and validation*

Glucose and oxygen are primary energy sources for cancer cells. Several lines of evidence support the idea that changes in gene expression levels (e.g. MCT1, HIF1) elicit metabolic reprogramming of cancer cells in nutrient-poor environments, promoting cancer cell survival and disease progression.

A more in-depth theoretical understanding of the evolutionary processes at the root of cancer cell adaptation to nutrient deprivation can be achieved through analysis and numerical simulation of structured-population models. The focus of this talk is on non-local partial differential equations modelling the adaptive dynamics of a population of cancer cells structured by the level of gene expression. First, I will present an experimentally-informed mathematical model of a well-mixed population, which was calibrated with data from in vitro experiments on glucose-deprived aggressive cancer cells. Then, I will present a spatially-explicit model and discuss the additional analytical challenges introduced by spatial movement, and how a formal Hamilton-Jacobi approach can be used to obtain weak solutions in appropriate asymptotic limits.

**Noemi David**

*Singular limits arising in mechanical models of tumor growth*

The mathematical modelling of cancer has been increasingly applying fluid-dynamics concepts to describe the mechanical properties of tissue growth. The bio-mechanical pressure plays a central role in these models, both as the driving force of cell movement and as an inhibitor of cell proliferation. In this talk, I will present how it is possible to build a bridge between models that have different pressure-velocity or pressure-density relations. In particular, I will focus on the inviscid limit from a visco-elastic model to a Darcy's law-based model, and the incompressible limit that links the latter to a Hele-Shaw free boundary problem with density constraint.

## **Jan-Frederik Pietschmann**

### *On a mathematical model for neuron growth*

We study a free boundary model to examine the effect of vesicle transport onto neurite growth. It consists of systems of drift-diffusion equations describing the evolution of the density of antero- and retrograde vesicles in each neurite coupled to reservoirs located at the soma and the growth cones of the neurites, respectively. The model allows for a change of neurite length depending on the vesicle concentration in the growth cones. After establishing existence and uniqueness for the time-dependent problem, we briefly comment on possible types of stationary solutions. Finally, we provide numerical studies on biologically relevant scales using a finite volume scheme. We illustrate the capability of the model to reproduce cycles of extension and retraction.

## **Bao Quốc Tăng**

### *An aggregation model of cockroaches with fast-or-slow motion dichotomy*

This talk presents a reaction–diffusion system describing social behaviour of cockroaches. An essential new aspect in this model is that the dispersion behaviour due to overcrowding effect is taken into account as a counterpart to commonly studied aggregation. This consideration leads to an intriguing new phenomenon which has not been observed in the literature for cockroaches. Namely, due to the competition between aggregation towards areas of higher concentration of pheromone and dispersion avoiding overcrowded areas, the cockroaches aggregate more at the transition area of pheromone. Moreover, the fast reaction limit is also considered where the switching rate between active and inactive subpopulations tends to infinity. By utilising improved duality and energy methods, together with the regularisation of heat operator, it is proved that the weak solution of the reaction–diffusion system converges to that of a reaction-cross-diffusion system. This is based on a joint work with Jan Elias, Hirofumi Izuhara, Masayasu Mimura.

## **Havva Yoldaş**

### *Run and tumble model for bacteria movement*

I will talk about run and tumble equations describing the movement of chemotactic bacteria under the effect of chemical stimulus. I will discuss some properties of the models, particularly the similarities and differences between the run and tumble equations and the kinetic equations arising in interacting gas dynamics, i.e. Boltzmann-type kinetic equations. We will look at the long-time behaviour of this class of equations and summarise the recent hypocoercivity results. The talk is based on papers in collaboration with Jo Evans from the University of Warwick.

## Laura Kanzler

### *Size-spectrum Evolution in Marine Ecosystems*

Trophic interactions between animals in the ocean were matter of interest since 6 decades. It was quickly discovered that the individuals' body size acts as 'master trait' in food webs of animals, giving rise to emergent distributions of biomass, abundance and production of organisms.

We propose and investigate a deterministic structural equation of Boltzmann type, aiming to capture this emergence phenomenon in aquatic ecosystems. The equation of interest is derived from individual based dynamics governed by a stochastic process. Following the observation that the body mass is the crucial trait in these dynamics, it is based on the assumption that binary interactions between individuals in the ecosystem take place: A predator feeding on a prey, which then results in growth of the predator with assimilating a certain (usually very small) amount of its prey's mass as well as production of a certain amount of organisms or nutrients, nursing the ecosystem at a very small scale. This model reproduces the so-called "cascade-effect", which is frequently observed in ecosystems and describes the suppression of specific trophic positions in the ecosystem as result of an indirect influence from one trophic level to the second next lower or higher.

Some analytical results in specific parameter regimes are discussed and numerical simulations underlying these observations are given.

## Pierre Haas

### *Impossible ecologies: interaction networks and stability of coexistence in ecological communities*

Does an ecological community allow stable coexistence? In particular, what is the interplay between stability of coexistence and the network of competitive, mutualistic, or predator-prey interactions between the species of the community? These are fundamental questions of theoretical ecology, yet meaningful analytical progress is in most cases impossible beyond two-species communities. In this talk, I will therefore show how we addressed this problem statistically: For all non-trivial networks of interaction types of  $N \leq 5$  species, we sampled Lotka-Volterra model parameters randomly and thus computed the probability of steady-state coexistence being stable and feasible with Lotka-Volterra dynamics. Surprisingly, our analysis reveals "impossible ecologies", very rare non-trivial networks of interaction types that do not allow stable and feasible steady-state coexistence. I will classify these impossible ecologies, and then prove, somewhat conversely, that any non-trivial ecology that has a possible subecology is itself possible. This theorem highlights the "irreducible ecologies" that allow stable and feasible steady-state coexistence, but do not contain a possible subecology. I will conclude by showing the classification of all irreducible ecologies of  $N \leq 5$  species. Strikingly, this indicates that the proportion of non-trivial ecologies that are irreducible decreases exponentially with the number  $N$  of species. Our results thus suggest that interaction networks and stability of coexistence are linked crucially by the very small subset of ecologies that are irreducible.



## Tuesday, 20th of June

**Antonio Esposito**

*A gradient flow approach to Cahn-Hilliard models*

In this talk I will discuss the analysis of Cahn-Hilliard equations using an optimal transport approach. These models have been recently obtained as approximation of nonlocal systems of PDEs describing cell-cell adhesion. Gradient flow techniques are indeed useful to show global-in-time existence of solutions for both the scalar and the systems case. This is based on an ongoing work in collaboration with J.A. Carrillo, C. Falcó, and A. Fernández.

**Lea Happel**

*Curvotaxis - The influence of curvature on (collective) cellular motion*

Cells sense and respond to local curvature, essentially by aligning the filaments with the principal curvature directions. This influences not only the shape of single cells, it also plays an important role regarding cellular motion. For example recent experiments for cylindrical epithelial tissues of MDCK cells point to a strong connection between (extrinsic) curvature and collective cell rotation.

Mathematically this has been modelled by a coarse-grained continuous active polar gel model with ad hoc added linear curvature terms, but not by an agent-based model. We therefore propose a multiphase field model which models each cell separately and hence takes cellular properties and cell-cell interactions into account, to close this gap and add a new part in the free energy for extrinsic curvature contributions. This extrinsic curvature energy is inspired by the theory of surface liquid crystals, where extrinsic curvature effects are well understood. We consider cylindrical shapes and compare our results with the experimental data of MDCK cells. Additionally we investigate the collective behavior of cells on different tori to take a first look into surfaces with non-constant Gaussian curvature.

**Sebastian Aland**

*Numerical simulation of active cell surfaces - from pattern formation to cell division*

Shape changes of single cells are governed by the actomyosin cortex, a thin layer of active material underneath the cell surface. Besides the imposed rigidity, the cortical surface exerts an active contractile tension, the strength of which being controlled by the concentration of force-generating molecules. The complex interplay of molecule transport and surface hydrodynamics gives rise to pattern formation and self-organized shape dynamics. Despite the biological importance of these phenomena, the system is far from being understood.

To improve this understanding, we present numerical simulations of such an active surface immersed in viscous fluids. The cortex is modelled as a viscoelastic surface material, described by a freely evolving Finite-Element grid. The dynamics are coupled to a surface concentration equation of force-generating molecules (e.g. actomyosin). We analyze the emerging mechanochemical patterns and shape changes and show that the activity of the surface can lead to cell division or cell migration.

**Joshua Bull**

*Mathematical Methods for Spatial Analysis of Medical Imaging*

New technologies in medical imaging have led to increased use of multiplexed histology: images of whole tissue samples at single-cell resolution, in which a cell's expression of up to 50 different markers can be identified at the same time. These images present an intriguing mathematical question - how can we best understand which markers are spatially co-localised or anti-correlated? How can we identify structure in the interactions between cells of different types, and what can this tell us about how to better understand and treat diseases? In this talk, we present an overview of mathematical approaches for the spatial analysis of such medical imaging data, with applications to cancer and covid-19 lung samples. We explore mathematical ideas ranging from spatial statistics, to topological data analysis, to networks, and beyond, and consider how best to combine all information from all of these fields to better describe medical images.

**Gissell Estrada-Rodriguez**

*Robust estimation of interaction potentials in nonlinear nonlocal gradient flow equations with noisy data*

When applying aggregation-diffusion equations to model real-life phenomena, a major challenge lies in the choice of the interaction potential. Previous numerical and theoretical studies typically required knowledge of the form of the potential and the goal is often to reproduce the observed dynamics qualitatively, not quantitatively. In this talk, we propose a robust variational approach to solve the inverse problem of estimating the interaction potential in nonlocal nonlinear PDEs from discrete observations of a single trajectory noisy data, based on regularised least squares. We investigate how to compute a faithful estimator that is close to the true interaction potential.

**Tomasz Dębiec**

*On some Hookean models of dilute polymeric fluids*

We consider the Hookean dumbbell model, a system of nonlinear PDEs arising in the kinetic theory of homogeneous dilute polymeric fluids. It consists of the unsteady incompressible Navier-Stokes equations in a bounded Lipschitz domain, coupled to a Fokker-Planck-type parabolic equation with a centre-of-mass diffusion term, for the probability density function, modelling the evolution of the configuration of noninteracting polymer molecules in the solvent.



The micro-macro interaction is reflected by the presence of a drag term in the Fokker-Planck equation and the divergence of a polymeric extra-stress tensor in the Navier-Stokes balance of momentum equation. In a simplified case where the drag term is corotational, we prove global existence of weak solutions and discuss some of their properties: we use the relative energy method to deduce a weak-strong uniqueness type result, and derive the macroscopic closure of the kinetic model: a corotational Oldroyd-B model with stress-diffusion.

In the general noncorotational case we consider “generalised dissipative solutions” — a relaxation of the usual notion of weak solution, allowing for the presence of a, possibly nonzero, defect measure in the momentum equation, which accounts for the lack of compactness in the polymeric extra-stress tensor. Joint work with Endre Suli (Oxford).

## **Chandrasekhar Venkataraman**

### *Multiscale modelling of cell signalling*

We consider homogenisation of a model for cell signalling processes in biological tissues. Such signalling processes are the primary mechanism by which cells interact and respond to external stimuli. Hence they play an important role in the majority of cell biological phenomena. The signalling model we consider includes diffusion and nonlinear reactions on the cell surfaces, and both inter- and intracellular signalling. We derive, under the assumption of a periodic cell distribution, the equations satisfied in the limit as the cell number tends to infinity with the volume fraction of tissue occupied by the cells constant. Furthermore, we present a finite element method for the limiting two-scale bulk surface system and report on numerical simulations of the model equations. Details of the implementation will be discussed with reference to a simplified model problem.

## **Luca Alasio**

### *Mathematical modelling of the visual cycle and related diseases*

The visual cycle is a fundamental bio-chemical process in the retina: it allows photoreceptors to convert light into electrical signals (phototransduction) and subsequently to return to the dark state. George Wald obtained the Nobel Prize in 1967 for his pioneering studies on this process and it has been an active field of research in Ophthalmology ever since. I will discuss the key aspects of the visual cycle in photoreceptors and present a new mathematical model for the visual cycle in rod cells. The model consists of a system of coupled ODEs and PDEs for the concentrations of relevant molecules and proteins in photoreceptor outer segments. The goal is to give a quantitative description of the kinetics of the main photo-sensitive molecules after exposure to light. I will explain how the model can be extended in order to account for the accumulation of toxic byproducts in the eye in connection to degenerative retinal diseases.



## Wednesday, 21st of June

**Franziska Matthäus**

*Continuous and agent-based modeling approaches capture different aspects and provide complementary insights into skin patterning*

The spatially regular patterns of hair follicles are formed during early embryogenesis by the interaction of two classical pattern formation systems. The outer skin layer, the epidermis, produces chemical components that interact and generate a Turing pattern by diffusion-driven instability. Motile cells in the lower skin layer, the dermis, use chemotaxis to form regularly spaced dermal aggregates guided by the chemical map. We consider two complementary modeling approaches to gain insight into this coupled system. Using a continuous PDE system to describe the cell density as well as the concentration of two interacting and diffusing chemicals, we explore, using linear stability analysis, the pattern region for the coupled system. This approach shows that a) the parameter region for patterning is generally enlarged by coupling the individual patterning systems, b) increasing the chemotactic sensitivity speeds up the patterning process, however on the cost of pattern regularity, and c) that the interaction of the two processes can generate patterns for parameters where neither of the individual systems is unstable, and, in some cases, can also extinguish patterning rather than reinforcing it.

In a second approach we use a hybrid agent-based model to describe the cells as discrete objects interacting with the chemical system given as continuous variables. The discrete description allows us to include mechanical cell-cell interaction which, as we show, can account for rotation of the dermal aggregates observed in the experimental data. With this hybrid agent-based model we show that cluster rotation arises when the cellular response to the chemical gradient dominates the response to the mechanical forces, and that cluster rotation also depends on chemical components controlling the cluster size.

Thus, while the PDE approach facilitates a rigorous mathematical analysis, the simulation-based discrete system can account for mechanical interaction, and by this help to understand certain aspects of the biological system that are not captured by classical PDE systems.

**Simon Syga**

*Natural selection under the go-or-grow dichotomy leads to the emergence of phenotypic and genetic heterogeneity*

Cancer is a significant global health issue, with treatment challenges arising from genetic and phenotypic heterogeneity in tumors. In this study, we examine the complex relationship between evolutionary processes and phenotypic plasticity, specifically focusing on the interplay between cell migration and proliferation. Our novel cellular automaton model takes into account the movement, growth, and death of cells, as well as a change between mobile and growing states controlled by inherited and mutation-driven genotypes and the cells' microenvironment, specifically the local cancer cell density. We observe that cells at the tumor edge evolve to favor migration over proliferation and vice-versa in the tumor bulk. However, we show that this phenotypic heterogeneity can be realized by completely distinct regulations of the phenotypic switch, and that parameters such as the apoptosis rate determine which go-or-grow strategy is most effective. We estimate the transition between the different evolutionary regimes using a mean-field approximation of the

evolutionary dynamics.

This new approach demonstrates that a specific pattern of phenotypic heterogeneity can result from different cell decision making processes that need to be distinguished in different microenvironments. It also indicates that equating phenotypic traits and genotype in theoretical models can hide the underlying complexity of the cell decision-making process required to produce any observed phenotypic heterogeneity. We expect that the explicit incorporation of decision-making processes in evolutionary models can shed light on various topics in the field of tumor ecology and evolution, such as the plasticity of metabolism, cell migration, and treatment resistance in the future.

## **William Duncan Martinson**

*Extracellular matrix remodelling by lead chick cranial neural crest cells is a major determinant of robust collective migration*

Collective cell migration plays an essential role in vertebrate development, yet the extent to which dynamically changing microenvironments influence this phenomenon remains unclear. Observations of the distribution of the extracellular matrix (ECM) component fibronectin during the migration of loosely connected neural crest cells (NCCs) lead us to hypothesize that NCC remodeling of an initially punctate ECM creates a scaffold for trailing cells, enabling them to form robust and coherent stream patterns. We evaluate this idea in a theoretical setting by developing an individual-based computational model that incorporates reciprocal interactions between NCCs and their ECM. ECM remodeling, haptotaxis, contact guidance, and cell-cell repulsion are sufficient for cells to establish streams *in silico*, however, additional mechanisms, such as chemotaxis, are required to consistently guide cells along the correct target corridor. Further model investigations imply that contact guidance and differential cell-cell repulsion between leader and follower cells are key contributors to robust collective cell migration by preventing stream breakage. Global sensitivity analysis and simulated gain- and loss-of-function experiments suggest that long-distance migration without jamming is most likely to occur when leading cells specialize in creating ECM fibers, and trailing cells specialize in responding to environmental cues by upregulating mechanisms such as contact guidance.

## **Axel Voigt**

*Fluid deformable surfaces*

Biological membranes, the cellular cortex or epithelia tissue are soft materials which show a solid-fluid duality. They store elastic energy when stretched or bent (as solid shells), but under in-plane shear, they flow as viscous two-dimensional fluids. We model this behaviour, solve the resulting system of surface PDEs numerically and discuss the biological implications of the solid-fluid duality.

## Tommaso Lorenzi

### *Spatial spread and evolutionary dynamics of heterogeneous cell populations: insights from partial integro-differential equation models*

In this talk, partial integro-differential equation models describing the spatial spread and evolutionary dynamics of heterogeneous cell populations will be considered. In these models, a continuous structuring variable captures intercellular variability in cell proliferation and migration rates. A formal derivation of such deterministic, continuum models from corresponding stochastic, individual-based models will be carried out, analytical and numerical results summarising the behaviour of the solutions to the model equations will be presented, and the insights generated by these results into the mechanisms that underpin collective cell migration will be briefly discussed.

## Benjamin M. Friedrich

### *A mechanism of branching morphogenesis inspired by diatom silica formation*

The formation of minerals by living organisms is a widespread biological phenomenon occurring throughout the evolutionary tree-of-life. The silica-based cell walls of diatom microalgae are impressive examples due to their intricate architectures and outstanding materials properties that still defy their reconstitution in vitro. Recent advances in imaging nascent diatom silica allow rationalizing possible mechanisms of their pattern formation, providing new recipes for bottom-up mineral synthesis.

We present a minimal mathematical model that explains the formation of branched patterns of silica ribs - a fundamental feature of the silica cell wall. We propose that silica deposition releases an inhibitor that slows down up-stream precursor conversion, thereby implementing a self-replicating reaction-diffusion system, akin to a non-classical Turing mechanism. The proposed mechanism highlights the role of geometrical cues for guided self-organization, providing a rationale for the presence of a single initial pattern seed, known as primary silicification site in diatoms.

The proposed model provides a generic mechanism of branching morphogenesis where communication between the branches is mediated chemically via concentration of released inhibitor. This provides a kinetic realization for a widely used phenomenological rule that tips start to branch whenever the distance to their neighbors exceeds a critical threshold, as employed in previous models of branching morphogenesis. The mechanism of branching morphogenesis characterized here is possibly generic and may apply in other biological systems, such as corals, bacteria biofilms or bronchial trees.

Joint work with Iaroslav Babenko and Nils Kröger.

The Swift-Hohenberg (SH) model is a mathematical framework used to study pattern formation in physical systems. It is represented by a partial differential equation that describes the evolution of a scalar field. This equation may be obtained as a non-conservative (L2) gradient flow of an energy functional, while the conservative (H-1) gradient flow of the same energy is often referred to as phase field crystal (PFC) model with the order parameter embodying essential properties microscopic densities in ordered systems. We begin with an overview of the main aspects of these models, as well as examples of their application to describe ordered systems. We then discuss the description of defects in such systems, introducing a unified field theory of topological defects and non-linear local excitations, which can be generally applied to smooth order parameters of different kinds. Finally, we consider the framework delivered by SH or PFC models to describe hyperuniform systems which feature long-wavelength fluctuations as in an ordered system (e.g., crystals) but without translational/orientational order.



# Locations

## Guest house: Gästehaus am Weberplatz, Weberplatz 3

Website: <https://www.gaestehausweberplatz.de/weberplatz/information-in-english>

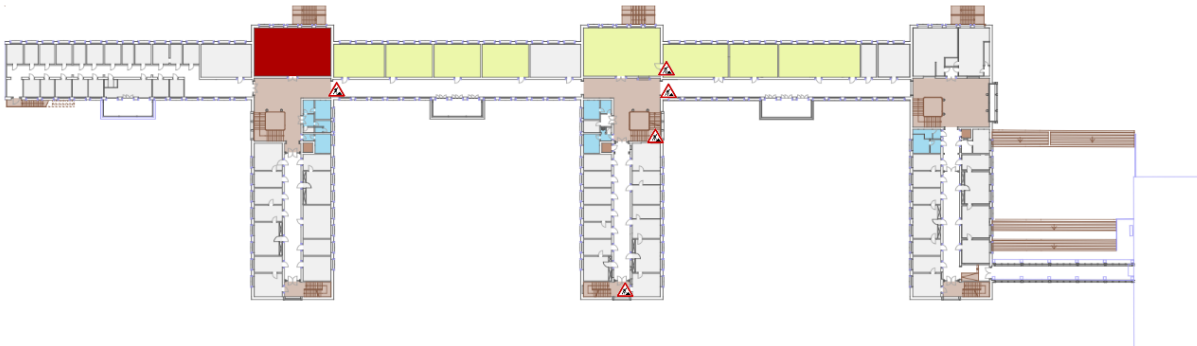
Google Maps: <https://goo.gl/maps/pbrTAKpyYe9MoyeZ6>

Front of the Guesthouse:

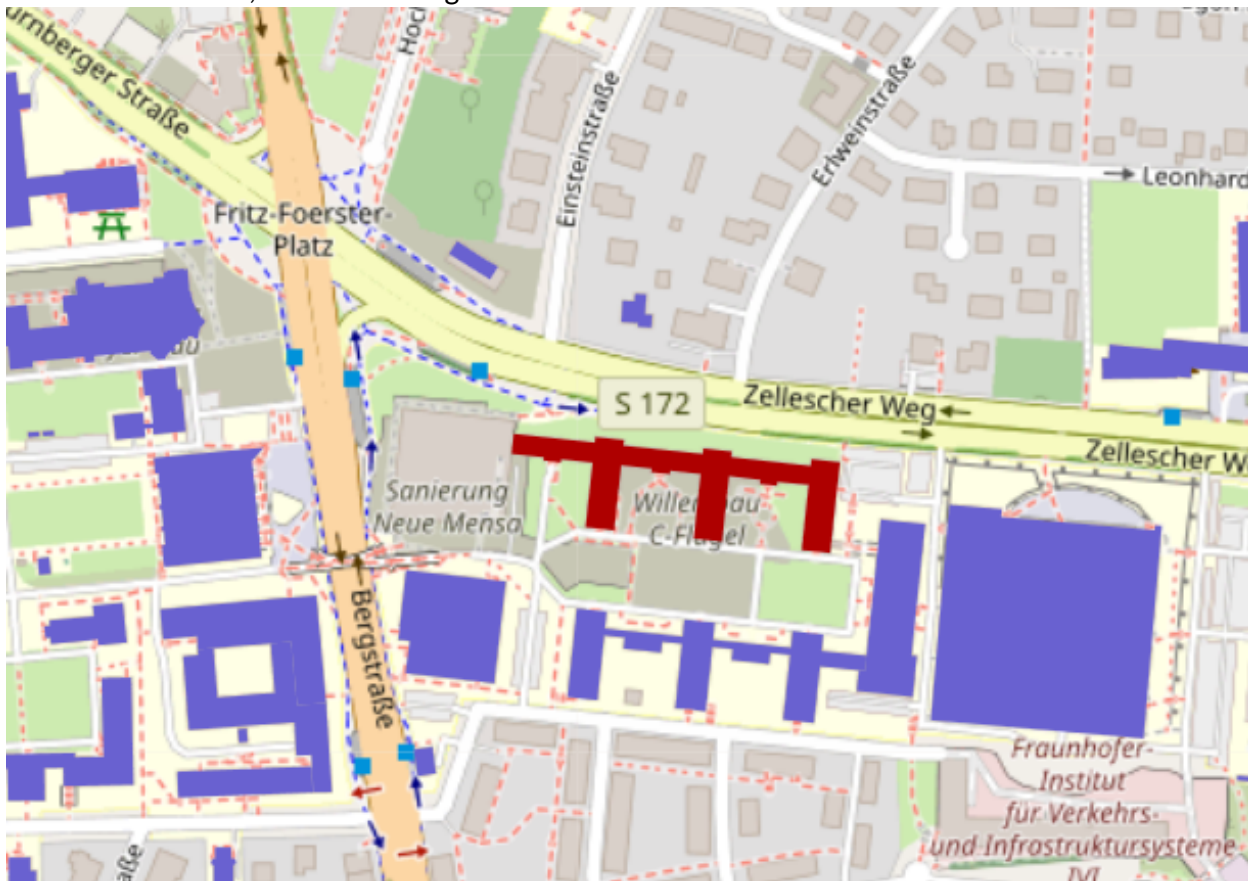


## C207, Willers-Bau Haus C, Zellescher Weg 12 - 14

Room C207, 2nd floor:



Willers-Bau Haus C, Zellescher Weg 12 - 14:



Coordinates to enter the building: 51.029061, 13.732934



## Library, Room 250, Zellescher Weg 25

Library, 2nd floor:



Zellescher Weg 25:



Coordinates to enter the building: 51.028950, 13.743255

## Dinner and Wine Tour

Augustiner an der Frauenkirche: An d. Frauenkirche 16-17, 01067 Dresden

Google Maps: <https://goo.gl/maps/wkEZP4yDQiRfuCJ8A>

PlanWirtschaft: Louisestraße 20, 01099 Dresden

Google Maps: <https://goo.gl/maps/Tw5dTyoQjjsBn8XL8>

Wine Tasting: Schloss Wackerbarth, Wackerbarthstraße 1, 01445 Radebeul

Google Maps: <https://goo.gl/maps/RJQYDTbtmBoFYx168>