Snapshots of modern mathematics
from Oberwolfach

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Emergence in biology and social sciences

Franca Hoffmann[1] • Sara Merino-Aceituno[2]

Mathematics is the key to linking scientific knowledge at different scales: from microscopic to macroscopic dynamics. This link gives us understanding on the emergence of observable patterns like flocking of birds, leaf venation, opinion dynamics, and network formation, to name a few. In this article, we explore how mathematics is able to traverse scales, and in particular its application in modelling collective motion of bacteria driven by chemical signalling.

1 Emergence: the big mystery

1.1 A gap in the scientific knowledge

Emergent phenomena are ubiquitous in nature: they correspond to the appearance of large-scale structures in underlying microscopic dynamics. At the microscopic level particles or agents interact following some rules, but as the macroscopic structures are not encoded directly into these rules it is a challenge to explain how the macroscopic or observable dynamics emerge from the microscopic dynamics (see Figure 1). Examples of emergence include

[1] Hausdorff Center for Mathematics, University of Bonn, Endenicher Allee 62, 53115 Bonn, Germany.
[2] Faculty of Mathematics, University of Vienna, Oskar-Morgenstern-Platz 1, 1090 Vienna, Austria.

collective dynamics (flocks of birds, school of fish, pedestrians, cell dynamics ...) [3, 8, 9, 10, 16, 18], network formation (capillary formation [1], leaf venation [13], formation of gullies, ant trails [6], ...), opinion dynamics [12], tumour growth [22], sperm dynamics and fertility [11], and tissue development [23].

Understanding emergence in science is central to explaining how observable phenomena arise. However, experimental techniques tend to be limited to one given scale (large or small) and in general it is not possible to experimentally study the link between the different scales.[3] That's where mathematics plays a crucial role. The mathematical tools for studying emergence come mainly from kinetic theory, originally developed to study problems in mathematical physics in the field of gas dynamics. The application of this mathematical framework to explore systems coming from biology and sociology poses many new and interesting challenges at the level of the modelling and mathematical analysis (partial differential equations and probability theory).

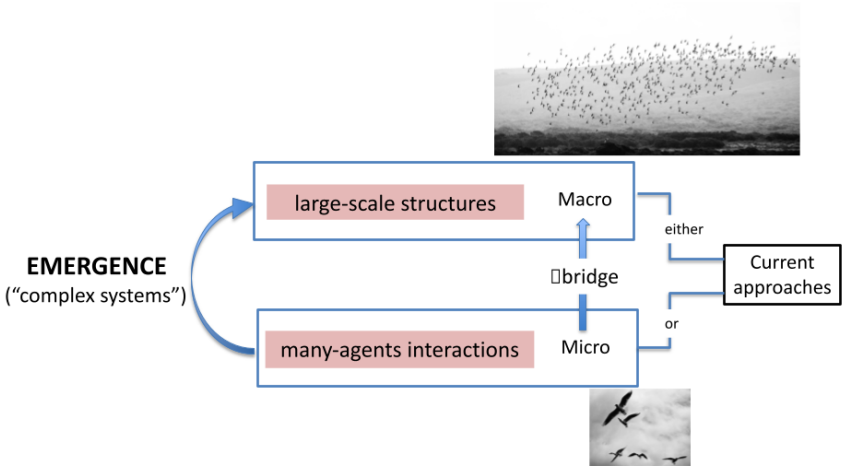

Figure 1: Schematic concept of emergence: macroscopic structures (like flocking) arise from underlying microscopic dynamics (interactions between individual birds). It is not obvious how the dynamics of the individual birds give rise to the large-scale patterns of flocking.

[3] For an interesting video showing the different scales in the universe see *Powers of ten* produced by Eames Office: https://www.youtube.com/watch?v=0fKBhvDjuy0

1.2 Our understanding of the world is "layered"

Scientific knowledge corresponds to a given "level of description" or scale. For example, to investigate the functioning of a cell, one can study the cell at a scale where the cell can be seen: how it grows, moves, divides and interacts with its environment. Alternatively, one can also look at the cell at a lower scale studying the composition of each part of its organelles, their chemical composition, mechanical properties and the relation between each. Or, one can consider a much larger scale where the cell is part of a given tissue. At this larger scale, one would like to know how this cell is affected and affects the tissue.

Each one of these levels of description (the molecular, the cellular, the tissue), and how they are linked, is necessary to fully understand the functioning of a cell. By traversing scales, we are able to answer questions like: how does the structure and functionality of a tissue arise? Answering questions about emergent phenomena is not easy: understanding the behaviour of a single cell is not sufficient to predict the behaviour of cell aggregates or tissue. Most of the approaches used so far to study problems in biology and social sciences do not focus on understanding the emergence of observable phenomena from the underlying microscopic structure, since this is extremely challenging.

2 How does maths help to explain emergent phenomena?

2.1 Reducing information to gain understanding

Sometimes, having a lot of information on a system does not increase our understanding of it. Suppose that you knew exactly what each individual in your country was going to vote in the next elections. That is a lot of information! You could list one by one the voting intentions of everyone. However, listing one by one the voting intentions of everyone (microscopic information) does not yield any useful information. What we would like to know typically is the percentage of votes each party gets ("kinetic" information) or the average vote in the different regions (macroscopic information). Such an analysis would provide us with meaningful information, that is to say, information we can both understand and utilize. This is exactly the idea of going from microscopic to macroscopic dynamics: to reduce information in a meaningful way.

Consider another example, that of opinion dynamics. Suppose we encode the opinions of individuals on given topic by assigning a value from the set $\{-1, 1\}$. A list of everyone surveyed containing their name, the neighbourhood in which they live and their opinion would be the microscopic information in this system. We can then organise it into a kinetic description, where we lose the information on the particular individuals, but we learn the proportion of

people with a given opinion in any neighbourhood. We can define a kinetic function f as follows:

$$f(\text{neighbourhood } Y,\ \text{opinion } x) = \frac{\text{number of people with opinion } x \text{ in neighbourhood } Y}{\text{total number of people}}.$$

For the macroscopic description we could instead ask for the average of all opinions in each neighbourhood, by dividing the sum of the opinions by the number of people in a neighbourhood. The reduction of information presented in this example looks very simple, but things become more complicated when we consider that people interact among each other and that their opinions evolve over time.

The original example was the study of gas dynamics. Here, at the microscopic scale we have Newton's law governing the position and velocity of each gas molecule; at the kinetic level we have Boltzmann's equations for gas dynamics; and at the macroscopic level we have the Navier-Stokes equations for fluid dynamics [26]. By deriving macroscopic equations from microscopic ones we validate the macroscopic equations and at the same time we understand how macroscopic phenomena arise. Through the process of deriving macroscopic equations we lose information, but we gain understanding!

2.2 Useful modelling and the scientific method

The starting point of all this mathematical process is, of course, a microscopic model. At the microscale we will have particles or agents characterised by some given properties (like the position and velocity of a particle or the voting intention of a person) that interact following some rules. At this point we need to model the system as simply as possible so that we capture what we are interested in investigating or the hypothesis that we want to test. Simplicity is important as very complicated models are neither controllable (there are too many variables and we lose sight of what is influencing what), nor tractable (the mathematical analysis would become too complicated). Therefore, finding a good modelling framework is a critical first step in this analysis, and when it comes to biological or social systems, this challenge defers substantially from physical systems.

The primary complication is that there do not seem to be first principles in biology and sociology as there are in classical mechanics (think of Newton's laws). This is very clear when we consider social interactions. What universal laws govern opinion dynamics, pedestrian dynamics, the collective motion of birds, and so on? How do we know that they will not differ between cultures, species or even two individuals? However, we can still do a lot by focussing

on "heuristic rules" to mathematically describe a phenomenon observed at the microscale without worrying about the particular mechanisms by which it occurs. Typically, these heuristic rules become hypotheses to be tested; for example, in [23] the authors hypothesize that mechanical factors between cells and fibres are enough to explain observable configurations in adipose (fat) tissue. We aim to test this by first developing a microscopic model for cells and fibres, and then using mathematical scaling techniques to derive a macroscopic model that enables us to study the emergence of adipose tissue. In this way, the mathematical connection between the scales allows one to establish a rigorous link between the suggested microscopic cause of a macroscopic phenomenon.

3 From micro to macro: an example in biology

3.1 Bacterial chemotaxis

One of the most important abilities that living beings have is to adapt themselves to the environment that surrounds them. Notwithstanding their adaptability, the survival of any life form, from the most microscopic bacteria to the largest mammals, strongly depends upon its capacity to understand whether an environment is suitable and to migrate towards better places if it is not. This could be for reasons of food availability, to avoid predators or to regroup into more efficient colonies.

This phenomena is evident in multicellular organism, but even many bacteria, such as *Escherichia Coli*, *Rhodobacter Sphaeroides* and *Bacillus Subtilus*, are able to respond to extracellular changes in their surrounding environment. Here we concentrate on *E. Coli*, because its biochemistry and movement are now rather well understood.

There are several reasons why bacteria seek to move to a different location; one of the most fundamental motivations being the search for food. Additionally, many types of micro-organisms prefer to stay together as a group to increase their chances of survival. So how is a single cell without eyes and ears able to determine where the other members of the colony are, or where to go to find nutrients? Questions like these are best answered at the micro scale. Cells such as *E. Coli* have receptors on their outer membrane that allow them to detect the change in concentration of chemical substances in their environment. This enables them to move towards attractants or away from repellents by means of a biased "random walk" (we will get back to this shortly). Generally speaking, the directed movement of cells and organisms in response to chemical gradients is called *chemotaxis*.

In the past 20-30 years, this mechanism has been an area of increasing interest for applied mathematicians as the cooperation between experts in both

mathematics and biology has allowed fruitful refinements to well-established models whose weaknesses had been accepted by many researchers for the benefit of their great simplicity and universality. From the mathematical viewpoint, research at the interface of these two subjects is especially interesting as it serves both the comprehension of biological phenomena and the development of mathematical tools that find application in a wide range of other subjects.

3.2 Macroscopic bacterial dynamics

Historically, the Keller–Segel model [25, 21] has been one of the principal approaches to describe bacterial motion mathematically. First introduced in [20] (1953) and [15] (1970) to describe aggregation of slime mold amoebae, this model has become one of the most widely studied models in mathematical biology. It is a system of equations that describes the macroscopic density of cells $\rho(t, x)$ at location x and at time t; it encodes how $\rho(t, x)$ changes due to the presence of nutrients and the chemoattractant[4] produced by the cells themselves. These three quantities – the density of cells, the concentration of nutrients, and the chemoattractant concentration – interact in a non-trivial way. Cells move around towards high concentrations of nutrients, and towards the majority of the rest of the cell population; the nutrient is consumed by the cells locally, and the chemoattractant both degrades and is produced by the cells locally. In addition, all three quantities diffuse at different speeds that can be measured experimentally. Together, these factors result in complicated dynamics. The Keller–Segel model tries to encapsulate, approximately, the dynamics when looking at the bacterial population as a whole. A number of more refined models for the collective behaviour of cell populations have been developed since, mostly inspired by the pioneering work of Patlak, Keller, and Segel. These models have helped to understand important characteristics of bacterial chemotaxis, but they have certain limitations. In particular, they rarely allow a detailed comparison with the underlying complex microscopic behaviour, and turn out to be less accurate in certain settings. For example, with the classical Keller–Segel model, it is possible that the solution $\rho(t, x)$ concentrates on a single point after a finite amount of time [5, 4], meaning that all cells move to the same location. In practice however, this is not possible as one bacteria cannot be exactly in the same point as another. Several approaches exist to refine the model to be more realistic, such as introducing terms in the equations that prevent overcrowding [7, 14].

Another example where the classical Keller–Segel model turns out not to be a suitable model is when we aim to describe cell populations moving towards a

[4] We refer here to the "chemoattractant" both as the chemical substance that is produced by the cells and that they are attracted to.

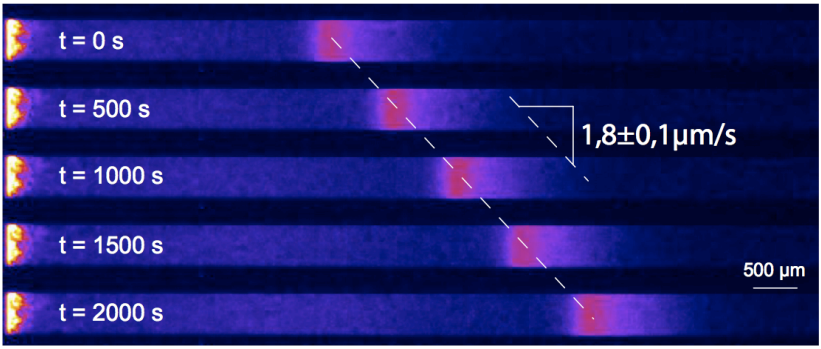

Figure 2: *E. Coli* travelling pulses: the band speed is constant and the profile is asymmetric. Source: [24], p2.

given food source. The seminal paper analysing *E. Coli*'s chemotaxis is Adler's *Chemotaxis in Bacteria* [2] where he experimentally demonstrates that motile strains of *E. Coli* respond to nutrient presence (oxygen, galactose, glucose etc.) by collective motion towards high concentration regions of these substances. The resulting moving bands are commonly referred to as travelling waves.

The travelling waves observed by Adler have later been reproduced by others, see for example the experiment described in [24] of a bacterial suspension in a one dimensional channel filled with a nutrient (Figure 2). These observations are in contradiction with predictions given by the Keller–Segel model, which does not allow for travelling waves to occur at all. In other words, functions $\rho(t, x)$ of a shape that would describe moving bands of bacteria are not solutions of the equations. As these moving bands are observed in practice however, this is an indication that the Keller–Segel model is over-simplified in certain settings, and fails to capture crucial dynamics of the system. To understand what is going on and which mechanisms are responsible for travelling waves to occur, one has to look at the microscopic scale.

If we understand the behaviour of a single cell in response to its environment, we can then develop a corresponding kinetic description. On the kinetic level, we do not expect to observe large scale phenomena such as travelling waves. This is where mathematical scaling techniques play a crucial role: they allow us to connect the kinetic model to a macroscopic model, and ultimately reveal the driving forces on the cellular level that are responsible for macroscopic patterns such as travelling wave to arise.

3.3 Microscopic bacterial dynamics

As mentioned above, when observing *E. Coli* under the miscroscope, it looks as if the cells perform a "random walk". This means that they travel for some time in a given direction and then "randomly" change to another direction of travel, and so on. If this was true, how then is it possible that we observe groups of cells moving at a constant speed towards the food source as in Figure 2?

By now, we understand much more about the behaviour of a single *E. Coli* cell. Motile strains of *E. Coli* swim by the mean of several flagella assembled into a coherent rotating bundle which turns counter-clockwise propelling the bacterium forward. In absence of any external stimulus *E. Coli* in fact do perform a random walk, alternating between two phases: *runs* and *tumbles*. During runs a cell undergoes a smooth straight line swim for about one second. Then it performs a completely random change of direction, a tumble, caused by the change of the rotational direction by one or more flagella.

Since individual bacteria are generally 1-3 μm in length, they are too short to detect changes in chemical concentration along their body, which is why they use a temporal mechanism instead. More precisely, they compare the average number of bound receptors over the past 1 second with their average number during the past 3 seconds [17]. In order to stay close to the majority of the bacterial population and to reach nutrients, a cell therefore simply tumbles more often if it is going in a bad direction, that is, away from the group/food source, and less often if it is going in a good direction, that is, towards the group/food source. On average, this will generate a movement in the desired direction. Hence, on the microscopic level, chemotaxis is a consequence of the fact that sensing the variation in a present chemical concentration biases the tumbling frequency, causing a *biased random walk*. Mathematically, we want to be able to build a rigorous link between the travelling waves observed on the macroscopic scale and this microscopic tumbling mechanism.

3.4 Kinetic framework

A kinetic model of the bacterial population describes the density of cells $f(t, x, v)$ located at position x, at time t and swimming with velocity v. The term "kinetic" here refers to the fact that we not only track the position of cells, but also their velocities. Taking inspiration from the kinetic theory of gases, we think of each cell as a gas particle, and instead of a physical model for collisions between gas molecules, we need instead a biological model for the "collision" of cells, that is, how a single cell responds to the presence of another. We can encode this behaviour in what is called a communication function, using our knowledge about the microscopic behaviour of a single cell. To do this, we adapt the

Boltzmann equation modelling gas dynamics for the purpose of modelling the kinetic dynamics of *E. Coli* [19].

In order to explain this equation, we need to introduce some more notation. First of all, we let the function $S(t,x)$ be the concentration of chemoattractant produced by the cells at a given time t and position x, and we let $N(t,x)$ denote the concentration of nutrients, also at time t and position x. We define the probability that a cell travelling at velocity v changes direction to a new velocity v' to be given by the function $\mathbf{T}_{S,N}(v,v')$, and summing over all possible new velocities v' gives the probability for a cell travelling at velocity v to tumble:

$$\lambda_{S,N}(v) = \int \mathbf{T}_{S,N}(v,v')\,dv'.$$

Note that these probability functions only depend on the functions S and N defined above. Now, we obtain the following equation:

$$\underbrace{\partial_t f + v\cdot\nabla_x f}_{run} = \underbrace{\int \mathbf{T}_{S,N}(v',v)f(t,x,v')\,dv' - \lambda_{S,N}(v)f(t,x,v)}_{tumble}\,. \qquad (1)$$

This equation expresses mathematically that we assume changes in the distribution of cells occur due to only two mechanisms: (i) bacteria changing position during a run phase, and (ii) bacteria changing velocity due to a tumble event. The left-hand side of the equation encodes the change in cell density along the trajectory of a cell at point x travelling with velocity v; this term represents the run phase of the bacterial motion. The term "$\partial_t f$" denotes the rate of change of the density function $f(t,x,v)$ with respect to time, and the term "$\nabla_x f$" denotes the rate of change of the density with respect to the position vector x. Note here that the velocity is not changing. On the right-hand side, we account for the change in $f(t,x,v)$ due to tumbling: we add all the cells that change direction from any other velocity to velocity v, and then subtract all the cells that tumble at velocity v to change to some other new velocity. Since the overall mass of cells is assumed to remain the same during this process, the changes due to runs have to be equal to the changes due to tumble events, resulting in the equation as stated.

3.5 Zooming-out to see the bigger picture

Once we have an accurate kinetic model, we can draw a mathematical connection to the macroscopic scale where large-scale patterns can be observed. The rigorous connection between the kinetic description and the macroscopic equations can be established by mathematically "zooming out" to change the viewpoint from

micro to macro[5]. In other words, we move from a scale where we observe the speed of a single cell, to a new, coarser scale, where we observe the speed of a whole group of cells in a travelling wave. We expect the ratio between these speeds to be small as a result of the fact that a single cell makes many turns in different directions when moving within the group, even if the group at a macroscopic level is moving at a constant speed in a straight line. The speed of the macroscopic travelling wave is then necessarily slower than the speed of the individual cells, as it results from an average of the microscopic movements. Experimental evidence indeed confirms that the bulk velocity of a moving bacterial wave is much lower than the speed of a single bacterium. More precisely, they differ by only one order of magnitude, as observed in the micro-channel experiment in [24]. So, we rescale time and space in our kinetic model (1) in such a way that the ratio between the macroscopic speed of a travelling wave and the microscopic speed of a single cell vanishes:

$$\frac{\text{speed of a macroscopic travelling wave}}{\text{speed of a single bacterium}} \to 0\,.$$

Sending this ratio to zero (when in reality it is small), provides an approximation of the dynamics when we are only interested in the macroscopic patterns. More precisely, making this simplification will reduce the complexity of the equation after going step by step through a mathematical limiting procedure, and will allow us to focus on changes in time and position only, neglecting changes in velocities. By changing our modelling framework, we lose some information (here, on the distribution in terms of microscopic velocities), and gain instead information on the bigger picture (here, being able to mathematically capture the macroscopic travelling wave).

This rescaling allows us to derive a new macroscopic model for the bacterial cell population, now interpreted as the kinetic cell density at location x summed over all possible velocities:

$$\rho(t,x) = \int f(t,x,v)\,dv\,.$$

In contrast to the classical Keller–Segel model, the equation we obtain by this procedure takes the microscopic dynamics into account; it also turns out to be much more complicated: the term describing the drift of the cell population due to the presence of the nutrient and the chemoattractant is *nonlinear* [24]. This means that the term is raised to at least a power of 2, and as a consequence, it is much harder to find an explicit solution for this model.

Linearizing this term as a simple approximation, the equation reduces to the classical Keller–Segel model. This explains why the Keller–Segel equations

[5] Technically, this is called *hydro-dynamic scaling*.

provide a sufficient model for bacterial chemotaxis on the macroscopic scale in most cases (when the linear terms represent a good approximation), and also why it fails to capture certain phenomena such as travelling waves (because it neglects the nonlinear dynamics that give rise to these patterns).

The bacterial chemotaxis models described here are just one example, illustrating how mathematical scaling techniques enable us to understand emerging behaviour at different scales. Multi-scale techniques enable us to test and identify which are the driving forces behind the dynamics we observe, and to make sure we work with the correct models to capture the key phenomena we are interested in.

4 A unique contribution of mathematics to science

This is an exciting time to be a mathematician working in applications of kinetic theory. It has been with the help of abstract thinking, in particular, mathematics, that we have been able to understand and write the laws of physics in simple, understandable and useful forms. The paramount example of this are Newton's laws. Now, mathematics is providing again a unique contribution to science: to bypass the limitations of experimental sciences and establish the links between the different layers of knowledge to explain how the world that we experience emerges from its underlying microscopic structure.

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Fig. 1 Diagram created by the authors using stock photos from the website https://www.pxfuel.com/.

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References

[1] P. Aceves-Sanchez, B. Aymard, D. Peurichard, P. Kennel, A. Lorsignol, F. Plouraboue, L. Casteilla, and P. Degond, *A new model for the emergence of blood capillary networks*, Networks and Heterogeneous Media **16** (2021), no. 1, 91–138.

[2] J. Adler, *Chemotaxis in bacteria*, Science **153** (1966), no. 3737, 708–716.

[3] N. Bellomo and J. Soler, *On the mathematical theory of the dynamics of swarms viewed as complex systems*, Mathematical Models and Methods in Applied Sciences **22** (2012), no. supp01, 1140006.

[4] A. Blanchet, J. A. Carrillo, and P. Laurençot, *Critical mass for a Patlak–Keller–Segel model with degenerate diffusion in higher dimensions*, Calculus of Variations and Partial Differential Equations **35** (2009), no. 2, 133–168.

[5] A. Blanchet, J. Dolbeault, and B. Perthame, *Two-dimensional Keller-Segel model: optimal critical mass and qualitative properties of the solutions*, Electronic Journal Differential Equations **44** (2006), 1–33.

[6] E. Boissard, P. Degond, and S. Motsch, *Trail formation based on directed pheromone deposition*, Journal of Mathematical Biology **66** (2013), no. 6, 1267–1301.

[7] V. Calvez and J. A. Carrillo, *Volume effects in the Keller-Segel model: energy estimates preventing blow-up*, Journal de Mathématiques Pures et Appliquées **86** (2006), no. 2, 155–175.

[8] J. A. Carrillo, Y.-P. Choi, and S. P. Perez, *A review on attractive–repulsive hydrodynamics for consensus in collective behaviour*, Active Particles, Volume 1, Springer, 2017, pp. 259–298.

[9] F. A. C. C. Chalub, P. A. Markowich, B. Perthame, and C. Schmeiser, *Kinetic models for chemotaxis and their drift-diffusion limits*, Nonlinear Differential Equation Models, Springer, 2004, pp. 123–141.

[10] P. Degond, A. Frouvelle, J.-G. Liu, S. Motsch, and L. Navoret, *Macroscopic models of collective motion and self-organization*, Séminaire Laurent Schwartz—EDP et Applications (2012), 1–27.

[11] P. Degond, S. Merino-Aceituno, F. Vergnet, and H. Yu, *Coupled self-organized hydrodynamics and stokes models for suspensions of active particles*, Journal of Mathematical Fluid Mechanics **21** (2019), no. 1:6.

[12] B. Düring and M.-T. Wolfram, *Opinion dynamics: inhomogeneous Boltzmann-type equations modelling opinion leadership and political segregation*, Proceedings of the Royal Society A: Mathematical, Physical and Engineering Sciences **471** (2015), no. 2182.

[13] J. Haskovec, P. Markowich, B. Perthame, and M. Schlottbom, *Notes on a PDE system for biological network formation*, Nonlinear Analysis **138** (2016), 127–155.

[14] T. Hillen and K. J. Painter, *A user's guide to PDE models for chemotaxis*, Journal of Mathematical Biology **58** (2008), no. 1, 183.

[15] E. F. Keller and L. A. Segel, *Initiation of slime mold aggregation viewed as an instability*, Journal of Theoretical Biology **26** (1970), no. 3, 399–415.

[16] B. Maury and J. Venel, *A mathematical framework for a crowd motion model*, Comptes Rendus Mathématique **346** (2008), no. 23-24, 1245–1250.

[17] N. Mittal, E. O. Budrene, M. P. Brenner, and A. van Oudenaarden, *Motility of E. Coli Cells in Clusters Formed by Chemotactic Aggregation*, Proceedings of the National Academy of Sciences (2003), 13259–13263.

[18] G. Naldi, L. Pareschi, and G. Toscani, *Mathematical modeling of collective behaviour in socio-economic and life sciences*, Springer Science & Business Media, 2010.

[19] H. G. Othmer, S. R. Dunbar, and W. Alt, *Models of dispersal in biological systems*, Journal of Mathematical Biology **26** (1988), no. 3, 263–298.

[20] C. S. Patlak, *Random walk with persistence and external bias*, The bulletin of mathematical biophysics **15** (1953), no. 3, 311–338.

[21] B. Perthame, *Transport equations in biology, chapter 5*, Frontiers in Mathematics, Birkhäuser Verlag, Basel, 2007.

[22] B. Perthame, F. Quirós, and J. L. Vázquez, *The Hele–Shaw asymptotics for mechanical models of tumor growth*, Archive for Rational Mechanics and Analysis **212** (2014), no. 1, 93–127.

[23] D. Peurichard, F. Delebecque, A. Lorsignol, C. Barreau, J. Rouquette, X Descombes, L. Casteilla, and P. Degond, *Simple mechanical cues could explain adipose tissue morphology*, Journal of Theoretical Biology **429** (2017), 61–81.

[24] J. Saragosti, V. Calvez, N. Bournaveas, A. Buguin, P. Silberzan, and B. Perthame, *Mathematical description of bacterial travelling pulses*, PLoS Computational Biology **6** (2010), no. 8.

[25] M. J. Tindall, P. K. Maini, S. L. Porter, and J. P. Armitage, *Overview of mathematical approaches used to model bacterial chemotaxis. ii. bacterial populations*, Bulletin of Mathematical Biology **70** (2008), no. 6, 1570–1607.

[26] C. Villani, *A review of mathematical topics in collisional kinetic theory*, Handbook of mathematical fluid dynamics **1** (2002), no. 71-305, 3–8.

Franca Hoffmann *is a Junior Professor at University of Bonn.*

Sara Merino-Aceituno *is Associate Professor at the Faculty of Mathematics at the University of Vienna.*

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