# Workshop: Tissue growth and movement

**Week 10-14th January 2022**
**(IHP, Paris)**

# Titles and abstracts

**Ardaseva Aleksandra** (University of Copenhagen)

**Bridging microscopic cell dynamics to nematohydrodynamics of cell monolayers**

**Abstract:** The emergence of liquid-crystalline features is increasingly realised in different cell monolayers. Here, we present a cell-based model of cell layer, based on phasefield formulation, that connects mechanical properties at the single cell level to the multiscale nematic and hydrodynamic properties at the tissue level. In particular, we present a minimal formulation that reproduces the well-known bend-splay hydrodynamic instabilities in the continuum nematohydrodynamic formulation of active matter, which is complemented by analytical description of the instability threshold in terms of activity and elasticity of the cells. Furthermore, we provide a quantitative characterisation and comparison of flows and topological defects for extensile and contractile stress generation mechanisms, and demonstrate the emergence of spontaneous gap formation within a confluent monolayer as a consequence of the interplay between activity and elasticity of the cells. Together, the results contribute to bridging the gap between micro-scale cell dynamics and tissue-scale collective cellular organisation.

**Bailleul Richard** (EMBL Heidelberg and University of Geneva)

**Deciphering bio-mechanical drivers of morphological diversity in early animal development**

**Abstract:** The development of an embryo occurs through the succession of timely orchestrated processes. Early-occurring mechanisms are critical in laying the foundations for all downstream developmental events, which progressively define the morphology of the animal that should fit with its environment. How from initially similar shapes are formed the huge diversity of body plans we can observe in the animal kingdom remains largely a black box. With their simple body structure and symmetry, cnidarians (sea anemones, corals, jellyfish) have emerged as a great model system to study the evolution of morphogenesis. Throughout development, the embryos lose their sphericity and transform into swimming larvae as they elongate along their axis of symmetry. The shapes of the larvae that result from this elongation process vary drastically between cnidarian species. In this talk, I will compare the transitions in shape, cell mechanics, and gene expression that occur during larval morphogenesis between two sea anemone species. Using a theoretical framework based on the continuum theory of active surfaces, I will reproduce shared and varying attributes of elongation dynamics between both species, and discuss from models predictions what early genetic and physical parameters drive shape changes in these animals.

**Ben Amar Martine** (Ecole Normale Supérieure and Sorbonne Université)

**Morpho-elasticity of thin living structures**

**Abstract:** Thin structures are ubiquitous in life mostly in botanic (flowers, leaves) but not only . In mammals or insects, they concern epithelia or the couple epithelium-extracellular matrix joined in a thin bilayer. The complexity of finite-elasticity with growth is then simplified, the main task being the adaptation of elasticity to the geometry of the system under study. I will present two different growing epithelia: first the cyst made by pluri-potent stem cells then the wing of the imaginal disc of genetically mutated drosophila. In the first case, the classical and simple model of a growing spherical shell reproduces perfectly the dynamics of proliferation of the stem cell assembly during more than 10 days with an extremely low level of stress. For the second case, I will show how the Von-Karman-Ciarlet modelling with growth explains the local collapse of the bilayer.

Ref.: 'Morpho-elasticity of human pluripotent stem cell cysts' Joseph Ackermann, Philippe J.R. Cohen, Kevin Alessandri, Andrea Leonard, Pierre Nassoy, Jean-François Joanny, Martine Ben Amar, preprint JMPS 2021

**Benzekry Sébastien** (Inria Sophia Antipolis–Méditerranée)

**Quantitative modeling of metastasis: cancer at the organism scale**

**Abstract:** In the majority of solid cancers, secondary tumors (metastases) are the main cause of death. Quantitative mathematical modeling could have important value to 1) test biological theories against empirical data and bring mechanistic insights and 2) design numerical tools predictive of metastatic relapse or the impact of therapies. I will present research efforts towards the establishment of such mathematical constructs descriptive and predictive of metastatic development. The general framework is based on a physiologically-structured partial differential equation for the time dynamics of a population of metastases. Results will be presented for model-based exploration of 1) spontaneous development of metastasis following surgery [1], 2) theoretical models of metastatic spread and growth [2], 3) tumor-tumor systemic interactions [3] as well as recent results modeling paradoxical pro-metastatic effects of anti-angiogenic therapies. If time allows, translation of the models will be presented in two clinical settings: brain metastasis from non-small cell lung cancer [4] and prediction of metastatic relapse in early-stage breast cancer [5]. Together, these results represent a step towards the integration of mathematical modeling for research on metastasis and as a predictive tool for personalized oncology.

[1] Benzekry, S. et al. Modeling Spontaneous Metastasis following Surgery: An In Vivo-In Silico Approach. Cancer Research 76, 535–547 (2016).

[2] Baratchart, E. et al. Computational Modelling of Metastasis Development in Renal Cell Carcinoma. PLoS Computational Biology 11, e1004626 (2015).

[3] Benzekry, S., Lamont, C., Barbolosi, D., Hlatky, L. & Hahnfeldt, P. Mathematical Modeling of Tumor-Tumor Distant Interactions Supports a Systemic Control of Tumor Growth. Cancer Research 77, 5183-5193 (2017).

[4] Bilous, M. et al. Quantitative mathematical modeling of clinical brain metastasis dynamics in non-small cell lung cancer. Scientific Reports 9, 13018–13 (2019).

[5] Nicolò, C. et al. Machine Learning and Mechanistic Modeling for Prediction of Metastatic Relapse in Early-Stage Breast Cancer. JCO Clinical Cancer Informatics 4, 259-274 (2020).

**Bruna Maria** University of Cambridge

**Phase separation in active Brownian particles**

**Abstract:** I will discuss models for active matter systems consisting of many self-propelled particles. These can be used to describe biological systems such as bird flocks, fish schools, and bacterial suspensions. In contrast to passive particles, these systems can undergo phase separation without any attractive interactions, a mechanism known as motility-induced phase separation. Starting with a microscopic model for active Brownian particles with repulsive interactions, I will discuss four possible macroscopic PDEs (ranging from a nonlocal model to a local cross-diffusion system). I will then present work concerning the stability and analysis of such models.

**Ciarletta Pasquale** (Politecnico di Milano)

**Mathematical models and tools for personalized medicine**

**Abstract:** In the first part, I will present the result of a research project in collaboration with Ospedale Neurologico Besta and Instituto IFOM in Milan funded by Associazione Italiana per la Ricerca sul Cancro. In this work, we have proposed a predictive math- ematical model and we have built simulation tools to describe the growth the most aggressive brain tumour, glioblastoma multiforme (GBM), using patient-specific data, together with its response to therapy, to assist medical doctors in optimiz- ing individual therapeutic strategies. The model has been calibrated and validated against the data collected by a clinical study performed at the Istituto Neurologico Besta on a cohort of 32 patients diagnosed with GBM that have been screened by the most advanced bio-imaging techniques during diagnosis, biopsy, surgery, adju- vant therapy and post-therapy follow-up. The computational tools use clinical data deriving from patient specific advanced Magnetic Resonance and Diffusion Tensor Imaging into to a mathematical model which is able to consider and exploit both chemical and mechanical phenomena driving GBM evolution. This multidis- ciplinary approach has proven to aid clinicians in the customization of therapeutic strategies in the new field of precision medicine and, in particular, of personalized neuro-oncology.
In the second part, I will present a mathematical model, based on pre-clinical observations performed at Ospedale San Raffaele in Milan, of tumor evolution in presence of adoptive cellular therapy, a type of immunotherapy that enhances the immune system natural response by means of genetic manipulations of autologous T cells. The diffuse-interface model is described by a Cahn-Hilliard type equation coupled with a reaction-diffusion equation for the local nutrient concentration, accounting for the interaction between engineered T cells and tumor cells through a Michaelis-Menten kinetics. The trafficking of engineered T cells into the tu- mor micro-environment is modeled through a reaction-diffusion-advection equation, while the spatio-temporal variation of the chemical signal is described by a reaction- diffusion equation. The model is solved numerically using the finite-element method, on a computational domain generated from the pre-clinical imaging data of mouse prostatic adenocarcinoma, and calibrated with data already available in literature. Finally, we performed numerical simulations in order to assess the efficacy of the therapy in combination with different modulations of the tumor microenvironment. The numerical results show how the local availability of engineered T cells modi- fies the tumor evolution, resulting in a slowed tumor growth or complete regression in four weeks after the treatment, in accordance with the experimental outcomes reported in literature.

**Collin Annabelle** (Bordeaux INP)

**Mathematical models of electroporation validated on medical data**

**Abstract:** Electroporation is a complex phenomenon that occurs when biological tissues are subjected to electric pulses. Electroporation makes it possible to either kill directly the cells in the target region (tumor) or to introduce molecules into living cells. However, one of the main limitation of using electroporation in the clinical routine comes from the technical difficulties raised by such therapies, in particular it is difficult to well determine the treated zone. Mathematical models and numerical simulations can be used either to better understand the effects of the electric field on the cells or to compute the distribution of the electric field allowing to provide a numerical evaluation of the treatment. In this context, the available medical data - volume and tumor heterogeneity extracted from experimental/medical imaging or measurements of the electric intensity that flows through needles - can be used to identify the model parameters through strategies of data assimilation. The full strategy will be illustrated on various examples.

**Etchegaray Christele** (University of Bordeaux)

**A fluid-based model for single-cell migration**

**Abstract:** In this talk, I will present a deterministic model of single-cell migration based on a fluid description of the intracellular actin cytoskeleton, and on its interaction with a molecular specie. The problem writes as a reaction-advection-diffusion for the molecular dynamics, and a Darcy law coupled with a Poisson problem for the fluid. A Dirichlet boundary condition on the fluid pressure accounts for both the molecular regulation and the active behavior of the cytoskeleton. The model accounts for both static and motile behaviors, as shown from mathematical analysis and numerical simulations. Furthermore, by adding stochastic fluctuations in the molecular advection term, the model provides numerical trajectories that share key-properties with their experimental counterparts. This is a joint work with Nicolas Meunier (LaMME, Évry) and Raphaël Voituriez (LJP, LPTMC, Sorbonne université).

**Girel Simon** (Université Côte d'Azur)

**A multiscale model of the CD8 T-cells immune response**

**Abstract:** Infection of an organism by a pathogen triggers the activation of the CD8 T-cells and the initiation of the immune response. The result is a complex program of proliferation and differentiation of the CD8 T-cells, controlled by the evolution of their molecular content. I will introduce two mathematical models of the CD8 T-cell response. The first one is presented as an impulsive differential equation by which we study the effect of unequal molecular partitioning at cell division on the regulation of molecular heterogeneity. The second one is an agent-based-model that couples the description of a discrete population of CD8 T-cells and that of their molecular content. This model can reproduce the different typical phases of the CD8 T-cell response at both the cellular and the molecular scales. These two studies support the hypothesis that the cell dynamics observed in vivo is a consequence of the molecular heterogeneity structuring the CD8 T-cell population.

**Goudon Thierry** (University Côte d'Azur)

**A PDE model describing the immune cells-tumor growth interactions.**

**Abstract:** We introduce a mathematical model intended to describe by means of a system of partial differential equations the early stages of the interactions between effector immune cells and tumor cells. The model is structured in size and space: on the one hand the tumor development is governed by growth and cell division mechanisms, and, on the other hand the immune cells, activated by the presence of the tumor, are directed towards the tumor micro-environment by chemotactic mechanisms. Remarkably, the model exhibits a possible control of the tumor growth by the immune response; nevertheless, the control is not complete in the sense that the asymptotic states keep residual tumors and activated immune cells, a situation corresponding to the equilibrium phase predicted by biologists.The equilibrium state can be interpreted in terms of an eigenvalue problem coupled to a constrained drift-diffusion equation, an interpretation which is fruitful to set up numerical approaches in order to investigate the features of the equilibrium.
The model can be extended to take into account protumor effects of the immune response which can be very important to switch to the escape phase, with an uncontrolled growth of the tumor. Such a modeling can be useful to guide the design of combined immunotherapy strategies.

**Grasselli Maurizio** (Politecnico di Milano)

**Nonlocal Cahn-Hilliard-Hele-Shaw systems**

**Abstract:** In a two-dimensional Hele-Shaw cell, provided that the viscous forces dominate the inertial ones, the well-known Navier-Stokes-Cahn-Hilliard system for an incompressible binary flow can be approximated by the so-called Cahn-Hilliard-Hele-Shaw (CHHS) system. In three dimensions, the CHHS system is used to describe fluid flow in a porous medium as well as it is a cornerstone of solid tumor growth modeling through diffuse interfaces. I intend to present some recent results on a nonlocal CHHS system characterized by degenerate mobility, singular potential, and nonconstant kinematic viscosity. "Nonlocal" means that the demixing effects in the free energy are represented by a (spatially) nonlocal term. Well-posedness and regularity issues will be discussed. Also, a comparison with the results obtained for similar systems will be made. This is a joint project with C. Cavaterra and S. Frigeri (Università degli Studi di Milano).

**Hubert Florence** (Aix-Marseille University)

**Microtubules (MT) a key target in oncology: mathematical modeling of anti-MT agents on cell migration**

**Abstract:** Microtubules (MTs) are protein filaments found in all eukaryotic cells which are crucial for many cellular processes including cell movement, cell differentiation, and cell division, making them a key target for anti-cancer treatment. In particular, it has been shown that at low dose, MT targeted agents (MTAs) may induce an anti-migratory effect on cancer and endothelial cells, leading to new prospects in cancer therapy. In that context, we propose to better understand the role of MT dynamics and thus of MTAs on cell migration using a mathematical cell centered model of cell migration taking into account the action of microtubules in the process. The model use a fluid based approach that describes, through level-set techniques, the deformation of the membrane during cell migration. The fluid part of the model is mainly composed of Stokes equations and the biochemical state of the cell is described using Reaction-Diffusion equations. Microtubules act on the biochemical state by activating or inactivating proteins of the Rho-GTPases family. The numerical simulation of the model is performed using Discrete Duality Finite Volume techniques. We describe the different schemes used for the simulation, focusing on the adaptation of preexisting methods to our particular case. Numerical simulation are performed, showing a realistic behavior of the simulated cells in term of shape, speed and microtubules dynamics. Different strategies for a depolymerizing MTA (Vincristin) mechanisms are investigated and show the robutness of our model.

**Jabin Pierre-Emmanuel** (Pennstate University)

**Tracking the adaptation and compensation processes of patients' brain arterial network to an evolving glioblastoma**

**Abstract:** We develop automated personalized system-level analysis methods to analyze the structure of a patient's complete brain arterial network, and to estimate its mean blood flow behavior, from magnetic resonance (MR) images. This allows us to track the compensatory arterial changes and mean blood flow behavior from a patient's clinical images. By analyzing time series data from a patient with aggressive brain cancer, we found significant variations in the structure of the arterial network, and the resulting changes in blood flow dynamics due to biomechanical mechanism. We simulated the evolution in the patients arterial network over time and find that the patients arterial network changes are due to the interplay between the development of the tumor and compensation processes of the brain. This implies that significant disease-related local changes cause global changes at the arterial network, which in return impacts the progression of the disease. We hope that such unique spatiotemporal patterns of the arterial network could assist in predicting the evolution of glioblastoma over time. This is a joint work with J. Zhu, S. Teolis, N. Biassou, A. Tabb, and O. Lavi.

**Joanny Jean-François** (College de France)

**Instabilities and geometry of growing tissues**

**Kanzler Laura** (Sorbonne University)

**Kinetic Modelling of Colonies of Myxobacteria**

**Abstract:** Myxobacteria are rod-shaped, social bacteria that are able to move on flat surfaces by 'gliding' and form a fascinating example of how simple cell-cell interaction rules, including alignment and reversal of individuals, can lead to emergent, collective behaviour. In this talk a new kinetic model of Boltzmann-type for such colonies of myxobacteria will be introduced and investigated. For the spatially homogeneous case an existence and uniqueness result will be shown, as well as exponential decay to an equilibrium for the Maxwellian collision operator. Further, model extensions and their analysis will be addressed and numerical simulations will be shown.

**Loy Nadia** (Politecnico di Torino)

**Direction-dependent turning leads to anisotropic diffusion and persistence**

**Abstract:** Cells and organisms follow aligned structures in their environment, a process that can generate persistent migration paths. Kinetic transport equations are a popular modelling tool for describing biological movements at the mesoscopic level, yet their formulations usually assume a constant turning rate. Here we relax this simplification, extending to include a turning rate that varies according to the anisotropy of a heterogeneous environment. We extend known methods of parabolic and hyperbolic scaling and apply the results to cell movement on micro-patterned domains also through numerical simulation of the transport model. We show that inclusion of orientation dependence in the turning rate can lead to persistence of motion in an otherwise fully symmetric environment, and generate enhanced diffusion in structured domains. (With Hillen, T. and Painter, K.).

N.Loy, T. Hillen and K.J. Painter (2021), Direction-Dependent Turning Leads to Anisotropic Diffusion and Persistence, *European Journal of Applied Mathematics*, 1-37. doi:10.1017/S0956792521000206

**Marciniak-Czochra Anna** (University of Heidelberg)

**Mathematical modelling of clonal dynamics of acute myeloid leukemia: Model-based approach to understand within-tumour heterogeneity**

**Munoz Jose J.** (Universitat Politècnica de Catalunya)

**On the stability of oscillations in rheological models with delay and viscous friction during morphogenesis**

**Abstract:** We present a rheological model based on a dynamic adaptation of the rest-length of the tissue during morphogenesis. This adaptation is dependent on previous strain levels by using a delay parameter. We show that the stability of the displacements is dependent on this delay and other material parameters. We demonstrate that, unexpectedly, an increase in frictional viscosity destabilises the oscillatory response. The extension of the model to non-linear strain measures is able to reproduce the observed sustained oscillations in embryonic tissues. The model is applied to the analysis of the Central Nervous System (CNS) in Drosophila fly. Increase in stiffness and a decrease in viscosity allows us to hypothesise a instability of the oscillations prior to CNS condensation. Although other reasons for the triggering of the morphogenetic movements cannot be discarded, the theoretical and experimental measurements point towards a possible source of tissue instabilities during morphogenesis.

**Painter Kevin** (Politecnico di Torino)

**The impact of flow and flow-oriented swimming on the capacity to aggregate**

**Abstract:** The gathering of cells or animals into large groups provides one of nature's most spectacular examples of self-organising behaviour. To achieve this within a fluid environment, though, could involve confronting strong and turbulent flows. Cells and animals can neutralise flows by swimming upstream, a phenomenon termed rheotaxis. But is it always advantageous to use rheotaxis? We look at the effects of flow both in the presence and absence of rheotaxis, teasing out scenarios under which group formation and maintenance is enhanced. Optimised behaviour results not when rheotaxis is always on, but modulated according to local population density.

**Piotrowska Monika Joanna** (University of Warsaw)

**Computational Modelling growth and response of multicellular EMT6/Ro spheroids to multi-fraction irradiation**

**Abstract:** In this talk, we describe the development, over several years, of a high-fidelity 'in-silico' multicellular EMT6/Ro spheroidal model which aims to offer a better understanding of cancer dynamics under irradiation and to improve therapeutic outcomes. Our earlier works [1-2] have shown that computational cellular automata (CA) models of EMT6/Ro spheroid growth can be successfully calibrated to a broad spectrum of tumour characteristics. The underlying coupled EMT6/Ro tumour growth—multi-irradiation model [3-4], was then further calibrated across 18 independent experimental multi-fraction studies, requiring the direct modelling of DNA damage and repair processes following known biological systems resulting in a high-fidelity computational model of tumour growth under irradiation. Finally, and recently, we have developed an advanced GPU-GA technique to conduct highly parallel search in a vast combinatorial space to identify promising irradiation protocol candidates for further clinical study.

References:

[1] M. J. Piotrowska and S. D. Angus. A quantitative cellular automaton model of in vitro multicellular spheroid tumour growth.Journal of Theoretical Biology, 258(2):165-178,2009.

[2] S. D. Angus and M. J. Piotrowska. The onset of necrosis in a 3d cellular automaton model of EMT6 multicellular spheroids. Applicationes Mathematicae (Warsaw), 37(1):69-88,2010.

[3] S. D. Angus and M. J. Piotrowska. A numerical model of EMT6/Ro spheroid dynamics under irradiation: Calibration and estimation of the underlying irradiation-induced cell survival probability. Journal of Theoretical Biology, 320:23-32, 2013.

[4] S. D. Angus and M. J. Piotrowska. A matter of timing: Identifying significant multi-dose radiotherapy improvements by numerical simulation and genetic algorithm search. PLoS ONE, 9(12):e114098, 2014.

**Pouchol Camille** (University of Paris)

**Asymptotic analysis for some selection-mutation models: locations and weights of limit singular measures**

**Abstract:** The goal of this presentation is the detailed asymptotic analysis of a simple integro-differential for a structured population growing logistically, through

$$\frac{\partial n}{\partial t}(t,x) = (r(x) - \rho(t))\, n(t,x),$$

where the coupling comes from the total mass $\rho(t) := \int_\Omega n(t,x)\,dx$. These equations are well-known to lead to concentration where the fitness function $r$ reaches its maximum, *i.e.*, any limit point $n^\infty$ is a sum of Dirac masses located on $\arg\max(r)$ [ref1].
However, if and how the limit depends on the initial condition is less clear. Thanks to Laplace's formula, I will explain how the limit measures (and more generally the asymptotics) can completely be characterised, and how they depend both on the initial condition as well as the local concavity of $r$ at the maximum points.
Alternatively, one can use a vanishing viscosity approach by adding a small mutation term $\varepsilon\Delta$. The asymptotic behaviour of the resulting PDE is easily analysed, and one recovers uniqueness for the limit $n_\varepsilon^\infty$. One may then pass to the limit $\varepsilon \to 0$ to find that the limit measure $n^\infty$ also is concentrated on $\arg\max(r)$. In contrast with the initial approach, semi-classical analysis shows that $n^\infty$ is typically unique and with a single Dirac mass.
All these results have been obtained in collaboration with Tommaso Lorenzi from Politecnico di Torino and may be find in [ref2]. Finally and if time permits, I'll present some early results on adding an advection term to the integro-differential equation (PhD work of Jules Guilberteau, together with Nastassia Pouradier Duteil, Sorbonne Université).
[ref1] B. Perthame, *Transport equations in biology*, Springer, 2006. [ref2] T. Lorenzi, C. Pouchol, *Asymptotic analysis of selection-mutation models in the presence of multiple fitness peaks*, Nonlinearity 33, 5791 (2020).

**Preziosi Luigi** (Politecnico di Torino)

**Modelling cell re-orientation under stretch**

**Saut Olivier** (CNRS Bordeaux)

**Early evaluation of cancer progression and treatments from radiological images using modeling and AI.**

**Abstract:** The main goal of this talk is to present examples of how mathematical modeling and AI may help clinicians following the evolution of solid cancer. As the main insight on the disease is obtained from routine radiological images, our models are image-driven. When longitudinal data is available (as standard follow-up), mechanistic models based on PDE may be built and offer useful predictions of the evolution of the disease. I will present the case of meningioma and the derivation of an accurate model for the description of their natural growths. For patient-specific simulations, models have to be personalized which adds an additional challenge: data assimilation. I will described some novel methods that we have developed to recover model parameters from radiological history of patients and eventually build decision-helping algorithms. In some other cases, a single exam is available in routine for each patient. In such cases mechanistic models tend to be replaced by AI algorithms (which could also be used for automatic segmentations of lesions). I will present how we hope to combine these two approaches to reach better prediction abilities.

**Schmidtchen Markus** (University of Dresden)

**On the Incompressible Limit for a Tumour Growth Model Incorporating Convective Effects.**

**Abstract:** In this work we study a tissue growth model with applications to tumour growth. The model is based on that of Perthame, Quirós, and Vázquez proposed in 2014 but incorporates the advective effects caused, for instance, by the presence of nutrients, oxygen, or, possibly, as a result of self-propulsion. The main result of this work is the incompressible limit of this model which builds a bridge between the density-based model and a geometry free-boundary problem by passing to a singular limit in the pressure law. The limiting objects are then proven to be unique.

**Schmeiser Christian** (University of Vienna)

**Reduction of the Filament Based Lamellipodium Model (FBLM) to an active curve**

**Abstract:** The lamellipodium is a flat cell protrusion typically adhering to flat substrates, where it acts as cell motility engine. It is supported by a network of filaments of polymerized actin. The FBLM is a two-dimensional continuum model based on the mechanical behavior of this network and its dynamics, including (de)polymerization, cross-linking, substrate adhesion, and other effects. I shall report about recent efforts to reduce this model to the dynamics of an active curve along the cell periphery.

**Tang Min** (Shanghai Jiao Tong University)

**Transition from fractional diffusion to normal diffusion in kinetic models**

**Trescases Ariane** (University of Toulouse)

**A multi-tissue viscous model for tissue growth with enforced segregation**

**Abstract:** During vertebrate embryo elongation, live imaging shows cellular turbulent behavior in the embryonic tissues. We propose a mechanical model in 2D to model the growth of the tissues during the embryo elongation. Viscosity of the tissues is taken into account through the Brinkman law, a choice which allows a non-trivial rotational for the velocities of the tissues. One more specificity of our model is that segregation between the tissues is enforced through a segregation pressure. After the incompressible limit is (formally) performed, the qualitative behaviour at the limit is studied, and a ghost effect is discussed.