J-Biot 2026, 9-10 novembre, Grenoble
Les 2èmes Journées de la Biologie Théorique

Les résumés et les présentations des participants peuvent être téléchargées ici : https://hal.science/JBIOT26

Jour 1 Lundi 9 novembre

8h30 Welcome-Accueil

9h00 Romain Yvinnec. *« A population dynamics approach to ovarian follicle homeostasis throughout the reproductive lifespan »*
Résumé : In this talk, we present a population dynamics approach to study the homeostasis of the ovarian follicle population throughout the reproductive lifespan. In mammals, the number of female germ cells continuously decreases over time, whereas in fish, oogenesis is self-renewing. Nevertheless, in both cases, the active germ cells within growing ovarian follicles exhibit remarkable robustness properties. We revisit a compartmental population dynamics model for ovarian follicle development by incorporating follicular interactions that arise (directly or indirectly) from the regulation exerted by reproductive hormones on their activation, growth, and death rates. The characteristics of the follicle population distribution are obtained thanks to a time scale separation between follicle activation and growth, which enables us to study the relationship between the growing follicle pool and the quiescent one. Depending on the ratio of activation to growth rates, we perform a full bifurcation analysis and show that the model of growing follicles can exhibit monostability, multistability, or stable oscillatory solutions, arising respectively through saddle-node and Hopf bifurcations. Finally, we discuss how to calibrate such models, either through a parametric approach using population count data from mice, or through a nonparametric approach using follicle size distribution data from medaka.

9h50 Giada Fiandaca. *« Decoding Cellular Plasticity and Resistance in Rhabdomyosarcoma: Bridging Population Models and Regulatory Networks »*
Résumé : Tumour phenotypic heterogeneity is a major contributor to treatment failure. Cellular plasticity allows cells to transition between states with different treatment sensitivities. Quiescent cells can evade chemotherapies targeting proliferating cells and subsequently re-enter the cell cycle, potentially driving tumour regrowth. Understanding the regulation of these transitions could reveal therapeutic targets to prevent recurrence. We investigate this question using patient-derived rhabdomyosarcoma organoids that preserve the cellular diversity of patients' tumours. To this end, we propose an approach combining mechanistic population modelling and gene regulatory network (GRN) inference in untreated and treated conditions. Ordinary differential equations describe proliferation, cell death and transitions between proliferative and quiescent states, including their modulation by vincristine. The population model then informs mechanistic GRN inference from time-resolved single-cell transcriptomic data, providing dynamical constraints to guide the identification of intracellular regulatory mechanisms underlying plasticity. This connection presents a methodological challenge: growth, viability and cell-state measurements describe population behaviour, whereas transcriptomic profiles capture molecular variation within cells. These observations differ in scale, temporal resolution and biological information. Beyond integrating heterogeneous data, our approach seeks to establish a consistent correspondence between the states and dynamics represented in the two mathematical frameworks. Our ultimate aim is to identify regulatory genes whose perturbation could impair plasticity-mediated resistance and explore drug combinations in silico. This would help prioritise strategies that target proliferating cells while limiting the persistence or reactivation of quiescent populations.

10h20 Baptiste Zuber. *« Mathematical model of the lactate shuttle between glioblastoma and neurons »*
Résumé : Gliomas, brain tumors that develop from glial cells, are classified by the WHO into different grades. Among grade IV gliomas are glioblastomas, which are the most aggressive brain tumors, for which no effective

therapies currently exist. Recent studies have highlighted the importance of lactate in tumor growth dynamics. Building on work carried out by a team of biologists at the University of Bordeaux on the lactate shuttle between glioblastoma cells and neurons, we are developing a mathematical model based on partial differential equations to describe the evolution over time of tumor cell density, lactate concentration, and glutamate concentration. In this new approach, tumor cell density is divided into two compartments representing two distinct cell phenotypes : glycolytic cells and oxidative cells. These two cell phenotypes affect lactate production and consumption. Variations in lactate levels, in turn, influence the concentration of glutamate produced by neurons. Finally, the glutamate produced promotes the proliferation and invasion of tumor cells. The calibration of the model parameters is currently underway. For this purpose, we use data from the literature, as well as experimental results obtained in vitro by the LIENSs laboratory in La Rochelle and the IBGC in Bordeaux on different glioblastoma cell lines. In this presentation, I will describe the mathematical model and present numerical simulations reproducing the evolution of a glioblastoma in vitro.

10h35 Coffee Break Posters

11h05 Samuel Bernard. *« Individual-based modelling approaches to probe interactions in complex cell populations »*
Résumé : Simuscale is a multiscale, individual-based modelling platform for performing numerical simulations of heterogeneous populations of individual cells evolving in time and interacting physically and biochemically. Models are described at two levels: cellular level and population level. The cellular level describes the dynamics of single cells, as defined by the modeller. Cells have an internal state that includes default properties such as cell size and position, and may also include any other cell-specific state, such as gene or protein expression. The population level describes the mechanical constraints and biochemical interactions between cells. Cells evolve in bounded 3D domain, and can divide or die. We illustrate usage with a recent model of neuroblastoma in ex vivo patient-derived tumoroids (PDT). We implemented multiscale PDT model in Simuscale. Model rules specify cell fate based on an intracellular stochastic gene regulatory network. The state of the network is modulated by intercellular signals from the local spatial environment. Two modes of intercellular communication were explored. In the first one, signalling by cell-to-cell contact resulted in a unstructured PDT. In the second one, short range diffusive coupling, where signalling has an radius of action of few cells, resulted in a highly structured PDT, recapitulating ex vivo tumoroid structures.

11h35 Sophie Hecht. *« Microscopic and macroscopic models for the growth of bacteria micro-colony »*
Résumé : Bacteria are abundant organisms that participate in many processes occurring in medicine, agriculture, ecology, industry. Starting from a single organism, they quickly develop into organized micro-colonies and biofilms. The formation of microcolonies, although widely studied over the past decade, is still poorly understood. We will first consider a microscopic model where each bacterium is modeled by a spherocylinder and where the bacteria interact through non-overlapping constraints. By taking into account the asymmetry of the poles of the bacteria, the model reproduces the mechanical characteristics of the growth of micro-colonies, and this without implementing either attraction or adhesion. Then we present the derivation of this model toward an aggregation model with growth and fragmentation.

12h05 Botao Dai. *« Quantifying Hypoxia-Induced Radioresistance: Experimental Analysis and Compartmental Modeling of Cell State Dynamics »*
Résumé : More than 50% of patients with cancer receive radiotherapy to kill tumor cells. Several mathematical models predicting post-irradiation cell survival are used clinically to plan treatment; however, they often neglect the tumor microenvironment, which can modulate radiotherapy response. A key driver of radioresistance in solid tumors is hypoxia. In this project, we focus on hypoxia-induced radioresistance. Our experiments are designed to quantify how reduced oxygen levels before and after irradiation affect survival and post-treatment dynamics. Specifically, we quantitatively analyzed the individual contributions of diminished free radical yield and hypoxia-induced quiescence to the overall radioprotective effect. We also present a biologically motivated compartmental model to analyze the dynamic transitions of cells between distinct states (e.g., repaired, senescent,

and unrepaired subpopulations) under different conditions. By fitting time-course data, the model estimates key transition rates and incorporates oxygen dependence, enabling accurate simulation of cellular responses under hypoxic conditions. This work provides insight into the oxygen-dependent responses of cancer cells to radiation, informing more effective and context-aware therapeutic strategies.

12h20 Felipe Olivares Fernandez. *« ATM-ApoE crown formation in Alzheimer's disease: A mathematical approach »*
Résumé : Alzheimer, neurons, radiation, PDEs Alzheimer's disease is the most common form of dementia, a neurodegenerative condition that is still not fully understood. While most mathematical models of Alzheimer's focus on amyloid plaques and the tau protein, this talk presents an alternative approach based on the RIANS response, specifically the interaction between two proteins, ATM and ApoE, inside neurons. We first introduce the biological mechanism and show how ordinary differential equations model perinuclear crown formation under stress, as well as the effects of radiation and drugs as potential treatments. We then study a reaction-diffusion-advection system with time-dependent coefficients that describes the spatial distribution of these proteins in a cell. Results on global well-posedness are presented: existence and uniqueness of a non-negative global solution.

12h35 Lunch Break Posters

14h00 Raluca Eftimie. *« Mathematical approaches to investigate keloid disorders »*
Résumé : In this talk, we present a class of non-local mathematical models developed to investigate normal and abnormal wound healing processes such as keloids (these abnormal processes lead to tissue overgrowth, remodelling and invasion similar to those observed in benign tumours). The models account for non-local cell-cell and cell-matrix interactions via different signalling molecules as well as long-distance cell protrusions. We discuss various analytical and numerical aspects associated with these non-local models from the perspective of biological applications. Due to the complexity of this model, and lack of appropriate data, it is difficult to parametrise it. Nevertheless, we discuss model parametrisation for a simplified local model.

14h50 Véronique Thomas-Vaslin. *« Le vivant comme système sensible historisé : émergence de l'inférence, de la mémoire et de la tolérance immunitaire dans l'organisation phylo-ontogénétique des holobiontes »*
Résumé : Comment les systèmes vivants maintiennent-ils communication, identité et résilience tout en demeurant exposés au désordre et aux perturbations de l'environnement ? Comment des organismes poly génomiques tels les holobiontes (hôte + microbiote) intègrent-ils histoire évolutive (y compris des transmissions non génétiques), développement et contexte écologique pour produire mémoire, cognition et tolérance immunologique assurant l'intégrité de l'organisme ? Ces questions invitent à un changement de paradigme : passer de descriptions réductionnistes d'objets à une conception du vivant comme organisation historisée et (auto)organisée, dotée d'une sensibilité (détecter, discriminer, pondérer des perturbations) et d'inférences (interprétations contextuelles conduisant à l'action). L'holobionte et le système immunitaire illustrent une co organisation multiéchelle façonnée par la phylo ontogenèse et par des temporalités intriquées — Aion (évolution), Chronos(développement) et Kairos (fenêtres critiques de décision). La génération stochastique de récepteurs, la dégénérescence et des interactions rares ou bruitées ne sont pas seulement des sources d'aléa, mais des ressources d'exploration qui, via des boucles de rétroaction, transforment des distorsions en diversité fonctionnelle puis en robustesse/résilience. Le cadre Generic Sensor Actuator (GenSA) propose une abstraction indépendante de l'échelle : des « holons » s'organisent en réseaux/holarchies pour percevoir, intégrer, mémoriser et répondre via des interactions fondées sur l'énergie ou l'affinité, des molécules aux écosystèmes. Sa contrepartie formelle, le Generic Sensory Automaton (GENSA), rend opératoire cette articulation en combinant deux modes complémentaires : (1) la détection de motifs biologiques conservés (invariants) hérités et sélectionnés et (2) la détection de distorsions lors de l'ontogenèse, lorsque l'exploration somatique (diversité stochastique des immunorécepteurs) permet d'évaluer des variations d'affinité/énergie et de signaler des changements au delà d'un seuil qui formalise une bascule de régime : en deçà, fluctuations intégrées et tolérance dominante (notamment sous contrôle Treg) ; au delà, rupture de tolérance et activation/inflammation.

15h20 Dominique Pastor. *« Le GENSA (GENeric Sensory Automaton) : un modèle mathématique des interactions dynamiques par affinité en biologie »*

Résumé : This presentation revisits the paper 'The Generic Sensory Automaton (GENSA) for modeling affinity-based dynamics and interactions in biology', by Pastor et Thomas-Vaslin. In this talk, for further dissemination and exchanges, we present and discuss the GENSA (GENeric Sensory Automaton), a mathematical framework aimed at modeling biological systems, like the immune system. The GENSA is an automaton capable of detecting and responding to environmental changes and thus, capable of perceiving and responding to events in its surrounding. The capacity of the GENSA to detect events in its environment, and modify its state accordingly, is formalized through mathematical notions of specificity and sensitivity. We give and discuss two fundamental GENSAs : the Neyman-Pearson GENSA (NP-GENSA) and the Random Distortion Testing GENSA (RDT-GENSA). These two GENSAs embed distinct decision types and satisfy functional redundancy, in a sense close to the biological notion of degeneracy. We will present some prospects this model opens, especially with degeneracy. The possibility to construct networks of GENSAs and the perspectives these networks open will also be discussed, beyond the conclusions of the initial paper.

15h50 Coffee Break Posters

16h20 Gabin Calmet. *« Model for evolution of multicellularity in bacterial colonies »*

Résumé : Multicellularity has evolved independently in several lineages, including bacteria. For instance, the obligate multicellular bacterium S. coelicolor specialises cells that secrete antibiotics at specific stages of its life cycle, and B. subtilis biofilms exhibit cell differentiation, division of labor and spatial organization. These colony-level developmental dynamics are governed by complex gene regulatory networks (GRNs) that enable robust colony growth. However, we currently do not understand well the selective drivers of bacterial multicellularity, and how bacterial development evolves. We investigate these questions using a computational evolutionary approach. We model a population of bacterial cells that can replicate, deposit antibiotics, and sporulate in a discrete two-dimensional plane. These behaviors are dynamically controlled by a GRN, which drives colony development. Genomic mutations, such as duplication, deletions and modification of TF binding strength, can rewire the network and change the course of development. Initial simulations coupling antibiotic production with population dynamics provided a first step towards validating the model, by assessing whether its dynamics were consistent with established microbiological observations. To summarise, in this model a developmental program can evolve to regulate growth, antibiosis and sporulation in a colony, driven by emergent colony-level selection and long-term accumulation of mutations.

16h35 Baptiste Rault. *« Fusion multimodale pour l'estimation du rythme cardiaque fœtal »*

Résumé : Le rythme cardiaque fœtal (RCF) est un indicateur précieux pour surveiller la bonne santé d'un fœtus pendant l'accouchement. Son suivi en continu permet une intervention rapide en cas de détection de détresse fœtale. La cardiotocographie (CTG) est de nos jours la technique de référence pour le suivi du RCF, mais souffre encore d'erreurs d'estimations, notamment à cause de confusions avec le rythme cardiaque maternel. Bien que prometteuses, d'autres alternatives à la CTG comme l'électrocardiographie abdominale (ECG) et la phonocardiographie abdominale (PCG) souffrent des mêmes problématiques et ne permettent pas encore de concurrencer la CTG. Afin de permettre un suivi fœtal plus robuste, nous proposons une approche multimodale d'estimation du RCF, en fusionnant une estimation de RCF issue de la modalité ECG et une autre issue de la modalité PCG. Cette fusion est réalisée à partir d'un algorithme basé sur du filtrage de Kalman commutatif (Switching Kalman filter). Ce type d'algorithme permet de faire des hypothèses sur chaque modalité, en supposant à chaque instant qu'elle est une estimation correcte du RCF, une estimation incorrecte, ou une confusion avec le rythme maternel. De plus, l'algorithme permet de considérer le rythme cardiaque maternel de référence, pour détecter les confusions maternelles des modalités. Testé sur 37 enregistrements de femmes en fin de grossesse, 79.3% des estimations issues de cette fusion multimodale sont concordantes avec le RCF de référence, contre 47.2% des estimations issues l'ECG seule, et 68.7% des estimations issues de la PCG seule. Par ailleurs, son temps de calcul est compatible avec une application de suivi fœtal en continu, en estimant en seulement 5min11s les RCF de 17h47min d'enregistrements fœtaux.

16h50 Pascale Calabrèse. *« Inspiratory and expiratory sinus arrhythmia in healthy human »*

Résumé : Breathing and ECG were recorded in 33 healthy human subjects at rest. The RR interval was labeled according to its occurrence in the respiratory phases: II (both R waves during inspiration), IE (first R wave in inspiration and following R wave in expiration), EE (both R waves during expiration), and EI (first R wave in expiration and following R wave in inspiration). For each subject, II and EE intervals were plotted versus normalized mean inspiratory duration and normalized mean expiratory duration, respectively, and correlation coefficients and slopes of regression lines were calculated. Statistical analyses were conducted to compare these slopes between and within individuals. These relationships appeared to be linear in most cases, although neither the decrease nor the increase in heart rate occurred at the same rate for all subjects. Overall, the slope associated with II intervals was not higher, in terms of absolute values, than the slope associated with EE intervals for the same subject. Our results underscore the difference between inspiratory and expiratory sinus arrhythmia and suggest that the effects of any stimulation presumed to induce changes in vagal cardiac activity should primarily be sought during expiration.

17h20 Pierre Auger and Norris Vick. *« Hommage à Michel Thellier »*

Résumé : À venir

18h00 Assemblée Générale (AG) de la SFBT

Jour 2 Mardi 10 novembre

8h30 Welcome-Accueil

9h00 Elias Ventre. *« Calibrating stochastic models of gene expression from single-cell data: from gene selection to trajectory and network inference »*
Résumé : Differentiation is the process by which a cell acquires a given phenotype through the expression of genes over time. This dynamic is widely understood to result largely from the action of a gene regulatory network (GRN). A particularly fruitful way to study it is to model the dynamics of a cell under the action of its GRN as a stochastic process: GRN inference then becomes the calibration of this model from experimental data. Temporal snapshots of scRNA-seq data make this problem both promising and challenging: as protein trajectories cannot be observed at the single-cell level, it's not straightforward to establish direct causal relationships between regulator activity and target responses. Two main strategies can be distinguished. Generic models, such as systems of stochastic differential equations, can be calibrated with non-parametric methods based on optimal transport and benefit from highly efficient existing tools. Mechanistic models, such as piecewise deterministic Markov processes directly parameterized by a GRN, are better suited to incorporating biological priors but require dedicated methods. They also raise the additional challenge of selecting a gene set that is both biologically relevant and tractable for the model. After discussing these issues, I will focus on CardamomOT, an algorithm that calibrates a PDMP-based model of gene expression within a novel mechanistic optimal transport framework. It jointly reconstructs the GRN and the unobserved protein trajectories. On in silico and experimental datasets, it accurately recovers the velocity fields driving cellular trajectories and the hidden protein levels, alongside reliable GRN structures. Finally, I will show that the calibrated model can serve as a generative model to predict cellular responses to unseen perturbations.

9h50 Arnaud Bonnaffoux. *« Bifurcations stochastiques dans les réseaux de régulation géniques : de la modélisation à la validation expérimentale »*
Résumé : Au sein du département de biologie computationnelle du CRCL (Centre Léon Bérard, Lyon), nous développons des modèles dynamiques exécutables des réseaux de régulation géniques (GRN), inférés à partir de données de cellule unique. Le cœur de notre cadre est un modèle mécaniste stochastique de type processus de Markov déterministe par morceaux (PDMP). Le promoteur de chaque gène y bascule entre deux états, l'ARN et la protéine suivent des équations différentielles entre les sauts, et les interactions du réseau modulent la fréquence des bursts transcriptionnels. Les états cellulaires sont alors des attracteurs métastables, et les décisions de destin des transitions entre bassins dont la probabilité dépend de la structure du réseau. Autour de ce modèle, nous avons construit une chaîne complète. L'analyse des données, sous hypothèse ergodique, isole les trajectoires, les bifurcations et les vagues d'expression. Ces vagues contraignent l'espace des topologies et rendent l'inférence traitable. La simulation confronte les candidats aux distributions observées. L'inférence restant non identifiable, un plan d'expériences choisit enfin la perturbation la plus discriminante. J'illustrerai ce cadre par deux cas d'usage validés expérimentalement. Dans l'initiation de la dystrophie musculaire de Duchenne, un double interrupteur à bascule rend compte d'une bifurcation où la mutation ne crée pas de destin nouveau mais déplace les probabilités de destins préexistants. Dans l'acquisition de résistance du mélanome aux thérapies ciblées (travaux non publiés), une sous-population pré-résistante émerge du franchissement stochastique d'un seuil par une boucle d'activation mutuelle. Dans les deux cas, les extinctions géniques prédites ont été testées. Je discuterai enfin la distinction entre bifurcation au sens strict et transition induite par le bruit.

9h Patricia Lamirande. *« Complexité des modèles et compréhension mécanistique : un compromis illustré par la dynamique tumeur–immunité »*
Résumé : Les modèles mathématiques mécanistiques sont largement utilisés pour approfondir notre compréhension des mécanismes biologiques, notamment la régulation de la croissance tumorale et sa réponse aux traitements. Cependant, la richesse des mécanismes de régulation biologique peut conduire à des modèles de grande dimension, exposés à des problèmes de non-identifiabilité des paramètres. Des critères statistiques tels que les critères d'information d'Akaike et bayésien permettent de comparer les modèles en équilibrant qualité

d'ajustement et complexité. Toutefois, un modèle plus parcimonieux n'est pas nécessairement suffisant pour capturer adéquatement les mécanismes biologiques pertinents. Le choix du niveau de complexité constitue ainsi un compromis entre parcimonie, interprétabilité et capacité à formuler des hypothèses mécanistiques. Nous illustrons ce compromis à l'aide de modèles de croissance du cancer ovarien et de réponse au traitement (cisplatine et immunothérapie) dans des modèles murins déficients ou compétents en recombinaison homologue. Nous développons une hiérarchie de modèles mathématiques de complexité croissante décrivant la croissance tumorale, la réponse aux traitements et les interactions avec le système immunitaire. Cette approche permet d'étudier comment la structure du modèle influence les dynamiques prédites et leur interprétation mécanistique. Les modèles reproduisent des dynamiques tumorales et des réponses aux traitements qualitativement distinctes entre les deux types tumoraux, et montrent que certains mécanismes ne peuvent être identifiés qu'à partir de modèles suffisamment complexes pour représenter les interactions pertinentes. Ces résultats illustrent les limites d'une sélection de modèle fondée uniquement sur des critères statistiques lorsque l'objectif est la compréhension biologique, et soulignent l'importance d'adapter la complexité du modèle à la question biologique étudiée.

10h50 Coffee Break Posters

11h20 Aurélien Tauzin, Phuong Thao Chu, and Antoine Frenoy. *« Revisiting Luria & Delbrück with stochastic simulations »*

Résumé : The fluctuation test from Luria & Delbrück is a cornerstone of molecular evolution. Originally designed to demonstrate the independence of mutations and selection, it is routinely used to estimate mutation rates, a key evolutionary parameter as it determines the speed at which genetic diversity is generated de novo. However, this method relies on a mathematical model that makes restrictive assumptions about the effect of mutations (no effect or fixed effect) and cell demographics (no cell death). This leads to biased estimates when these assumptions are not met, which is often the case for many questions of interest, such as effect of stress on mutation rate, estimating mutation rates on non-neutral loci, or estimating mutation rates in vivo. We propose a more general simulated model for the emergence of mutations in bacterial populations. We show that coupling this model with a Bayesian MCMC parameter inference method permits simulation-based estimation of both the mutation rate and the fitness effect of the mutation from fluctuation tests in complex ecological conditions. We found that it works in a broader range of scenarios than existing methods for fluctuation test analyses. We further use our simulation method to consider situations where the phenotype of interest can be conferred by several alternative mutations with different effects on fitness. This permits reconciliation of this framework with modern molecular knowledge.

11h50 Gouled Souleiman-Dahir. *« Mathematical Modeling, Calibration, and Optimal Control of Forest Regeneration under Anthropogenic Pressures: The Day Forest, Djibouti »*

Résumé : Forest ecosystems in arid and semi-arid regions face increasing threats from climate change and human pressures, making active restoration strategies a scientific and policy priority. In these environments, plant–plant facilitation, particularly the nurse role of certain species is a key mechanism for sustaining endemic vegetation. This presentation investigates the optimal control and calibration of a compartmental forest regeneration model, with Acacia seyal as the selected nurse species, applied to the Day Forest ecosystem (Djibouti), a semi-arid habitat harboring a major declining population of East African junipers (Juniperus procera). The model incorporates facilitative plant–plant interactions and two time-dependent control variables representing management interventions aimed at reducing human-induced disturbances. Optimal controls are characterized via Pontryagin's Maximum Principle, and their existence and uniqueness are formally established. The model is then calibrated through a structured procedure grounded in field studies and ecosystem reports from the Day Forest. Numerical simulations are subsequently performed to evaluate a range of management and protection strategies under realistic conditions. Results show that simultaneously activating both human-disturbance reduction in- terventions yields the most effective restoration outcomes. These findings provide a replicable quantitative framework for designing cost-effective restoration policies in semi-arid ecosystems under human pressure, with direct applicability to comparable ecosystems across the Horn of Africa and beyond.

12h05 Nicolas Noirault. *« Prise en compte de la diversité évolutive cachée »*

Résumé : En écologie, on s'intéresse à des espèces liées par un parcours évolutif commun, représenté par un arbre phylogénétique daté, dont les feuilles représentent les espèces observées au temps présent, et les branches ont une longueur proportionnelle au temps évolutif séparant les différents organismes. Ainsi, une mesure de la diversité évolutive est la longueur totale de l'arbre (somme des longueurs de branches). Les indices de diversité permettent d'attribuer une part de cette diversité totale à chaque feuille de l'arbre. Cela permet de classer les espèces selon l'importance de leur contribution à la diversité de l'arbre. Les politiques de conservation peuvent ainsi concentrer leurs efforts vers les espèces dont la disparition entraînerait le plus de perte de diversité. Cependant, certaines espèces, bien que présentes dans le milieu, n'apparaissent pas dans l'arbre parce qu'elles n'ont pas été observées. La prise en compte de ces espèces peut avoir un impact important sur le classement des espèces observées. Nous proposons ici une méthode pour simuler des arbres complets, conditionnellement à l'arbre observé. La méthode se base sur la dérivation de la distribution conditionnelle des lignages non observé dans le cadre d'un modèle classique de diversification de type naissance et mort. Nous pouvons ainsi échantilloner la diversité totale, attribuée aux espèces observée et non-observée, et fournir une mesure d'incertitude sur le classement des espèces à conserver. Nous avons appliqué cette méthode sur un jeu de données d'amphibiens d'Amazonie. Sur 10000 simulations d'arbres complets, nous observons une forte variabilité des classements. Par exemple, l'espèce Pipa Pipa, classée première dans l'arbre observée ne reste première que dans 46% des cas dans l'arbre complet.

12h20 Lunch Posters

14h00 Karin John. *« Unraveling Microtubule Lattice Dynamics: Mechanisms of Tubulin Turnover Beyond the Extremities »*

Résumé : Microtubules are key structural elements of living cells that are crucial for cell division, intracellular transport and motility. They are dynamic polymers, which grow and shrink by addition and removal of tubulin dimers at their extremities. Within the microtubule shaft, dimers adopt a densely packed and highly ordered crystal-like lattice structure, which is generally not considered to be dynamic. Recent experiments have shown that microtubules exhibit a lattice dynamics far away from the extremities. This dynamics manifests itself as localized incorporation of free tubulin into the microtubule shaft, either spontaneously or facilitated by microtubule associated proteins (MAPs), e.g. ATP dependent MAPs such as molecular motors and severing enzymes, but also ATP independent MAPs such as tau. A major biological corollary of such fresh tubulin incorporation events is the increased stability of dynamic microtubules. The mechanisms underlying tubulin incorporation into the microtubule lattice remain poorly understood. Given the inherent complexity of microtubule dynamics and the limitations of experimental observations, multiple incorporation scenarios are plausible. To address this, we develop minimal coarse-grained models that capture the essential time and length scales of the system. These models allow us to test mechanistic hypotheses in silico and generate predictions to guide experimental efforts. In this presentation, I theoretically explore mechanisms of lattice dynamics, integrating experimental findings with our existing knowledge from tip dynamics. I focus on two scenarios for lattice turnover: (1) spontaneous turnover at topological defects, where lattice irregularities facilitate dimer exchange, and (2) motor-stimulated dynamics, where molecular motors drive turnover, potentially via long-lived dimer vacancies.

14h50 Lisa Blum Moise. *« Mechanistic models of social foraging »*

Résumé : Understanding how animals extract information from sensory inputs and use it to guide decisions is central to biology and cognitive science, a challenge that becomes even more complex when social interactions are involved. Foraging behavior offers a powerful paradigm for addressing this issue: it is widespread across species, experimentally tractable, and ideally suited for studying how individuals make collective decisions (Garg et al., 2026). Patch-foraging tasks are particularly valuable because they require repeated decisions to stay or move based on accumulated evidence. We developed a mechanistic framework grounded in evidence accumulation theory, using coupled stochastic differential equations (drift-diffusion model) to capture these decisions (Davidson et al., 2019). Through analytical analysis, with statistical physics tools, and numerical simulations, we uncovered oscillatory dynamics in how groups distribute across patches over time, eventually reaching steady-state values. We quantified these dynamics using metrics such as mean residence time, group accuracy, and cohesion,

and extended our framework across diverse environments, group structures, and social interaction types (Blum Moyse & El Hady, 2025, 2026a, 2026b). Critically, different cognitive mechanisms; including distinct modes of gathering social information (counting, pulsatile, diffusive, reward) and integration strategies (threshold or belief modulation), produce markedly different group-level dynamics. Our analysis of model selection and parameter identifiability revealed key guidelines for applying these models to empirical data, including which experimental conditions and metrics are most informative. These results provide a roadmap for identifying the cognitive mechanisms underlying collective foraging decisions across species and experimental systems.

15h20 Dorothée Bertin. *« Geometric incompatibility in vertex models »*
Résumé : This work focuses on how geometric incompatibility influences the deformation of epithelial-like structures. We use simplified polyhedral models and a vertex model to study how geometry and cellular mechanics interact. We focus on two geometries, a tetrahedron and a dodecahedron, and investigate how their deformation changes with the target shape index. We observe different deformation behaviours depending on the starting geometry.

15h35 Christelle Margossian. *« Influence of Mechanical and Biochemical Cues on Endothelial Cell Migration and Traction Forces »*
Résumé : Endothelial cell migration plays a key role in angiogenesis and is influenced by mechanical and biochemical signals from the surrounding environment. This project investigates the effects of substrate stiffness, extracellular matrix proteins, VEGF stimulation, and cell–cell interactions on endothelial cell migration and traction forces. EA.hy926 endothelial cells are cultured on polyacrylamide gels of different stiffnesses (4.5 and 14 kPa), coated with either collagen I or fibronectin. Cell migration is analyzed using time-lapse microscopy and mean squared displacement (MSD), while traction force microscopy (TFM) is used to quantify the forces exerted by cells on the substrate. Preliminary results show that fibronectin promotes more persistent migration, particularly on softer gels, while increasing substrate stiffness tends to enhance traction forces. Ongoing studies focus on the effects of VEGF stimulation and mechanical interactions between pairs of endothelial cells. Overall, this work aims to better understand how mechanical and biochemical cues regulate endothelial cell behavior during angiogenesis.

15h50 Bertrand Fourcade. *« Protein multiphosphorylation and cellular signaling: nanoclusters, diffusion, and noise in a physics-based modeling approach »*
Résumé : Nanoclustering is a characteristic feature of the activated state of proteins and is essential for forming numerous subcellular structures. The formation of these nanoclusters is highly dependent on a series of post-translational modifications, such as mono- and multi-phosphorylation and dephosphorylation of residues. We theoretically simulate how a protein can be either mono- or multi-phosphorylated on several residues in functional nanoclusters, depending on effective biophysical parameters (diffusion, dwell time, etc.). Moving beyond a binary view of phosphorylation, this approach highlights the interplay between mono- and multi-phosphorylation, the cooperative effects generally associated with multi-phosphorylation networks, and stresses the role of phosphatases in transforming graded phosphorylation signals into almost switch-like responses. We will also show how competition between intrinsic and extrinsic noise can influence signaling pathways for multiphosphorylated states. The results are discussed in light of experiments that probe the distribution of phosphoresidues.

16h20 Coffee Break Posters

16h50 Olivier Destaing. *« Revisiter le transfert d'information en signalisation cellulaire pour comprendre les principes moléculaires de la redondance et de la prise de décision. »*
Résumé : Understanding specificity and redundancy among Src Family Kinases (SFKs) challenges how cells achieve signaling specificity using closely related enzymes and paves the way to revisit basic properties of any cell signaling network. A way to decipher the molecular mechanisms of redundant or specific signaling activity is to directly manipulate them in order to induce specific decision-making activity that supports the selection between

distinct cellular responses. We developed optogenetic probes to control SRC family kinases (SFKs) signaling in order to revisit decision-making. Optogenetic control of the adaptor properties led us to uncouple their kinase and adaptor functions and uncovers a unique mechanism driven by oligomerized- and SH3-dependent specific binding that enables inducing distinct signaling outputs for each kinase. Based on this principle, it was possible to generate two different decision-making events (leading to different acto-adhesive structures, i.e. lamellipodia or invadosome), upon the modulation of the dynamics of optoSRC nanoclusters at plasma membrane that were dependent on its lateral diffusion, SH3 properties and associated intrinsically disordered region (IDR, and named unique domain). This approach has been used in numerous cell type such as epithelial cells, fibroblasts, T lymphocyte and macrophages. Optogenetic activation of the most redundant SFK members, SRC and HCK, can trigger specific cellular responses, despite only supposed overlapping functions. Optogenetically controlled nanoclustering of optoHCK even unveils a novel role in activating polarized clathrin hotspots essential for directional migration. Redundancy could even be rebuilt within optoHCK through stepwise optogenetic reconstruction of adaptor modules revealing the essential role of both membrane anchoring and SH2-domain. This deconstructive approach allowing to control SFKs nanoclustering elucidated the molecular basis of their pleiotropic, their shared and unique signaling activities and supports a model of cooperative functional entanglement between SFK members, rather than simple redundancy.

17h20 Nathan Shourick. *« Hydromechanical model of wood formation »*
Résumé : The cambium is the meristematic tissue that produces wood in trees. In a cross-section of the tree, the cambium gives birth to regularly-shaped cells, organized in radial rows, especially in gymnosperms - a group of woody plants that includes conifers. The growth of plant cells relies on water uptake by osmosis, thereby generating turgor pressure. Next, the volume of cells increases as their walls stretch, eventually causing permanent deformation. Unlike other plant tissues, the cambium is mechanically confined between the phloem (including the bark), the already formed wood and the adjacent tangential cells. Moreover, the xylem (wood in the making) is not homogeneous and is generally divided into multiple zones with different hydromechanical roles. The mechanical role of confinement, zonation, and cell divisions are all coupled mechanisms that have not yet been fully elucidated. In this talk, we present a hydromechanical model of wood formation that extends the Lockhart-Ortega model to the multidimensional and multicellular case. We discuss the geometric, kinematic and mechanical conditions for the stability of cellular patterns and tissue growth.

17h50 Remise du Prix Delattre