NVTB annual Schoorl meeting 2026
Book of Abstracts

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KEYNOTE 1 · 1 JUNE

Emergent spatiotemporal correlations and mechanochemical feedback in model epithelial tissues

Henkes S.
Presented by Silke Henkes

In active materials, forces are produced at the local scale instead of imposed from the outside. Life has found a way to harness these mechanisms create collective motion such as flocks of birds, and more microscopically move and shape organisms through the properties of biological tissues. In this talk, I will show how active forces, feedback, and the mechanics of dense materials interact to give rise to emergent spatial and temporal correlations, self-organised patterns, and exotic mechanical properties. First, a very simple model, active Brownian particles, provides a minimal description of such diverse systems as epithelial tissues, colloidal packings and crowds of people. The interactions of elasticity and uncorrelated activity gives emergent spatiotemporal 'swirly' correlations that fit experimental data for cell sheets and their boundaries when combined with the vertex model of epithelial mechanics. Adding self-alignment with the force on the agent gives rise to spectacular oscillations in confinement, recently also observed in human crowds and engineered active solids. I will show how they can be explained as driven oscillations of the low-energy phonons of a solid. Second, mechanochemical stress feedback in cell-cell junctions arises from the catch bond dynamics of the actomyosin cortex. It allows a junction to generate a contractile force that can overcome external pulling and thus allow for an active rearrangement or T1. In vertex and continuum models, for strong enough feedback this gives rise to convergence-extension flows where the flow is opposite the direction of mechanical polarisation, effectively generating materials with negative viscosity. I will give an overview of this effect combining experimental data, detailed vertex models simulations and how simplified continuum and mean field models can provide insight into the mechanisms at play.

TALK 1 · 1 JUNE

Catch-bond dynamics of cell-cell adhesion in the Cellular Potts model predicts dynamics in cell clustering, sorting and invasion

Rens, E.G.
Presented by Lisanne Rens

Cell–cell adhesion is crucial for tissue organization and collective migration. Cells also adhere to the extracellular matrix (ECM) that surrounds them. Both cell-cell and cell-ECM adhesions can be catch-bonds, which strengthen under increasing force. Although recent mathematical studies have explored how models of catch bonds in cell–ECM interactions influence cell dynamics, the effects of catch-bond behavior of cell–cell adhesions remains understudied. Here, I extend the Cellular Potts Model with catch-bond dynamics for cell–cell and cell–ECM adhesions. I report three key findings: 1) The model predicts the relative adhesion binding rates required for cells to cluster rather than disperse. 2) Decreasing either contractility or adhesion binding rates can reverse engulfment patterns of two cell types. 3) In a specific parameter regime, the model generates short chain-like invasive protrusions into the ECM, indicative of enhanced invasive potential arising purely from catch-bond mechanics. In summary, incorporating catch–slip bond dynamics into a Cellular Potts Model reveals complex relationships between adhesion, cell forces, and collective cell behaviors. This framework offers mechanistic explanations for phenomena such as unexpected cell-sorting outcomes that cannot be resolved by classical differential adhesion models.

TALK 2 · 1 JUNE

Predicting Invasiveness of Oral Cancer by Mechanistic Computational Modelling

Bukman M.C., Rens E.G.
Presented by Maaike Bukman

Oral Squamous Cell Carcinoma (OSCC) is the most common form of oral cancer, accounting for over 90% of all cases and ranking among the most prevalent cancers worldwide. Despite advances in treatment, patient survival remains poor. Recent studies indicate that changes in the organization and orientation of collagen fibers in the extracellular matrix (ECM) are a promising prognostic risk factor for metastasis. In aggressive tumors, the cancer cells interact with the aligned collagen fibers to facilitate invasion into surrounding tissue. Mechanistic computational models that integrate patient data can help predict invasiveness. To this end, we extend the cellular Potts model (CPM) to include cell migration along the orientation of fibers. We simulate tumor invasion in tissue environments based on experimental data. The Menzel lab at TU Delft provides us with data to initialize and calibrate simulations. Fiber orientations and cell shapes were derived from Computational Scattered Light Imaging (ComSLI). By systematically varying model parameters, we investigate potential invasion patterns, identify likely invasion regions and assess how tissue structure influences tumor spread. In the future, model predictions will be evaluated against pathological assessment of the invasive tumor front and correlated with patients known prognosis, including recurrence and metastasis. This approach aims to provide a step towards objective and patient-specific prediction of tumor invasion, with potential application in risk stratification and clinical decision making.

TALK 3 · 1 JUNE

Spatial evolution of viruses

Ashby, B., van Dijk, B.
Presented by Sultan Ahmed Nazir

Viruses are the most abundant biological entities on Earth and play a central role in the ecology and evolution of life across all domains. While they have traditionally been studied as isolated pathogens or parasites, there is a growing recognition that viral populations are better understood as communities of interacting entities, with diverse forms of social interactions between viruses shaping their dynamics and evolution. This shift in perspective opens new avenues for understanding viral ecology beyond host–virus interactions alone. In this work, we used mathematical modelling to investigate the evolutionary origins of hyperparasitism (where viruses exploit other viruses) in temperate phages. We show that, in spatially structured populations, multilevel selection can drive evolutionary branching, leading to the emergence of distinct parasite and hyperparasite strategies. These results highlight how spatial structure and viral interactions can generate novel evolutionary outcomes beyond those predicted by classical models.

TALK 4 · 1 JUNE

Evolution of Suicidal Altruism and the Dispersal of Spores

Prikšāns N., Hogeweg P.
Presented by Nauris Prikšāns

Spatial assortment is usually seen as playing a key role in the evolution and persistence of altruistic behavior in a population. The slime mold Dictyostelium discoideum exhibits a social life cycle where starving cells commit altruistic suicide to promote the dispersal of others, thus seemingly counteracting assortment. We have developed a spatial eco-evolutionary model to

investigate the role of dispersal—and its interaction with assortment—in the evolution of altruism in a Dictyostelium-like system. The results indicate that suicidal altruism that promotes dispersal of nearby cells is beneficial for the system and highly evolvable, with the benefit of broader dispersal outweighing the cost of altruist death.

TALK 5 · 1 JUNE

Computational modeling of accessory chromosome transfers and pathogenicity in soil-borne fungi

Kóródi L., van Dijk B.
Presented by Lőrinc Kóródi

Soil-borne fungi fill several different niches in soil ecology, from saprophytes to parasites. Interestingly, the ecological role of fungi as either a pathogen or a commensal is not set in stone. Instead, several fungal species have now been reported to horizontally transfer pathogenicity genes, and with them the ability to infect plants. An intriguing mechanism of horizontal transfer that has been identified is the transfer of accessory chromosomes — chromosomes that are not essential and can therefore be lost and gained dynamically. Although horizontal chromosome transfer in fungi was reported as early as 1998, its evolutionary consequences, especially in relation to pathogenicity and disease in agriculture, are not well understood. To that end, we present a spatial, individual-based model of soil-borne fungi, with which we investigate the eco-evolutionary effects of horizontal chromosome transfer. We primarily focus on how horizontal transfer affects the prevalence of pathogenicity in a natural fungal–plant ecosystem, and how agricultural crop rotation shapes these complex dynamics. Our results suggest that the horizontal transfer of accessory chromosomes helps the survival of parasites by two distinct mechanisms. First, the ability to lose and regain accessory chromosomes increases evolvability, an effect that leads to a pronounced benefit of horizontal transfer during long-term crop rotation. Second, we observe that horizontal transfer allows pathogenicity genes to escape the effects of weak selection or drift, an effect that is relevant even in a relatively stable environment. Finally, horizontally transferred chromosomes become enriched in effector genes, as observed in nature. These insights show us how horizontal transfer — a process that is known to massively impact prokaryotic evolution — has important consequences for fungal evolution and disease epidemiology, highlighting the need for more research into this phenomenon.

TALK 6 · 1 JUNE

Natural Selection in Transcription Factor–DNA Interaction Motifs: A Comparative and Population Genomics Perspective

Joshi M, Duchen P, Kapopoulou A, Laurent S
Presented by Manas Joshi

Natural selection heavily influences the evolutionary trajectories of species by impacting their genotype-to-phenotype transitions. On the molecular level, these transitions are shaped by the regulatory sequences. In this study, we employed a combination of population and comparative genomics to investigate how natural selection affects specific regulatory sequence classes involved in the regulatory transcription factor–DNA interactions. These interactions consist of two motifs, namely: transcription factor-binding domains and transcription factor-binding sites. Using publicly available annotation data for Homo sapiens, Arabidopsis thaliana, and Drosophila melanogaster, we first constructed the species-specific lists of the transcription factor-binding domain regions. On applying some of the commonly used summary statistics, we found signals of purifying selection acting on transcription factor-binding domains, consistent with their functional importance. Next, using the biochemical assay-based annotations, we identified potential

transcription factor-binding site regions and used variants within them as nonsynonymous equivalents. Interestingly, we also observed that noncoding transcription factor-binding site regions showed similar levels of constraint to that of coding regions for populations with large Ne. Signals of positive selection were limited. Nevertheless, McDonald–Kreitman estimates revealed that, in both fruit-fly and thale-cress, α for transcription factor-binding domains was consistently higher than for adjacent nonbinding domains, whereas no such difference was apparent in humans. Taken together, our comparative analysis shows that the efficiency of negative—and to a lesser extent positive—selection on transcription factor–DNA interface elements scales with effective population size. The dataset and analysis pipeline provide a baseline for future studies of regulatory evolution across coding and noncoding regions.

TALK 7 · 1 JUNE

Selection for Robustness: When Plateaus are Slippery and Peaks are Safe

Luiselli J., Beslon G., van den Berg P.
Presented by Juliette Luiselli

Theory predicts that populations should evolve mutational robustness − the ability to maintain a constant phenotype despite mutations. This means that populations are expected to occupy broad, flat plateaus in the fitness landscape, where most mutations are neutral, while populations on narrow peaks are more fragile ("survival of the flattest"). However, selection does not operate on single mutations but on phenotypes resulting from entire replication events, which may entail multiple mutations. We therefore distinguish mutational robustness from replicative robustness, defined as the probability that a replication event produces similarly fit offspring. Mutational and replicative robustness may seem closely aligned at first sight, but we show with an evolutionary simulation model that they can be selected in opposite directions. Our results show that while genomes with high redundancy and/or large amounts of non-coding DNA are mutationally robust, they are replicatively fragile because their size increases the number of mutations per replication. This is especially relevant when genomes are at risk of severe disruption from chromosomal rearrangements. Our results expose limits of the fitness landscape metaphor in the context of robustness evolution: neutral plateaus may be mutationally robust but replicatively unstable and therefore disfavoured by selection, whereas narrow peaks arising from densely encoded genomes can replicate more reliably. Our results identify replicative robustness as a distinct target of selection and show that whether populations persist on plateaus or peaks depends not only on tolerance to individual mutations but also on their overall likelihood of mutating.

TALK 8 · 1 JUNE

Phosphorylation dynamics of membrane-actin linkers could explain contradicting experimental findings in the C. elegans intestine

Tan N., Tsingos E.
Presented by Nienke Tan

Tubular epithelial tissues are present in various organs such as the intestine. Membrane-actin linkers of the Ezrin, Radixin, and Moesin (ERM) family are essential for sculpting the apical surface of cells to ensure proper formation of the lumen and the microvilli that are typical for these tissues. The binding activity of ERM membrane-actin linkers is thought to require phosphorylation of a highly conserved residue, which is mediated by LOK/SLK kinases. However, recent experimental findings in the nematode C. elegans have cast doubt on the importance of this phosphorylation. In this model organism, the only linker protein is ERM-1. A C. elegans mutant ERM-1 that cannot be phosphorylated (phosphomutant) still retains some functional activity. More intriguingly, the phosphomutant can partially rescue an otherwise lethal phenotype of the C.

elegans LOK/SLK ortholog GCK-4. Additionally, GCK-4 has been shown to be essential in the formation of the intestine and microvilli but surprisingly does not phosphorylate ERM-1. Here, we study the dynamics of ERM-1 in a minimal ODE model that incorporates the actin and membrane binding reactions of ERM-1 as well as its phosphorylation. The aim of this work is to find a model system that might explain contradicting observations on phosphomutants of ERM-1. We find that this contradiction can be explained by a possible role of GCK-4 in actin accumulation which is severely inhibited in the gck-4 null background. In addition, dynamics are consistent with overall high phosphorylation rates and low actin binding rates. In summary, we show that the dynamics of ERM-1 could explain the contradicting observations and might point to the potential role of GCK4 in actin accumulation. This brings us closer to understanding the dynamics underlying the formation of tubular tissues.

TALK 9 · 1 JUNE

From quiet to quick recall response: a key role for quiescent memory T cells?

Zwerink, M.G.C.M., de Boer, R.J., Borghans, J.A.M.
Presented by Madelon Zwerink

Although the overall CD8+ T cell immune response is reproducible, individual naïve CD8+ T cells vary significantly in their contributions. A single naïve CD8+ T cell and all its descendants constitute a "family". Interestingly, while the size of a family in the primary response shows little correlations with its size in the secondary response, there is a strong correlation between family sizes across recall responses. Recent findings using the DivisionRecorder system have revealed that recall responses are primarily driven by lowly divided memory T cells with stem-cell like properties. Understanding how cells responsible for immunological memory are generated and maintained is crucial for improving immune protection post-vaccination. We developed a model in which activated CD8+ T cells initially undergo a few rapid, highly synchronized burst divisions. After a burst, the resulting daughter cells have diversified in branches. Some branches continue to divide and combat the current infection, while others return to quiescence and provide future protection by generating recall responses upon re-infection. The inclusion of these quiescent cells in the model accounts for the observed improved correlation between family sizes across recall responses. This is because the number of quiescent cells formed during the primary response is stochastic but correlated with the number formed during the subsequent response. In conclusion, a growing body of research highlights the crucial role of lowly divided memory T cells in long-term protection against pathogens.

TALK 10 · 1 JUNE

Modelling the Robustness of Squid Tentacle Extension to Spatiotemporal Variations in Muscle Activation

van Leeuwen J., Kier W.M.
Presented by Johan van Leeuwen

Using a previously developed forward dynamics model of tentacle extension in the squid Doryteuthis pealeii, we investigated how spatial patterns of muscle activation influence the distribution of muscular power, mechanical work, and kinetic and elastic energy along the tentacle. The simulations show that peak extension velocity is remarkably robust to variations in activation timing. Specifically, delays in activation propagation along the tentacle stalk—even up to 50% of the total extension duration—have only a limited effect compared to a synchronous activation pattern. Small positive delays, where distal regions activate slightly later than proximal regions, can marginally enhance peak extension velocity. In contrast, negative delays (with distal activation preceding proximal activation) consistently reduce performance. Furthermore, the

extension dynamics are largely insensitive to superimposed random variability in activation timing, particularly when delays are small and positive—conditions consistent with estimates derived from measured motor neuron axon diameters. This robustness to variability in activation patterns suggests that precise neural timing is not critical for effective tentacle extension. Instead, the non-linear mechanical properties of the tentacle musculature likely play a key role in making performance robust against spatio-temporal variability in motor control.

TALK 11 · 2 JUNE

3D Multiscale Modelling of Plant Biomechanics

Ramos, J. R. D., Merk R. M. H.
Presented by João Ramos

The genetic underpinnings that define tissue shape and function are still an open question. Part of the answer is in the biomechanics of cells and tissues and in the cross-talk between biomechanics and biochemistry. In particular, plants exhibit differential growth mediated by the viscous and plastic properties of the cell wall and the spatial distribution of chemical patterns. We are working on a 3D version of the plant modelling package VirtualLeaf, i.e., a quantitative multiscale three-dimensional vertex model of plant development. The final goal is to combine cell wall mechanics with known models of growth, genetic regulation, and water transport, to model plant tissues developing through regulated cell divisions and growth. We will show that our model is capable of capturing the mechanical frustration arising naturally from models of plant cell growth, when accounting for spatial heterogeneity of mechanical parameters and multicellularity. We also explore the possible three-dimensional structures arising from different cells division rules, and whether the three-dimensionality and cell division rules change known relationships between cell volume, pressure and number of neighbours, typically measured only on the epidermis.

TALK 12 · 2 JUNE

Studying root morphogenesis through a mechanical framework designed for simple model prototyping

Brunet A., ten Tusscher K.H.W.J.
Presented by Armand Brunet

Plant root morphogenesis is a complex process involving the interplay between regulatory networks and cell mechanics. To better understand this process, mechanobiology relies on various computational methods. We present a framework based on a hybrid vertex-and-mass-spring model approach that enables the simulation of root tissue growth and development under different signaling conditions. The proposed tool is designed to provide a simple and flexible platform for studying root development while accounting for the reciprocal interactions between mechanics and signaling. By applying this framework, we aim to investigate how mechanical forces influence cell division, expansion, and differentiation during processes such as lateral root formation. This tool is expected to facilitate the generation of testable hypotheses on root development for subsequent experimental validation.

KEYNOTE 2 · 2 JUNE

Evolution from mixed to fixed handedness in mirror-image flowers: insights from adaptive dynamic

Saltini M., Barrett S.C.H., Deinum E.E.
Presented by Marco Saltini

Mirror-image flowers (enantiostyly) involve a form of sexual asymmetry in which a flower's style is deflected either to the left or right side, with a pollinating anther orientated in the opposite direction. This curious floral polymorphism, which was known but not studied by Charles Darwin, occurs in at least 11 unrelated angiosperm families and represents a striking example of adaptive convergence in form and function associated with cross-pollination by insects. In several lineages, dimorphic enantiostyly (one stylar orientation per plant, both forms occurring within populations) has evolved from monomorphic enantiostyly, in which all plants can produce both style orientations. We use a modelling approach to investigate the emergence of dimorphic enantiostyly from monomorphic enantiostyly under gradual evolution. We show using adaptive dynamics that depending on the balance between inbreeding depression following geitonogamy, pollination efficiency, and plant density, dimorphism can evolve from an ancestral monomorphic population. In general, the newly emergent dimorphic population is stable against invasion of a monomorphic mutant. However, our model predicts that under certain ecological conditions, for example, a decline of pollinators, dimorphic enantiostyly may revert to a monomorphic state. We demonstrate using population genetics simulations that the observed evolutionary transitions are possible, assuming a plausible genetic architecture.

TALK 13 · 2 JUNE

The risk of reduction: modeled HIV-1 dynamics after antibody treatment depend on dimensionality

Kutler Dodd, G., Hogeweg, P., de Boer, R.J.
Presented by Griffin Kutler Dodd

Rapidly evolving viruses form diverse quasispecies that enable escape from immune responses and treatments. For example, HIV-1 can rebound within weeks after broadly neutralizing antibody (bNAb) treatment due to the emergence of high-fitness antibody-escape mutants in the quasispecies. However, most existing models of viral dynamics include only a small number of virus variants and either assume an arbitrary fitness distribution for mutants or require extensive fitting to sparse clinical data. To overcome this, we constructed an extremely high-dimensional HIV-1 quasispecies model parameterized with in silico binding affinity predictions, capturing the dynamics of 3.2 million viral strains. We show that this model qualitatively reproduces observed rebound dynamics after bNAb administration without fitting to experimental data. To compare with lower-dimensional models, we explored different dimensionality reduction methods and found only models in which the included variants are selected based on quasispecies properties, such as total mutational flux, can accurately predict antibody responses. Finally, we introduce an eco-evolutionary design framework that uses our high-dimensional model to optimize antibody binding towards the most important strains in the quasispecies, identifying antibodies that can effectively suppress viremia.

TALK 14 · 2 JUNE

Isolation as a dynamic driver of eco-evolutionary change

Kaminas A., Van Doorn G.S., Etienne R.S.
Presented by Alexandros Kaminas

Isolation is one of the most pervasive forces in biology, yet its consequences remain paradoxical: it can both drive diversification and lead to extinction. Existing theoretical frameworks typically address these patterns separately, focusing either on colonisation–extinction dynamics or on trait evolution in isolation. The aim of this project is to connect these perspectives within a single framework, asking how eco-evolutionary diversification unfolds under mainland gene flow and the persistent risk of local extinction. Building on population-dynamic models of trait evolution, we extend an existing framework to better capture key features of insular systems. The framework introduces three key extensions. It first incorporates an explicit spatial structure via a mainland–island model, allowing isolation to be represented as a variation in colonisation rates. It then allows speciation to arise from ecological and evolutionary feedbacks, rather than being imposed externally as fixed phylogenies. Finally, it further relaxes the assumption of purely competitive interactions, enabling alternative interaction types such as mutualism or parasitism to influence community structure and evolutionary patterns. This framework links trait distributions, species abundances, and phylogenetic structure under varying degrees of mainland connectivity, examining how isolation can either promote diversification or increase the likelihood of extinction. Ongoing work focuses on examining model behaviour across isolation gradients and identifying the conditions that lead to these contrasting regimes. Although motivated by oceanic islands, the approach is broadly applicable to systems characterised by restricted immigration and strong interactions, including fragmented habitats and host-associated microbial communities.

TALK 15 · 2 JUNE

Evolution of interspecific territoriality between species

Boog D., Kokko, H.
Presented by Dajo Boog

Many species establish territories. Despite the costs of defending against intruders, territoriality can be selected due to benefits such as exclusive access to resources or mates. Although territories are commonly defended only against conspecifics, some species also defend their territory against heterospecific individuals. This interspecific territorial behaviour has been argued to be due to the individual misidentifying a heterospecific as a conspecific. However, various studies have shown that the behaviour is not solely caused by misidentification, but that it can be adaptive due to factors like niche overlap or hybridization. However, how strong should territorial defence against other species be? This question is especially relevant because niche overlap between species may be asymmetric. Previous theoretical work has focused primarily on the evolution of recognition of heterospecifics as a competitor, while assuming a fixed territory size and the same level of defence as against conspecific intruders. Here, we investigate interspecific territorial behaviour by allowing the defence efforts of two species against heterospecifics to evolve. The space may either be shared between the species or claimed as a territory, depending on the defensive efforts of both species. We show the conditions under which species do not defend against heterospecifics and fully share the space, as well as when strong interspecific territorial defence evolves. Higher niche overlap between the species increases defensive efforts for a larger territory. However, if the other species has a high interspecific territorial aggression and strongly defends their territory, the best response may likewise be to maintain a high defence effort. Even when both species have no niche overlap and would benefit from sharing the space, this effect may still maintain interspecific territorial aggression.

TALK 16 · 2 JUNE

Anisogamy, then what?

Long, X, Henshaw M.J.
Presented by Xiaoyan Long

The empirical distribution of sex roles is complex. Broad patterns (e.g., mating competition is typically more intense among males while parental care is more commonly provided by females) coexist with numerous exceptions. This complexity has driven sustained controversy over the causal relationship between anisogamy (i.e., sex differences in gamete size) and sex differences in parental care and mating competition. If anisogamy is the fundamental cause of these sex differences, then why are they so variable? Using mathematical models, we show that anisogamy can drive the evolution of sex differences along various contrasting evolutionary trajectories, depending on the ecological context. Anisogamy can thus explain both broad patterns in sex-role evolution and exceptions to these patterns.

KEYNOTE 3 · 2 JUNE

From static snapshots to fluxes: approaches for integrating omics data with genome-scale metabolic models

Adriaens M.E.
Presented by Michiel Adriaens

Pathway analysis remains the dominant lens through which omics data are interpreted. But in the context of metabolism, it offers a static snapshot in which metabolic functions are forced into predefined boundaries and where deciding whether a pathway is "up" or "down" is rarely straightforward. Genome-scale metabolic models (GEMs) provide a way out, by representing metabolism as a network of reactions that can be queried for fluxes rather than enrichments. In my keynote, I will discuss how omics data can be integrated with GEMs to move from descriptive snapshots toward mechanistic, flux-based interpretations, touching on where current approaches still fall short and where the field is moving next. I will first introduce metabolic task analysis as a tractable middle ground between full flux-based analysis and pathway enrichment, scoring functional sub-units of the network from transcriptome data to yield a metabolic fingerprint. The second part focuses on a methodological shift that is reshaping the field: enzyme-constrained GEMs (ecGEMs) and the role of machine learning in making them tractable. Turnover numbers, long the bottleneck for kinetic constraints at genome scale, are now being predicted directly from sequence and substrate structure by deep-learning frameworks such as UniKP, CatPred, and DeepEnzyme. Combined with protein-to-mRNA ratios, these predictions allow transcriptomics to be translated into reaction activities that carry kinetic meaning. I will close by broadening the scope and argue that a single omics dataset can sustain far more analysis than is typically extracted from it, and that combining tools across disciplines will provide more comprehensive insights.
