SCIENTIFIC REPORT :
WORKSHOP MECHANICS and GROWTH of TISSUES :
From Development to Cancer
INSITUT CURIE PARIS France
January 13-17th 2014.

1. Summary

The four days workshop on "Mechanics and Growth of Tissues: From Development to Cancer brought together physicists and biologists, with an interest in tissue behavior during development and cancer. There had been many new developments over the last three years such as optogenetics, new theoretical concepts or studies of the interplay between mechanics and signaling and the meeting was therefore very timely. The meeting was held over 4 days at the Institut Curie in Paris and covered 5 themes: Tissue homeostasis and Cancer, Development, Dynamics and gene networks, Cell division, Novel approaches. 194 researchers attended the meeting. We had 23 Invited Speakers and 12 short talks selected from abstract submissions. 113 posters were presented in 4 distinct poster sessions of 2 hours. Finally Uri Alon gave a general audience talks on Science interaction.

2. Description of the scientific content of and discussions at the event.

The past years have shown that physical biomechanical and biochemical concepts play an increasing role in understanding complex phenomena governing the dynamic organization of cells in healthy and pathological tissues. The behavior of a cell is not an intrinsic property: in a tissue, it strongly depends on its interactions with neighboring cells and of the microenvironment of the cells. The properties of tissues often result from a collective behavior of the cells which self-organize and communicate via different cellular signaling systems. When the physiological conditions vary, drastic changes of behaviors are observed that can be described in terms of bifurcations between different states of the tissue or in terms of dynamic phase transitions, a typical example being the epithelial-mesenchymal transition. It is thus essential to find the right level of description of the collective cell behavior for understanding tissue and organ development, tissue morphogenesis and cancer progression. In order to provide an overview of the advance of the field we have selected 13 oral presentation that covered the following 5 themes:

- The first theme on "Tissue homeostasis and Cancer" covered the mechanics of tissues, signaling with tissues and importantly the feedback between mechanics and signaling.
- The second theme on "Development" covered various aspects of tissue patterning and shape, of cell segregation in tissues, actin flows and dynamics, and of cell migration within tissues.
- The third theme on "Dynamics and gene networks" focused on tissue dynamics and time dependent effects in tissues.
- The 4th theme discussed various aspects of cell division.
- The 5th theme covered new experimental aspects with an emphasis on imaging and more specifically optogenetics.

The X invited oral presentations were given in the following format: : 35 minutes talk + 10 minutes discussion. The format gives the opportunity to the speakers of a long introduction to describe the state of the art in the field and to introduce the necessary concepts to grasp the unpublished and published findings in such a mixed audience. We have selected from 113 abstract 13 oral presentation of a 15 minutes talks + 5 minutes discussion. The 4 posters sections of 2 hours provided the attendees with a sufficient time for extensive discussions. Overall the 5 themes were well covered. The 3rd theme “Dynamics and Gene networkd” was not fully covered and we believe that it will be important to reinforce the characterization of the dynamics of gene network in relation with the mechanical properties of tissues.

3. Assessment of the results and impact of the event on the future directions of the field

In our view the meeting was a great success. The number of participants exceeded our expectation and we had to refuse abstract submissions and participants. The 45 minutes speakers' oral presentation offered the possibility of long introduction so that people from different field could grasp the novelty and impacts of the presented findings. We have selected oral contributions from abstracts of young scientist to promote their work in front of this rather large audience. The attendances of the oral presentation and of the poster sessions were both excellent. The on site lunches and dinners that we provided were key to the interaction between participants. Overall, we believe that the meeting has provided an rich overview of the advance of the field of tissue mechanics in both normal and pathological conditions. The future direction in the field are (i) a better description and characterization of the different length- and time-scales;(ii) the need of a better interaction between classical genetics approaches and physical modeling (iii) a better integration of the "omics" data and physical models in the field of tissue development.

4. Annexes:

4. 1. Program of the meeting

Monday January 13 (Burg Building)	
8.30	Registration
9.45	Welcome and Opening Remarks
SESSION I	Chaired by Yohanns Bellaïche
10.00	**Uri Alon** I1: Evolutionary tradeoffs and the geometry of phenotype space
10.45	**Buzz Baum** I2: The importance of being well-rounded
11.30	**Zeiss Presentation**
11.40	*Coffee Break*
12.00	**Nadine Peyriéras** I3: A complex system approach of embryonic morphogenesis
12.45	*Lunch*
SESSION 2	Chaired by Françoise Brochard
14.00	**Thomas Gregor** I4: Reproducibility of developmental processes
14.45	**Benoit Ladoux** O1: Epithelial bridges maintain tissue integrity during collective cell migration
15.05	**Matthieu Piel** I5: A cell's life under confinement: growth, division and migration when space is limited
15.50	*Coffee Break*
16.30	**Anne Classen** O2: Disruption of epithelial integrity by local boundary mechanisms in Drosophila imaginal discs
16.50	**Ben Simons** I6: Dynamical stem cell heterogeneity in the maintenance of adult tissues
20.00	*Dinner*

Tuesday January 14
(Burg Building)

SESSION 3 Chaired by Vincent Hakim

9.00	**Thomas Lecuit** I7: Biomechanical control of tissue shape changes
9.45	**Boris Shraiman** I8: Mechanics of epithelial morphogenesis: from theory to experiment and back
10.30	*Coffee Break*
11.00	**Manuel Théry** I9: Redistribution of contractile forces during epithelial to mesenchymal transition correlates with polarity reversal
11.45	**Julien Vermot** O3: Endocardial mechanosensitivity is mediated by a trpv4 dependent signaling pathway during valvulogenesis
12.30	*Lunch*

SESSION 4 Chaired by Frank Jülicher

14.00	**Enrico Coen** I10: Polarity, Plants and Picasso: The role and mechanism of tissue cell polarity in plant morphogenesis
14.45	**Jochen Guck** I11: The regulatory role of cell mechanics in differentiation and cancer
15.30	*Coffee Break*
16.00	**Martin Howard** I12: How fission yeast cells sense their size: cortical regulation by a sizer Cdr2
16.45	**Christof Aegerter** O4: Growth control via mechanical feedback in the Drosophila wing imaginal disc
17.05	**Alexis Maizel** O5: Morphogenesis without cell migration: how plants do it
18.00	Poster Session 1- Odd numbers *(BDD Building)*
20.00	*Dinner*
21.00	Poster Session 1- Odd numbers *(BDD Building)*

Wednesday January 15
(ENSCP Building)

SESSION 5 Chaired by Martine Benamar (TBC)

9.00 **David Nelson**
I13: Population Genetics of Three Dimensional Range Expansions

9.45 **Jody Rosenblatt**
I14: Epithelial cell turnover—new roles for mechanical tension driving cell death and division

10.30 *Coffee Break*

11.00 **Daniel Needleman**
I15: Self-Focusing of the Ran Gradient in Mitosis: Signaling, Mechanics, and Spindle Size

11.45 **Jean-Léon Maître**
O6: Cell-autonomous increase in contractility drives compaction of the mouse embryo

12.30 *Lunch (Burg Building)*

SESSION 6 Chaired by Suzanne Eaton

14.00 **Shigeru Kondo**
I16: Pigmentation pattern

14.45 **Jennifer Zallen**
I17: Shaping the embryo: Cellular dynamics in development

15.30 *Coffee Break*

16.00 **Tim Mitchison**
I18: Cell Division in Very Large Cells

16.45 **Paulina Strzyz**
O7: Apical mitosis depends on interkinetic nuclear migration but not centrosome position and ensures controlled tissue development

17.05 **Enrique Martin-Blanco**
O8: Histoblast expansion dynamics during metamorphosis: from kinetics to forces

18.00 Poster Session 2 – Even numbers *(BDD Building)*

20.00 *Dinner (Burg Building)*

21.00 Poster Session 2 - Even numbers *(BDD Building)*

Thursday January 16
(ENSCP Building)

SESSION 7	Chaired by Pascal Silberzan (TBC)
9.00	**Bob Goldstein** I19: Mechanisms of apical cell shape change
9.45	**Yoshihiro Morishita** O9: Bayesian inference of whole-organ deformation dynamics from limited space-time point data
10.25	**G. Wayne Brodland** O10: Can Cell Shape Reveal Which Property Changes Allowed a Cell to Become Metastatic?
10.30	*Coffee Break*
11.00	**Lars Hufnagel** I20: Bio-imaging across scales: from cells to embryos
11.45	**Bo Dong** O11: Mechanical coupling of ECM tension and apical membrane expansion determines epithelial tube size
12.30	*Lunch (Burg Building)*

SESSION 8	Chaired by Jean-François Joanny
14.00	**Christian Dahmann** I21: Signals and mechanics guiding cell segregation in tissues
14.45	**Sirio Dupont** O12: A mechanical checkpoint controls the growth of cells within a monolayer by regulating YAP/TAZ activity
15.05	*Coffee Break*
15.30	**Olivier Pourquié** I22: Towards Physical principles of vertebrate development
16.15	**Jacques Prost** I23: Tissue Mechanics and Multicellular Spheroids
17.00	Conclusions

2. Speakers List and contributions :

I1 - URI ALON

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Weizmann Institute of Science, Rehovot, Israel

Evolutionary tradeoffs and the geometry of phenotype space

Organisms, tissues and molecules often need to perform multiple tasks. But usually no phenotype can be optimal at all tasks at once. This leads to a fundamental tradeoff. We study this using the concept of Pareto optimality from engineering and economics. Tradeoffs lead to an unexpected simplicity in the range of optimal phenotypes- they fall on low dimensional shapes in trait space such as lines, triangles and tetrahedrons. At the vertices of these polygons are phenotypes that specialize at a single task. We demonstrate this using data from animal and fossil morphology, bacterial gene expression and other biological systems.

I2 - BUZZ BAUM

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MRC laboratory for Molecular Biology, London, UK

The importance of being well-rounded

As cells progress through mitosis they undergo profound changes in cytoskeletal organisation and cell shape when dividing in vitro and in a tissue context. These include cell rounding upon entry into mitosis, alignment of the metaphase spindle with cortical cues in response to asymmetries in the environment, and polar relaxation and cell division following the onset of anaphase. Here, I will discuss our attempts to uncover the molecular, cellular and physical mechanisms driving the changes in the actin-based cortex that underlie these events, and the mechanisms used to couple cortical remodelling to mitotic progression. Finally, I will explore potential roles for the metaphase cortex and mitotic rounding in cancer progression.

I3 - NADINE PEYRIÉRAS

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CNRS Gif-sur-Yvette, France

A complex system approach of embryonic morphogenesis

We approach the understanding of tissue growth in normal and pathological conditions through the quantitative analysis and biomechanical modeling of model organisms' embryonic morphogenesis, based on in vivo imaging. The cellular level of organization is taken as resulting from the integration of sub-cellular and supra-cellular processes. Cell dynamics are investigated through 3D+time imaging of developing embryos with fluorescent nuclear and membrane staining. The automated reconstruction of the cell lineage tree, annotated with nucleus and membrane segmentation, provides measurements for cell behavior: identity, fate, displacement, division, shape and contact changes. This quantitative data is sufficient to find statistical models for cell proliferation and cell descriptors evolution in time and space, and characterize the spatial and temporal length scale of cell displacements and tissue deformations. Confronting numerical simulation derived from a multi-agent based biomechanical model with empirical measurements extracted from the reconstructed digital specimens, is the basis for testing hypotheses for processes underlying zebrafish gastrulation and early neurulation, in normal and mutant fish lines. Further correlating cell behavior, tissue biomechanics and biochemical activities by comparing the patterns revealed by cell fate, velocity, strains or gene expression, is a step toward the integration of multi-level dynamics. This overall framework lays the ground for a transdiciplinary approach of living systems' morphogenesis.

I4 - THOMAS GREGOR

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Princeton University, Princeton, USA

Reproducibility of developmental processes

The macroscopic structures of developing multicellular organisms display an extraordinary level of reproducibility from one individual to the next, all the way to the exact location of individual hair bristles on the surface of the skin. The patterns that lead to this reproducibility get established during the earliest stages of embryonic development, and are often governed by individual molecular events. How do we reconcile the precise and reproducible outcomes of development with the uncertainty arising from the fluctuations inherent to low copy number events? I will present our current strategies to make progress, utilizing a number of novel measurement techniques, to understand how macroscopic structures in multicellular organisms are so reliably reproducible.

I5 - MATTHIEU PIEL

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Institut Curie, Paris, France.

A cell's life under confinement: growth, division and migration when space is limited

In tissues, cells have their physical space constrained by neighboring cells and extracellular matrix. In the recent years, we have developed simple and versatile devices to precisely and dynamically control this confinement parameter in cultured cells. I will present results we obtained on the effect of forces and confinement on dividing and migrating cells. Early in mitosis, cells round up by pushing on their surroundings. This is essential to ensure enough space to assemble a proper mitotic spindle. In prometaphase and metaphase, cell shape, as well as forces acting on the cell, together affect the orientation of the mitotic spindle, setting the division axis. Later in the division process, daughter cells re-adhere and spread, while the cytokinetic process is not yet finalized. During this late step, forces cells exert on each other and on the substrate regulate the final abscission process. Confinement also strongly affects the mode of migration, eliciting fast amoeboid migration of weakly adherent cells, including mitotic cells. Finally I will present a new method to accurately follow volume of single cells on long timescales. We found, for all cell types we measured, that cell volume homeostasis is mostly based on the fact that smaller cells have a longer cell division cycle, a process well established for yeast cells, but which was thought to be absent in animal cells. We also found that when cells enter mitosis, they very significantly increase their volume, due to osmotic swelling, a feature that, together with cortical stiffening, might help them to push on their neighbors and round up when dividing under confinement in a tissue.

I6 - BEN SIMONS

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Cavendish Laboratory, Cambridge, UK

Dynamical stem cell heterogeneity in the maintenance of adult tissues

In adult, tissues are maintained and repaired by stem cells, which divide and differentiate to generate more specialized progeny. The mechanisms that control the balance between stem cell proliferation and differentiation promise fundamental insights into the organization of tissues, and the factors leading to their dysregulation in disease. However, stem cells are difficult to distinguish from their more differentiated progeny, and resolving these mechanisms has proved challenging. In recent years, the quantitative analysis of static lineage tracing assays, based on the study of transgenic mouse models, have revealed conserved patterns of stochastic stem cell fate across different tissues and organisms. However, the cellular basis of stochastic fate choice has remained elusive. Here, by combining novel in vivo live-imaging assays with static marker based assays, we show that the long-term maintenance of both mouse germ line and intestinal epithelium involve the reversible transfer of stem cells between states primed for renewal and differentiation. By showing that stem cell function is shared among a dynamically interconverting heterogeneous pool, these studies offer a new perspective on the maintenance of adult tissues.

I7 - THOMAS LECUIT

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Biomechanical control of tissue shape changes

Epithelial tissues exhibit a remarkable dual property of robustness and fluidity. This operates on different time

scales and relies on unique mechanical properties of the cell cortex and on adhesive interactions between cells. We seek to understand the fundamental molecular mechanisms responsible for this property. This is essential to understand morphogenesis of developing embryos and organs, and is severely affected in a number of disease, in particular cancer progression. To that end we develop a range of approaches, from the genetic and pharmacological perturbations of molecular components, the quantitative imaging of proteins using a variety of photonic methods, probing of the physical properties of cells within intact tissues, and computational modelling of morphogenesis at different scales (molecular to tissue scales). I will present our recent progress in understanding how adhesion and cortical tension regulate the dynamic remodelling of cell contacts in the primary epithelium of Drosophila embryos. I will first focus on the regulation of tensile activity driving cell shape changes. I will also address how E-cadherin-actin interactions control force transmission at cell interfaces. Last I will address how biochemical signals control the spatial patterns of actomyosin contractility.

I8 - BORIS SHRAIMAN

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Mechanics of epithelial morphogenesis: from theory to experiment and back

I9 - MANUEL THÉRY

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Redistribution of contractile forces during epithelial to mesenchymal transition correlates with polarity reversal

Mammary gland development as well as breast cancer progression relie on the tissue plasticity associated to epithelial to mesenchymal transition (EMT) and its reversion by mesenchymal to epithelial transition. EMT has been shown to involve a profound reprograming of gene expression as well as cell adhesion and cell cytoskeleton remodelling. These changes allow the highly connective epithelial cells to orchestrate their modification into an alternate morphology more conductive to migration. During the transition, cells switch from an apico-basal polarity to a front-rear polarity. Here we used a minimal tissue made of cell doublets, on micropatterned and deformable substrates, to investigate the relationship between cell mechanics and cell polarization during EMT. We found that cells undergo polarity reversal as revealed by the repositioning of the centrosome/Golgi on the opposite side of the nucleus. In parallel cells reverse the balance of intra versus intercellular tensional forces. These results suggest that the reorientation of mammary epithelial cell function, from a lumen-oriented cohesive and static state, to a stroma-oriented mesenchymal and migratory state, is powered by intracellular organelle repositioning coupled to mechanical force rebalancing.

I10 - ENRICO COEN

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John Innes Centre, Norwich , UK

Polarity, Plants and Picasso: The role and mechanism of tissue cell polarity in plant morphogenesis

Development involves highly oriented cell behaviours, such as anisotropic growth and asymmetric cell divisions. It is unclear how these orientations are specified and how they lead to particular cellular or tissue outcomes. We have been addressing this problem using a combination of genetic, morphological, computational and imaging. The results provide new insights into how genes interact to specify orientations of growth and division, leading to particular shapes. The talk will illustrate how integrating biological and computational methods may lead to a quantitative mechanistic framework for development.

I11 - JOCHEN GUCK

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Technische Universität Dresden, Dresden, Germany

The regulatory role of cell mechanics in differentiation and cancer

The mechanical properties of cells are increasingly being investigated as they prescribe the response to external

forces and define the physical limits of a cell's interaction with its three-dimensional tissue environment. Largely determined by the cytoskeleton, an internal polymer network regulated by intricate biochemical processes, cell mechanics also has an important biological component. The cytoskeleton is central to many biological functions, specifically evolves during the normal differentiation of cells, and is characteristically altered in many pathologies, including inflammation and cancer. We have shown that during the course of differentiation of human myeloid precursor cells into three different lineages, the cells alter their viscoelastic properties to suit their ultimate fate and function. Myeloid cells circulating in blood are compliant at short time-scales as they have to be advected through constrictions in blood vessels. In contrast, cells required to migrate through tissue pores at long time-scales (> minutes) have reduced steady-state viscosity. Apparently, a reduction in steady-state viscosity is a physiological adaptation for enhanced migration through tissues and rationalizes our earlier finding that metastatic cancer cells have particularly high compliance. Our results indicate that the material properties of cells define their function, can be used as a cell differentiation marker, and could serve as target for novel cancer therapies.

I12 - MARTIN HOWARD

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John Innes Centre, Norwich UK

How fission yeast cells sense their size: cortical regulation by a sizer Cdr2

Cells can in principle control their size by growing to a specified size before commencing cell division. How any cell actually senses its size remains poorly understood. Fission yeast (Schizosaccharomyces pombe) are rod-shaped cells that grow to ~14 μm in length before entering mitosis. Here, we find that a peripheral membrane protein kinase cdr2p has properties of a dose-dependent "sizer" that controls mitotic entry. As cells grow, the local cdr2p concentration in nodes at the medial cortex accumulates as a measure of cell size. Our findings, which challenge a previously proposed pom1p gradient model, suggest a new model where cdr2p reads out cell size by probing the surface area over the whole cell and relaying this information to the medial cortex.

I13 - DAVID NELSON

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Harvard University, Cambridge, USA

Population Genetics of Three Dimensional Range Expansions*

We develop a simple model of genetic diversity in growing spherical cell clusters, where the growth is confined to the cluster surface. This kind of growth occurs in cells growing in soft agar, and can also serve as a simple model of avascular tumors. Mutation-selection balance in these radial expansions is strongly influenced by scaling near a neutral, voter model critical point and by the inflating frontier. We develop a scaling theory to describe how the dynamics of mutation-selection balance is cut off by inflation. Genetic drift, i.e., local fluctuations in the genetic diversity, also plays an important role, and can lead to the extinction even of selectively advantageous strains. We calculate this extinction probability, taking into account the effect of rough population frontiers. *Joint work with Max Lavrentovich

I14 - JODY ROSENBLATT

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University of Utah, Utah, USA

Epithelial cell turnover—new roles for mechanical tension driving cell death and division

Cells growing on their own in a culture dish, divide or die depending upon whether growth factors or apoptotic stimuli are present. However, cells comprising the epithelia that coat organs need to maintain constant numbers so that they preserve their primary function as a barrier without amassing into tumors. We have found that epithelia maintain homeostatic cell numbers through mechanical tensions. When cells become crowded, they activate the stretch-activated channel, Piezo 1, to trigger cells to extrude out of the layer and later die. When there are too few, stretching, conversely induces cells to rapidly divide by ramping up Cyclin B levels. Thus, stretch-activated channels could regulate both cell death and division once epithelia reach homeostatic densities to maintain constant cell numbers.

I15 - DANIEL NEEDLEMAN

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Harvard University, Cambridge, USA

Self-Focusing of the Ran Gradient in Mitosis: Signaling, Mechanics, and Spindle Size

During spindle assembly, microtubules are highly enriched near chromatin by a process which, in many systems, is driven by the GTPase Ran. The Ran pathway has been proposed to establish a reaction-diffusion network that generates gradients in the behaviors of soluble proteins around chromatin, but the manner in which this happens is poorly understood. To better characterize the behavior of the Ran pathway, we developed a novel form of fluorescence fluctuation spectroscopy capable of quantitatively measuring the concentration, diffusion, and interactions of soluble proteins simultaneously at hundreds of locations throughout cells. We use this technique to study the behaviors of soluble Ran, importin-alpha, importin-beta, RanBP1, RanBP2, RanGAP, and a variety of downstream cargo proteins throughout mitotic human tissue culture cells, and we investigate how the spatial organization of this network changes in response to perturbations. Our results suggest that a self-focusing of the Ran pathway is produced by an interplay between soluble gradients of upstream signaling molecules and the mechanics of the microtubule network they generate. This feedback has interesting implications for models of spindle assembly and the maintenance of spindle size.

I16 - KONDO SHIGERU

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Osaka University, Osaka, Japan

Pigmentation pattern

The reaction-diffusion mechanism, presented by AM Turing more than 60 years ago, is currently the most popular theoretical model explaining the biological pattern formation including the skin pattern. This theory suggested an unexpected possibility that the skin pattern is a kind of stationary wave (Turing pattern or reaction-diffusion pattern) made by the combination of reaction and diffusion. At first, biologists were quite skeptical to this unusual idea. However, the accumulated simulation studies have proved that this mechanism can not only produce various 2D skin patterns very similar to the real ones, but also predict dynamic pattern change of skin pattern on the growing fish. Now the Turing's theory is accepted as a hopeful hypothesis, and experimental verification of it is awaited. Using the pigmentation pattern of zebrafish as the experimental system, our group in Osaka University has been studying the molecular basis of Turing pattern formation. We have identified the genes related to the pigmentation, and visualized the interactions among the pigment cells. With these experimental data, it is possible to answer the crucial question, "How is the Turing pattern formed in the real organism?" The pigmentation pattern of zebrafish is mainly made by the mutual interactions between the two types of pigment cells, melanophores and xanthophores. All of the interactions are transferred at the tip of the dendrites of pigment cells. In spite of the expectation of many theoretical biologists, there is no diffusion of the chemicals involved. However, we also found that the lengths of the dendrites are different among the interactions, which can substitute the difference of diffusion constant in the RD model. Therefore the real mechanism we found in the zebrafish skin is not the classic RD mechanism, but is mathematically equivalent to the original Turing mechanism.

I17 - JENNIFER ZALLEN

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Sloan-Kettering Institute, New York, USA.

Shaping the embryo: Cellular dynamics in development

A major challenge in biology is to understand how large-scale changes in tissue structure are generated on a cellular and molecular level. In the fruit fly *Drosophila*, the characteristic elongated shape of the head-to-tail axis is achieved through the rapid and coordinated movements of hundreds of cells. We found that these movements are oriented by cellular asymmetries in the localization of the molecular machinery that generates contractile and adhesive forces between cells. Using quantitative imaging, we showed that these asymmetries result in higher-order collective cell behaviors in which groups of cells assemble into multicellular rosette structures that form and resolve in a strictly polarized fashion, promoting efficient elongation. Rosettes form through a combination of biochemical and mechanical signals that orient actomyosin contractile activity. An initial asymmetry in the localization of the myosin II motor protein is amplified by mechanical tension, promoting the formation of

multicellular contractile networks that contract to promote efficient elongation. In addition, the dynamics of cell adhesion proteins are controlled by the spatially regulated activation of tyrosine kinase signaling at cell-cell junctions that are targeted for disassembly, demonstrating an essential role for tyrosine kinase signaling in spatially regulated cell interactions during development. Multicellular rosette behaviors have since been shown to occur during epithelial elongation in vertebrates and may represent a general mechanism linking cellular asymmetry to tissue elongation. We are currently using molecular genetic and live imaging approaches to understand how genes encode the forces that generate polarized cell behavior, and developing biophysical methods to elucidate the mechanotransduction mechanisms that allow cells to modify their behavior in response to their mechanical environment.

I18 - TIM MITCHISON

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Harvard Medical School, Boston, USA

Cell Division in Very Large Cells

One of the big challenges in biology is to understand how cells physically self-organize using molecules that are much smaller than the cell. This challenge is epitomized by frog eggs and early embryos, where cell are hundreds of microns in diameter. After fertilization frog eggs cleave in the middle, and then cleave again at right angles, on their way to becoming embryos. The question of how these cleavage planes are accurately positioned has interested biologists for centuries. We have studied this problem using microscopy and biochemistry in frog and fish eggs, and in cell free extracts made from frog eggs. The answers lie in the behavior of starburst-like arrays of microtubules called asters that grow out of centrosomes, and in how these asters grow and interact inside the egg. I will describe our progress in understanding how large asters grow to fill the cell, what happens when two asters meet, how asters move within the cell, and how these processes together determine cleavage plane geometry.

I19 - BOB GOLDSTEIN

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Mechanisms of apical cell shape change

Apical constriction changes cell shapes, driving fundamental morphogenetic events, including gastrulation in diverse organisms and neural tube closure in vertebrates. Apical constriction is thought to be triggered by contraction of apical actomyosin networks. I will present results from my lab showing that such actomyosin contractions begin before cell shape changes in both C. elegans and Drosophila, demonstrating that such contractions must not be not sufficient for cell shape change in vivo, and that other events must trigger cell shape change in response to actomyosin contractions. In C. elegans, actomyosin networks are initially dynamic, contracting and generating significant cortical tension without substantial shrinking of apical surfaces at first. Apical cell-cell contact zones and actomyosin only later move increasingly in concert, with no detectable change in actomyosin dynamics or in cortical tension. Thus, apical constriction appears to be triggered not by a change in actomyosin dynamics or cortical tension, but by dynamic linking of apical cell- cell contact zones to an already contractile apical cortex. I will also present our work seeking to identify molecular mechanisms of cell shape change that have been conserved across the bilaterally symmetrical animals.

I20 - LARS HUFNAGEL

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Bio-imaging across scales: from cells to embryos

I21 - CHRISTIAN DAHMANN

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MPI-CBG, Dresden, Germany

Signals and mechanics guiding cell segregation in tissues

Segregating cell populations with distinct functions and fates is crucial for animal development. Maintaining straight boundaries between different cell populations within tissues requires mechanisms to counteract cell rearrangements and cell mixing caused by cell division and tissue reshaping. Local increases in mechanical tension are important in segregating cell populations at boundaries within tissues, yet the signals that control increases in mechanical tension and the mechanisms by which mechanical tension influences cellular dynamics to segregate cell populations remain unknown. Here we demonstrate that the Hedgehog signaling pathway is necessary and sufficient to increase mechanical tension along the boundary between anterior and posterior cell populations in Drosophila wing imaginal discs. Moreover, by quantitatively analyzing cellular dynamics in the vicinity of tissue boundaries in pupal Drosophila histoblasts, we show that cell mixing within the same cell population involves multiple cell intercalations. Cells also intercalate along boundaries between different cell populations, junctional rearrangements during intercalation, however, are biased to disfavor cell mixing. Simulations of tissue growth with two cell populations suggest that local increases in mechanical tension can account for the observed bias in junctional rearrangements during intercalation. We propose that Hedgehog signaling induces local increases in mechanical tension and that mechanical tension guides cell segregation at tissue boundaries by biasing cell intercalations.

I22 - OLIVIER POURQUIÉ

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Towards Physical principles of vertebrate development

I23 - JACQUES PROST

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Tissue Mechanics and Multicellular Spheroids

After introducing the notion of homeostatic pressure, I will subsequently introduce dynamical equations, which exhibit fluid like behavior on time scales long compared to duplication and apoptosis times, in the vicinity of homeostatic conditions. Subsequently, I will describe stress-clamp experiments, which provide numbers on the effects of stress on cell division and apoptosis and discuss what do we learn from these experiments.

4.3. Participant List:

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