FONDATION LOUIS-JEANTET

2016 LOUIS-JEANTET SYMPOSIUM

12 October 2016
Centre Medical Universitaire (CMU), Geneva
Auditorium 250

Alexander Stark

The Research Institute of Molecular Pathology (IMP), Vienna, Austria

Wednesday, 12 October 2016, 09:00 – 09:25

Decoding transcriptional regulation

In higher eukaryotes, genes are expressed dynamically in complex spatial and temporal patterns, which are progressively refined to set up body plans and define specific cell-types. Each cell is able to read the regulatory information in promoter- and enhancer sequences to express specific sets of genes. In contrast, we cannot decipher this regulatory code – a large knowledge gap compared to our detailed understanding of the genetic code, which allows us to seamlessly translate DNA into protein sequences and to determine the protein-coding gene content of any newly sequenced genome.

I am presenting our work towards understanding the regulatory code in Drosophila and human cells by an interdisciplinary approach. We functionally characterize regulatory sequences by enhancer screens in Drosophila embryos and fly and human cell-culture and assessing core promoter activities of large candidate libraries. Computational motif analyses coupled to supervised machine-learning methods are powerful tools that allow us to determine motifs that are shared in functionally related sequences and are promising candidates to explain regulatory function. We also study how enhancers activate different types of promoters and how enhancer – core-promoter specificity is encoded in the two elements' sequences. We finally dissect the combinatorics of transcription factors and transcriptional cofactors at enhancers by directed tethering in enhancer complementation assays, which revealed functionally distinct classes of transcription factors and cofactors.

Biography

After having completed his diploma studies in biochemistry at the University of Tuebingen and his PhD studies at the EMBL and the University of Cologne in 2004, Alexander Stark moved to the Broad Institute (MIT and Harvard) and CSAIL (MIT) for his postdoc. Returning to Europe in 2008, he took up a group leader-position at the Research Institute of Molecular Pathology (IMP) in Vienna. In early 2015, Stark was promoted to Senior Scientist and became part of the scientific lead team at the IMP. Stark's work focuses on studying regulators of gene expression by combining systematic genome-wide experiments and computational analyses.

http://www.starklab.org/

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12 October 2016
Centre Medical Universitaire (CMU), Geneva
Auditorium 250

Eileen Furlong

Head of the Genome Biology Unit/Dept, EMBL Senior Scientist
Genome Biology Unit, EMBL, Heidelberg, Germany

Wednesday, 12 October 2016, 09:25 – 09:50

Genome regulation during developmental transitions

Embryonic development requires the coordinated expression of genes in both a space and time manner. This complex regulation is controlled through the binding of transcription factors (TFs) to enhancer elements, sometimes located at great distances from their target gene. Chromatin conformation studies have shown that gene activation by remote enhancers is associated with the establishment of a chromatin loop to the promoter element. However, the dynamics and general properties of loop formation during embryonic development, as well as their relationship to TF occupancy and chromatin state of developmental enhancers remains poorly understood. This talk will discuss our ongoing efforts to resolve the interplay between chromatin topology, enhancer activity and the regulation of gene expression during embryonic development. We are integrating information from 4C HiC, CRISPR-Cas9 deletions and more. This talk will discuss these results, and how we are complementing these efforts with genome engineering and DNA FISH and at multiple stages of development.

Biography

Eileen Furlong studied biochemistry at University College Dublin, where she obtained her Ph.D. in the regulation of immediate early response genes. She studied developmental biology during her postdoctoral studies at Stanford University, and became a group leader at the European Laboratory for Molecular Biology in Heidelberg in 2002. Since 2009, she is head of the Genome Biology Unit/Dept at EMBL, and Senior Scientist. She is an advanced ERC investigator, elected EMBO member, and a member of many review boards.
Her research spans the areas of transcription/chromatin regulation and developmental biology using the integration of genetics, genomics and computational biology. In particular, her laboratory is associated with dissecting general principles by which developmental enhancers function and how robustness is imparted within developmental programs.

http://furlonglab.embl.de/

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Centre Medical Universitaire (CMU), Geneva
Auditorium 250

Jean-Paul Vincent

The Francis Crick Institute, London

Wednesday, 12 October 2016, 09:50 – 10:15

Signals that control growth and patterning in developing epithelia

Numerous signals control tissue growth during development. It has become clear that, in addition to systemic signals such as insulin-like peptides, patterning signals significantly contribute to tissue growth. For example, the two key signals that pattern wing imaginal discs of Drosophila, Wingless and Dpp, are required for tissue growth. Both proteins have been suggested to form a concentration gradient that positions patterning elements. However, the role of graded signalling in growth control is still a subject of debate. Our long term aim is to determine how patterning signals cooperate with each other and with systemic signals to ensure timely growth. I will first discuss how the range of Wg and Dpp is established and modulated. I will then outline our attempts to control the range, intensity and timing of these signals in order to assess how signalling activity contribute to tissue growth.

Biography

Jean-Paul Vincent studied applied physics at the University of Louvain before completing a PhD in biophysics at UC Berkeley. There he showed that a subcortical rotation in the frog egg specifies the embryonic axis. During his post-doc at UCSF, he helped devise the first photoactivatable cell lineage tracer. In1993, Jean-Paul became a principal investigator at the LMB in Cambridge before moving to the National Institute for Medical Research (London). He is now at the Francis Crick Institute where his interests span the signaling processes that control growth, apoptosis and patterning in developing epithelia, using Drosophila as a model system.

http://www.crick.ac.uk/research/a-z-researchers/researchers-v-y/jean-paul-vincent/

http://jpvincentlab.com

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Centre Medical Universitaire (CMU), Geneva
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Jürgen A. Knoblich

IMBA – Institute of Molecular Biotechnology, Vienna, Austria

Wednesday, 12 October 2016, 10:15 – 10:40

Modelling human brain development and disease in 3D organoid culture

The human brain is highly unique in size and complexity. While many of its characteristics have been successfully studied in model organisms, recent experiments have emphasized unique features that cannot easily be modelled in animals. We have therefore developed a 3D organoid culture system derived from human pluripotent stem cells that recapitulates many aspects of human brain development. These cerebral organoids are capable of generating several brain regions including a well-organized cerebral cortex. Furthermore, human cerebral organoids display stem cell properties and progenitor zone organization that show characteristics specific to humans. Finally, we use both RNAi and patient specific iPS cells to model microcephaly, a human neurodevelopmental disorder that has been difficult to recapitulate in mice. This approach reveals premature neuronal differentiation with loss of the microcephaly protein CDK5RAP2, a defect that could explain the disease phenotype. Our data demonstrate an in vitro approach that recapitulates development of even this most complex organ, which can be used to gain insights into disease mechanisms.

Biography

Jürgen Knoblich did his PhD in the laboratory of Christian Lehner at the Friedrich Miescher Institute of the Max Planck Society in Tübingen. After a postdoctoral period in the laboratory of Yuh Nung Jan at UCSF, San Francisco, he joined the IMP in 1997 as a junior group-leader. In 2004, he moved to IMBA where he is now senior scientist and deputy director.

The Knoblich lab uses Drosophila and mouse genetics, as well as 3D culture models derived from human ES or iPS cells to identify the molecular mechanisms that control asymmetric cell division and the balance between proliferation and differentiation in neural stem cell lineages.

http://www.imba.oeaw.ac.at/research/juergen-knoblich/

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Maria-Elena Torres-Padilla

Institut de Génétique et de Biologie Moléculaire et Cellulaire, CNRS/INSERM U964, U de S, F-67404 Illkirch, CU de Strasbourg, France, Institute of Epigenetics and Stem Cells, Helmholtz Centre Munich, D-81377 Munich, Germany.

Wednesday, 12 October 2016, 11:10 – 11:35

Heterochromatin remodeling is essential for reprogramming at fertilization

Upon fertilization in mammals the highly differentiated gametes are reprogrammed to create a totipotent zygote. This reprogramming process is highly efficient in contrast to artificial reprogramming. One of the major features occurring during preimplantation development is the dramatic remodeling of constitutive heterochromatin, although the functional relevance of these chromatin changes is unknown. Here we show that heterochromatin remodeling is essential for natural reprogramming at fertilization. Enforcing the maintenance of constitutive heterochromatin, through ectopic expression of heterochromatin modifiers, results in the failure to generate blastocysts and the failure of nuclear transfer-based reprogramming. Heterochromatin propagation in vivo is prevented by regulation through RNA and acts as a determining signal for parental epigenetic asymmetry. Overall, we document the functional importance for the restricted transmission of constitutive heterochromatin during reprogramming.

Biography

Maria-Elena did her undergraduate studies at the Faculty of Sciences of the UNAM, Mexico and obtained her Ph.D at the Institut Pasteur in Paris in 2002. She was a postdoctoral fellow at The Gurdon Institute, University of Cambridge, UK between 2002 and 2006. She then worked as senior scientist with Laszlo Tora until 2008. She leads the team “Epigenetics and cell fate in early mammalian development’ at the IGBMC in Strasbourg, France since december 2008 and has recently become the Director of the newly established Institute of Epigenetics and Stem Cells at the Helmholtz Centre Munich, in Germany. Maria-Elena is an EMBO Young Investigator, holds an ERC starting grant and was elected EMBO member in 2015.
Research in her laboratory focuses on understanding how early mouse development is regulated by chromatin-mediated changes in gene regulation. In particular, she is interested in understanding how the transitions in cell potency and cell fate are regulated by chromatin-mediated processes. She has become a leader in the fields of developmental biology and epigenetics. By using early mouse embryo as a unique model system she studies totipotency in vivo and the de novo establishment of chromatin domains and how chromatin regulates cell fate decisions and the consequent generation of pluripotent cells in the embryo.

www.hmgu.de/ies

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Auditorium 250

Christof Niehrs
Mainz Professor

Institute of Molecular Biology, Mainz, Germany
DKFZ, Heidelberg, Germany

Wednesday, 12 October 2016, 11:35 – 12:00

Not so canonical Wnt signaling

Canonical Wnt signaling is thought to regulate cell behavior mainly by inducing b-catenin-dependent transcription of target genes. In proliferating cells Wnt signaling peaks in the G2/M phase of the cell cycle, but the significance of this 'mitotic Wnt signaling' is unclear. I will introduce Wnt-dependent stabilization of proteins (Wnt/STOP), which is independent of b-catenin and peaks during mitosis. Wnt/STOP plays a critical role in protecting proteins from GSK3-dependent polyubiquitination and degradation. Wnt/STOP signaling increases cellular protein levels and cell size. Wnt/STOP rather than b-catenin signaling is the dominant mode of Wnt signaling in several cancer cell lines and gametes.

Biography

Christof Niehrs did his PhD at EMBL in Heidelberg, Germany in 1990. He then moved to the University of California Los Angeles for his postdoc before starting his lab as head of the Division of Molecular Embryology at the German Cancer Research Center (DFKZ) in Heidelberg. In 2010 he founded and was appointed director of the Institute of Molecular Biology (IMB) in association with the University of Mainz. He has been awarded numerous awards, including the Gottfried Wilhelm Leibniz Prize from the DFG (German Research Foundation) and is elected member of several learned societies, including the American Academy of Arts and Sciences.

http://www.dkfz.de/en/mol_embryology/index.php
https://www.imb.de/research/niehrs/research/

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Edith Heard
Professor of Epigenetics and Cellular Memory at the Collège de France

Unité Génétique et biologie du développement, Institut Curie, France

Wednesday, 12 October 2016, 12:00 – 12:25

Developmental dynamics of X-chromosome inactivation: how to establish, maintain and reactivate gene silencing on a chromosome-wide scale

Understanding the regulation of gene expression during normal development is crucial for our comprehension of the alterations that can lead to cancer. Using the process of X-chromosome inactivation as a model system, we are developing approaches that allow us to gain insights into the fundamental mechanisms that underlie the dynamics of gene expression during development and cellular differentiation, as well as during tumorigenesis.
During development, specialized cell types emerge from a common single progenitor thanks to the expression of specific sets of genes, and the silencing of other genes. The differences between cell types are not due to DNA sequence differences but rather to what is often referred to as epigenetic variation.
The aim of our group is to understand how cells can express their genomes differentially and in a stable, although sometimes reversible, manner during development, using X-chromosome inactivation as our model. X inactivation is a normal process, entailing the silencing of one of the two X chromosomes in female mammals. Once established the silent state is stably maintained through cell divisions and throughout the adult life, but can be reversed at certain stages of development, in the germ line and possibly in cancer cells. The inactive X provides a unique model of chromosome-wide epigenetic silencing.

Biography

Edith Heard obtained her PhD at Imperial Cancer Research Fund laboratory in London. She then moved to the laboratory of Philip Avner at Pasteur Institute for her posdoc before setting up her lab at Institut Curie in 2001. She is now director of the Genetics and Developmental Biology Unit at Institut Curie and a Professor of Epigenetics and Cellular Memory at the Collège de France since 2012.

http://ugbdd.curie.fr/en/team_heard

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Joanna Wysocka
Professor of Chemical and Systems Biology and of Developmental Biology

Stanford University School of Medicine, Stanford, CA, USA

Wednesday, 12 October 2016, 13:40 – 14:05

Gene regulatory mechanisms in human development and evolution

Interactions between the genome and its cellular and signaling environments, which ultimately occur at the level of chromatin, are the key to comprehending how cell-type-specific gene expression patterns arise and are maintained during development or are misregulated in disease. Research in our laboratory is focused on understanding how cis-regulatory information encoded by the genome is integrated with the transcriptional machinery and epigenetic regulation to allow for emergence of form and function during embryogenesis. Central to the cell type-specific transcriptional regulation are distal cis-regulatory elements called enhancers, which function in a modular way to provide exquisite spatiotemporal control of gene expression during development. We are using a combination of genomic, genetic, biochemical, and single-cell approaches to investigate how enhancers are activated in response to developmental stimuli, how they communicate with target promoters over large genomic distances to regulate transcriptional outputs, and what is the role of chromatin modification and remodeling in facilitating or restricting enhancer activity.
While studies in model organisms have led to great progress in unveiling the conserved mechanisms of gene regulation, many aspects of development that are unique to humans and other primates remain unexplored, as are regulatory principles underlying emergence of human-specific traits. I will discuss some of our recent progress in understanding transcriptional mechanisms governing human development and evolution, such as our recent quantitative analyses of cis-regulatory divergence in the human and chimpanzee neural crest, an embryonic cell population that is most relevant for evolution of human craniofacial form.

Biography

Polish-born biochemist Joanna Wysocka obtained her Ph.D. at Cold Spring Harbor Laboratory in New York. After her post-doctoral fellowship at Rockefeller University, where her research focused on chromatin biology, Wysocka established an independent laboratory in the Departments of Chemical and Systems Biology and Developmental Biology at Stanford University. There she shifted her focus to stem cell biology, and, in 2010, she won the Outstanding Young Investigator Award from the International Society for Stem Cell Research. Honored with a Vilcek Prize for Creative Promise in Biomedical Science, in 2013, and an HHMI Investigator appointment in 2015, Wysocka continues to build on her work, uncovering crucial insights into cell fate and lineage.
https://med.stanford.edu/profiles/joanna-wysocka?tab=bio

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Auditorium 250

Thomas Lecuit

IBDM – Institut de Biologie du Développement de Marseille, France

Wednesday, 12 October 2016, 14:05 – 14:35

Biomechanical control of tissue morphogenesis

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.
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 polarization of cortical tension underlies the dynamic cell shape changes and tissue morphogenesis. I will report recent findings delineating a novel GPCR signalling pathway responsible for the spatial regulation of cortical tension by the Rho1 pathway during tissue invagination and tissue extension. Evidence of mechanical feedbacks will be reported and discussed.

Biography

Thomas Lecuit is a group leader at IBDM, Marseille and a tenured research director from CNRS. His research aims at understanding how biological forms emerge from information and mechanics.
He made contributions to cellular and biophysical understanding of developmental processes. In an effort to foster interdisciplinary research in biology he coordinates a network on the quantitative biology of signalling in Marseille (*Labex InForm*) and is the director of a new centre, the Turing Centre for Living Systems to develop computational and theoretical approaches to biology in Marseille.
He is an elected member of the EMBO, member of French Académie des Sciences and of Academia Europaea.

http://www.ibdm.univ-mrs.fr/equipe/cell-polarity-and-tissue-morphogenesis/

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Cédric Blanpain

WELBIO, IRIBHM, UNIVERSITE LIBRE DE BRUXELLES (ULB)

Wednesday, 12 October 2016, 14:30 – 14:55

Cancer cell of origin and tumor heterogeneity

Different theories have been proposed to explain tumour heterogeneity including the cancer cell of origin. Here, we developed genetically engineered mouse models allowing lineage tracing together with oncogenic activation in different cell lineages of the skin epidermis and the mammary gland and assessed the role of the cancer cell of origin in regulating tumour heterogeneity. I will present different evidence that the cancer cell of origin controls tumour heterogeneity in epithelial cancers. I will discuss the underlying molecular mechanisms that promote tumor differentiation, propagation, EMT, metastasis and resistance to therapy. These results have important implications for the understanding of the mechanisms controlling tumor heterogeneity and the development of new strategies targeting essential cancer cell functions.

Biography

Cédric Blanpain, MD/PhD, is full professor and WELBIO investigator at the IRIBHM, Université Libre de Bruxelles, Belgium. His lab is studying the role of epithelial stem cells during development, homeostasis and cancer. His lab uncovered new populations of stem cells and progenitor cells in the epidermis, prostate and mammary gland, and identified the cancer cell of origin in these different tissues. Using novel genetic approaches, they demonstrated the existence of cancer stem cells within their natural microenvironment and identified intrinsic and extrinsic mechanisms regulating their functions. Cedric Blanpain is supported by the Belgian FNRS, WELBIO and the European Research Council. He received several important awards including the EMBO Young investigator, outstanding young investigator award of the International Society of Stem Cell Research (ISSCR), or the Liliane Bettencourt award for Life Sciences. He is EMBO member since 2012.

http://blanpainlab.ulb.ac.be/index.htm

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Auditorium 250

Silvia Arber
Professor of Neurobiology

Biozentrum, University of Basel and Friedrich Miescher Institute, Basel, Switzerland

Wednesday, 12 October 2016, 14:55 – 15:20

Disentangling neuronal circuits for motor control

Movement is the behavioral output of the nervous system. Animals carry out an enormous repertoire of distinct actions, spanning from seemingly simple repetitive tasks like walking to more complex movements such as forelimb manipulation tasks. This talk will focus on recent work elucidating assembly, organization and function of neuronal circuits at the core of choosing, maintaining, adjusting and terminating distinct motor behaviors. It will show that dedicated circuit modules within different brainstem nuclei and their interactions in the motor system play key roles in action diversification.

Biography

Silvia Arber is Professor of Neurobiology at the Biozentrum of the University of Basel and Senior Investigator at the Friedrich Miescher Institute for Biomedical Research (FMI) in Basel, Switzerland. Arber graduated in Pico Caroni's laboratory at the FMI in 1995. After a postdoc in Thomas Jessell's laboratory at Columbia University, she established her research group at the Biozentrum and the FMI in 2000. She has been awarded numerous prizes, including the National Latsis Prize (2003), the Schellenberg Prize and the Friedrich Miescher Award (2008), the Otto Naegeli Prize (2014), and the Premio Remedios Caro Almela (2015).

http://www.biozentrum.unibas.ch/research/groups-platforms/overview/unit/arber/

http://www.fmi.ch/research/groupleader/?group=2

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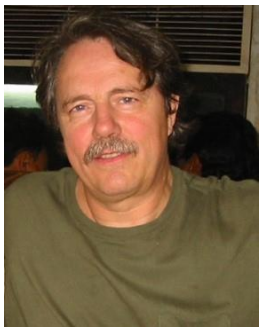

Claude Desplan
Silver Professor of Biology, Director

Center for Developmental Genetics, Department of Biology, New York University, New York.

Wednesday, 12 October 2016, 15:50 – 16:15

Generation of neuronal diversity through temporal and spatial patterning

The Drosophila visual system is composed of the retina and the optic lobes, lamina, medulla, and lobula complex. These structures receive retinotopic inputs from photoreceptors specialized in motion vision (lamina), or color and polarized light vision (medulla). At least 100 types of neurons in the optic lobes process these inputs for extracting visual information.
The medulla contains 40,000 neurons of more than 80 cell types that are organized in 800 columns corresponding to the 800 unit eyes in the retina (ommatidia). How is this variety of neurons generated and how is retinotopy established? Medulla neurons are born from 800 neural stem cells that sequentially express five transcription factors in a temporal manner, similar to the sequence of transcription factors observed in embryonic neural stem cells. Different neurons emerge in each temporal window, therefore generating a series of 800 neurons of each type: These 'Uni-columnar neurons' are generated throughout the neuroepithelium and have a 1:1 stoichiometry with the photoreceptors that innervate the medulla. We will describe the mechanisms controlling the transition from one neural stem cell stage to the next.
How are the less numerous 'multi-columnar' neurons that have larger receptor fields and are present at a ~1:10 stoichiometry with photoreceptors generated? We will show that these subtypes emerge from the same neural stem cells that also produce uni-columnar neurons, but differ in different regions of the medulla neuroepithelium, which is highly patterned with each region contributing to producing different multi-columnar neurons. In spite of their restricted origins, these neurons still contribute to the entire retinotopic map through migration of their cell bodies. Therefore, the generation of 80 cell types involves the integration of temporal and spatial patterning that preserves retinotopy of neurons present at different stoichiometry.

Biography

Claude Desplan was trained at the Ecole Normale Supérieure in St. Cloud, France. He received his D.Sc at INSERM in Paris in 1983 before joining Pat O'Farrell at UCSF as a postdoc. In 1999, Dr. Desplan moved to NYU where he investigates the neural basis of color vision in Drosophila. Dr. Desplan serves on multiple scientific advisory boards and in funding agencies. He is an elected member of the American Association for the Advancement of Science, an elected foreign member of EMBO, and an elected fellow of the New York Academy of Sciences.

http://www.nyu.edu/projects/desplan/

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Auditorium 250

Anne Ferguson-Smith
Arthur Balfour Professor of Genetics

Department of Genetics, University of Cambridge, UK

Wednesday, 12 October 2016, 16:15 – 16:40

Murine metastable epialleles: Epigenetic control of variable phenotype, developmental programming and transgenerational epigenetic inheritance

Genetic models of epigenetic inheritance can provide useful insights into mechanisms, stability and heritability of modified states. Endogenous retroviruses (ERVs) are a class of LTR retrotransposons representing around one quarter of the repetitive elements in the murine genome. Most are epigenetically silenced. However, in two classic mouse models, Agouti viable yellow (Avy) and Axin fused (Axin(Fu)), a member of the IAP class of ERV has inserted in the vicinity of the agouti and axin genes respectively, and been variably DNA methylated between individuals; this is associated with transcriptional variability of these genes and non-genetically conferred phenotypic variation. Such alleles are known as metastable epialleles. We report a genome-wide systematic screen for novel metastable epialleles. The establishment of variable epigenetic states during development and their heritability from parent to offspring has been explored providing useful insights into mechanisms of transgenerational epigenetic inheritance.

Biography

Anne Ferguson-Smith is Professor of Genetics and Wellcome Trust Senior Investigator at the University of Cambridge. She obtained her BSc from the University of Glasgow and subsequently her PhD from Yale University. After a postdoc in Azim Surani's laboratory, she established her research group at the University of Cambridge, where she is now head of the Genetic Department.

http://www.gen.cam.ac.uk/research-groups/ferguson-smith

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Alexander Schier

Leo Erikson Life Sciences Professor of Molecular and Cellular Biology

Department of Molecular and Cellular Biology, Harvard University

Wednesday, 12 October 2016, 16:40 – 17:05

Lineage tracing by genome editing

Multicellular systems develop from single cells through a lineage, but current lineage tracing approaches scale poorly to whole organisms. I will discuss studies with the Shendure lab that show how genome editing can progressively introduce and accumulate diverse mutations in a DNA barcode over multiple rounds of cell division. The barcode, an array of CRISPR/Cas9 target sites, records lineage relationships in the patterns of mutations shared between cells. The rates and patterns of editing are tunable and thousands of lineage-informative barcode alleles can be generated. Sampling hundreds of thousands of cells from individual zebrafish reveals that most cells in adult organs derive from relatively few embryonic progenitors. I will discuss how genome editing of synthetic target arrays for lineage tracing (GESTALT) will help generate large-scale maps of cell lineage in multicellular systems.

Biography

Alex Schier was a graduate student in Walter Gehring's lab, where he studied the transcriptional regulation of homeobox genes. As a postdoctoral fellow in Wolfgang Driever's lab, he performed a large-scale screen for mutations affecting zebrafish development. As an Assistant and Associate Professor at the Skirball Institute of NYU School of Medicine and Professor at Harvard University, he has contributed to the understanding of the molecular basis of vertebrate embryogenesis and the establishment of zebrafish as a model system.

http://www.schierlab.fas.harvard.edu