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Sebastian Greiss
Genetic Code Expansion in C. elegans and its applications for precise spatiotemporal control of proteins with light

Lloyd Davis 1, Zhiyan Xi 1, Kieran Baxter 1, Angeliki Goutou 1, Ailish Tynan 1, Sebastian Greiss 1

1 Centre for Discovery Brain Sciences, University of Edinburgh, United Kingdom – United Kingdom

We have developed methods to expand the genetic code of C. elegans, allowing the site-specific introduction of chemically synthesized non-canonical amino acids (ncAA) into chosen proteins in vivo. For this we modify the worm’s translational machinery by adding an archeal aminoacyl tRNA synthetase/tRNACUA pair. The archeal tRNACUA delivers the ncAA to the ribosome, where it is incorporated into the nascent polypeptide chain at an amber stop codon introduced into the gene of interest. Examples of available ncAA include photo-caged amino acids, amino acids containing post-translational modifications, and bioorthogonal linkers.

In our lab we currently focus on photo-caged amino acids to engineer light activatable versions of existing proteins. Photo-caged amino acids contain an aromatic group attached to the amino acid side-chain, which is stable in visible light, but can be cleaved off by illumination with 365 nm UVA light. When such a caged amino acid is introduced into the catalytic site of an enzyme in lieu of the native residue, the enzyme is rendered inactive. Short exposure to UVA light can then be used to remove the caging group and activate the enzyme. The light can be delivered either globally by illuminating the entire animal, or cell specifically using a microscope mounted laser.

We demonstrate the utility of photo-caged amino acids for precise spatio-temporal control of gene expression, and for inducing apoptosis with light.
Session 1
Proteolytic activation of endo-siRNA pathways

Rajani Gudipatti * 1

1 Friedrich Miescher Institute for Biomedical Research (FMI) – Switzerland

Hundreds of proteases process and/or degrade proteins to control physiological processes in the animal body. Members of the DPPIV family are clinically important, as illustrated by the control of type-II diabetes through pharmacological inhibition of DPP4. To explore the physiological functions of DPP IV proteins, we deleted each of six DPP-Four family (pf) genes in C. elegans. We observed that deletion of dpf-3, orthologous to mammalian DDP8/9, causes temperature-sensitive male sterility. This phenotype is characteristic of small RNA pathway defects, and we find a specific subset of endo-siRNAs depleted in dpf-3 mutant animals. Concomitant transposon activation and DNA damage result in a loss of sperm and sperm precursor cells. Endo-siRNAs mediate transposon silencing by guiding Worm ArGOnaute (WAGO) proteins to their targets, and we provide evidence that DPF-3-mediated proteolytic processing is important for WAGO protein function. Thus, we have identified a new level of control of endo-siRNA activity.

*Speaker
Nanopore sequencing of C. elegans native transcriptome reveals the existence of a novel splice leader targeting specific genes.

Denis Dupuy * 1, Florian Bernard 2,3

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2 Acides Nucleiques : Régulations Naturelle et Artificielle – Université de Bordeaux, Institut National de la Santé et de la Recherche Médicale : U1212, Centre National de la Recherche Scientifique : UMR5320 – France
3 Sagol School of Neuroscience - Tel Aviv University – Israel

Our recent meta-analysis of alternative exon usage in Caenorhabditis elegans based on publicly available RNA-seq dataset (Tourasse et al., Genome Research, 2017) refined our comprehension of C. elegans transcriptome, especially regarding the quantitative aspects of alternative splicing in messenger RNAs. However, sequencing technologies (NGS) such as Illumina technology are known to introduce amplification bias affecting the overall distribution of mRNAs detected in one experiment and short-reads are not suited to accurately predict the frequency of isoforms derived from multiple alternative splicing events. In this study, we are exploiting the long read and amplification-free analysis made possible by Oxford Nanopore Technology (ONT) to overcome those limitations. Nanopore-based sequencing allow to directly sequence nucleic acids without any prior amplification step and generates long-reads covering up to the full-length of the molecule. Hence, we are aiming to further characterize C. elegans transcriptome by providing a more accurate measure of isoforms ratios, a better comprehension of exons associations during alternative splicing and by characterizing differentially transspliced mRNAs.

We assessed the efficiency of three different sequencing kits commercialized by ONT that are recommended for transcriptomics. Our results suggest that direct cDNA sequencing is most suited for transcriptome analysis in C. elegans, in regard to the quantity of data generated while preserving the quality of the dataset. Our experimental results support our recent observation that trans-splicing is more pervasive than was previously believed with ~90% of genes with detectable splice-leaders Additionally, we have identified a novel splice leader with extremely narrow gene specificity.

*Speaker
Gene Regulatory Network analysis reveals new ageing genes

Celia Raimondi *, Manusnan Suriyalaksh 1, Abraham Mains 1, Simon Andrews 1, Marta Sales 2, Olivia Casanueva† 1

1 Babraham Institute – United Kingdom
2 Universitat Rovira i Virgili – Spain

The nematode Caenorhabditis elegans is a versatile platform for investigating ageing and its modulation by various factors ranging from insulin IGF1 signalling (IIS) to dietary intake and reproduction. Interestingly, researchers had discovered that reproductive system regulates the lifespan of C. elegans (Hsin H and Kenyon C, 1999). Indeed, removal of the germ line by laser ablation or by mutation of glp-1 gene (Notch signalling pathway) leads to increase the lifespan of C. elegans. This suggest that germline must produce signals that coordinate reproduction with aging. Without the germline, the worm not only copes with the infertility, it also successfully re-establishes metabolic homeostasis and converts the drawback into a favourable lifespan increase.

Knowing that ageing is a multivariable process, the inter-relationships between actors, rather than individual gene potentially control the longevity phenotype. To understand this adaptability mechanism, we decided to perform a Gene Regulatory Network (GRN) analysis of the sterile and long-lived mutant, glp-1. The use of GRN allows to give a comprehensive view of this process as well as to predict new important actors. For the first time, time-resolved transcriptomics measurements combined with public multiomics data were used to reverse-engineered the GRN of the worm. Network properties were then used to predict causality relationships (edges) and central genes (actors) that potentially control ageing. Using RNA interference combined with high-throughput qPCR and lifespan assay we validated the GNR and unveil new regulators of ageing upon germline loss of function.

*Speaker
†Corresponding author: Olivia.Casanueva@babraham.ac.uk
The transcriptome of germline P-bodies in C. elegans

Szilvia Ecsedi *†1,2, Zhou Yi 2, Andres Cardona 3, Mokrane Khier 3, Alia Bahri 3, Agnes Loubat 3, Arnaud Hubstenberger 3

1 Szilvia Ecsedi – Université Côte d’Azur (UCA), Institute of Biology Valrose, UMR7277, CNRS, Univ. Nice Sophia-Antipolis – France
2 Université cote d’Azur Institute of Biology Valrose – Université Côte d’Azur (UCA), Institute of Biology Valrose, UMR7277, CNRS, Univ. Nice Sophia-Antipolis – France
3 Université cote d’Azur – Université Côte d’Azur (UCA), Institute of Biology Valrose, UMR7277, CNRS, Univ. Nice Sophia-Antipolis – France

Coordinated gene expression networks play a central role in cellular adaptation. As a part of this process, RNAs are covered by proteins and form ribonucleoproteins (RNPs) that regulate the fate of RNAs. RNPs can further co-assemble into diverse, non-membranous granules. To study how RNP assembly coordinates RNA regulatory networks and test the functionality of RNA aggregates in adapting gene expression, we purified oocyte P-bodies starting from whole C. elegans. For this purpose, we successfully implemented a modified Fluorescent Activated Particle Sorting (FAPS) method coupled with RNA sequencing and systematically characterized the RNA regulatory networks of oocyte P-bodies.

To determine mRNA enrichments in oocyte P-bodies, we compared the P-body transcriptome to the oocyte transcriptome. We saw the wide continuum of fold change distributions, which suggests that RNAs do not localize in a binary manner either in granules or in the cytosol. To validate RNA enrichment in the granules, we used single-molecule Fluorescence in Situ Hybridization (smFISH) and computed the absolute number of RNA molecules within condensates versus the dispersed soluble phase. The partition coefficient strongly correlated with our RNA-Seq results suggesting that mRNA targeting to P-bodies is finely tuned. Gene set enrichment analysis identified core regulatory pathways among P-body mRNAs, while RNAs encoding structural and housekeeping cellular functions were depleted.

Using large-scale RNA-protein interaction data sets, we demonstrate that mRNAs targeted to oocyte P-bodies are translationally repressed, protected from decay, and transiently stored before re-entering in translation later, during oocyte activation or embryonic development. Thus, the RNA content of P-body is developmentally regulated. Altogether, oocyte P-bodies are storing centers for maternal RNAs that will be sequentially translated to control embryonic development.

*Speaker
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Session 2
Syndecan is an organizer of the C. elegans neuromuscular junction.

Camille Vachon *, 1, Xin Zhou 1, Maëlle Jospin 1, Méllisa Cizeron 1, Hannes Bülow 2, Jean-Louis Bessereau† 1

1 Institut NeuroMyoGène (INMG) – CNRS : UMR5310 – France
2 Albert Einstein College of Medicine – United States

The extracellular matrix (ECM) is an integral component of chemical synapses, where it plays structural and functional roles. In C. elegans, body-wall muscle cells receive excitatory and inhibitory innervation from cholinergic and GABAergic motoneurons, respectively. Ce-punctin (also known as MADD-4) is an ECM protein secreted by motoneurons in the synaptic cleft. Specific combinations of Ce-punctin isoforms trigger the clustering of acetylcholine or GABA receptors at synaptic sites. Other components of the ECM, such as heparan sulfate proteoglycans (HSPG), are present at neuromuscular junctions (NMJs) but their functions remain elusive.

Using a knock-in allele we found that the HSPG syndecan is a synaptic protein at NMJs and is enriched at cholinergic neuromuscular synapses. Syndecan is a glycoprotein composed of a core transmembrane protein carrying 3 polysaccharidic side chains. In C. elegans the core protein is encoded by a single gene sdn-1. It was previously shown to be required for gonad arm migration and neuritic growth and guidance.

sdn-1 disruption affects the localization and the levels of ACh and GABA receptors at NMJs. Electrophysiological recordings further confirmed that more than 60 % of synaptic AChRs are removed from synaptic regions in sdn-1 mutants.

Insertion of a SL2-Scarlet reporter cassette in the sdn-1 locus indicates that sdn-1 is broadly expressed, including epidermis, neurons and body-wall muscles. We tagged SDN-1 with a DEGRON sequence by CRISPR and used the Auxin Inducible Degradation system to identify the cellular origin of SDN-1 at NMJs. About 80 % of SDN-1 originates from muscle and 20 % from neurons. The synaptic localization of SDN-1 strictly depends on Ce-Punctin. Mutation of the glycosylation sites on the core protein shows that SDN-1 heparan sulfate stabilize SDN-1 at synapses. Furthermore, we demonstrated that the intracellular domain of SDN-1 recruits intracellular scaffolding proteins that, in turn, localize AChRs at NMJs.

*Speaker
†Corresponding author: jean-louis.bessereau@univ-lyon1.fr
LSM2-8 and XRN-2 contribute to the silencing of H3K27me3-marked genes through targeted RNA decay

Anna Mattout * 1, Dimos Gaidatzis 2, Jan Padeken 2, Christoph Schmid 2, Florian Aeschlimann 2, Véronique Kalck 2, Gasser Susan† 2

1 CBI Toulouse – Université Paul Sabatier - Toulouse III – France
2 FMI – Switzerland

Heterochromatin, correlates with repressed gene expression. Transcriptional repression is believed to be the main - if not the only - mechanism responsible for the silencing of promoters in heterochromatin in higher eukaryotes. However, in a genome-wide derepression screen of a heterochromatic reporter, we identified unexpectedly three RNA binding proteins, members of the LSM complexes. We examined their function in order to identify potential heterochromatin silencing mechanisms at the co-/post-transcriptional level in metazoan. The C. elegans LSM proteins share 60-90% homology with the human LSMs, and assemble into two major complexes, LSM1-7 and LSM2-8. The LSM1-7 is known primarily to be involved in cytoplasmic mRNA decay while LSM2-8 regulates both nuclear mRNA decay and U6 snRNA stability. Combining microscopic, genetic and genomic approaches we could show that LSM2-8 silences specifically heterochromatic reporters. Importantly, the complex also silences endogenous regions selectively enriched for the heterochromatic mark H3K27me3. Developmental defects and premature death were observed in worms lacking LSM-8. LSM-8-mediated silencing seems to be at least partially dependent on mes-2, the Polycomb-like HMT responsible for H3K27me3 deposition, whereas it is independent of H3K9me2/me3. LSM-8-mediated silencing not only targets H3K27me3-marked genes but also appears to favor H3K27me3 deposition on those genes. LSM8-mediated silencing is detectable from early embryonic stages through adulthood, in every somatic cells. Importantly, we also found that the LSM2-8 complex works cooperatively with XRN-2, a 5'-3' exonuclease. Together, our results suggest that the LSM2-8 complex recognize selectively transcripts arising from heterochromatic H3K27me3-enriched domains and promote their silencing through degradation by XRN-2. LSM8-mediated silencing of H3K27me3 genomic regions through RNA silencing, is a new mechanism, at a new level of regulation, which was not previously shown for higher eukaryotes.

* Speaker
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When choosing food, *Caenorhabditis elegans* only cares about density (and not bacterial species)

Gabriel Madirolas 1, Leslie Marie-Louise 1, Lydia Gaouar 1, Andrea Garza-Enriquez 1, Mikail Khona 2, Jeff Gore 2, Alfonso Pérez-Escudero *

1 Centre de Recherches sur la Cognition Animale – CNRS : UMR5169 – France
2 Massachusetts Institute of Technology (MIT) – United States

As choosing the right food is key for survival, animals dedicate a wide range of sensors and processing power to make the right choice-finding and selecting the best food is probably one of the key drivers behind the evolution of taste and olfaction. In this context, *Caenorhabditis elegans* should pick its food carefully, taking into account different characteristics of each bacterial species, such as nutritive value, ease to consume (based on size and hardness of the bacteria) and potential pathogenicity. Indeed, previous studies show that worms take these factors into account, preferring some bacterial species over others.

We studied the response of *C. elegans* to different types of food (different bacterial species and different densities for each species). Thanks to a high-throughput experimental pipeline, we were able to map these preferences systematically, covering a wide range of bacterial densities for each species and for combinations of several species.

Contrary to previous studies, we found that the response of *C. elegans* is highly stereotyped and largely independent of the bacterial species. *C. elegans*’ preference for a food patch increases with its density following a well-defined sigmoidal shape, whose central region (for intermediate densities) implements Weber’s law and a version of the Ideal Free Distribution. Remarkably, the single variable determining this preference is food density measured in number of bacterial cells per mm², regardless of the bacterial species or the total amount of biomass (so the size of the cells had no impact). The differences found in previous studies can be attributed in the most part to different species reaching different densities on the experimental plates. We also evaluated the fitness benefit that the worms obtain from each food source. This fitness benefit also increases with food density, but in this case we also found a moderate influence of the bacterial species.

These results indicate that *C. elegans* uses a so-called ”rule of thumb” to make foraging decisions, focusing on the single-most relevant parameter to measure the quality of a food patch and neglecting the rest. They also suggest that the basic circuitry governing foraging in *C. elegans* may be simpler and depend on fewer sensory inputs than previously thought.

*Speaker*
Dystrophin-Associated Protein Complex (DAPC) and planar cell polarity (PCP) pathways pattern membrane domains of C. elegans muscles

Alice Peysson *, Nora Zariohi *, Marie Gendrel †, Thomas Boulin‡

1 Institut NeuroMyoGène CNRS UMR 5310 - INSERM U1217 (INMG) – Université Claude Bernard Lyon 1, Université Claude Bernard Lyon 1 – France
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Potassium channels form a large family of well-conserved ion channels that play a key role in controlling cellular excitability. However, the precise mechanisms that control the expression, the biosynthesis and the subcellular distribution of potassium channels are still insufficiently elucidated. By combining forward genetics, gene editing, and live imaging, we have identified novel mechanisms that control the subcellular distribution of potassium channels in C. elegans. 81 and 72 genes code for potassium channel subunits in the genomes of mammals and C. elegans, respectively. Recent single cell/RNAseq analyses have revealed that 21 potassium channels are co-expressed in each body wall muscle cell of the worm (Cao et al., 2017). To begin to understand how each channel contributes to the regulation of muscle excitability, we investigated the subcellular distribution of these channels by tagging nine of them with fluorescent proteins using CRISPR/Cas9-based gene editing. We found that these nine channels have distinct subcellular distributions. Most notably, TWK-28, a two-pore domain (K2P) potassium channel, is confined to the anterior tip of muscle cells, while another K2P channels, TWK-24, is localized to midsection of each cell. Finally, we found that the BK channel SLO-1 shows a punctate pattern, restricted to the posterior half of the cell. These highly reproducible subcellular distribution patterns offer an original model system to study the cellular mechanisms that compartmentalize membrane proteins.

In a forward genetic screen, we found that genes belonging to the Dystrophin-Associated Protein Complex (DAPC), such as dystrophin/dys-1, control the localization of TWK-28 at the muscle cell surface. This well-conserved protein complex is composed of at least 10 intra- and extracellular protein, which link the extracellular matrix to the intracellular actin cytoskeleton. By systematically tagging DAPC components, we found that they adopt striking distribution patterns at the muscle surface. Notably, some of these proteins are also highly compartmentalized, defining distinct anterior and posterior domains within individual muscle cells. These

*Speaker
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patterns are highly reproducible and are repeated along the antero-posterior axis of the worm. Our working model is that TWK-28 is recruited to the anterior tip of each muscle cell by physically interacting with some of the DAPC components.

How is this striking membrane compartmentalization achieved? **Planar cell polarity pathways (PCP)** are conserved mechanisms that generate asymmetries at the scale of cells and tissues. Using a candidate gene approach targeting conserved PCP genes, we found that TWK-28 channels are no longer restricted to the anterior tip of each muscle cell in mutants of ROR/cam-1 and the Wnt ligand egl-20. In these mutants, TWK-28 is now found both at the anterior and the posterior tips of muscle cells. Understanding the molecular mechanisms linking DAPC and PCP pathways to pattern membrane domains of *C. elegans* muscles will be the focus of our ongoing work.
Session 3
The Polo-like kinase (PLK-1) merges the parental genome into a single nucleus by triggering lamina disassembly in the C. elegans zygote

Griselda Velez Aguilera *1, Sylvia Nkombo-Nkoula 2, Lucie Van Hove 2, Jana Link 3, Nicolas Joly 2, Batool Ossareh-Nazari 2, Jean Marc Verbavatz 4, Verena Jantsch 3, Lionel Pintard†2

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After fertilization, the haploid gametes of egg and sperm are replicated into separate pronuclei surrounded by a nuclear envelope but before the first zygotic division, the nuclear envelopes disassemble allowing both sets of chromosomes to be incorporated into a single nucleus in daughter cells after mitosis. In the C. elegans zygote, partial inactivation of the mitotic polo-like kinase PLK-1 prevents NEBD and the mixing of the parental chromosomes. However, the critical PLK-1 targets in this process are not known. Here we show that PLK-1 phosphorylates the single C. elegans lamin LMN-1 to promote lamina disassembly. We demonstrate that expression of non-phosphorylatable versions of LMN-1 prevents Lamina depolymerization, which is sufficient to induce the formation of embryos with a paired nuclei phenotype, containing either the maternal or the paternal chromosomes in each daughter cell at the two-cell stage. Finally, we reconstitute the depolymerization of pre-assembled LMN-1 filaments by PLK-1 in vitro and demonstrate the importance of LMN-1 phosphorylation sites in this process. Our findings indicate that PLK-1 is a crucial regulator of lamin dynamics in C. elegans and possibly also in other organisms.

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The polarity protein PAR-4/LKB1: a novel regulator of C. elegans gut development

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Intestinal epithelial cells can absorb food through their highly specialized apical surface, the so-called brush border, which is composed of many microvilli. Ectopic activation of the kinase and tumor suppressor PAR-4/LKB1 has been shown to be able to induce the formation of an apical brush border with microvilli-like structures in intestinal epithelial cancer cell lines. However, the function of PAR-4/LKB1 in intestinal cells has so far not been characterized in vivo. In order to determine whether it is involved in brush border formation, we used confocal and transmission electron microscopy to observe the intestinal epithelium in C. elegans par-4 thermosensitive mutant embryos. Surprisingly, we have found that PAR-4/LKB1 loss-of-function does not inhibit the formation of microvilli in intestinal cells, even if they present mild defects. However, par-4 mutant embryos show striking defects of the intestinal epithelium architecture with the appearance of strong intestinal lumen defects, supernumerary apical junctions and an increased number of intestinal cells. Altogether, these results indicate that PAR-4/LKB1 is required to control the intestinal cell number and maintain the correct architecture of the intestinal epithelium. We are currently investigating the mechanisms by which PAR-4/LKB1 plays these functions, in particular whether it regulates the cell cycle through the CDC-25.1 phosphatase. Moreover, further experiments will allow us to understand if the intestinal lumen defects are due to the excess of intestinal cells or to a separate function of PAR-4/LKB1.

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The Argonaut NRDE-3 and MET-2 redundantly target SET-25 to full length transposable elements

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Establishing, maintaining and erasing chromatin domains are fundamental processes that shape the epigenetic memory of a cell. Lysine 9 methylation on histone H3 (H3K9me) is one of the defining histone modifications of heterochromatin. Silencing of satellite repeats, transposable elements and developmentally regulated genes, through H3K9me is essential to ensure genome stability (Zeller and Padeken et al., 2016). To understand how H3K9me2/me3 domains are established and maintained we investigated the dependencies of the 2 H3K9 specific histone methyl transferases (HMTs) in C. elegans. SET-25 (SUV39h1/h2) catalyzes H3K9me3, while MET-2 (SetDB1) deposits H3K9me1/me2 (Towbin et al., 2012). ChIPseq experiments in met-2 and set-25 mutant embryos showed that while the majority of H3K9me3 domains are completely dependent on MET-2 for the recruitment of SET-25, however ~10% of H3K9me3 domains, particularly evolutionary young transposons, can be methylated by SET-25 independently from MET-2. A RNAi screen showed that SET-25 targeting to these sites is dependent on the somatic Argonaut NRDE-3 and the MBT domain protein LIN-61. Analysis of the nrde-3, lin-61, met-2 and set-25 single and double mutants showed that LIN-61 functions by reinforcing SET-25 binding to existing H3K9me2/me3 regions. In contrast NRDE-3 is redundant with MET-2 for the targeting of SET-25 to full length transposable elements. Loss of nrde-3 and met-2 results in the synergistic upregulation and expansion of transposable elements leading to embryonic lethality.

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Specificity of sperm-derived mitochondria autophagy and the urge for new answers

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During the last decade, significant progress were made in our understanding of the mechanisms insuring uniparental maternal mitochondrial genome transmission and C. elegans turned-out to be a powerful experimental system to tackle this universal biological question. Sperm derived mitochondria are eliminated following fertilization by selective autophagy, but the mechanisms that restrict this process to sperm-derived organelles are not well understood. Recently, the loss of mitochondria membrane potential (ΔΨm) in sperm-derived mitochondria after their entry has been proposed as the initial factor to trigger paternal mitochondria elimination in C. elegans (Sato M et al, 2018 and Zhou Q et al, 2016). Using a cationic fluorescent dye we followed, in sperm-derived mitochondria, the membrane potential at the time of fertilization and during autophagosome formation. We will present our intriguing findings that challenge the current view of the triggering signal of sperm mitochondria degradation and suggest, that beyond the autophagy degradation, additional mechanisms exist to prevent the inheritance of paternal mitochondrial genome.

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Posters
Re-defining a GABAergic neuron

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The balance between excitation and inhibition is critical to the proper function of neural circuits.

GABA, the main inhibitory amino-acid neurotransmitter in mature neurons, is a remarkably multi-functional neurotransmitter: it can bind to either ionotropic GABAA (mediating fast neurotransmission) or metabotropic GABAB receptors (mediating slow neurotransmission) that may be localized extra-, peri-, pre- and postsynaptically.

The GABAergic phenotype in vertebrates and invertebrates has been defined classically by the presence of three key players in the presynaptic neurons: (i) glutamic acid decarboxylase (GAD), the enzyme needed to synthetize GABA from glutamate, (ii) the H+-coupled transporter (VGAT) that packages GABA in synaptic vesicles, and (iii) the Na+-coupled transporter (GAT) that recaptures GABA at the nerve terminal after its release in the synaptic cleft. For over 20 years, the C. elegans GABAergic nervous system was thought to be composed of only 26 out of the total 302 neurons. However, an in-depth revision of the GABAergic nervous system in C. elegans was performed. In particular, we have shown that additional neurons contain GABA but do not always express GAD/unc-25, VGAT/unc-47 and GAT/snf-11, the landmark gene portfolio for classical GABAergic neurons. Indeed, 22 new GABA-positive cells that do not conform to this classical definition were identified. They can be categorized into 4 different types of neurons expressing different combinations of these factors. Two of these types show evidence of alternative modes of GABA transport because they lack expression of known GABA transporters, VGAT/unc-47 and/or GAT/snf-11, and they do not synthetize GABA. Deciphering these new mechanisms of GABA transport will shed light into the regulation of neural circuits through inhibition.
Programmed genome elimination in Mesorhabditis nematodes

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We discovered that nematodes from the Mesorhabditis genus undergo programmed genome elimination during somatic cell differentiation, similar to what has been described in the parasitic nematodes Ascaris. Using cytological observations we found that chromosomes start to fragment in the ABa, ABp and EMS cells at the 4cell stage. in the subsequent cell divisions, DNA fragments lag on the mitotic spindle and thus stay in the cytoplasm before being degraded. Only the genome of the germline precursor cell P4 remains intact. Programmed genome elimination in the soma has been observed in at least 100 species belonging to 9 different phyla. In each case, the eliminated portions are satellite repeats and germline genes. This process has emerged several time independently over the course of evolution but how and why sequences are recognized to be physically eliminated remains a mystery. One reason for this is that the species in which these observations have been made are difficult to study in laboratory conditions. Gene inactivation by RNAi is possible in Mesorhabditis worms, their embryos can be easily manipulated and the genome of these species is small, making this group of nematodes an interesting system to start exploring the mechanisms of elimination. To this end, we are using a combination of genomic approaches, cytological description and functional analysis.
Still exploring fungal virulence using *C. elegans*

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To understand better fungal pathogenesis in an *in vivo* setting, we use the interaction between *C. elegans* and its natural pathogenic fungus *Drechmeria coniospora*. We have selected 12 candidate virulence factors and directly express them in *C. elegans* as chimeric proteins (with fluorescent and affinity tags) under the control of the epidermis-specific *col-19* promoter. To modulate tightly their expression, we have also incorporated degron tags and use the auxin inducible degron system.

One group of candidates are heat-labile enterotoxins, which are expanded in the genome of *D. coniospora* compared to other pathogenic fungi. We selected three enterotoxins (g6833, g2819 and g7949) from a total of 19. Surprisingly, each of them has a distinct localisation pattern in the *C. elegans* epidermis: g2819 goes to the nucleus, g6833 has a punctate cytoplasmic distribution, while g7949 accumulates close to the membrane specifically adjacent to the neighbouring seam cells. The transgenic worms expressing g2819 and g7949 are sick and die precociously with or without *D. coniospora* infection, while g6833 has no effect on survival. Normally, infection provokes the induction of expression antimicrobial peptide genes of the *nlp* and *cnc* families. Interestingly, expression of the single enterotoxin g7949 blocks the transcription of both. In contrast, worms expressing enterotoxin g2819 exhibit a STAT-dependent elevation of *nlp-29*. Finally, g6833 has little or no effect on AMP gene expression. We are currently exploring the molecular basis of these differences and the relative importance of these factors during infection.

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Intestinal mitochondrial activity modulates epidermal innate immunity

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A previous RNAi screen for antifungal innate immunity genes revealed a suppression of antimicrobial peptide gene expression in the nematode epidermis through cross-tissue signalling [1]. The suppression of the antimicrobial peptide gene nlp-29 after Drechmeria coniospora infection is specifically triggered by mitochondrial dysfunction in the intestine. This suppression, best characterised for the knockdown of the mitochondrial inner membrane metalloprotease, SPG-7, has been found to be independent of the mitochondrial UPR transcription factor, atfs-1, thus pointing to a role for mitochondrial activity per se in regulating nlp-29 expression.

We first took a candidate approach to try to identify the signal leading to the inhibition of epidermal nlp-29 expression. By reverse genetics we excluded ROS production, detoxification pathway and intestinal p38 signalling as being involved in the generation of the signal. Synaptic vesicle exocytosis is not involved in the transmission of the signal.

To uncover the mechanisms responsible for the mitochondrial-dependent cross-tissue signalling, we therefore carried out an EMS screen. We mutagenized a strain carrying an nlp-29p::GFP construct, with a gain of function mutation for gpa-12 that activates reporter gene expression in adults. When this strain is grown on spg-7(RNAi), nlp-29p::GFP expression is suppressed. Using the Biosort, we singled GFP+ F2 adults that had been cultured on OP50 into 48-well NGM plates seeded with spg-7 RNAi bacteria. We screened their F3 progeny, looking for populations in which the spg-7 suppression of GFP expression was relieved. From ca. 4,600 F2 worms, we isolated 51 candidates and retained 8 for in-depth study. Progress on this screen will be reported.


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Decrypting the mechanism of action of Drechmeria’s virulence factors; a biochemical approach

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Drechmeria coniospora is a nematophagous fungal pathogen that infects nematodes though the adhesion of its conidia to the worm’s cuticle. After penetrating the worm’s body, Drechmeria is predicted to secrete a broad range of proteins leading to the death of its host after as little as 48h. Although the host’s immune response to Drechmeria is well characterised, very little is known about how Drechmeria kills its host so efficiently.

In this project, we have therefore focused on deciphering the molecular mechanisms of Drechmeria’s pathogenesis. To do so, we have designed a strategy to express tagged chimeric proteins from Drechmeria in a controlled manner directly in the epidermis of C. elegans (see abstract “Still exploring fungal virulence” by Xing Zhang et al.).

Each of the candidate virulence factors that we have expressed is associated with a unique pattern of intracellular localisation, suggesting that they might interact with different host proteins. In order to further characterise the mechanism of action of the fungal virulence factors, we are implementing an approach based on co-immunoprecipitation followed by mass spectrometry in the hope of identifying their specific host protein targets. Establishing the interactome of some of these candidate virulence factors should give insight into their mode of function and be a starting point to unravel the complexity of fungal pathogenesis.

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Phosphorylation of an essential Microtubule Severing AAA+ Enzyme (Katanin) regulates its stability and activity during C. elegans embryo development

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Microtubules (MTs) are dynamic cytoskeletal polymers with instrumental functions in cell division (meiosis and mitosis), morphogenesis, motility and signaling. MTs constantly polymerize and shrink and this dynamic behavior, which is critical for their function, is regulated by a large family of MT-interacting proteins. Whereas most of these proteins interact with the microtubule plus or minus ends, another class interacts with the MT lattice and severs MTs along their length, thereby controlling MTs size and density. Three evolutionarily conserved MT-severing enzymes have been identified: Fidgetin, Spastin and Katanin. Mutation of these enzymes has been linked to various defects and pathologies including developmental defects, neurodegenerative disorders such as hereditary spastic paraplegia (HSP) and the Fidget disease. Nevertheless, in spite of their importance, the mode of action and the regulation of these enzymes are poorly understood.

To directly address the question about the regulation of Microtubule Severing Enzyme activity we used C. elegans Katanin as model. Indeed, in C. elegans, Katanin MT-severing activity is essential for meiotic spindle assembly but is toxic for the mitotic spindle, suggesting a strong regulation, in space and time, of this enzyme activity.

Here, we deciphered the role of Katanin phosphorylation in the regulation of its activity and stability. We demonstrate unequivocally that Katanin phosphorylation at a single residue is necessary and sufficient to target Katanin for proteasomal degradation after meiosis, while phosphorylation at the other sites only inhibits Katanin ATPase activity stimulated by microtubules.

*Speaker
Studying the role of H3K9 methylation at tissue specific genes during C. elegans development.

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Constitutive heterochromatin, defined by methylation of histone H3 lysine 9 (H3K9me), is found at both repeat elements and a subset of tissue specific genes. H3K9me is critical to the silencing of repetitive elements in order to protect genomic integrity. The role of H3K9me at genes is less well defined; however, there is emerging evidence that H3K9me can correlate with gene silencing. In mammals, many histone methyltransferases (HMTs) target H3K9 and are required for viability, which makes the analysis of H3K9-specific regulation of transcription challenging. In contrast, Caenorhabditis elegans has only two H3K9 HMTs, MET-2 that deposits H3K9me1/me2 and SET-25, which catalyzes H3K9me3. We have begun to characterize the role(s) of H3K9me2 and/or me3 in tissue-specific gene expression, by asking which genes becomes de-repressed in purified tissues from met-2 set-25 (or single) mutant worms. Our results indicate that a subset of tissue specific genes are de-repressed in the absence of H3K9me2 but not in the absence of H3K9me3. Furthermore, cells from different tissues (embryos, muscle cells and seam cells) have distinct subsets of de-repressed genes in the absence of H3K9me, suggesting that the de-repression is dependent on available transcription factors. Finally, we generated a MET-2::degron fusion to induce degradation of MET-2 in differentiated tissues. Acute loss of MET-2 in muscle cells results in subsequent loss of H3K9me2 and de-repression of tissue specific genes, demonstrating that heterochromatin remains dynamic in non-dividing, differentiated tissues.

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Characterizing the organization and dynamics of the cytoskeleton during the first asymmetric cell division of Diploscapter pachys embryos

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Cell diversity arises from asymmetric cell divisions that differentially segregate fate determinants to daughter cells that are usually different in size. The first division of the Caenorhabditis elegans embryo is well studied, and has become a standard model for understanding asymmetric cell division. Upon sperm entry, a cue initiates polarity establishment via anterior directed actomyosin contractility and differential localisation of polarity proteins at the embryo poles. Shape changes observed during polarity establishment and spindle positioning of the C. elegans embryo have been well characterized. Interestingly, asymmetric cell division of the one-cell embryo is highly conserved across evolution of different nematode species, but the shape changes accompanying division are divergent.

The objective of this project is to characterize these morphological differences in a parthenogenetic species evolutionary distant from C. elegans, Diploscapter pachys. My preliminary results show that D. pachys embryos have exaggerated shape changes prior to cleavage as compared to C. elegans. Moreover, there is an asynchrony between the protrusions and retractions of the posterior and anterior pole of the embryo as observed by DIC microscopy. Currently, I have established a working technique in staining the embryo membrane via feeding with liposomes encapsulating membrane dye. Fluorescently labelled membrane will allow more precise tracking of membrane deformations. My next goal is to use this method to load SiR-actin and SiR-tubulin into D. pachys embryos in order to visualize the actin and microtubule cytoskeleton during morphological changes. Subsequently I will use the same method to apply photoactivatable actin and microtubule inhibitory drugs, such as PST-1, in order to observe the effects on the asymmetric cell division of the embryo when the inhibitory activity is turned on with light. To complement these observations and obtain a molecular picture of D. pachys division, I will perform RNAi against cytoskeleton players. RNAi has recently been shown to work in D. pachys, although so far in my hands, it has not proven to be effective.

All together the results of this study will shed light on fundamental physical and molecular aspects of asymmetric cell division, and in comparison with C. elegans, will bring to light alternate mechanisms of asymmetric cell division.

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Mechanical sensing of damage

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The cuticle, tightly linked to the epidermis, forms a physical barrier that protects *C. elegans* from injury and insults. It is a complex structure of different annuli and furrows collagens. Breaching the cuticle triggers an immune and a repair response. A new cuticle is then formed at each moult through sequential expression of collagen genes, including a subset of *dpy* genes (*dpy-2, 3, 7, 8, 9 and 10*) that correspond to collagens found only in furrows. We previously shown that furrow-less cuticular collagen mutants exhibit constitutive immune, osmotic and detoxification responses. Atomic force microscopy revealed that these mutants have a softer cuticle. We hypothesis that furrow collagens could be part of an epidermal sensor, activated by mechanical stress.

To address this hypothesis, we have focused on a poorly characterised organelle that bridges the cuticle and the epidermis. These organelles were first described in 1988 but their function remains unknown. There structure appears as 20 nm repeat folded parallel plasma membrane stack, spanning over 1 micron using transmission electronic microscopy. Serial block-face scanning electron microscopy revealed a high density in all hyp7 but underneath the muscle. Interestingly, sparse and abnormally folded organelles are found only in furrow-less cuticular collagens mutants.

To complement these analyses, we are taking advantage of the V-type proton ATPase subunit *a*, VHA-5 that was identified as a marker of this organelle. We have generated a variety of VHA-5::GFP expressing strains, in wild type and *dpy* backgrounds. Moreover, using live imaging, we are exploring the dynamic organisation of these membrane organelles upon different stresses.

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Role of MADD-4/Ce-Punctin processing at the neuromuscular junction

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In C. elegans, body-wall muscle cells receive both excitatory (cholinergic) and inhibitory (GABAergic) inputs. MADD-4/Ce-Punctin is an extracellular matrix protein, secreted by motoneurons, which specifies the receptors to cluster at each type of neuromuscular junction (NMJ). Two types of MADD-4 protein are expressed: i) long isoforms (MADD-4L) are expressed by cholinergic motoneurons and are required for the correct localization of ACh receptors; ii) the short isoform (MADD-4S) is expressed at both types of synapses and is required at GABA synapses to recruit GABA receptors and at cholinergic synapses to prevent the recruitment of GABA receptors by MADD-4L. Recent data have shown that MADD-4S, but not MADD-4L, is cleaved. Cleavage results in the localization of the MADD-4S N-terminal fragment at GABA synapses, where it probably assembles with NLG-1 to recruit GABA receptors. The C-terminal fragment diffuses to cholinergic synapses where it interacts with MADD-4L to inhibit GABA receptor recruitment. Cleavage can occur in the linker region of MADD-4S between the immunoglobulin-like domain and the thrombospondin domain 8 and is coupled to trafficking, as mutations in this region cause the trapping of MADD-4S in cell bodies. MADD-4 cleavage likely generates two polypeptides with different functions, as the expression of the C-terminal fragment can rescue the localization of GABA receptors in madd-4S(0), as opposed to the N-terminal fragment. A second cleavage, yet to be analyzed, might occur between thrombospondin domains 4 and 5. Altogether, these proteolytic events seem critical to expand the functions of MADD-4 depending on the synaptic contexts.

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Characterization of Katanin-microtubules interaction required for meiotic spindle assembly in C. elegans embryo

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Microtubules (MT) are dynamical polymers of cytoskeleton, crucial, for example, for cell division, morphogenesis, motility and signaling. Microtubules dynamic is regulated by a large family of MT-associated proteins (MAPs) including the Microtubules Severing Enzymes (MSE). This sub-family is composed of three conserved AAA+ ATPases (ATPase Associated with diverse cellular Activities): Spastin, Fidgetin and Katanin which regulate MTs length and density by severing MTs. Mutation of these enzymes might cause defects and human pathologies. Nevertheless the mode of action and their regulation are still unclear.

The nematode C. elegans represents an attractive model to study MSE function. Indeed, in this system, the conserved AAA+ ATPase Katanin, which is composed of MEI-1 and MEI-2 subunits, is essential for the assembly of the acentrosomal female meiotic spindle. A default of activity of this enzyme is thus easily trackable.

Recently, we have shown that MT severing activity is essential to meiotic spindle assembly, most likely by generating more dynamic MT polymers. We also showed that MEI-2 is responsible for MT binding of Katanin in vitro but the molecular determinants are still unknown.

Combining biochemical and genetics approaches, we aim to identify the precise region of MEI-2 required for MT binding and its role for Katanin activity. Here, we present the preliminary results concerning the identification and the characterization of this motif. In addition, we report the localization of MEI-2 (Katanin) during worm development, using live-imaging in a C. elegans strain containing sGFP::MEI-2. Strikingly, we confirmed the presence of Katanin during the formation of the meiotic spindle but more unexpectedly we revealed the persistence of the enzyme during later embryonic stage.

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Polo-like kinase 1 interacts with phospho-NPP-19 to drive nuclear pore complexes disassembly during mitosis

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Nuclear envelope breakdown (NEBD) is an essential step of mitosis ensuring proper chromosome segregation between the daughter cells. NEBD occurs in prophase and involves three steps: (i) the nuclear pore complexes (NPCs) composed of more than thirty nucleoporins (NPPs) are disassembled, (ii) the nuclear lamina is depolymerized, and (iii) microtubules tear apart the nuclear envelope. NEBD is triggered by evolutionarily conserved mitotic kinases notably the Polo like kinase 1 (Plk1) and Cyclin dependent kinase 1 (Cyclin-Cdk1). How these kinases coordinate their activity in space and time to regulate NEBD is not fully understood. We have shown recently that Plk1 is recruited to the nuclear pore complexes in prophase in human cells and C. elegans embryos to promote NEBD. More specifically, the nucleoporins NPP-1, NPP-4 and NPP-11, which form a trimeric complex located in the central channel of the NPC, recruit Plk1. Once localized at the central channel of nuclear pore complexes, Plk1 must reach structural nucleoporins to trigger NPC disassembly. NPP-19 is a structural nucleoporin, part of the Inner Ring complex (IRC), where it interacts with NPP-22, NPP-13 and NPP-8. We found that PLK-1 physically interacts with NPP-19 in a phosphorylation-dependent manner in vitro and we have identified the critical phosphosites implicated in this interaction. To evaluate the functional importance of this interaction in vivo, we have generated a C. elegans line expressing NPP-19, mutated on the critical phosphosites and thus unable to interact with PLK-1. We then followed IRC disassembly using strains expressing endogenously fluorescently tagged NPP-8, NPP-13 or NPP-22. We found that IRC disassembly is defective when the interaction between PLK-1 and NPP-19 is abrogated indicating that PLK-1 recruitment to the IRC is required for proper disassembly of this complex. Overall this project will help to better understand the molecular mechanisms by which PLK-1 promotes NPC disassembly to ensure accurate chromosome segregation during mitosis.

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Large-scale non-invasive imaging of C. elegans enables reliable measurements of ageing, neurodegeneration and behaviour

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Our beloved model organism C. elegans has been very successful in many areas of fundamental biology including the science of ageing and neurodegeneration. However, the assays commonly used in these disciplines can be time-consuming and tedious, making experimentation and screening difficult. For example, the lifespan assay is relatively simple but it takes a long time to be proficient at it and it can be subjective and reproducible results can be difficult to achieve. We have developed a method that quantifies ageing by near-continuously measuring worm movement on standard 6cm petri dishes. This technology allows us to measure the rate of decay of movement over time using several parameters, including distribution of speeds. The method involves an array of small cameras each controlled by a single board computer, allowing easy scaling to multiple plates. Automated data analysis facilitates reporting of data. This method is more sensitive and quantitative than lifespan analysis. In addition, it can be used to measure other aspects of worm movement including chemotaxis and paralysis. The non-invasive nature of the technology removes sources of variation and allows worms to be later used in other experiments, such as molecular or biochemical analysis or in manual lifespan assays. The application of this technology will allow large and more quantitative experiments in several fields of C. elegans research.

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Heterochromatin integrity preserves C elegans resilience to extreme temperature stress

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Segregation of genomic regions into accessible euchromatin and inaccessible heterochromatin is essential for temporal and tissue-specific gene transcription. These compartments are associated with post-translational modifications of histones. Methylation of histone H3 lysine 9 (H3K9me) silences heterochromatic satellite repeats, transposable elements and tissue-specific genes. In C. elegans, the SETDB1 homolog MET-2 catalyzes mono- and di-methylation of H3K9 and is required for proper developmental timing and the preservation of fertility. MET-2’s ability to preserve genome integrity requires concentration in nuclear foci during embryogenesis. These foci are sensitive to 1,6-hexanediol and dynamic in their recovery from photobleaching, both characteristics of phase separated condensates. Further, temperature stress leads to the reversible dissociation of MET-2 from nuclear foci in embryos, and recovery from heat shock requires MET-2. Paradoxically, transcriptional profiling reveals a striking correlation between embryos that undergo heat stress and embryos lacking met-2, while MET-2 is required for robust induction of HSF-1 nuclear concentration. The sensitivity of MET-2 foci to heat stress is uncoupled from canonical signaling pathways and may reveal a feature of the heterochromatic compartment broadly, as the HP1 homolog HPL-2 colocalizes with MET-2 and also dissociates from foci upon exposure to elevated temperature, coincident with chromatin-wide reorganization. Finally, we demonstrate an organismal ‘memory’ of heat stress, as larvae that experience heat shock survive better if previously conditioned as embryos. Taken together, these findings suggest an organism’s tolerance and recovery from early life stress may require both establishment and subsequent remodeling of the heterochromatic compartment.
ACR-16 clustering at putative neuro-neuronal synapses

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At the Caenorhabditis elegans neuromuscular junction, homomeric ACR-16 acetylcholine receptors form postsynaptic clusters in register with acetylcholine release sites and mediate excitatory transmission. We created a knock-in strain which expresses ACR-16 receptors tagged with a red fluorescent protein. Unexpectedly, we observed that ACR-16 receptors form a line of round and sharp clusters that parallels the neuromuscular junctions at the ventral cord. To identify the cells generating these clusters, we used the auxin-inducible degron system and used CRISPR/Cas9 to introduce a degron sequence in the acr-16-rfp locus. Muscle-specific expression of TIR-1 caused the disappearance of ACR-16-RFP from neuromuscular junctions. However, round punctae remained unchanged, suggesting a neuronal expression. To better investigate in which cells acr-16 is expressed, we built a transcriptional reporter. We observed that several neurons expressed acr-16, including 4 neurons in the head and several others along the ventral cord. We plan to identify these neurons, in particular using the NeuroPAL system. Together, these results suggest that ACR-16 clusters might correspond to neuro-neuronal synapses, which we plan to characterize at the cellular and molecular levels. First, we will confirm that these clusters correspond to bona fide synapses and does not result from random accumulation of tagged ACR-16 receptors. Second, we will perform a candidate gene approach, by testing a set of genes that are known to control ACR-16 clustering at the neuromuscular junction. We already showed that MADD-4/Punctin, the main synaptic organizer of neuromuscular junctions, is not required, suggesting the existence of distinct molecular mechanisms. Third, we will set up a forward genetic screen to isolate mutants in which the distribution of ACR-16-RFP is compromised. Altogether, our project should identify new regulators of synaptogenesis.

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A neuronal thermostat controls membrane fluidity in C. elegans

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Cells adapt to temperature shifts by adjusting lipid desaturation levels and the fluidity of membranes in a process dependent on fatty acid desaturase enzymes, and which is thought to be controlled cell autonomously. We have discovered that subtle, step-wise increments in ambient temperature can lead to the conserved heat shock response being activated in head neurons of C. elegans. This response is exactly opposite to the expression of the lipid desaturase FAT-7 in the worm’s gut with respect to temperature. We use neuronal overexpression of hsf-1, the master regulator of the heat shock response, as a tool to study the consequences of the heat shock response in neurons. We find that over-expression hsf-1, in head neurons, causes extensive fat remodeling to occur across tissues. These changes include a decrease in fat-7 expression as well as a shift in the levels of unsaturated fatty acids in the plasma membrane. These shifts are in line with membrane fluidity requirements to survive in warmer temperatures. We have identified that the cGMP receptor, TAX-2/TAX-4, expressed in a subset of sensory neurons, as well as TGF-β/BMP signaling, as key players in the transmission of neuronal stress to peripheral tissues. This suggests that a thermostat-based mechanism can centrally coordinate membrane fluidity in response to warm temperatures across tissues in multicellular animals.

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Probiotic Bacillus subtilis protects against α-synuclein aggregation in a C. elegans model

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Recent discoveries have implicated the gut microbiome in the progression and severity of Parkinson’s disease (PD), however, how gut bacteria affect such neurodegenerative disorders remains unclear. We identified a probiotic bacterial strain, Bacillus subtilis PXN21, which not only inhibits α-synuclein aggregation but also clears preformed aggregates in an established C. elegans model of PD. This protection is seen in young and ageing animals and is partly mediated by DAF-16. We found that multiple B. subtilis strains can trigger the protective effect via both spores and vegetative cells, partly due to biofilm formation in the gut of the worms and the release of bacterial metabolites. We identified several host metabolic pathways differentially regulated in response to probiotic exposure, including sphingolipid metabolism. We further demonstrated functional roles of the sphingolipid metabolism genes lagr-1, asm-3, and sptl-3 in the anti-aggregation effect. Our findings provide a basis for exploring the disease-modifying potential of B. subtilis as a dietary supplement.

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Linking instantaneous behavior with global outcomes in Caenorhabditis elegans chemotaxis

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We aim to link the instantaneous behaviors of *Caenorhabditis elegans* (those that happen in the timescale of seconds) to their consequences in longer timescales (hours or days). One of the most relevant among such consequences is the foraging success of worms in complex environments. Our previous work showed that the distribution of worms across food patches of different qualities is described to great accuracy by a simple rule: The number of worms inside each patch is proportional to a power of the amount of food contained by each patch. This was found independent of the number of patches (up to 4) and the food ratio between patches (up to 1/16). This distribution of individuals, known in Ecology and Game Theory as the Ideal Free Distribution, is predicted to be an evolutionary stable strategy.

To understand how this distribution emerges from individual behavioral rules, we studied how worms can use chemotaxis to find food patches in complex environments. Most of the previous work done in chemotaxis, both in *C. elegans* and in other species, focuses on how an individual can detect and climb a gradient of chemoattractant that increases monotonically towards its source. In contrast, we studied the more realistic case in which sources of chemoattractant with the same composition but different density create a complex landscape of chemical cues.

We first focused on the physical properties that drive the diffusion of chemoattractants. The concentration profile presented by a diffusive substance is of an exponential nature. This imposes strong constraints to the ability to distinguish from a distance the quality of different sources. These constraints provide parameter-free predictions, and we found two particularly striking results. First, in a wide range of conditions the probability to reach each of two different sources is almost independent on their relative quality. Second, we found a surprising trade-off: Being more sensitive to a given chemoattractant makes an individual less capable to distinguish a high-quality source from a low-quality one.

To test our predictions experimentally, we studied how *C. elegans* chooses between two drops of NaCl of different concentrations. As predicted by our theory, we find that worms detect the difference more easily when the concentrations are in a range near their detection threshold than when they are higher. This result cannot be attributed to the loss of attraction to NaCl at high concentrations, because a control with a single drop shows monotonically increasing

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attractiveness in our whole range of concentrations (which is always below 50 mM). Our results highlight the importance of disentangling which aspects of behavior are attributable to the characteristics of the animal, and which ones are due to the constraints imposed by the environment or the experimental situation, and are therefore generalizable to other organisms and situations.
Development of a methodology to screen the biological activity of cheese fractions, using the nematode Caenorhabditis elegans

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Natural products have been a successful source of new drugs over the last 30 years(1). If approximately 15% of medicinal plants have been investigated, only less than 1% of the microbial biodiversity has been explored. In this context, the complex microorganisms diversity found in fermented food, especially cheese, represents a great interest as a source of new bioactive molecules. The goal of this study is to develop and validate a methodology to identify bioactive fractions extracted from cheese and having beneficial biological effects and to identify molecules of interest.

Caenorhabditis elegans is an in vivo model that is used in ecotoxicological studies(2) and to screen the probiotic effect of bacteria(3). However, C. elegans is under-exploited to characterize the biological properties of extracts or potentially beneficial molecules. In that way, our laboratory has developed a methodology to screen the biological activity of cheese fractions using the nematode.

For this purpose, a process of chemical extraction from cheese has been developed. The activity of the resulting fractions was then characterized measuring the lifespan of the nematode. A specific methodology has been developed to evaluate its survival in presence of the different fractions added in its growth medium. Preliminary results are promising, showing an impact of cheese fractions on C. elegans survival.

Related publications:


*Speaker

Curative Treatment of Candidiasis by the Live Biotherapeutic Microorganism Lactobacillus rhamnosus Lcr35® in the Invertebrate Model Caenorhabditis elegans: First Mechanistic Insights

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The resistance of Candida albicans to conventional drug treatments, as well as the recurrence phenomena due to dysbiosis caused by antifungal treatments, have highlighted the need to implement new therapeutic methodologies. The antifungal potential of live biotherapeutic products (LBP) has already been demonstrated using preclinical models (cell cultures, laboratory animals). Understanding their mechanisms of action is strategic for the development of new therapeutics for humans. In this study, we investigated the curative anti-C. albicans properties of Lactobacillus rhamnosus Lcr35® using the in vitro Caco-2 cell and the in vivo Caenorhabditis elegans models. We showed that Lcr35® does inhibit neither the growth (p = 0.603) nor the biofilm formation (p = 0.869) of C. albicans in vitro. Lcr35® protects the animal from the fungal infection (+225% of survival, p < 2 10–16) even if the yeast is detectable in its intestine. In contrast, the Lcr35® cell-free supernatant does not appear to have any antipathogenic effect. At the mechanistic level, the DAF-16/Forkhead Box O transcription factor is activated by Lcr35® and genes of the p38 MAP Kinase signaling pathway and genes involved in the antifungal response are upregulated in presence of Lcr35® after C. albicans infection. These results suggest that the LBM strain acts by stimulating its host via DAF-16 and the p38 MAPK pathway.

*Speaker
Proteolytic activation of endo-siRNA pathways

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Hundreds of proteases process and/or degrade proteins to control physiological processes in the animal body. Members of the DPPIV family are clinically important, as illustrated by the control of type-II diabetes through pharmacological inhibition of DPP4. To explore the physiological functions of DPP IV proteins, we deleted each of six Dipeptidyl Peptidase Four (IV) family (dpf) genes in C. elegans. We observed that deletion of dpf-3, orthologous to mammalian DDP8/9, causes temperature-sensitive male sterility. This phenotype is characteristic of small RNA pathway defects, and we find a specific subset of endo-siRNAs depleted in dpf-3 mutant animals. Concomitant transposon activation and DNA damage result in a loss of sperm and sperm precursor cells. Endo-siRNAs mediate transposon silencing by guiding Worm ArGOnaute (WAGO) proteins to their targets, and we provide evidence that DPF-3-mediated proteolytic processing is important for WAGO protein maturation and function. Thus, we have identified a new level of control of endo-siRNA activity.

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The balance between excitation and inhibition is critical to the proper function of neural circuits. GABA, the main inhibitory amino-acid neurotransmitter in mature neurons, is a remarkably multi-functional neurotransmitter: it can bind to either ionotropic GABAA (mediating fast neurotransmission) or metabotropic GABAB receptors (mediating slow neurotransmission) that may be localized extra-, peri-, pre- and postsynaptically. The GABAergic phenotype in vertebrates and invertebrates has been defined classically by the presence of three key players in the presynaptic neurons: (i) glutamic acid decarboxylase (GAD), the enzyme needed to synthesize GABA from glutamate, (ii) the H+-coupled transporter (VGAT) that packages GABA in synaptic vesicles, and (iii) the Na+-coupled transporter (GAT) that recaptures GABA at the nerve terminal after its release in the synaptic cleft. For over 20 years, the *C. elegans* GABAergic nervous system was thought to be composed of only 26 out of the total 302 neurons. However, an in-depth revision of the GABAergic nervous system in *C. elegans* was performed. In particular, we have shown that additional neurons contain GABA but do not always express GAD/*unc-25*, VGAT/*unc-47* and GAT/*snf-11*, the landmark gene portfolio for classical GABAergic neurons. Indeed, 22 new GABA-positive cells that do not conform to this classical definition were identified. They can be categorized into 4 different types of neurons expressing different combinations of these factors. Two of these types show evidence of alternative modes of GABA transport because they lack expression of known GABA transporters, VGAT/*unc-47* and/or GAT/*snf-11*, and they do not synthetize GABA. Deciphering these new mechanisms of GABA transport will shed light into the regulation of neural circuits through inhibition.
A system for the high-throughput analysis of acute thermal avoidance and adaptation in C. elegans

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Avoiding damage is essential for survival. In animals, this is achieved via nociceptive sensory neurons, which trigger avoidance behaviors in response to noxious stimuli. Remarkably, prolonged noxious stimulations may engage plasticity mechanisms, including adaptation, where nocifensive responses are dampened. Despite the potential for breakthroughs in clinical pain management, we know surprisingly little about nociceptive adaptation processes.

Our group studies thermal nociception in C. elegans, which is a well-suited model in neuroscience, given the evolutionary conservation of molecular and neural functions, its amenability to genetic manipulations and its well-described nervous system and behavioral repertoire. C. elegans produces robust heat avoidance responses, which are dampened upon prolonged heat stimulation.

In order to fully exploit this promising paradigm, we developed a methodology suitable for the high-throughput analysis of C. elegans heat-evoked reversals and the adaptation to repeated stimuli. We introduce two platforms: the INFERNO (for infrared-evoked reversal analysis platform), allowing the quantification of the thermal sensitivity in a petri dish containing a large population (> 100 animals), and the ThermINATOR (for thermal adaptation multiplexed induction platform), concomitantly running an adaptation protocol in up to 18 worm populations at the same time.

Combining the INFERNO and ThermINATOR platforms with the powerful genetic and circuit analysis tools available in C. elegans will enable an efficient and comprehensive analysis of the mechanisms underlying nociceptive adaptation.

*Speaker
Argonaute slicer activity is required for maternal mRNA clearance in C. elegans embryos

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Argonaute proteins and their interacting small RNAs play a key role in regulating complementary mRNA targets during metazoan development. Despite the conserved slicer activity of Argonautes, their endogenous catalytic functions remain largely unknown. Here, we show that the depletion of the Caenorhabditis elegans Argonaute CSR-1 during embryonic development leads to embryonic lethality in a catalytic-dependent manner. The embryonic CSR-1 interacts with endogenous small RNAs antisense to hundreds of cleared maternal mRNAs, and preferentially cleaves those no longer engaged in translation. Thus, the slicer activity of CSR-1 is required for maternal mRNA clearance during the maternal-to-zygotic transition in C. elegans embryos. Given the conservation of Argonaute catalytic activity, we propose that a similar mechanism operate to clear maternal mRNAs during maternal-to-zygotic transition across species.

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Role of the SIN3/HDAC histone deacetylase complex in the C. elegans germline

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Histone post-translational modifications contribute to the regulation of gene expression through complex context dependent mechanisms. In the germline of *Caenorhabditis elegans*, transcription from the single X in males and both Xs in hermaphrodites is repressed by a mechanism dependent on histone modifications and distinct from the dosage compensation complex (DCC) operating in the soma. X silencing in the germline relies on global repression mediated by the PRC2-like MES complex, which deposits H3K27me3. Lack of MES/PRC2 activity results in derepression of the X and sterility. The X chromosome in the germline is also globally hypoacetylated, but the mechanism leading to hypoacetylation, and its contribution to X silencing, remains unknown. The histone deacetylase complex SIN3/HDAC is associated with gene repression or activation depending on the chromatin context, by poorly understood mechanisms. The lab recently obtained transcriptomic data showing that in the absence of SIN-3, the main component of the SIN3/HDAC complex in *C. elegans*, the X chromosome is globally derepressed, with the vast majority of genes being upregulated. SIN-3 inactivation also leads to sterility. Therefore, SIN-3/HDAC contributes to X-chromosome silencing, and plays an important role in the *C. elegans* germline. This provides a unique opportunity to study the role of this conserved complex in a specific developmental context. Through recently developed protocols, we will delve into the impact of the absence of SIN-3 on histone post-translational modifications, specifically in the worm’s germline.

*Speaker
Formin dynamics and actomyosin contractility at the cell cortex in the early C. elegans embryo

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Proper development and morphogenesis relies on fine-tuned spatial and temporal deployment of forces inside cells. The actomyosin cytoskeleton is determinant for the deployment of these forces and so, for the mechanical properties of embryonic cells and tissues. We investigated on how the actin network is organized during contractility events, such as the pulsed contractions in the two cell stage embryo, and analyzed the processive formin CYK-1 as a proxy. Formins are responsible for the polymerization of new actin filaments. We used HILO (near-TIRF) live imaging and single molecule automatic tracking of CYK-1::GFP to quantify the anisotropy of the actin network during pulsed contractions. Our results dress up a new model of both spatial and temporal organization of the actin network during cortical pulsed contractions in the C. elegans early embryos.

*Speaker
Identification of actin cytoskeletal content variation throughout the early lineage of C. elegans embryo

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C. elegans embryo follows a well-defined developmental pattern in which invariant cleavages establish reproducible patterns of cellular interactions. In addition to the rapid reduction in volume, many cellular events like polarization and asymmetries appear biochemically and physically in the daughter cells. This will lead in some divisions to a reproducible but diverse collection of founder cells. That will give rise to the establishment, step by step, of different cell identity, which is acquired through correct cell polarization, division and interaction patterns. All these processes rely in part on actin architectures, which is involved in cell shape and organization of the intracellular space while responding actively to the environment. To do so, actin is organized in a variety of dedicated architectures in a spatiotemporally regulated manner such as the thin cortex, filopodia and stress fibers. Cells modulate the composition, dynamics and architecture of these actin networks. It is known that specific actin architectures can affect cell morphology, mechanics and gene expression profiles and can thus modulate and feedback into the process of determination of cell identity. In my PhD project, I am studying how actin architecture and its nucleation are spatiotemporally controlled in the different cell types found in the early C. elegans embryo and how these actin specificities could impact cell commitment in the differentiation process. With the help of CRISPR GFP Knock-In strains, Spinning Disk Microscopy and quantitative image analysis, I am following two of the main actin nucleators (Arp2/3 complex and the formin CYK-1) which have cell-specific spatiotemporal distribution at the cell cortex, in specific subcellular structures and the cytosol. Comparison are made between sister cells or along the AP axis for example. This leads to heirloom questions: Are cells equal in terms of actin related content? Are the properties of actin cortex inheritable? How important is maternal inheritance of actin content for these early specifications? Is the composition of APBs scaling with the cell size?

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VerMidi XXIII - Sorbonne University
Friday March 13, 2020 – Sorbonne University, Paris

Invited Speaker
SEBASTIAN GREISS
UNIVERSITY OF EDINBURGH

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IECB
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IBV
Rajani Gudipatti
FMI
Anna Mattout
CBI
Jan Padeken
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