Young

Embryologist

Network

2026

Conference booklet

15th May 2026 | The Francis Crick Institute

YEN 2026 Programme

8:15-9:00 Registration

9:00-9:15 WELCOME ADDRESS

9:15-9:45 Diana Pinheiro (IMP, Vienna) - *Linking fate and shape in vertebrate gastrulation*

9:45-11:15 SHORT TALKS:

Yisha Lan (University of Cambridge) - *Nodal organized cell dynamics for building tissue shapes in vertebrate gastrulation*

Ioakeim Ampartzidis (University of Cambridge) - Planar Cell Polarity Drives Tissue Remodelling During Mammalian Body Plan Establishment

Laura Bader (The Francis Crick Institute) - Nuclear mechano-adaptation in a developing beating heart

Anh le (University College London) - Tissue flow acts as a guidance cue for immune cell polarisation and directional migration

10:45-11:15 Break/Posters (even numbers)

11:15-11:45 Christopher Thomas (IBDM, Marseille) - *Revealing the Secrets of Ovulation*

11:45-12:45 SHORT TALKS:

Shayma Abukar (University College London) – Deciphering cardiac lineages at single-cell resolution

Eleonore Ocana (University of Cambridge) - Uncovering temporal regulation mechanisms in mouse diapause

Meng Zhu (University of Cambridge) - Maternal hypoxia regulates limb heterochrony during mammalian embryogenesis

Tjaša Šentjurc (Royal Veterinary College, London) - Hyperleptinemia in obese mice compromises maternal-fetal crosstalk during placental developmental and function

12:45-13:45 Lunch

13:45-14:15 SCIENTIFIC PERSPECTIVES I: **Cambridge Reproduction Sciart- *Inside Pregnancy: Creative Perspectives on Reproductive Biology (Art exhibition)***

14:15-15:15 SHORT TALKS

Ignacio Rodriguez Polo (The Francis Crick, London) - Primate Gastruloids Model Early Embryonic Patterning and Endothelial Emergence

Bowen Chen (King's College London) - Shaping the Ear: Exploring the Physical and Mechanical Cues

Marc-Eric Perrin (Institute of Developmental Biology of Marseille)- Encoding neuronal shape in the stochastic dynamics of branching processes

Krishnanand Padmanabhan (Karolinska Institute) - In utero transduction enables clonal lineage tracing and conditional gene manipulation of the developing mouse gut including the enteric nervous system *(online presentation)*

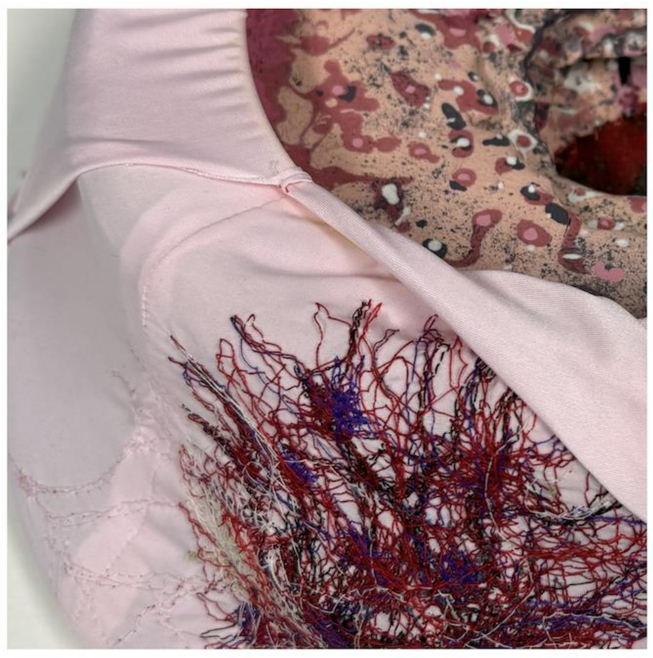
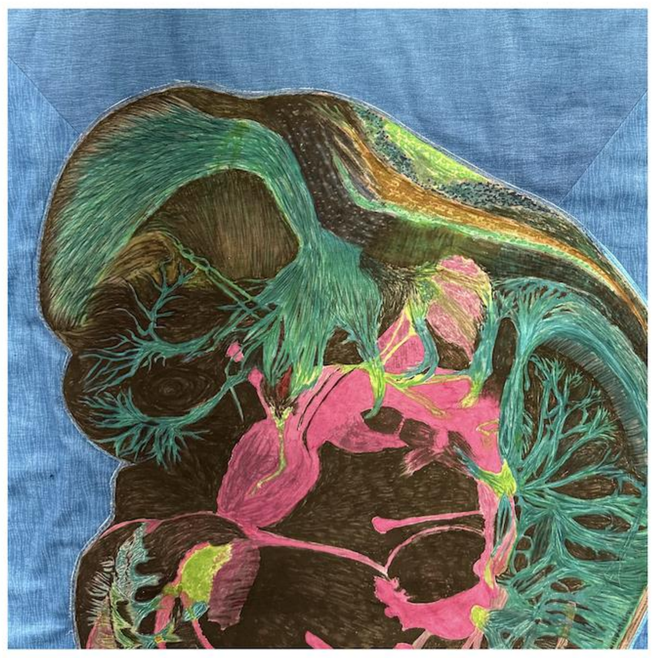
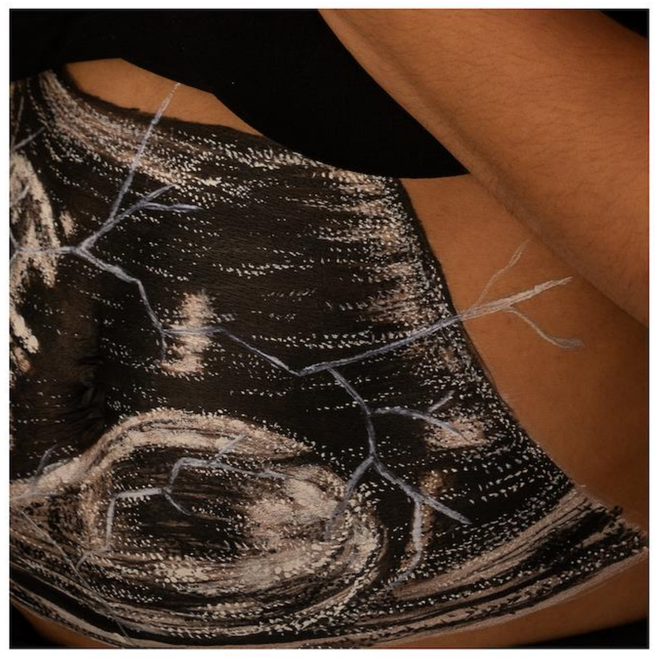
15:15-15:45 Break/Posters (odd numbers)/Art Exhibition

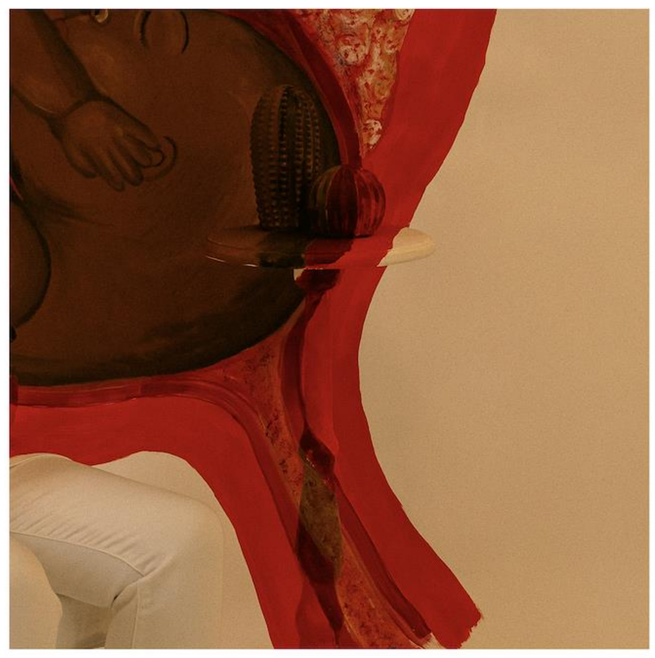
15:45-16:45 **The Sammy Lee Memorial Lecture: Kay Elder (Bourn Hall Cambridge) - *In-vitro culture of human preimplantation embryos, 1969-2026*.**

16:45-17:15 SCIENTIFIC PERSPECTIVES II **Marzia Munafò (EMBL, Rome) - *Creative science (and) illustrations - when molecular biology meets art***

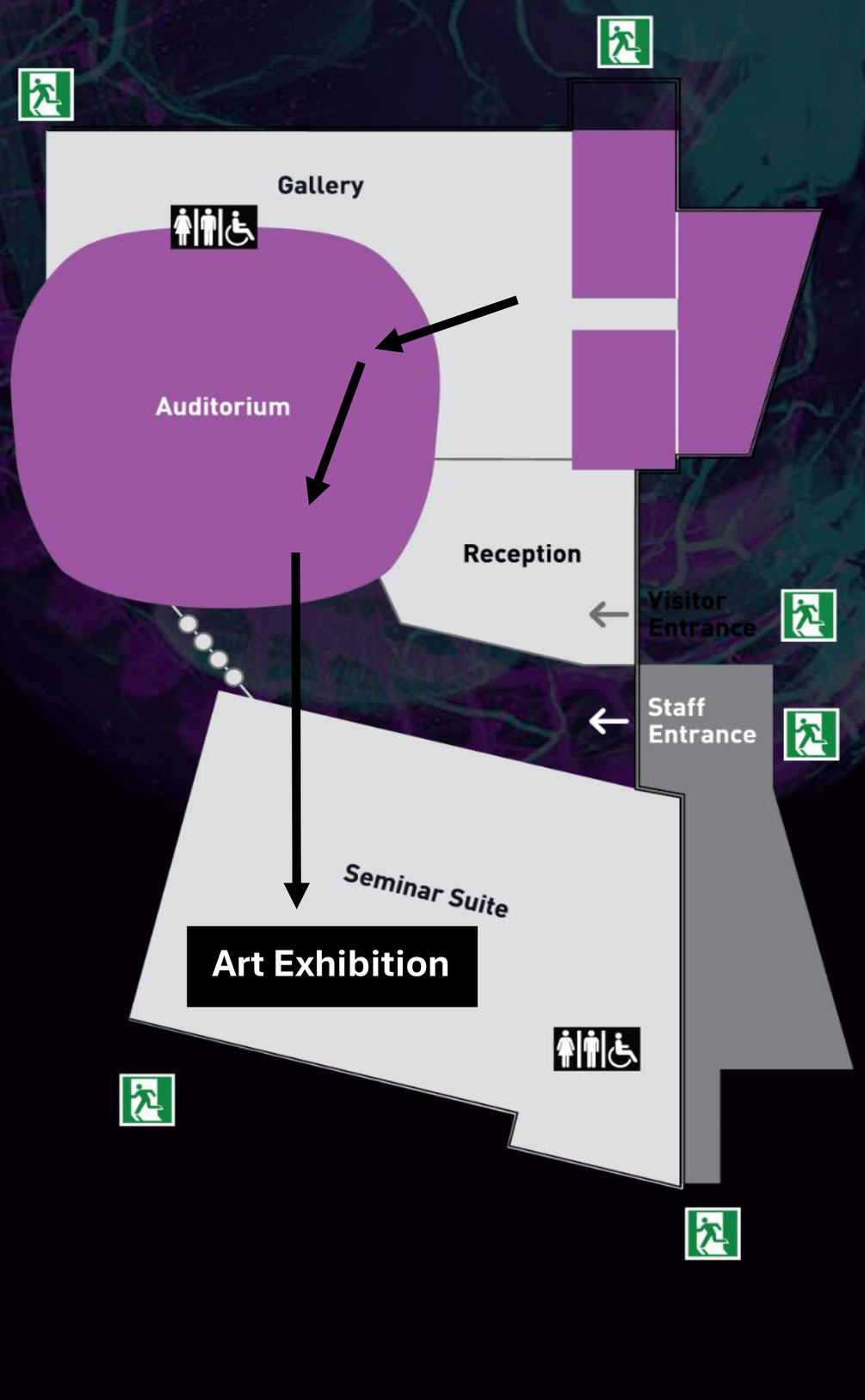
17:15-19:00 Poster and Abstract Prizes – Final remarks and Networking

Featured Art Exhibition:
The Science of Reproduction through Art
by Cambridge Reproduction Sciart

Come and see this exciting exhibition showcasing collaborations between artists and scientists!



About the Young Embryologist Network Conference 2026:

Welcome to YEN 2026! The Young Embryologist Network was founded in 2008 making this the 18th annual developmental biology meeting!

YEN 2026 is hosted in person at **The Francis Crick Institute** in London and online via Zoom. The event has a broad scope in developmental and reproductive biology research. We believe in the benefits of networking and strengthening collaborative work for early-career researchers such as PhD students and postdocs. We therefore encourage you to meet new people, ask questions, participate in the discussions and keep in touch after the conference. This is a crucial part of the development of any scientist and is why we do our best to ensure YEN conferences are free to attend and as accessible as possible.

We are excited to have invited PhD students, postdocs and young PIs, to present short talks and posters. These were selected from over 30 abstracts we have received, which we highly appreciate.

This year we have invited three phenomenal scientists who will share their research and experience in their fields.

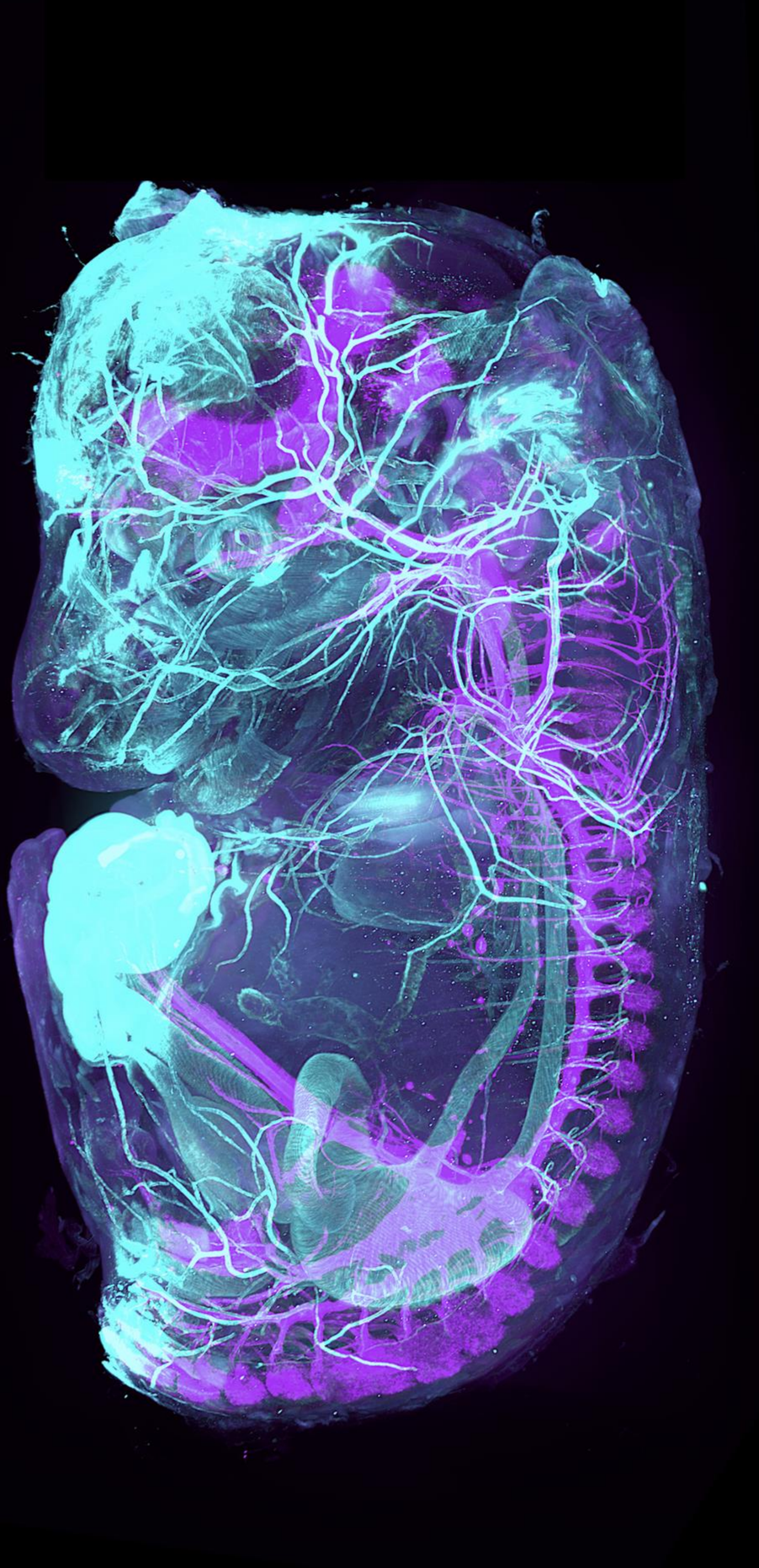
We will first hear from **Diana Pinheiro (IMP, Vienna),** who will talk about how, during the process of embryogenesis, cells acquire distinct identities and coordinated mechanical behaviours to form organized tissues and body structures. Next, we will have **Christopher Thomas (IBDM, Marseille),** who will share his insight into uncovering the cellular and mechanical mechanisms that govern ovulation. We will conclude the day with the **Sammy Lee Memorial Lecture** with our keynote speaker **Kay Elder (Bourn Hall, Cambridge),** who will take us on a journey through how in vitro culture of human preimplantation embryos has evolved from 1969 to the present day. This year, our **Scientific Perspective** session will explore the intersection of art, medicine, and scientific research, with a particular focus on how artistic practices and visual communication can represent, interpret, and communicate complex biological concepts. We will host an art exhibition from **Cambridge SciArt** and a talk by **Dr. Marzia Munafò (EMBL, Rome),** who has successfully combined her passion for molecular biology with her passion for illustration.

Young
Embryologist
Network

About the cover image:

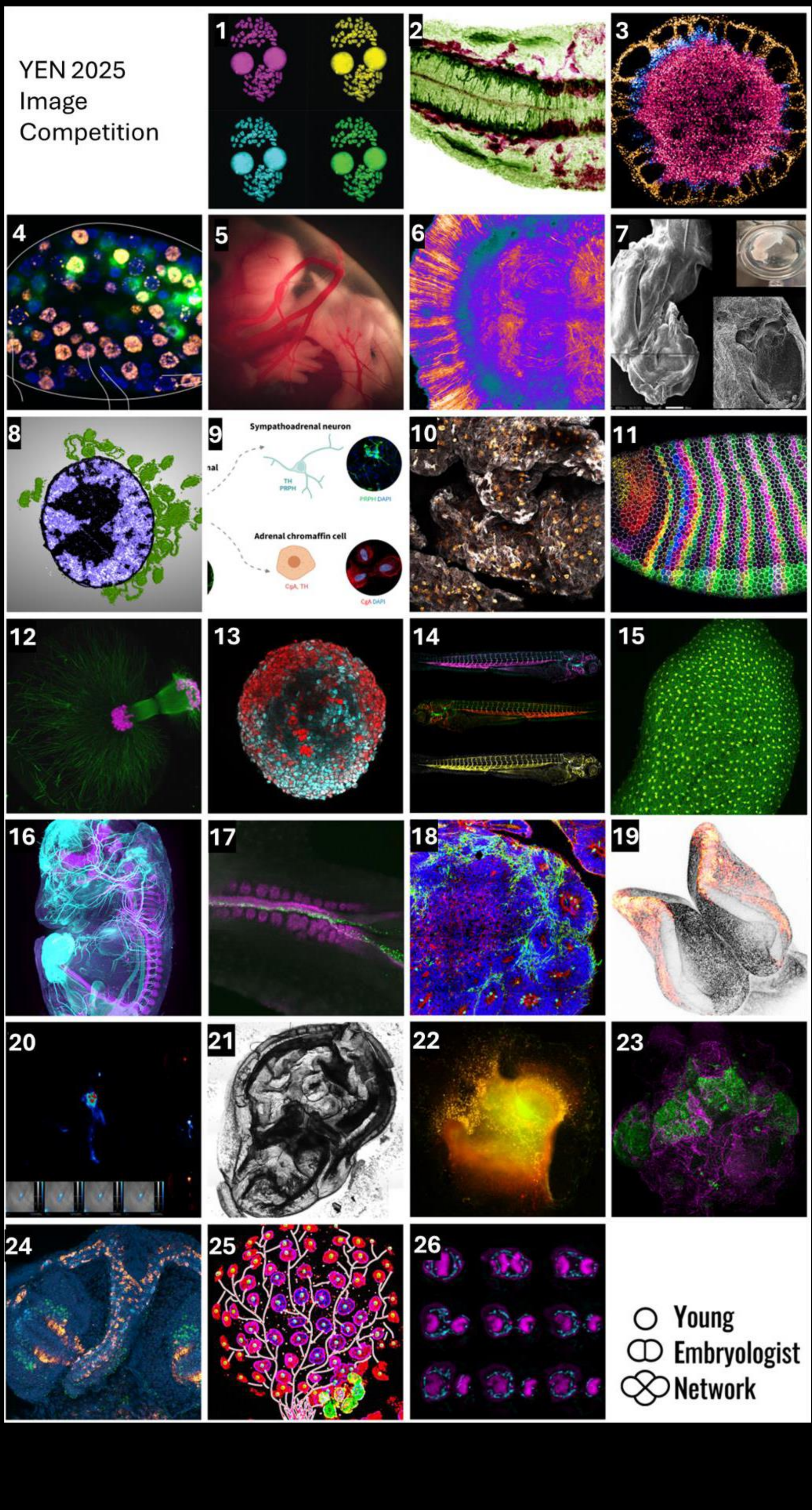
This year's cover image is the winning entry from the YEN 2025 Imaging Competition and was created by **Théo Morel (INSERM, France),** working on vascular development and homeostasis**.** The image shows a mouse embryo co-stained for arteries and sympathetic nervous system and imaged on a light-sheet microscope. We are delighted to showcase his image as the visual identity of YEN 2026 and to celebrate the creativity and scientific excellence of our community.

The imaging competition highlights the beauty of developmental and reproductive biology research and provides an opportunity for early-career researchers to share their work through scientific imaging. We are excited to continue this initiative in the years to come. As part of this tradition, the winner of the 2026 Imaging Competition will have their image featured on the cover of the YEN 2027 conference booklet and promotional materials.

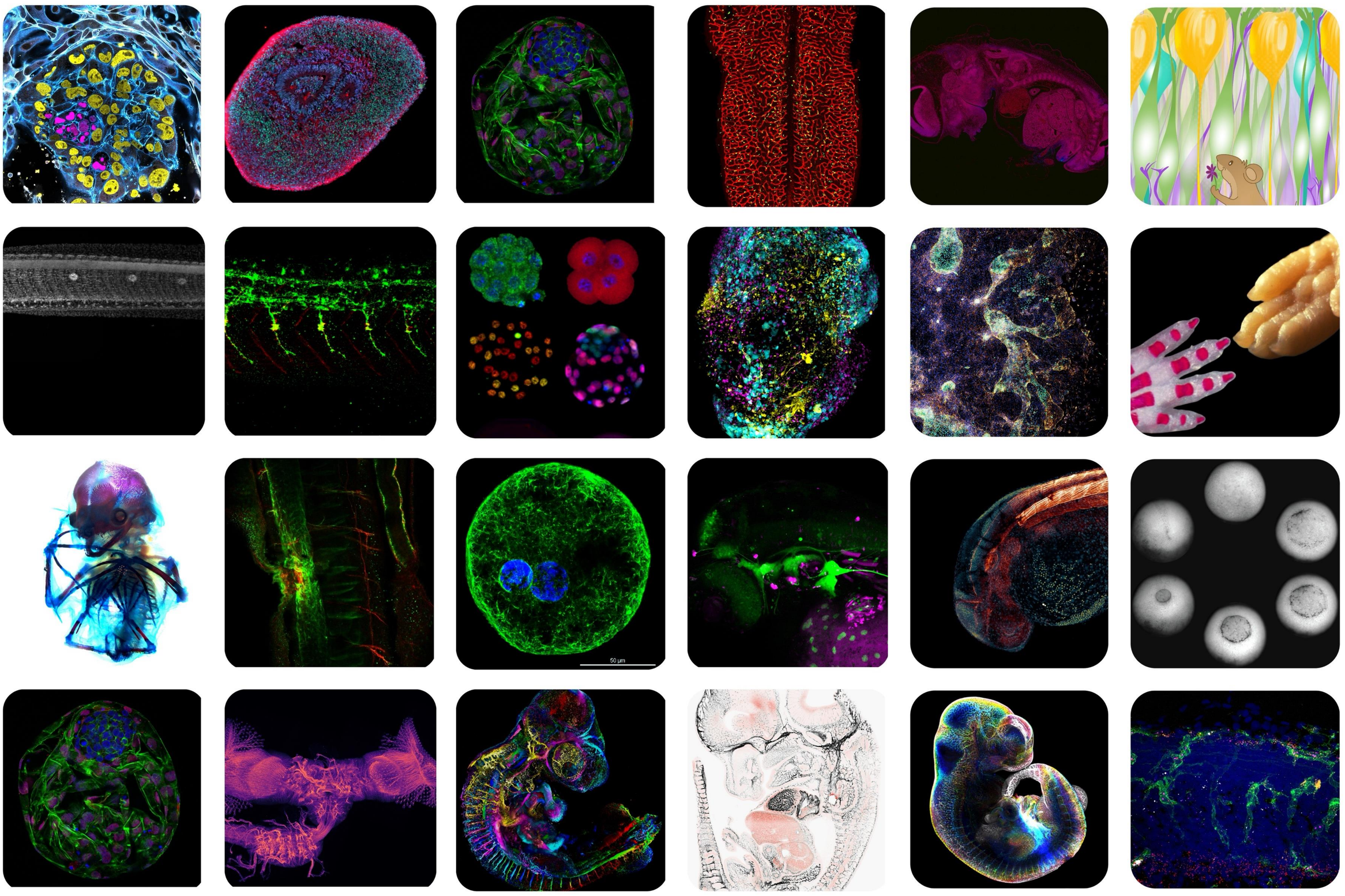


Théo Morel – *A 3D view beyond the skin*

Other images from YEN2025 imaging competition:



Don’t forget to vote for YEN2026 imaging competition!



Scan the QR Code to
vote for your favourite image, or
follow the link below:

https://docs.google.com/forms/d/e/1FAIpQLSeug5JWgF0LNwobRv2dIJN3mo4bd85ySsA9QXklKR9qkHaOfQ/viewform

Other useful information:

Follow us on social media:

Instagram: @YEN_network

https://www.instagram.com/yen_network/

Bluesky: @ yen-network.bsky.social

https://bsky.app/profile/yen-network.bsky.social

Twitter/X: @YEN_community

https://x.com/YEN_community

Website: https://www.youngembryologists.org/

Young
Embryologist
Network

Acknowledgements:

First, we would like to thank all the speakers, talk and poster presenters without whom YEN 2026 would not be possible. We would also like to thank everyone who helped us bring you YEN 2026 which includes our international representatives, the judges, Karen Lee and all the guests and attendees.
YEN 2026 Committee members
Co-chairs: Anita Bichisecchi and Timothy Wise
Committee members: Alejandra Guzman-Herrera, Ana Alex, Eleftheria Parasyraki, Catarina Ferreira, Ceren Canse, Laurine Jacob, María Perez Millet, Marta Perera Perez, Matyas Bubna-Litic, Helen Kiik, Irina Balaguer Balsells, Valeria Conde de Rafael, Jiaying Liu, Juqi Zou.
International reps: Francesca Boffa, Marc-Eric Perrin, Clara Ornella.
Judges: Karen Lee, Courtney Hanna, and Dillan Saunders.
Sponsors:
Hosting YEN and ensuring it is free of charge would not be possible without the generous support of our sponsors. We thank the following organisations: The Company of Biologists, The Francis Crick Institute, The Genetics Society and Imperial College London, REGEN, Society for Reproduction and Fertility.
We also are very fortunate to have amazing industrial sponsors who you can visit: CliniSciences, ThermoFisher, VectorBuilder, Promega and 10x Genomics.

SHORT TALKS ABSTRACTS:

Nodal organized cell dynamics for building tissue shapes in vertebrate gastrulation

By Yisha Lan (University of Cambridge)

During development, complex tissue shapes arise from collective cell behaviours, yet how molecular signals translate into tissue-scale mechanics remains unclear. Nodal, a TGF-β family morphogen, induces mesoderm and controls cell movements during gastrulation, but how it alters cell dynamics and mechanics is not well understood. To investigate this, we ectopically expressed Nodal in zebrafish embryos. Nodal+ cells induce blastoderm thickening at the animal pole and, at gastrulation onset, internalize to form an ectopic, radially symmetric “volcano”-like blastopore. This mimics normal blastopore formation in a simplified context.
Using an AI-based pipeline to reconstruct 3D cell shapes from live imaging, I found that Nodal-expressing cells have increased volume and surface area, are more elongated along the z-axis, and move in coordinated spatiotemporal patterns. Mechanical measurements (FLIM and micropipette aspiration) show that the overlying cell layer has reduced tension, initiating thickening. Cells then undergo radial intercalation toward the animal pole while spreading is restricted, leading to cell accumulation at the animal pole. This accumulation, together with increased cell size and cohesion, results in jamming and elevated pressure that further promotes thickening. Cells with the highest Nodal levels internalize and pull neighbouring cells to form the blastopore-like structure.

Planar Cell Polarity Drives Tissue Remodelling During Mammalian Body Plan Establishment

By Ioakeim Ampartzidis (University of Cambridge)

During mammalian gastrulation, the primitive streak emerges through coordinated tissue remodelling driven by apical constriction and convergent extension (CE). Epiblast cells shrink their apical membranes, intercalate to narrow and elongate the tissue, and ultimately establish the body axis. Although the planar cell polarity (PCP) pathway regulates these behaviours, the mechanisms by which PCP signalling controls mammalian body axis formation remain unknown. Here we use in vivo mouse models, co-evolutionary analyses, and human stem cell-based gastrulation systems to dissect PCP signalling during mammalian body axis establishment. Using the Looptail (Vangl2 mutant) mouse model, we identify axial elongation defects by E8.5 accompanied by epiblast- and cell-level abnormalities, hallmarks of disrupted CE and impaired apical constriction. To further dissect VANGL2 functional domains, we performed co-evolution analysis to identify regions evolving in concert with potential conserved signalling roles. Lastly, we performed an siRNA-mediated knockdown screen targeting most PCP genes in human pluripotent stem cell-derived 2D gastruloid micropatterns. Notably, VANGL2, DVL2, and FZD6 knockdown disrupt germ layer specification, supporting a conserved role for PCP signalling in early developmental patterning. Together, our findings uncover a previously underappreciated conserved role for PCP signalling in coordinating the tissue mechanics and cell fate decisions that drive body axis formation.

Nuclear mechano-adaptation in a developing beating heart
By Laura Bader (The Francis Crick Institute)

The embryonic heart starts beating while undergoing complex morphogenetic changes. The dynamic, but continuous nature of cardiac contractions in a developing heart presents a unique challenge for cardiomyocyte nuclei integrity. However, little is known about how nuclei maintain integrity throughout heart development. Using live imaging, 3D nuclei analysis and genetic perturbations, we report that differential deformation of heart chambers – ventricle and atrium – results in distinct nuclear protection mechanisms. While ventricle nuclei become increasingly indented and wrinkled, atrium nuclei remain taut throughout cardiac development. In ventricle, these nuclear indentations scale with the magnitude of cardiac forces while atrial nuclei remain unaffected. Of note, ventricle nuclei show higher expression of nuclear filament Lamin A/C and depletion leads to increased nuclear indentations. Atrial nuclei almost completely lack Lamin A/C expression and are unaffected by Lamin A/C perturbation but rely on a perinuclear pool of microtubules to sustain nuclei shape. Chamber specific nuclear morphology and Lamin A/C expression appear to be downstream of differential chamber deformation, as ventricle undergoes more deformation as atrium. Accordingly, genetically increasing atrium contractions leads to indented nuclei and higher Lamin A/C expression. Together, our findings provide new insights into how mechanical forces modulate nuclear mechanics in a developing organ.

Tissue flow acts as a guidance cue for immune cell polarisation and directional migration
By Anh le (University College London)

Embryonic myeloid (EM) cells are the first immune cell population to emerge during development and must disperse throughout the embryo to act as the first line of defence against infection. Although EM cells migrate directionally toward wounds, how they navigate toward unwounded tissues during early colonisation remains unclear. Here, we show that EM cell dispersion in Xenopus embryos is driven by cell-on-cell migration, an important yet underappreciated phenomenon, guided by dynamic tissue flows. By taking advantage of the easy-to-culture nature of Xenopus embryo tissue, we established an ex vivo EM cell migration system combining live imaging, computational analyses, and optogenetic manipulation. We find that local ectodermal tissue flows repolarise EM cell protrusions and bias their directional migration. Disrupting these flows in vivo, either genetically or mechanically, impaired EM cell dispersion. Our findings reveal that mechanical cues generated by surrounding tissue flows coordinate immune cell migration during development, highlighting an overlooked mechanism by which collective tissue dynamics guide individual cell behaviour.

Deciphering cardiac lineages at single-cell resolution
By Shayma Abukar (University College London)

The heart is the first organ to develop during embryogenesis, and its formation relies on the complex specification of distinct cardiac lineages. Recently, we have shown that chamber-specific progenitors have discrete spatial and temporal origins during gastrulation. What remains unclear is whether cardiac progenitors leaving the primitive steak are restricted in their fate or can contribute to other mesodermal derivatives. Here, we use a live-imaging and single-cell tracking approach to identify the point at which cardiac precursors, that contribute solely to the heart tube, first arise in the embryo. Through analysis of 5 live-imaging datasets, spanning 22-40 hours, we tracked the fate and behaviour of mesodermal progeny to determine when cardiac progenitors are specified in the nascent mesoderm, and if they exhibit distinct migratory behaviours.
Our study shows that the generation of the heart tube relies on at least two independent groups of early and late mesodermal progenitors that contribute to the ventricular and atrial regions of the heart tube respectively. Analysis of the migration of unipotent and bipotent mesoderm progenitors reveals greater dispersion of progeny of differing fates compared to those of the same fate, suggesting there is coordination in the migration of progenitors that are fate-committed.

Uncovering temporal regulation mechanisms in mouse diapause
By Eleonore Ocana (University of Cambridge)

The precise molecular mechanisms that control how developmental processes unfold at a specific tempo are largely unexplored and are essential for the formation of a healthy organism.
In stem cell models of mouse and human motor neuron differentiation, changes in the pace of development have been associated with differences in protein stability. However, we do not yet know whether this relationship is a general principle for changes in developmental rate or whether protein stability regulates developmental rate. To address this, we are using diapause as a model for pace modulation within a single species. Diapause is a reversible state of suspended animation induced in response to environmental stressors that results in delayed blastocyst implantation. We can artificially mimic this process in mouse embryos via mTOR inhibition. We hypothesize that during diapause, global protein stability is increased.
Our data suggest that the pluripotency factor OCT4 is stabilised under mTOR-mediated pausing ex vivo. We have developed methods to measure protein stability in mouse blastocysts. Global proteome stability appears to be differentially regulated in response to mTOR inhibition in different lineages of the embryo. We are now exploring the role of the proteasome in diapause induction and maintenance.

Maternal hypoxia regulates limb heterochrony during mammalian embryogenesis
By Meng Zhu (University of Cambridge)

Alterations in tissue growth timing, known as heterochrony, is thought as a driving force for evolution, but has been poorly addressed mechanistically. A prominent example is the limb heterochrony, where different tetrapod clades exhibit distinct temporal patterns for forelimb and hindlimb growth. In general, mammalian species display delayed hindlimb development compared to the forelimb. However, this feature is absent in avian species, where forelimbs and hindlimbs grow simultaneously. Such a distinction has been hypothesised as an adaptation to the energy supplies. Yet, the regulatory mechanisms of limb heterochrony remain elusive.
To comprehend limb heterochrony regulation in mammals, I compared the timing of key limb developmental events in chicken to that in mouse and opossum embryos. I found that the mammalian limb heterochrony starts at the limb initiation, characterised by the Epithelial-to-Mesenchymal Transition (EMT).
The varying timing of EMT is in turn associated with the timing of limb outgrowth regulators. Unexpectedly, I found that this heterochronic expression is not due to changes in cis-regulatory elements or the timing of growth factor induction but is linked to the physiological conditions experienced by the growing embryo. Functional navigation revealed that, the differential oxygen levels to which avian and mammalian embryos are exposed mediate the heterochronic expression of limb outgrowth factor. Specifically, hypoxic environment in early mammalian embryogenesis suppress hindlimb initiation. By integrating RNA-sequencing analyses with gain- and loss-of-function assays, I determined that hypoxia's impact on hindlimb development is at least partially implemented through the expression of NFKB transcription factors. Taken together, these results provide a comprehensive mechanistic account of developmental heterochrony, and exemplifies how tissue growth alters the timing during evolution.

Hyperleptinemia in obese mice compromises maternal-fetal crosstalk during placental developmental and function
By Tjaša Šentjurc (Royal Veterinary College, London)

Maternal obesity (MO) increases the risk of adverse pregnancy outcomes, with long-term implications for offspring health. Studies in mouse models of MO have shown that morphological changes in the placenta result from altered trophoblast differentiation. We previously reported that impaired decidualisation and placentation in obese mice are associated with the upregulation of leptin signalling modulators PTPN2 and SOCS3 in the uterus. We presently investigate the effects of altered uterine leptin signalling on trophoblast differentiation and placental function in diet-induced obese mice.
Western blot analysis of placentas from gestational day (E) 18.5 demonstrated increased SOCS3 and PTPN2 protein expression in obese mice, with immunofluorescence staining (IF) revealing higher expression of both proteins in the junctional zone.

RNA sequencing identified 136 differentially expressed genes (DEGs; p<0.05; FC>0.5), predominantly comprising genes upregulated in glycogen trophoblast cells (GlyT) and downregulated in the decidua. Deconvolution of bulk RNAseq data from E13.5 placentas also revealed an upregulation of GlyT-expressed genes (43 genes among the 270 DEGs), linked to leptin signalling and cell cycle regulation. Finally, IF of blastocysts from obese mice at E3.5 showed a downregulation of the trophoectoderm marker CDX2, as well as PTPN2 (p<0.05), suggesting developmental delay. These findings indicate that structural and functional defects in placentas from obese mice are largely associated with altered leptin signalling, likely compromising trophoblast development during embryo implantation and establishment of the maternal-fetal interface.

Primate Gastruloids Model Early Embryonic Patterning and Endothelial Emergence
By Ignacio Rodriguez Polo (The Francis Crick, London)

Gastruloids are three-dimensional pluripotent stem cell (PSC) aggregates that model key aspects of early development, including symmetry breaking, axial elongation, and germ layer formation, providing an in vitro system to study post-gastrulation fate decisions and early tissue interactions that are otherwise challenging to access in vivo. Here, we adapted the human gastruloid protocol to generate human, macaque, and baboon gastruloids under identical conditions, producing organised peri-gastrulation cell types consistent with published in vivo human and macaque single-cell RNA sequencing embryo benchmarks and establishing a platform for comparative analysis of primate developmental programs. Characterisation of primate gastruloids revealed variability across cell backgrounds in both human and macaque models, including a bias toward endoderm formation. Notably, endoderm-enriched gastruloids showed the emergence of endothelial-like cell populations by day 4, suggesting endothelial fate commitment driven by endoderm–mesoderm interactions and contributing to early vascular plexus-like structures.
Together, these findings establish a pan-primate gastruloid platform and provide new insights into cell composition and fate transitions in human and non-human primate gastruloids, offering future opportunities to dissect interspecies differences and generate PSC-derived advanced medical products for cell replacement therapies across multiple fate transitions, species, and maturation states within a physiologically relevant niche.

Shaping the Ear: Exploring the Physical and Mechanical Cues
By Bowen Chen (King’s College London)

Mechanical forces have recently emerged as central regulators of embryonic development, influencing processes such as cell fate specification, tissue differentiation, and the shaping of organ architecture. However, how mechanical cues integrate with molecular signalling to guide early morphogenesis remains unclear in many systems. The developing inner ear provides a powerful context for addressing this question because its intricate three-dimensional structure depends on precisely coordinated physical and biochemical inputs.
This study investigates how the mechanical microenvironment influences the initial patterning and outgrowth of the cochlear duct in chick embryos. The inner ear originates from the otic vesicle (OV), a simple epithelial sphere that remodels into the complex adult organ. Early in development, the OV generates two asymmetric protrusions, the dorsal endolymphatic duct and the ventral cochlear duct, which we hypothesize arise from mechanical heterogeneity in surrounding tissues. Brillouin microscopy reveals a pronounced stiffness, with a rigid neural tube dorsally and a more compliant mesenchyme ventrally.
To test how this asymmetry shapes OV morphogenesis, we developed a hydrogel-based ex vivo culture system that recreates dual stiffness conditions. This platform supports OV development and promotes directional outgrowth. By investigating the underlying mechanotransduction pathways, we observed spatially distinct responses to mechanical cues. Dorsally, YAP activation is enriched, reflecting exposure to the stiffer neural tube.
Ventrally, single-cell RNA sequencing and in situ hybridization chain reaction identified localized expression of the mechanosensitive ion channels PIEZO2 and TRPM3 precisely at the tip of the OV, where cochlear outgrowth initiates.
Together, these findings suggest that mechanical forces act alongside molecular pathways to guide early inner ear morphogenesis. This work provides foundational insight into how tissue mechanics contribute to organ shaping and establishes a versatile platform for studying mechano-regulated development with relevance to regenerative and bioengineering strategies for hearing disorders.

Encoding neuronal shape in the stochastic dynamics of branching processes
By Marc-Eric Perrin (Institute of Developmental Biology of Marseille)

Cell shape is central to cellular function, yet how reproducible morphologies emerge from stochastic cellular dynamics remains unclear. We addressed this question by studying dendrite development in two morphologically distinct classes of Drosophila mechanosensory neurons. Combining in vivo live imaging, quantitative analysis, cytoskeletal perturbations, and computational modeling, we compared dendritic branch dynamics across cell types. Although branches in both neuron classes follow similar local stochastic behaviors, their arbors develop along distinct trajectories that cannot be explained by standard diffusive growth models. We show that this divergence arises from long-timescale subdiffusive dynamics in Class I neurons, which are absent in Class IV neurons.

Guided by these observations, we developed a minimal four-parameter model that separates rapid exploratory fluctuations from slower branch dynamics and reproduces both developmental growth and final arbor architecture in the two classes. Cytoskeletal perturbations reveal complementary roles for actin and microtubules: actin promotes short-term branch exploration and arbor expansion, whereas microtubules regulate long-term branch mobility and class-specific morphology. These findings provide a general framework linking local cytoskeletal control to global neuronal architecture and show how stochastic branch dynamics can generate reproducible cell shapes.

In utero transduction enables clonal lineage tracing and conditional gene manipulation of the developing mouse gut including the enteric nervous system
By Krishnanand Padmanabhan (Karolinska Institute)

Understanding the molecular mechanisms governing development of the mouse central nervous system (CNS) has been greatly facilitated by efficient in-utero electroporation approaches. In contrast, enteric nervous system (ENS) research has lacked a robust in vivo gene-manipulation platform capable of targeting the developing ENS, which is embedded within the gut wall. This limitation has impeded functional interrogation of candidate regulators and contributed to a substantial knowledge gap relative to CNS development. To address this challenge, we developed an ultrasound-guided in-utero gene-delivery approach that enables precise targeting of the developing ENS. Lentiviral vectors are injected into the amniotic cavity of E7.5 mouse embryos, allowing transduction of the enteric anlage, the primordial neural crest while it remains contiguous with the neural plate. Preliminary data demonstrate successful targeting not only of the ENS, but also of additional neural crest derivatives, including dorsal root ganglia that innervate the gut, thereby directly linking ENS and CNS development. Notably, this system also labels progenitors that give rise to fibroblast, myoblast, endothelial, and immune lineages of the developing gastrointestinal tract. Using lentiviral barcoded lineage tracing, we identify previously unappreciated lineage decisions that contribute to fibroblast and epithelial diversification and regional identity. We further show that cell type–specific genetic manipulation can be achieved by delivering Cre-inducible double-inverted-orientation (DIO) viral constructs into transgenic Cre lines. Using the Sox10-CreERT2 model, we demonstrate the ability to direct enteric progenitors toward neuronal differentiation through Ascl1 overexpression. This versatile in utero gene manipulation platform represents a significant technical advance for ENS research, enabling high-throughput and mechanistically focused functional analyses of the growing set of candidate regulators emerging from single-cell genomic studies.

Young
Embryologist
Network

Poster abstracts

Even numbers present at break 1
Odd numbers present at break 2

Poster 1. Functional characterisation of FRIZZLED5 variants associated with coloboma and microphthalmia.

By Marina Adjei City (St George's University of London)

Ocular Coloboma (OC) and microphthalmia are congenital eye malformations. Mutations in the WNT receptor FRIZZLED5 lead to the pathogenesis of OC and microphthalmia in humans. To date, twenty-one mutations in the WNT receptor FZD5 have been reported, but only two have been functionally analysed. My project aims to determine the function of the remaining nineteen variants so we can precisely correlate genotype with phenotype in the affected patients, advancing our understanding of FRIZZLED5 function during eye formation.

Poster 2. Characterising EphB3b in zebrafish spinal cord regeneration

By Patrick Kaluzny (University Of Portsmouth)

Spinal cord injury (SCI) causes severe functional disability and often results in paralysis, yet effective treatments remain limited to rehabilitation. In contrast, zebrafish display a remarkable capacity for regeneration following complete SCI, achieving full recovery of motor and sensory function. Despite evolutionary differences, zebrafish and mammals share many similar cell types, but only zebrafish can regenerate their spinal cords. The mechanisms underlying this difference are not fully understood, although several proteins have been implicated in regulating regeneration. Eph receptors and their contact-dependent ligands, Ephrins, are involved in multiple regenerative processes, including axon guidance, cell migration through repulsion-based signalling, and tissue restructuring. EphB3b, a member of the Eph receptor family, remains largely uncharacterised, particularly in the context of SCI. Previous studies have shown that EphB3b plays roles in liver development and compartmentalisation, as well as synapse formation and axon guidance during nervous system development. Unpublished data from our lab indicate that EphB3b expression is upregulated following spinal cord injury. Furthermore, zebrafish lacking EphB3b recover sensory and motor function faster than control fish. These findings suggest that EphB3b may act as a regulator of spinal cord regeneration and could represent a potential target for understanding mechanisms that limit recovery after SCI.

Poster 3. The role of Eph/Ephrin in inflammation and regeneration following spinal cord injury

By Haijun Li (University of Portsmouth)

This project aims to elucidate the mechanism of neural regeneration after traumatic spinal cord injury (TSCI), which affects over 210,000 people in the UK. The inflammatory process is central to the regenerative response and has been proposed to play both positive and negative roles in tissue repair.

Notably, erythropoietin-producing hepatocellular carcinoma (Eph) receptors and Eph receptor-interacting proteins (Ephrin) ligands form a large family of receptor tyrosine kinases in mammals, which participate in the development, activation, and migration of immune cells. To deepen understanding of the function and regulation of neuroinflammation in spinal cord regeneration, we aim to investigate the role of the Eph/Ephrin signalling pathway in zebrafish. Analysis comprised two neural functional tests, assessing sensation and behaviour, as well as inflammation after TSCI, which compromises various pathways. Interestingly, EphA4 regulates functional recovery after TSCI, and its functions differ between canonical forward signalling and reverse signalling downstream of Ephrin ligands.

Poster 4. The relationship between neural tube closure and neural crest migration in mammalian development

By Achira Karunaratna (Univerity College London)

Neural tube defects are common congenital abnormalities that arise from incomplete neural tube closure. Neurocristopathies are defects that result from abnormal neural crest development. Remarkably almost all mouse mutant models of neurocristopathy develop exencephaly, a severe brain defect. This significant co-occurrence infers a relationship between two spatiotemporally congruent processes in early embryonic development: neural tube closure and neural crest migration. Moreover, pharmacological inhibition of methylation in the neural fold stage mouse embryo causes exencephaly and neural crest migration defects. The current study investigates the methylation-dependent mechanisms of neural crest migration using a cranial neural crest explant culture method. Using established protocols, it is shown that neural crest cells exhibit cytoplasmic protein methylation, distinctly elevated at cell protrusions including filopodia and lamellipodia. Cytoskeletal proteins such as F-actin and beta tubulin required for cell migration are found to be methylated via colocalization with a lysine-specific methyl mark. Inhibition of transmethylation reactions in explant culture show migration is retarded in a dose-dependent manner and is accompanied by a significant loss in cellular methylation. This current study provides evidence for methylation-dependent neural crest migration in the mouse and provides a mechanism that could explain how neural tube defects arise due to methylation cycle insults.

Poster 5. Understanding the mechanism of endoderm patterning

By Juliette Gracia (The Francis Crick Institute)

In vertebrates, the specification of endoderm and mesoderm progenitors is thought to depend on a morphogen gradient formed by Nodal, a ligand of the TGF-□ family, together with Nodal-induced long-range Fgf/Erk signalling. However, read-out of these gradients alone cannot explain the pattern of intermingled mesoderm and endoderm observed in early zebrafish embryos and 2D mouse/human gastruloids. Fgf/Erk inhibits the switch to endoderm fate in zebrafish embryos, and our lab has shown that Erk activity and downstream target phosphorylation are rapidly extinguished as cells approach mitosis and are reactivated in daughter cells with variable kinetics. We hypothesise that mitotic erasure of Erk activity is key for endoderm identity and, together with cell-to-cell differences in Erk reactivation rates, provides a source of heterogeneity that explains the spotty pattern of endoderm progenitors.

My PhD project aims to elucidate the molecular mechanism that couples the cell cycle-associated dynamics of Fgf/Erk signalling with endoderm patterning in zebrafish embryos. I am combining unbiased and candidate-based approaches to identify regulatory factors of the endodermal master regulator sox32 that are sensitive to Erk-dependent phosphorylation, thereby preventing sox32 expression. I am validating an Erk-dependent phosphosite identified in Gata5 and generating a phosphoproteomics dataset of Erk targets in zebrafish embryos.

Poster 6. Investigating the Role of Usp7 in Early Ovary Development

By Maria Pantelidou (Imperial College London)

Ovarian development relies on coordinated germ-somatic interactions that establish the primordial follicle pool, a determinant of female reproductive lifespan. USP7, a deubiquitinase implicated in transcriptional regulation, was reported to interact with FOXL2 in the ovary. Previous work showed that USP7 loss disrupts early follicle formation and granulosa cell differentiation, leading to small, dysfunctional ovaries where gametes fail to survive. Characterising USP7's role in this process is critical for understanding early ovarian development and how disruptions may contribute to reproductive disorders.

To investigate the developmental origin of these defects, we examined embryonic ovaries from an SF1-Cre conditional Usp7-knockout mouse model using immunofluorescence of markers of granulosa cells (FOXL2), Notch signalling (HES1), meiotic progression (SYCP3) and cell proliferation (Ki67).

We found that Usp7 deletion leads to reduced granulosa cell proliferation, indicated by reduced Ki67 expression, and disrupted germ cell meiosis, suggested by altered SYCP3 staining patterns. Mutant ovaries displayed disrupted HES1 localisation, consistent with altered Notch signalling, while granulosa cell specification was not affected.

These findings suggest a role for USP7 in maintaining rather than initiating granulosa cell differentiation and indicate a potential involvement in oocyte meiotic progression. These promising data provide evidence that USP7 contributes to somatic cell function and influences germ cell development, warranting further investigation.

Poster 7. Unveiling Early ALS Biomarkers: Insights From Neuromuscular Junction Dynamics

By Rebecca Bozzi (University of Portsmouth)

Amyotrophic lateral sclerosis (ALS) is the most common form of motor neuron disease consisting of the degeneration of upper and lower motor neurons, leading to muscle atrophy and death by respiratory failure. Despite extensive research, the pathogenic mechanisms of ALS remain poorly defined, due to the heterogeneous clinical presentation, late disease onset and the absence of clearly defined causative factors. Studies on ALS patients suggest that neurodegeneration may start at the neuromuscular junction (NMJ) prior to the onset of clinical symptoms. Rodent and zebrafish models with ALS mutations exhibit defects that appear at early embryonic stages, suggesting that ALS could be a neurodevelopmental condition. The membrane kinase EphA4 receptor is an important regulator of axonogenesis, axon guidance, development and maintenance of NMJ stability. EphA4 has been postulated as an ALS modulator in patients and animal models; however, the mechanism for this remains elusive.

We propose that EphA4 interacts with ALS mutations during embryonic stages, affecting NMJ development and inducing axonal defects seen in ALS. Through imaging the formation of NMJs during early development, behavioural analysis and gene editing techniques, our project aims to understand the relationship between EphA4 and neurodegeneration and find early markers of disease.

Poster 8. NMGN Congenital Anomalies Cluster: Patient-led functional genomics

By Lisa Leinhos University of Oxford

Approximately 1 in 20 babies is born with a severe anatomical malformation. Each year, this corresponds to 8 million affected newborns worldwide, of whom around 300,000 die within the first four weeks of life. Advances in high-throughput sequencing and analysis have accelerated the identification of potentially disease-causing changes in these patients. However, a major challenge remains in determining which of these genetic changes, or variants, are truly causative and in understanding the cellular and molecular mechanisms through which they disrupt normal development. The MRC National Mouse Genetics Network (NMGN) Congenital Anomalies Cluster, in partnership with the MRC Mary Lyon Centre, has been generating and characterising genetically engineered mouse models carrying patient-derived variants. Variants are assessed and prioritised by a Clinical Advisory Board; so far, 16 variants have entered a pipeline for gross morphological and organ-specific phenotypic analyses to support improved genetic diagnosis for patients, and to increase understanding of pathogenic mechanisms. Here, we summarise progress and highlight several examples, including evidence supporting variants in KCTD15 and RARG as novel human disease genes associated with frontonasal dysplasia and keratinising desquamative squamous metaplasia, respectively. We also discuss progress in establishing a high-throughput, low-cost phenotyping pipeline for congenital anomaly models through deconvolution of bulk RNA-sequencing data from mouse embryos. The NMGN Congenital Anomalies Cluster aims to strengthen understanding of genotype–phenotype relationships through close collaboration between developmental biologists, clinical geneticists, healthcare teams, and patient groups. Ultimately, the work seeks to improve the diagnosis, prognosis, and clinical management of families affected by congenital anomalies.

Poster 9. The mitochondrial NAD^+ transporter slc25a51b regulates ventricular morphogenesis and growth dynamics in zebrafish

By Anna de Boer (University of Oxford)

The heart is among the most metabolically active tissues per gram, with cardiomyocytes switching from glycolysis to fatty acid oxidation upon maturation. Nicotinamide adenine dinucleotide (NAD^+) is a vital metabolic coenzyme, with up to 70% residing in the mitochondria. Recently, SLC25A51B was characterised as a mitochondrial NAD^+ transporter that maintains the mitochondrial NAD^+ pool, yet its role in cardiac development and function remains largely unexplored. A comparative analysis of zebrafish strains with varying regenerative capacities identified slc25a51b as a genetic candidate for regulating cardiac regeneration. Given that zebrafish are a powerful vertebrate model renowned for their cardiovascular regenerative capacity and genetic tractability, CRISPR-Cas9 technology was employed to generate cardiomyocyte-specific slc25a51b knockout (cKO) and overexpression (cOE) models.

Strikingly, quantification of AFOG-stained ventricles revealed dramatic ventricular hypertrophy in adult slc25a51b cKO zebrafish. Further, GFP nuclear quantification in cardiomyocytes revealed increased numbers and density, indicative of hyperplasia. Importantly, no significant change in cell-cycle activity was detected, suggesting that this hyperplasia may have an embryonic origin. In contrast, slc25a51b cOE hearts exhibited hypotrophy without altered cell cycle activity. Preliminary imaging of embryonic hearts indicates these opposing phenotypes emerge early in cardiac development, implicating slc25a51b as a novel regulator of ventricular morphogenesis and growth dynamics.

Poster 10. Genetic requirements for GSK3b hyperactivation in embryogenesis

By Delia Capatina (King's College London)

Glycogen synthase kinase 3 (GSK3) is a serine/threonine kinase encoded by two genes: Gsk3a and Gsk3b. Tyrosine phosphorylation in the kinase domain is associated with a hyperactive state, the function of which remains poorly understood. GSK3a knockout mice are viable with no gross phenotype. GSK3b knockouts are not viable, showing liver degeneration and cleft palate. Double knockouts are embryonically lethal, suggesting GSK3 is essential for development, with the two isoforms having distinct functions. Given the lack of in vivo data on the role of GSK3 phosphorylation in embryogenesis, we aim to investigate the role of hyperactive GSK3b in this context. We generated a non-phosphorylatable GSK3b p.Y216F mouse line. Heterozygous animals show no gross phenotype. Homozygous mutants have phenotypes of varying severity, with several organs affected: the brain, the eye, and the cardiovascular system. Previous work knocking out GSK3 in the brain results in significant overgrowth. Our results suggest that there are targets specific to the GSK3b hyperactive state which are important in neurodevelopment. Future bulk RNA sequencing and phosphoproteomic analysis will pinpoint affected signalling pathways. The interaction of GSK3 mutants with downstream targets will be compared. This will help distinguish specific roles of GSK3 isoforms and phosphorylation sites in development.

Poster 11. Exploring genotype-phenotype correlations in Sox10 mutations in zebrafish

By Jennika Bates (University of Bath)

SOX10 is essential for the development of the neural crest and many of its derivatives, including melanocytes, enteric neurons, and glia, as well as some non-crest derived cell types including cells of the otic vesicle and CNS oligodendrocytes. Effects of SOX10 mutation are conserved across vertebrates and include defects in pigment, myelinating glia, and the enteric nervous system. In humans, SOX10 mutations are associated with neurocristopathies (neural crest disorders). Genotype-phenotype correlations are unclear, with the same mutation sometimes associated with variable disease classifications. It is not obvious if this variation is caused by specific mutations themselves, or influence from the genetic background and regulatory elements. CRISPR/Cas9 precise genome editing (PGE) techniques present an opportunity to investigate this discrepancy. So far, Homology-Directed Repair methods have had limited success, but now Prime Editing shows significant promise for the creation of zebrafish point mutations zebrafish.

By utilising a Cas9 nickase/reverse transcriptase fusion protein, only one strand is severed, resulting in lowered rates of unwanted insertions and deletions seen in other PGE methods. Here, we utilise prime editing to create a series of zebrafish sox10 mutant alleles representing disease variants observed in humans. Their characterisation will allow for definition of the sox10 structure-function relationship.

Poster 12. Investigating molecular timers regulating the emergence of radial polarity caps in early C.elegans embryos

By Norin Bhatti (The Francis Crick Institute)

PAR (partitioning defective) proteins form a network that is fundamental for metazoan cell polarity, generating distinct cellular domains through their mutual antagonism. During development, the PAR network is deployed to generate cell polarity in diverse contexts, but how this redeployment is regulated in space and time remains elusive. The early C. elegans embryo is an ideal model to study this as it presents several different polarity states; anterior-posterior polarity first emerges at the 1-cell stage, while the less studied radial polarity is seen from 7-cell stage. Radial polarity specifies an outside-inside axis for the early embryo and in this way is similar to polarity in the early mouse blastocyst. Here, I investigate potential mechanisms that time the emergence of the radial polarity state - the number of cell divisions, the onset of the maternal-to-zygotic transition, and maternal protein degradation.

Poster 13. Endogenous Nodal signalling governs primitive streak fate specification and ratio-controlled endoderm formation in human gastruloids

By Pauline Konsa (The Francis Crick Institute)

Gastrulation is a developmental process during which epiblast cells make coordinated fate decisions, thought to be directed by extraembryonic tissues acting as upstream ‘organisers’. However, Nodal, a central regulator of primitive streak (PS) fates, is initially produced by epiblast before becoming restricted to the anterior PS, suggesting that differentiating cells themselves play an active role in the emergent signalling landscape regulating fate decisions. Dissecting these intrinsic and dynamical mechanisms is complicated in the embryo. Gastruloids, a stem cell-based embryo model lacking extraembryonic tissues while still establishing organised fate patterning, offer an alternative for studying these mechanisms. We show that human gastruloids establish an endogenous Nodal signalling landscape, with the switch between anterior and posterior PS being controlled by early exposure, whilst the duration determines the specific aPS fates. Furthermore, the proportion of definitive endoderm cells scales with aggregate size, implying that the number of endoderm cells may be maintained through downregulation of Nodal, potentially via a negative feedback loop. Together, our findings demonstrate how emergent Nodal signalling is sufficient to drive coordinated PS fate decisions, even in the absence of extraembryonic tissues. We further show that human gastruloids establish a ratio-controlled endoderm via endogenous regulation of the Nodal signalling window.

Poster 14. The form and function of stochastic asymmetry in embryonic neural development

By Paula Richter (University College London)

Stochastic divergences from symmetry between left-right (LR) embryonic structures provide an intrinsic metric to assess morphogenetic variability in bilaterians. Neural tube (NT) closure is a paradigm of superficially-symmetrical morphogenesis, with the left/right halves of the neural plate elevating and bending quasi-independently to meet at the embryonic midline. We compared left/right NT symmetry to identify the limits of morphogenetic 'control' within individual mouse embryos. Molecularly, apical F-actin is symmetrical, such that right-side intensity correlates with the left-side of the same embryo. Similarly, at the cell level, left/right mitotic index also correlate. In contrast, tissue-level bending at distinct dorsolateral hinge points (DLHPs) is highly variable within embryos such that there is no correlation between embryos. Bending asymmetry is stochastic—neither side is predictably greater—and varies between individuals. We developed a 3D surface-mapping approach to map asymmetries across the open spinal NT, identifying regional 'hotspots' of asymmetry corresponding to shape transitions from flat to elevated, and elevated to medially bent at DLHPs. Short-term actomyosin inhibition significantly increases asymmetry in elevation, but not in DLHP bending. Recovery from inhibition rapidly restores physiological (a)symmetry. Thus, tissue-level NT asymmetry arises at shape transitions and is regionally restricted by actomyosin contractility. We are now exploring whether stochastic variability in morphogenesis underlies partially-penetrant malformations including spina bifida.

Poster 15. Visualising the Cytoskeleton in Developing Tendon Cells: The Effects of Embryo Immobilisation on Cell Structure and Organisation

By Claire Kavanagh (Trinity College Dublin)

Tendon development is highly dependent on mechanical stimulation generated by embryonic movement. This project investigated how mechanical immobilisation affects cytoskeletal organisation in developing chick tendons, with a particular focus on F-actin structure and cellular organisation. Embryos were subjected to either rigid or flaccid paralysis, alongside untreated controls, to assess how differing mechanical environments influence tendon cell morphology during development. An optimised immunofluorescence protocol was developed using phalloidin staining to visualise F-actin, alongside DAPI nuclear staining. Confocal microscopy and image analysis in ImageJ were used to quantify phalloidin intensity and nuclear spacing across treatment groups. Both immobilisation conditions altered cytoskeletal organisation compared to controls, with rigid immobilisation producing the most pronounced changes in F-actin structure and cellular arrangement. These findings support the role of embryonic movement in regulating tendon maturation and cytoskeletal organisation during development. Understanding how mechanical forces shape tendon formation may provide insight into developmental disorders and future regenerative medicine approaches targeting musculoskeletal tissues.

Poster 16. Dynamics of cardiovascular and endoderm morphogenesis using gastruloids

By Ornella Clara (Institute of Developmental Biology Marseille)

Gastruloids provide a complementary approach to in vivo studies by enabling highly perturbative experimental manipulation of developmental processes. They allow precise control of chemical signalling, physical constraints, and extracellular matrix interactions, offering a powerful model to investigate early organogenesis. Our laboratory has developed a protocol that biases gastruloid differentiation toward anterior endodermal and cardiovascular mesodermal lineages. These gastruloids recapitulate the emergence of tissue-like structures, including gut-like lumens, vascular networks, and cardiomyocyte-like cells, revealing a finely coordinated spatiotemporal organization during early development. In this context, my research focuses on how cells coordinate fate decisions, collective movements, and morphological changes during cardiovascular and endodermal morphogenesis. We investigate both global tissue organization and local single-cell dynamics through quantitative image analysis and single-cell RNA sequencing. Ultimately, we aim to connect the biophysical principles of organ formation with the signalling mechanisms governing cell fate decisions during cardiovascular and endodermal development.

Poster 17. Optimising Multiplexed RNAscope for Co-expression Profiling of Pigment Cell Fate Determinants via Spectral Unmixing

By Oleksandr Meshchanin University of Bath

How do individual neural crest progenitors commit to distinct pigment cell fates, and what transcriptional states drive these lineage decisions during zebrafish development? While scRNA-seq approaches are used to profile cell fate determinants, they underestimate fate potential by missing low-abundance transcripts such as tfec and ltk, and require tissue dissociation, thereby eliminating the spatial context needed to map co-expression patterns within intact embryonic tissue. Single-molecule FISH addresses the limitation of lost spatial context by detecting transcripts without dissociating the embryo, but it remains challenged by low-abundance targets due to limited signal amplification. Unlike traditional smFISH, RNAscope uses paired probes that initiate a branched amplification cascade only upon simultaneous co-hybridisation to adjacent target sequences, thereby suppressing background and enabling sensitive detection of low-abundance mRNA targets. Yet sequential imaging cannot resolve a dense fluorochrome panel in which closely spaced dyes have broad emission spectra with overlapping primary and secondary peaks. Linear spectral unmixing addresses this by decomposing mixed per-pixel emission spectra against a reference spectral image dataset, enabling reliable simultaneous spatial detection of pigment cell fate determinants at single-molecule resolution, providing the methodological foundation for directly interrogating transcript co-expression dynamics during neural crest lineage commitment.

Poster 18. Developing a versatile tool to investigate the role of GATA6 in human naïve epiblast plasticity

By Emily Davis (Living Systems Institute, Exeter University)

Cell fate decisions are critical for the successful development of the embryo. How these decisions are made in the early embryo is still relatively unknown, with limited studies on human embryos due to ethical concerns. Human naïve pluripotent stem cells (hnPSCs), representing the epiblast, can give rise to all three founding lineages of the blastocyst in vitro, unlike mouse. They are a highly tractable, high throughput platform by which to study early cell fate transitions. GATA6 is expressed in the inner cell mass, and later in the hypoblast and trophectoderm. In in vitro extraembryonic differentiation protocols, GATA6 is among the first genes to be upregulated, and is hypothesised to be one of the driving transcription factors for hypoblast fate. Here, we present a GATA6:HaloTag cell line and a doxycycline-inducible GATA6 cell line in hnPSCs. HaloTag is a self-labelling tag which can elicit different effects upon the addition of specific ligands, including fluorescence and degradation. GATA6:HaloTag fluorescence intensity can discriminate between trophectoderm and hypoblast cells by both microscopy and flow cytometry. These cell lines provide a useful platform for investigating lineage plasticity in hnPSCs and cell fate determination in human embryonic development.

Poster 19. Spatiotemporal neurogenesis and gliogenesis programs in the human fetal medulla oblongata

By Kai Boon Tan (University College London)

The medulla oblongata (MO) is a vital relay center for myriads of ascending and descending neural pathways between the spinal cord and higher brain regions essential for autonomic and motor functions. Previous studies reported that distinct MO progenitor domains along the dorsoventral axis, demarcated by different transcriptional factors, give rise to different neuronal cell types. However, the spatial and temporal programs governing cell type diversification during MO development remain poorly understood. We report, for the first time, that the expansion of the human lower rhombic lip with a subventricular zone-like progenitor niche that biases towards glial lineage in the early first trimester after the first wave of neuronal production. Moreover, the proliferation dynamics in the medullary neuroepithelium and parenchyma exhibited a high medially and low laterally spatial bias up to the late second trimester. Finally, we discovered a persistent SOX2+ basal progenitor pool that exhibits diverse division behaviors in the medullary parenchyma and persists throughout fetal development. Together, human fetal MO progenitors diversify into neuronal and glial cell types to support early circuitry assembly during development. Temporally and spatially expanded neurogenesis and gliogenesis dynamics sustain the functional assembly of the neural pathways in the developing central nervous system.

Poster 20. Reinterpreting the Role of Microtubules in Symmetry Breaking in the C. elegans Embryo
By Lucy Helyar (The Francis Crick Institute)

The C. elegans embryo relies on a conserved network of antagonistic PAR polarity proteins to establish asymmetric domains at the anterior and posterior cell cortex. The kinase PKC-3 is known to phosphorylate posterior PAR proteins, including PAR-2, to exclude them from the membrane. It has been proposed that the formation of an initial posterior domain involves local, transient protection of PAR-2 from PKC-3 by microtubules. This was based on key results: (1) PAR-2 binds microtubules, (2) microtubule binding inhibits PKC-3-dependent phosphorylation, and (3) mutations that affect microtubule binding show polarisation defects. However, direct disruption of microtubules had little effect on polarity, raising questions about this model. We found that the microtubule mutations overlap predicted membrane association domains as well as a binding site for PP1 phosphatase known to counter PKC-3 phosphorylation, suggesting that defects in polarisation could instead arise from defects in membrane binding and/or phosphatase efficiency. We have shown that one of these mutants shows intrinsic defects in membrane association, even when PKC-3 is absent, and mutations introduced to destabilise membrane binding show near identical phenotypes. Although we cannot formally rule out microtubule involvement, our findings suggest that the current model of microtubule-dependent symmetry breaking should be revisited.

Poster 21. Investigating Chromatin Remodelling Events During Spinal Cord Development
By Nick New (LMS)

Despite sharing the same genetic information, distinct cell types arise during development. Cell identity is determined by the coordinated regulation between chromatin accessibility and transcription. However, how chromatin landscapes are established and contribute to cell identity control remains a key question in developmental biology, with major relevance to regenerative medicine. We previously showed that CDX transcription factors play a central role in spinal cord formation by regulating chromatin accessibility. We hypothesise that CDX factors remodel the chromatin landscape by interacting with chromatin remodellers at key genomic sites. To identify potential interactors, we are employing both in-silico and ChIP-MS based approaches, combined with genome engineering in mouse embryonic stem cells. In parallel, we have established pharmacological perturbation assays in an in vitro model of spinal cord development to functionally test candidate factors. Our preliminary data support the view that CDX2 can interact with the SWI/SNF remodelling complex. These findings begin to reveal how transiently acting transcription factors coordinate chromatin remodelling events that underpin correct cell fate specification during development.

Poster 22. Patterna: A Shape-Agnostic Python-Based Suite for Robust Quantification of Micropattern Stem Cell Cultures
By Grezcia Payan (Cambridge Stem Cell Institute)

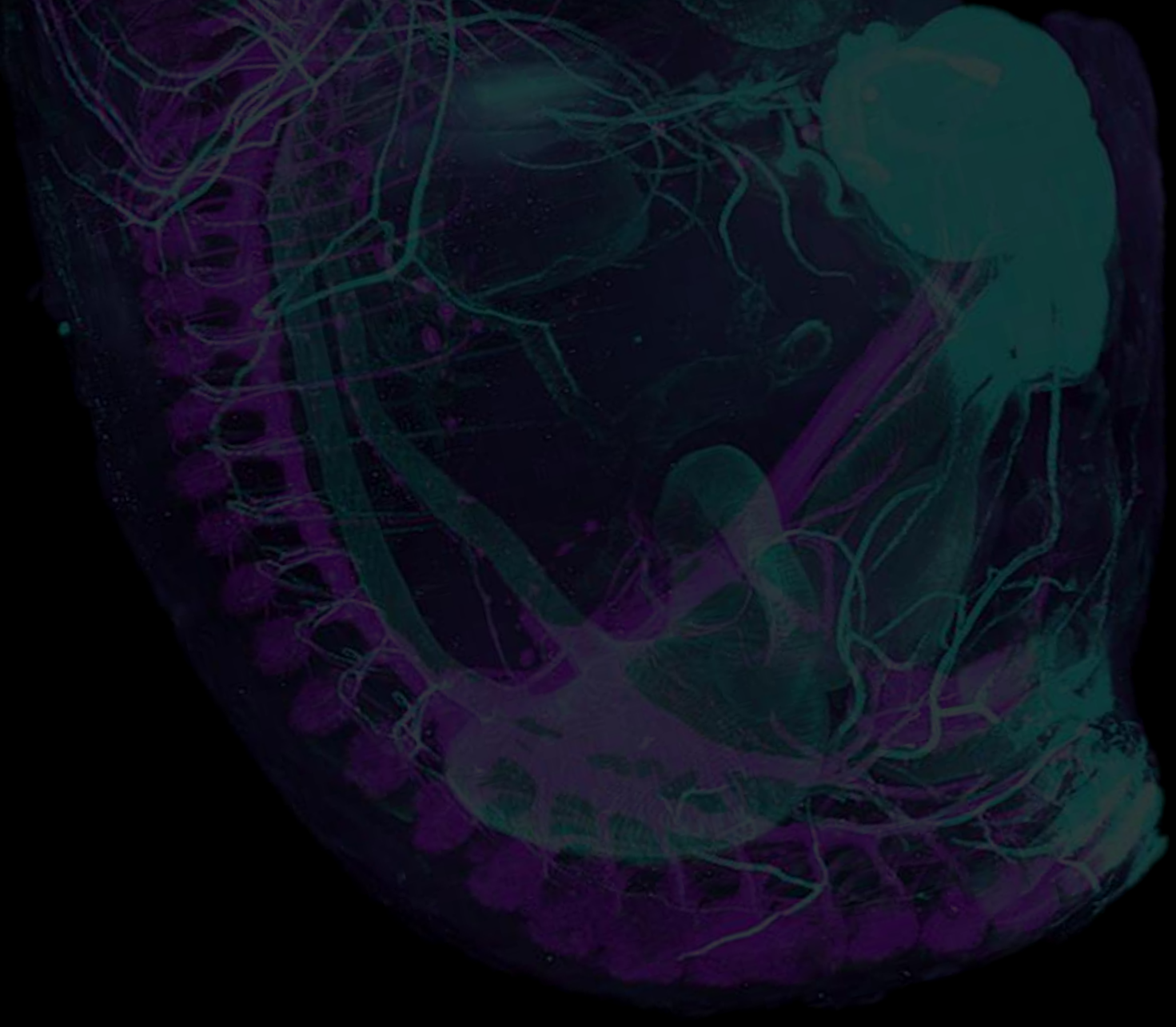
Micropatterned pluripotent stem cell cultures are widely used to study early developmental patterning, but their analysis is often limited by manual line profiles, visual interpretation, or methods optimized for simple circular geometries. This makes it difficult to compare micropatterns of different shapes or capture spatial heterogeneity across entire colonies. Here, we present Patterna, a shape-agnostic image analysis pipeline designed to quantify immunofluorescence patterns in micropatterned stem cell colonies. Patterna converts each colony into a normalized radial coordinate system using a “spiderweb” sampling strategy. Instead of measuring fluorescence along selected axes, the pipeline samples intensities from the colony centroid to the true boundary across many angular directions, defining radial position consistently from center to edge across circular, oval, triangular, or irregular geometries. From this representation, Patterna generates radial profiles, angle–radius maps, polar visualizations, orthogonal profiles, and quantitative pattern fingerprints. These outputs summarize marker organization through metrics including radial enrichment, peak position, radial spread, edge-to-center ratios, and anisotropy between orthogonal axes. Patterna also incorporates StarDist-based single-cell segmentation, enabling individual nuclei to be mapped onto the same normalized coordinate system.Together, Patterna replaces sparse manual sampling with whole-pattern, shape-normalized quantification, reducing user bias and enabling reproducible comparisons across geometries and conditions.

Poster 23. Uncovering Microtubule Function During Nuclear Deformation Through Confined Cell Migration In Vivo
By Minjia Pan (University of Cambridge)

Cell migration through confined environments requires cells to deform their nucleus—the largest and stiffest organelle—to pass narrow spaces without rupturing the nuclear envelope, which could otherwise lead to DNA damage and cell death. However, the cytoskeletal mechanisms regulating nuclear deformation in vivo remain unclear. Here, I demonstrate that dynamic microtubules are crucial for maintaining nuclear shape stability during confined migration. Using advanced live imaging of zebrafish embryos, I show that partial microtubule depolymerization with low-dose nocodazole increases nuclear deformation and accelerates the translocation of trunk neural crest (TNC) cells through narrow interstitial spaces, whereas cranial neural crest (CNC) cells migrating in non-confined environments are unaffected. Further analysis revealed that global actomyosin contractility is not substantially altered in interphase TNCs following microtubule depolymerization, and pharmacological inhibition of ROCK, despite reducing mitotic rounding, fails to rescue the enhanced nuclear deformation phenotype. These findings establish a context-dependent requirement for microtubules in maintaining nuclear integrity under physical confinement and highlight the potential for coordinated microtubule–actomyosin crosstalk during collective confined migration.

Poster 24. Mechanochemical Feedback Couples Cranial Neural Crest Migration and Neural Tube Morphogenesis via Mechanosensitive Fibronectin Remodelling
By Kai Weissenbruch (University College London)

Cranial neurulation and collective migration of cranial neural crest cells are two major processes during embryonic morphogenesis. Although often considered distinct and functionally independent, spatiotemporal overlap exists between both to a various degree across many species. Using Xenopus laevis as a model, we show that collective cranial neural crest migration is a prerequisite for cranial neural tube closure. Neural crest delamination and invasion into extracellular matrix, secreted by the cranial mesoderm, leads to fibronectin remodelling along the neural crest-neural plate border. The emerging fibronectin sheet not only segregates neural crest and neural plate but is also necessary for neural tube morphogenesis. Mechanistically, neural crest cells express the membrane-bound matrix metalloproteinase MMP14, which is necessary and sufficient for this fibronectin remodelling. Thus, the mechanism depends on spatially localised MMP14 activity, facilitated by collective neural crest cell movement, rather than diffusion. Finally, our data reveal that the neural crest-specific expression of MMP14 is mechanosensitive and amplified by compressive forces. Strikingly, such forces arise in vivo due to neurulation itself, as the bending motion of the neural folds spatially confines neural crest cells between the underlying mesoderm and the superficial ectoderm.



Poster 25. PAX3 Orchestrates Migration, Proliferation, Axial Identity and Fate Decisions in Neural Crest Cells During Mouse Development
By Sarah Chebouti (Mondor Institute of Biomedical Research)

Neural crest cells (NCCs) are multipotent embryonic progenitors that migrate from the dorsal neural tube to generate diverse derivatives, including neurons, glia, melanocytes, and mesenchymal tissues. Although PAX3 is a key neural plate border transcription factor required for NCC formation, its role in coordinating NCC lineage progression and fate decisions in vivo remains poorly understood. Here, we combine Pax3 loss-of-function in the mouse, lineage tracing, and single-cell RNA sequencing of Pax3-expressing derivatives to define PAX3-dependent developmental programs across embryonic lineages, with a focus on NCCs. Pax3 deletion results in disrupted NCC migration, defective dorsal root ganglia formation, and impaired peripheral nervous system formation. Single-cell transcriptomic analysis at E9.5 and E10.5 reveals loss of undifferentiated NCCs and a concomitant expansion of NCC-derived mesenchymal cells in Pax3 mutant embryos. Moreover, Pax3-deficient NCCs exhibit impaired migration, defective axial identity, and reduced proliferation. Importantly, loss of PAX3 leads to decreased neuronal differentiation and a shift toward mesenchymal fates. Together, our findings position PAX3 not only as an inducer of neural crest identity but as a safeguard of lineage progression that coordinates migratory capacity and fate determination. These results redefine PAX3 as a key dynamic regulator of neural crest cell states during embryonic development.

Poster 26. Gene-Regulatory Mechanisms in the Evolution of Human Brain Size
By Alex Donovan (MRC Laboratory of Molecular Biology)

In early corticogenesis symmetrically dividing neuroepithelial cells must undergo a cell state transition to become asymmetrically dividing neurogenic radial glia. This transition is characterised by a phasic cell shape change; from an epithelial to partially mesenchymal state, via an intermediate transient state known as the transitioning neuroepithelium. Concomitantly, the identity of the cell undergoes a switch in identity to enable acquisition of neurogenic capacity. The timing of this cell state transition is very precise, with subtle evolutionary shifts leading to dramatic alterations in brain size.
Mapping gene expression and chromatin dynamics at fine resolution during this transition in human cerebral organoids, we find that the timing of cell morphological change and aspects of radial glial identity acquisition are separable during this process. We then map dynamics in 3D genome architecture across transition to identify candidate transcriptional regulators. Employing newly established organoid NanoDam tools we then map the binding targets of one of these factors, ZEB2, genome-wide, finding that it regulates genes related to cell shape change but not radial glial functional identity. Together, these findings define key aspects of the gene-regulatory program underlying neuroepithelial to radial glial transition and suggest epigenetic mechanisms through which brain size evolution may be mediated.

Poster 27. Identification of Novel Genes Involved in Fovea Formation
By Jack Nicholls (City St George's, University of London)

The fovea is a specialised region of the retina responsible for visual acuity. It possesses several unique features, including a high density of elongated photoreceptors. Little is known about the genetic network underlying foveal development, and many individuals with foveal hypoplasia, a condition characterised by an underdeveloped fovea, lack a confirmed genetic diagnosis. The zebrafish retina has a fovea-like region known as the high acuity area. Gaining a better understanding of how the high acuity area develops may shed light on the mechanisms underlying human foveal formation and help identify novel genetic variants associated with foveal hypoplasia. To investigate high acuity area development, a zebrafish strain carrying a mutation in foxd1 has been generated. These mutants lack a high acuity area and are visually impaired. Transcriptomic analysis of foxd1 mutants has identified a set of genes potentially involved in the development of the high acuity area. To follow up on this analysis, CRISPR-Cas9 is being used to knock out these candidate genes. Once mutant strains have been generated, the structure of their retinas will be examined, and visual performance tests conducted to assess how each gene contributes to the development of the high acuity area.

Poster 28. Bioengineering Stem Cell Organoids to Model Human Neural Crest Development
By Carmen Moreno Gonzalez (King's College London)

The neural crest (NC) is a transient stem cell population which migrates throughout the developing embryo to contribute to diverse tissues throughout the body. Perturbations in neural crest development can lead to severe congenital anomalies and cancers, with over 700 neurocristopathies reported. In humans, early NC development remains poorly understood due to the inaccessibility of tissue samples and the scarcity of appropriate in vitro models. Currently, a limited number of NC organoid protocols are available, but these mainly focus on cranial NC and lack relevant tissue architecture. Here, we describe a novel bioengineered pipeline to derive hPSC-derived neuroepithelial organoids, “neurocrestoids” capable of producing NCCs with HOX gene expression along the anterior-posterior axis and featuring a physiologically relevant tissue architecture. We show that neurocrestoids recapitulate dynamic induction, delamination, and migration of human NCCs, and can be directly compared to murine NC explants for cross-species validation. Moreover, we have integrated our neurocrestoid with a customised micropatterned substrate suitable for live visualisation and guided separation of SOX10-positive migratory human NCCs.

Our “neurocrestoids on-a-chip” are reproducible across multiple hPSC lines and should be scalable for future diagnostic and therapeutic applications, significantly improving our ability to study human NC pathologies.

Poster 29. Decoding and Rewriting Fetal Hematopoiesis: Multi-Omics–Guided In Utero Genome Editing for Congenital Blood Disorders
By Fatima Al-Shemary (King's College London)

Inherited hematologic disorders can be reframed as disruptions of gene regulatory networks governing fetal hematopoiesis, positioning early development as a critical window for therapeutic intervention. Recent advances in multi-omics profiling—including whole genome sequencing, single-cell transcriptomics, and chromatin accessibility mapping—are enabling high-resolution reconstruction of fetal hematopoietic stem and progenitor cell (HSPC) states and the regulatory elements perturbed by pathogenic variants. These approaches are particularly powerful for resolving non-coding and structural variation, including enhancer dysregulation and higher-order chromatin interactions. We propose that integrating these datasets can directly inform in utero genome editing strategies. Precision editing platforms, including CRISPR-Cas variants, base editing, and prime editing, enable targeted correction of disease-causing alleles or modulation of key regulatory loci, such as enhancers controlling globin switching. Within the uniquely permissive fetal environment—characterised by immune tolerance and active stem cell expansion—these interventions may achieve durable, lineage-specific correction. However, translating this paradigm requires overcoming key barriers, including efficient delivery to fetal HSPCs, control of mosaicism, and mitigation of off-target and epigenomic effects. Bridging multi-omics insights with functional validation in developmental systems will be essential to establish safe, mechanism-driven in utero therapies for genetic blood disorders.

Poster 30. CDX Instruct Neural Crest Regionalization Programmes
By Irene Amblard (Imperial College London)

During development, neural crest cells (NCCs) generate distinct subtypes along the head-to-tail axis, but how these subtypes are positioned and specified remains an open question. Using a strategy to track early regionalisation events in mouse embryos, combined with spatial transcriptomics and quantitative stem cell models, we find that dynamic changes in CDX transcription factors explain NCC subtype identity at discrete positions in the body. The data demonstrate that different CDX factors preconfigure NCCs to adopt discrete HOX codes, explaining how distinct subtypes emerge at the correct position in the body. Together, these findings define a CDX-driven mechanism that couples early epiblast regionalisation to NCC subtype positioning, and support a hybrid origin for vagal NCCs at the head-to-trunk transition.

Poster 31. Cell Proliferation Regulates Pancreatic-Specific Chromatin Remodeling in Ventral Foregut
By Alberto Dinarello (Novo Nordisk Foundation Center for Stem Cell Medicine (reNEW), University of Copenhagen)

The ventral foregut is a proliferative region of the embryonic gut tube containing multipotent precursors of pancreas and liver. In vitro, ventral foregut stem cells (VFG) can be generated from pluripotent stem cells and maintained in culture for several passages. VFG cultures proliferate and self-renew, gradually shortening cell cycle over passages. Although the morphology and the transcriptome of these cells do not change during expansion, we observed a marked alteration of chromatin landscape, with later cultures (more than passage 6) priming pancreatic- and hepatic-specific enhancers for later differentiation and decommissioning those associated with alternative lineage fate. Consistent with these changes, late VFG cultures differentiate more efficiently and produce better pancreatic endoderm than earlier passage cells (3 or fewer passages). The priming of enhancers regulating pancreatic differentiation correlates with an increased binding of FOXA1 and GATA6. In parallel, histone modifications associated with enhancer commissioning such as H3K27ac and H3K4me1 are progressively more abundant in the same regions. This suggests that time and/or proliferation is required to prepare enhancers for later activation through the gradual erasure of repressive chromatin. Taken together, our findings demonstrate that proliferation of intermediates in differentiation is not only about increasing cell number but is required to correctly configure chromatin at enhancers, enabling efficient and effective differentiation.

Poster 32. Heterochrony of Axis Segmentation Underlies Extreme Morphogenesis in the Japanese Eel
By Ali Seleit (University of Freiburg, Faculty of Biology (CIBSS))

Heterochrony is a major mode of vertebrate body plan evolution, yet its molecular and cellular basis remains poorly characterized. Here, we show that axis segmentation in Anguilla japonica—a species that forms 120 vertebrae—is driven by the temporal extension of somitogenesis. This is achieved through the prolonged maintenance of axial progenitors in the tail beyond the hatching stage, coupled to an extreme segment scaling regime that operates under the constraint of minimal axis growth. We identify delayed Hox13 activation and sustained Oct4 expression as molecular signatures of the prolonged segmentation program. Furthermore, we describe two spatially distinct axial progenitor pools that expand stemness in the tail. These findings reveal how the modulation of stemness in time and space drives extreme morphological evolution in vertebrates.

Poster 33. Post-Translational Modification of Proteins Shapes Cortical Development
By Janina Koch (Charité – Universitätsmedizin Berlin)

The development of the mammalian neocortex from a pool of neural stem cells requires correct execution of many cellular processes, including cell division, fate acquisition, differentiation, migration, and establishment of complex cellular morphologies. These rapid transitions undertaken by developing cells are enabled by precise regulation of the proteome, to dynamically control protein function and fate within the cell. One such regulatory mechanism occurs via post-translational modification of proteins, to fine-tune their activity, stability, and sub-cellular localisation. In this study we explore the role of a small ubiquitin-like modifier, UFM1, which is conjugated to its target proteins in the UFMylation pathway. Dysregulation of UFMylation leads to microcephaly and epileptic encephalopathy, suggesting its importance during cortical development. To understand how protein UFMylation shapes cortical development, we are investigating the effects of Ufm1 loss in the murine cortex, using conditional knockout mice and in vivo labelling of developing neurons during embryogenesis. We find that Ufm1 cKO cortices contain fewer spinally projecting neurons, as well as disrupted neuronal morphology and polarity. To uncover the biochemical processes underlying this phenotype, we are performing proteomics to identify putative targets of UFMylation and to map the downstream pathways through which these targets may contribute to the observed phenotype.

Poster 34. Functional States of Sperm-Derived Centrioles in Androgenetic Sheep Embryos
By Aurora Scudieri (Università degli Studi di Teramo)

Centrioles are essential regulators of spindle organization and chromosome segregation during early embryonic development. In most mammals, the spermatozoon provides the functional centriole to the zygote, while the oocyte centriole is inactivated. Dispermic fertilization introduces two sperm-derived centrioles, frequently resulting in abnormal spindle assembly and defective cleavage. Interestingly, androgenetic embryos generated by fertilizing enucleated oocytes with two spermatozoa can still develop to the blastocyst stage, suggesting the existence of regulatory mechanisms controlling centriole activity. This study investigated sperm-derived centriole behaviour in ovine androgenetic embryos. Embryos were produced by injecting two spermatozoa into enucleated metaphase II oocytes followed by in vitro activation. IVF- and ICSI-derived embryos were used as controls. Centriole activity was assessed through α-tubulin immunofluorescence and confocal microscopy analysis of microtubule aster formation. Analysis performed 6–7 hours after activation revealed heterogeneous patterns of aster nucleation: two asters were detected in 24% of embryos, one aster in 43%, and no asters in 33%. Notably, the proportion of embryos displaying a single aster closely matched the cleavage rate (46%) observed in androgenetic embryos. These preliminary findings suggest that selective repression of one sperm-derived centriole may represent a prerequisite for developmental progression.

Poster 35. Enhanced Full-Term Developmental Competence of Sheep Embryos Produced In Vitro by TGF-β3
By Marika Domenicone (Università degli Studi di Teramo)

In vitro embryo culture is a critical component of assisted reproductive technologies, enabling the zygote to develop into blastocyst for subsequent uterine transfer. In vitro-produced embryos generally show lower developmental competence than in vivo-derived embryos. In sheep, blastocyst formation rates remain limited and implantation efficiency is reduced. Impaired activation of essential signalling pathways during in vitro culture is considered a major cause of compromised embryo quality. In this study, the canonical TGF-β/SMAD pathway was activated at the morula stage through TGF-β3 supplementation. Treated embryos showed an approximately twofold increase in blastocyst development compared with controls. Embryo quality was also enhanced, as indicated by increased numbers of CDX2-positive cells, suggesting improved trophoblast expansion. RNA-seq analysis revealed upregulation of genes involved in cell adhesion in TGF-β3-treated blastocysts, supporting improved implantation competence, further confirmed by in vitro outgrowth assays. To evaluate in vivo developmental potential, embryos were transferred into recipient ewes. From 160 fertilized oocytes, 16 blastocysts developed in the TGF-β3 group compared with 6 in controls. Pregnancy was achieved only with TGF-β3-treated embryos, and ultrasound confirmed normal fetal development at four months of gestation. These preliminary findings support TGF-β3 as a promising factor for improving sheep in vitro embryo production.

Poster 36. Differential Regulation of β-Catenin and Plakoglobin During the Naïve-to-Primed Pluripotency Transition
By Clara Andiazabal (University of Cambridge)

Naïve-to-primed pluripotency transitions mark a critical window in early embryonic development, during which cells of the epiblast acquire lineage competence. While β-catenin's role in regulating pluripotency and Wnt signalling is well studied, its homologue plakoglobin remains largely unexplored. We previously identified plakoglobin as being uniquely expressed in the pre-implantation epiblast, vanishing upon implantation, suggesting a stage-specific function. Here, we elucidate plakoglobin's dynamics during in vitro differentiation of mouse ESCs to EpiLCs, using wild-type and plakoglobin-overexpressing lines. Immunofluorescence analysis reveals a sharp downregulation of plakoglobin during the naïve-to-primed transition, contrasting with stable β-catenin levels. Despite this dramatic shift at the protein level, RT-qPCR shows a modest reduction in plakoglobin transcripts, pointing to post-transcriptional regulation. Given plakoglobin's conserved N-terminal destruction box, we hypothesise that it undergoes proteasome-mediated degradation akin to β-catenin. To examine whether this is a conserved feature across species, our work expands to early human development using human pluripotent stem cells (hPSCs). We have currently generated a plakoglobin overexpression hPSC line and identified conserved phenotypes between species. Our findings open up questions about plakoglobin's potential to modulate Wnt/β-catenin signalling. Elucidating its role promises to deepen our understanding of how pluripotency is fine-tuned at the molecular level during early development.

Poster 37. Can In-House Media Suite Support Consistent Embryo Development?
By Shilpa Doultani (National Dairy Development Board)

The application of Ovum Pick-Up and In Vitro Embryo Production (OPU-IVEP) technologies has seen global expansion as a means to accelerate genetic progress and reproductive efficiency in bovine herds. Despite their benefits, adoption in many developing dairy regions remains limited, primarily due to the high costs associated with commercially available media. To address this, the present study assessed the performance of a cost-effective, in-house developed media suite, comprising IVM, IVF, IVC, and Wash components, for bovine IVEP. The media suite was tested over four experimental rounds. In Rounds 1–3, cumulus-oocyte complexes (COCs) retrieved via ultrasound-guided ovum pick-up (OPU) were subjected to in vitro maturation (IVM) for 22 hours at 38.5°C in 5% CO_2 and high humidity. In vitro fertilization (IVF) was performed using frozen-thawed semen at a final concentration of 2 million sperm/mL for 18 hours. Presumptive zygotes were cultured in IVC medium for 7 days post-IVF at 38.5°C in a tri-gas environment (5% CO_2, 5% O_2, 90% N_2) without media change. The IVC media was supplemented with bovine serum albumin (BSA) in these rounds. A total of 1,760 oocytes were subjected to IVEP, resulting in 340 embryos, with a mean blastocyst rate of 19.3%.In Round 4, the same protocol was followed with the addition of 10% fetal bovine serum (FBS) in the IVC media. A total of 257 oocytes were subjected to IVEP, yielding 79 embryos with a mean blastocyst rate of 30.7%, representing a statistically significant improvement in embryo development. Statistical analysis was performed in Python using a Chi-square test, confirming this difference ($p = 0.000036$). These findings support that the addition of FBS augments the blastocyst rate, making it a potential in-house media for bovine embryo production.

Poster 38. Otic Vesicle Patterning in Human iPSC-Derived Inner Ear Organoids
By Jingyan Yang (King's College London)

The vertebrate inner ear is a complex three-dimensional organ responsible for hearing and balance. Within the inner ear, sensory hair cells detect sound and movement, and this information is transmitted to the brain by cochlear-vestibular neurons. Other cell types, such as supporting cells, are essential for maintaining ionic balance, structural organisation, and overall ear function. Genetic mutations or environmental damage can cause irreversible loss or malfunction of any of these cells, leading to hearing impairment or balance disorders. Understanding how these cell types arise during development is essential for regenerative and disease-modelling strategies.
During development, most inner ear cell types derive from a simple epithelium, the otic placode, which invaginates to form the otic vesicle (OV). Under the influence of signals from surrounding tissues, the OV becomes regionalised into distinct domains, each giving rise to specific components of the mature inner ear. Despite invaluable studies in animal models, many fundamental questions remain unanswered, and our understanding of human-specific developmental mechanisms remains limited.

While human iPSC-derived in vitro models offer an opportunity to study the human inner ear, they poorly recapitulate normal development. Currently, there is no approach to generate both cochlear and vestibular structures simultaneously.
Using human iPSC-derived inner ear organoids, we have generated a Pax2-reporter line to monitor OV development, characterised OV formation over time and assessed OV patterning using 3D imaging and gene expression analysis. We find that although OV-like structures reliably form in organoids, they do so asynchronously, and key patterning markers exhibit disorganised and non-regionalised expression. Highlighting a frequently neglected developmental window, this study exposes a limitation in current inner ear organoid models and lays the foundation for improving in vitro reconstruction of the human inner ear.

Poster 39. Autologous Blood-Derived Novel Rhythmic Thermogenic Melanogenic (RHYTHM) Adipocytic Niches with Pro-Neuro-Hematovasculogenic Competence

By Rhythm Arora (Post Graduate Institute of Medical Education and Research (PGIMER), Chandigarh, India)

Postmitotic cells such as neurons require healthy mitochondrial functioning, as active RNA-transport defects can contribute to integrated stress response (ISR), translational insults resulting in neuroinflammation and neurodegeneration. Heme-regulated inhibitor (HRI), a unique eIF2α kinase and heme-sensor, is central to regulating stress-erythropoiesis and hippocampal memory. Based on these insights and our preliminary findings, we investigated whether heme-mediated stress in adult blood cells (ABCs) could modulate HRI-dependent ISR and erythropoiesis-linked cellular homeostasis through de novo reprogramming of ABCs into bipotential neural crest-like progenitors with neuro-vasculo-immunogenic competence. RNA transcription of beta hemoglobin at chromosome 11 is driven by chromatin looping and activation of the beta locus control region (LCR). Importantly, metabolic insulin gene loci and the largest olfactory gene cluster flank the beta-LCR. While several neuro-immune metabolic diseases co-evolve, the co-expression of metabolic stress-induced neuro-immune pathways involving beta-LCR-driven chromatin looping linked neuronal gene regulation remains completely unexplored. We demonstrate establishment of pigmented MITF+MSX2+Perilipin+APOM+ thermogenic multilocular adipocytic niches from autologous CD45+ blood cells under controlled RBC-contributed heme stress. These precursors drive neurogenesis and hematopoiesis; controlled RBC escalation generates higher-order neurovascular tissues exhibiting epitranscriptomic memory-driven neurovascular coupling-mediated synaptic deregulation in patient-derived cultures. This platform provides a novel precision-medicine model to investigate neuro-immune development and brain activity-related disorders.

Poster 40. The Role of Folate Metabolism in Embryonic Development and Congenital Defects

By Swang Liang (UCL Great Ormond Street Institute of Child Health)

Congenital anomalies affect approximately 1 in every 20 births worldwide, represent the leading cause of infant mortality and frequently result in severe long-term disability. These conditions, including neural tube defects, heart defects and orofacial clefts, often result from interplay between genetic and environmental factors. However, precise mechanisms underlying most congenital disorders remain poorly understood. Impaired folate metabolism has been associated with neural tube defects and other anomalies. Novel mouse models in the lab implicate specific folate-metabolising enzymes including AMT (aminomethyltransferase) in neural tube defects, craniofacial malformations and heart defects. We aim to elucidate how loss-of-function mutations in AMT cause multiple structural abnormalities during embryonic development. At the molecular level, we are evaluating stage- and tissue-specific localisation of AMT, and its relative distribution in/out of complexes with other components of the glycine cleavage system. We hypothesise lack of AMT may lead to altered localisation of partner enzymes like GLDC. At the cellular level, we are investigating whether impaired function of AMT affects neural crest cell specification, migration or differentiation using in situ hybridisation and immunohistochemistry. Elucidating these mechanisms will provide insight into the developmental basis of congenital defects and offer the potential for improved strategies for diagnosis, treatment and prevention.

Poster 41. Embryo Origami: Investigating the Mechanism of Head Fold Formation in Chick Embryos

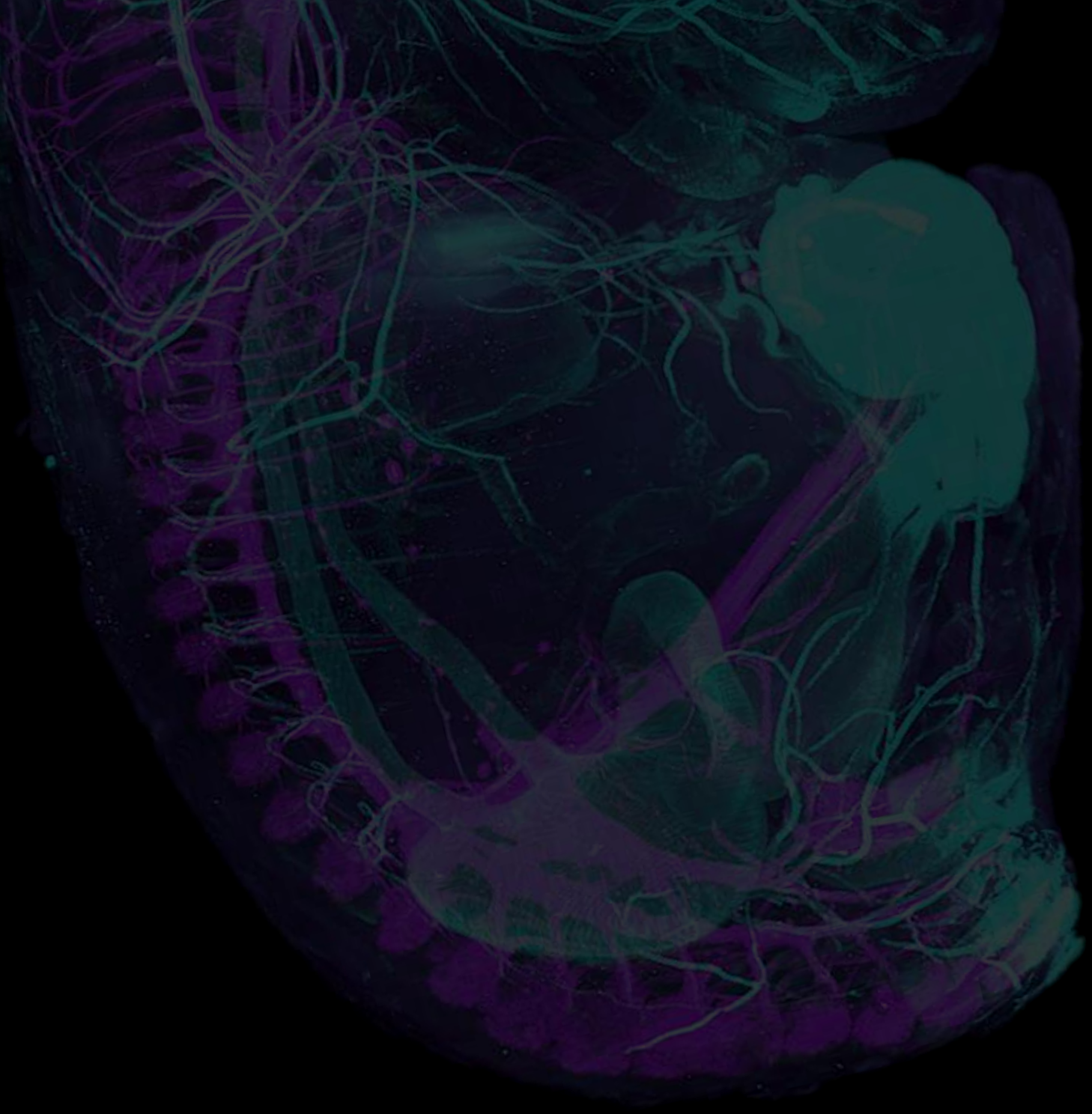
By Ruoheng Li (University of Cambridge)

During development, the avian embryo transforms from a flat disc into a three-dimensional structure through a series of body folds. The head fold (HF) is the first large-scale out-of-plane tissue deformation in the chick blastoderm, yet the mechanisms underlying its morphogenesis remain poorly understood. A previous computational simulation recapitulated folding dynamics by prescribing specific active deformations, but lacked direct experimental validation of the proposed cellular activities. Here, we use cryosectioning to examine HF at cellular resolution and show that actomyosin activity and tissue patterning are inconsistent with the predictions of this model, motivating the search for an alternative mechanism. Through live imaging, we find that the anterior blastoderm moves anteriorly against extraembryonic tissue prior to folding, and propose that HF arises as a patterned mechanical buckling response to compression.
Consistent with this, the boundary between the blastoderm and extraembryonic tissue is enriched in extracellular matrix (ECM) proteins, and enzymatic digestion of this boundary with dispase disrupts HF formation. Furthermore, direct mechanical compression of the anterior blastoderm confirms its capacity to buckle out-of-plane. To probe the molecular regulation of tissue mechanics, we inhibited ROCK and found that this abolishes HF and produces ectopic crinkles along the neural plate border. Mechanical measurements show that ROCK inhibition reduces anterior blastoderm stiffness, suggesting that folding defects may arise because a softer, more compressible tissue fails to respond to in-plane compressive stress through out-of-plane deformation. Together, these results reveal a mechanically driven buckling mechanism for HF initiation and implicate ROCK-dependent tissue stiffness as a key parameter.

Poster 42: Integrins pattern the Drosophila embryonic neuroepithelium by influencing progenitor morphodynamics, division and position

By Lamiya Dohadwala (Tata Institute of Fundamental Research)

Gene regulatory networks that confer cell fate and intracellular force generation mechanisms that drive cellular morphodynamics guide the patterning of tissues, but the nature of their interplay is poorly understood. We explore this during Drosophila embryonic neurogenesis whose first step, the delamination of a neuroblast from the surface epithelium, resembles anepithelial to mesenchymal transition (EMT). Using real-time, 3D confocal microscopy combined with quantitative morphometry, we identify robust morphometric state transitions that culminate in the basal delamination of the neuroblast (NB) from an equipotent epidermal proneural cluster, and find that mitotic rounding occurs concomitantly with delamination. We uncover marked heterogeneity and reduced collectivity in the morphodynamics of the NB and its nearest neighbours, and suggest their origins in the morphogenetic activity in the extending germband. We show that the appearance and distribution of the NB and GMC fate determinants Deadpan/dpn and Prospero/pros respectively correlate temporally with distinct morphometric states. We identify changes in cell-cell and cell-substrate adhesion that accompany neuroblast delamination and division and show that the adhesive microenvironment provided by integrin-ECM interactions patterns the neuroepithelium by influencing the spatiotemporal control of cell shape, division and position.



Young
Embryologist
Network

The Sammy Lee Medal

The medal is presented annually to an outstanding piece of research at the YEN meeting. The bronze medal was designed by the late Felicity Powell and is an artwork with depth and meaning both for Sammy's family and her own. Sammy Lee, Visiting Professor in Cell and Developmental Biology at UCL, passed away suddenly on 21 July 2012, aged 54. Sammy was a great friend to many in the community; a gregarious person who could and would happily talk to everyone he met. He was a lateral thinker whose enthusiasm was infectious. Sammy's scientific journey began in the 1970s. He chose to study Physiology at Chelsea College, KCL based on the fact he was a Chelsea Football supporter.

Sammy developed numerous new successful techniques including pioneering the first UK gamete Intra-fallopian Transfer (GIFT) program and in later years, whilst head of the lab in the Chelsea and Westminster hospital he developed a successful technique, allowing infected patients to give birth to HIV-free babies. When he returned to academic work, Sammy's focus at UCL was very much on the students who he was always willing to help. He enjoyed teaching the next generation of scientists both undergraduate and postgraduate. He also wanted to continue his research in stem cell and regenerative medicine research which included sponsoring a PhD studentship through his charity REGEN. It was his wish to present a medal to a young scientist to encourage them in their career.

With that in mind, since 2013, the Lee Family and REGEN have been great supporters of YEN. At each annual conference, Sammy's family presents the medal to the best short talk, celebrating the recipient's research achievements.

This medal was designed by Felicity Powell in memory of Sammy Lee with his warm smile on side and representation of an embryo on the other.

Young
Embryologist
Network