Prospectus 2018

The Wellcome Trust / Cancer Research UK
Gurdon Institute
Welcome to our new Prospectus 2018 and Annual Report 2017. Inside you will find out who we are, what we do, and why we believe the Gurdon Institute is a wonderful place to do research on development and disease.

Our International Scientific Advisory Board, chaired by Professor Judith Kimble, visited for two days in March 2017 and subsequently gave us a glowing audit. They stated that the research at the Institute is at the cutting edge, making it “one of the elite research institutions internationally”, and noted that we maintain “exceptional core facilities”. In this Prospectus you can read about the diverse range of research fields being pursued by our labs (pp. 14–45), the scientific highlights of 2017 (pp. 04–07), and get an insight into the many collaborative and social events that bring our members together (pp. 70–71) and engage the public in our research (pp. 68–69).

As well as hosting a truly international community of researchers and staff (p. 09), we continue to work on improving equality and diversity, creating opportunities for our people to thrive and develop. I am delighted that this has recently been recognised with our renewed Athena SWAN Bronze Award. We can wear our updated Bronze logo with pride for another four years.

Every year is marked by transitions. Hansong Ma moved into her lab in January 2017 and is now fully established with her mitochondrial genome experiments in full flow. We sadly said goodbye to Eugenia Piddini and her lab as they departed in June 2017 to the University of Bristol, with many congratulations for Eugenia’s Professorial Research Fellowship. We also said goodbye to Ann Cartwright, who retired in October after 17 years of fantastic service as our Institute Administrator; in her place we welcome Helen Lonergan as our new Business & Operations Manager.

Further movements are afoot for 2018 as Rick Livesey takes up a Chair of Stem Cell Biology at University College London’s Great Ormond Street Institute of Child Health in June. His research team will not physically move until the new Zayed Centre for Research into Rare Disease in Children opens in several months’ time; for now, we offer our congratulations on this exciting development that will take Rick’s stem cell research closer to translational outcomes and potential new treatments.

Another significant change for the Institute is due on 1st January 2019, when Senior Group Leader Julie Ahringer will become the Institute’s first female Director, and I step down to become a Senior Group Leader again. I am delighted to be handing the reins over to Julie after a very rewarding 10 years, and I wish her every success in her new role. This therefore marks my last introductory piece for the Prospectus. I hope that you enjoy reading about our research and other activities in 2017.

Daniel St Johnston
Director
May 2018
UK GSN Medal for Steve Jackson
The UK Genome Stability Network Medal 2017 was presented to Professor Jackson at the Network’s annual conference on 8th January 2017, for his “contributions to the field of genome stability and particularly for the realisation of the therapeutic potential of targeting the DNA damage response”. Steve received the medal from Professor Jessica Downs of the Institute of Cancer Research, who was a former PhD student and postdoc in the Jackson lab!

The big read
Drs Christy Hung and Rick Livesey appeared in a Financial Times Big Read article in November as part of a Seasonal Appeal for Alzheimer’s Research UK, the charity that funds the lab’s ‘Alzheimer’s in a dish’ research. Rick was interviewed and demonstrated the lab’s exploration of microglia, the brain’s immune cells, commenting: “in Alzheimer’s, do they overreact or under-react to damage in the brain?”.

Prestigious career development award
Dr Ufuk Günesdogan won a 2017 Sofja Kovalevskaja Award of €1.65 M to run his own research group for five years at the University of Göttingen, Germany. During his postdoctoral studies in Azim Surani’s group, Ufuk researched the development of primordial germ cells, work that he is continuing with an emphasis on regulation of gene activity in his new lab.

Storm Therapeutics is a winner
One of our most recent spinout companies, Storm Therapeutics, won the accolade of top Biotech Start-up at the Cambridge Independent’s Entrepreneurial Science and Technology Awards in September 2017. The company was founded by Professors Eric Miska and Tony Kouzarides, and is developing small molecule inhibitors of RNA-modifying enzymes for the treatment of cancers.

Revealing chromatin remodelling during brain development
The Brand lab use Targeted DamID to show that large-scale chromatin remodelling occurs during Drosophila neural development. Polycomb-mediated repression regulates cell lineage-specific transcription factors that control spatial and temporal gene expression patterns of the developing brain.

Chromatin state changes during neural development revealed by in vivo cell type specific profiling

METTL3 is a new drug target for AML
Searching for new treatments for Acute Myeloid Leukaemia (AML), the Kouzarides lab and colleagues have identified the methyl transferase enzyme METTL3 as a highly viable target. Inhibiting the METTL3 gene destroys AML cells without harming normal blood cells, and this will enable pharmaceutical efforts to find drugs that are specific METTL3 inhibitors, to treat AML.

Promoter-bound METTL3 maintains myeloid leukaemia by m6A-dependent translation control

Suppressor screen in yeast identifies mechanism for anti-cancer drug resistance
Using yeast as a model system to understand how cells might acquire resistance to the anti-cancer drug camptothecin, the Jackson lab define a mechanism that involves control of DNA topology and appears to be conserved in other eukaryotic cells, including human cells.

Chromatin determinants impart camptothecin sensitivity
A unifying theory for branching morphogenesis

Using statistical simulations compared with datasets from developing breast, prostate, pancreas and kidney, the Simons lab propose that growing ducts create networks by alternately elongating and bifurcating, but stopping when they contact another branch.

A unifying theory for branching morphogenesis


Epigenetic memory inhibits reprogramming in nuclear transfer

The Gurdon lab show that nuclear transfer embryos retain the memory of a past state of active gene transcription. This can be countered by demethylation treatment, which thereby improves the efficiency of nuclear transfer and successful demethylation treatment, which thereby improves gene transcription. This can be countered by embryonic nuclear transfer embryos.

Epigenetic memory inhibits reprogramming in nuclear transfer


The function of Chk1 in early frog embryo development

The Zegerman lab with ex-Gurdon Institute Director Jim Smith have examined the signals in the early dividing frog embryo to demonstrate why the cell cycle lengthens at the Xenopus laevis mid-blastula transition. The limiting replication factor Drf1is specifically inhibited by checkpoint kinase Chk1, leading to longer replication cycles. Chk1 inhibition of the replication factor Drf1 guarantees cell cycle elongation at the Xenopus laevis mid-blastula transition.

The function of Chk1 in early frog embryo development


Building the tails of comets

Using fluorescence microscopy to follow the biochemistry of actin assembly with artificial liposomes and human cell lines, the Gallop lab show how specific phospholipids PI(3)P and PI(4,5)P2, coupled with high membrane curvature, trigger actin polymerisation. The findings suggest a therapeutic strategy for Lowe syndrome, a multi-system genetic disease.

Building the tails of comets


The origin of human germ cells pinpointed with new pig model

New insights into early human development are provided by the Surani lab. With Cambridge and Nottingham colleagues they use pig embryos and human stem cells to simulate human embryo development, and pinpoint the origin of primordial germ cells.

The origin of human germ cells pinpointed with new pig model


Analysing the diversity of RNA modifications

The Miska lab, with Chemistry’s Balasubramanian lab and other colleagues, apply a suite of chemical techniques to analyse the complete C. elegans transcriptome (i.e. all the species of RNA molecules) for modifications, and then track how these respond to the environmental stressors of heat or starvation.

Analysing the diversity of RNA modifications


Screening potential Alzheimer’s drugs in vitro

The Livingey lab have developed an in vitro stem cell model of Alzheimer-type dementia using neurons derived from Down syndrome patients’ cells. They now demonstrate the feasibility of using these cells to screen small molecules that modulate production of toxic forms of the amyloid beta protein.

Screening potential Alzheimer’s drugs in vitro


Organoids for functional experiments on human embryonic lungs

While embryonic mouse lung has been the standard model for studying human lung development, the Rawlins lab now show that human embryonic lung distal tip epithelium can be grown in vitro, in long-term self-renewing cultures. This 3D-organoid model is accessible to genetic experiments, allowing more direct study of features unique to human lung and supporting research into lung diseases.

Organoids for functional experiments on human embryonic lungs

The Gurdon Institute is funded by Wellcome and Cancer Research UK to study the biology of development, and how normal growth and maintenance go wrong in cancer and other diseases.

The Gurdon Institute is a world-leading centre for research at the interface between developmental biology and cancer biology. We focus on several related topics: cell division, proliferation and genome maintenance; function and regulation of the genome and epigenome; mechanisms of cell fate determination; multipotency and plasticity; and the cell biological processes underlying organ development and function. We investigate these areas in both normal development and cancer using several model systems, from yeast to human organoids.

Since our formation in 1991, our research has led to major insights into the molecular and cellular defects that give rise to cancer and other diseases of ageing, and several findings have been successfully translated into drug discovery through spinout companies.

We publish around 100 papers each year, in the leading journals in our fields (pp. 72–77).

We currently have 14 group leaders and two associate group leaders (pp. 14–45). At any one time there are around 240 scientists at the Institute and up to 60 core support staff (pp. 50–63). There are typically over 40 nationalities represented among research staff (see chart on p. 08) and dozens of languages spoken.

As a research institute within the University of Cambridge School of the Biological Sciences we enjoy scientific collaborations and joint activities with departments close by, where our group leaders are members and contribute to teaching. But beyond being part of the local intellectual environment, our researchers gain additional benefits from our institute status:

- Exposure and access to a diverse range of research questions, model organisms and techniques, all under one roof (see pp. 14–45)
- Purpose-built laboratories with shared core facilities and access to state-of-the-art equipment
- Expert technical support for imaging and image analysis (pp. 46–47), next-generation sequencing and bioinformatics
- Central services providing administration, computing and IT support, stores, and media preparation and glass washing
- Frequent internal and external seminars and masterclasses, an annual retreat, and Institute associations for postdocs and PhD students (pp. 70–71)
- Public engagement programme for exchange and reciprocal learning between our scientists and the wider public (pp. 68–69)
- On-site catering and a variety of social events and sports groups, including basketball, badminton, weekly ‘happy hours’ and the Christmas party, to promote our inclusive, family atmosphere.
The Institute’s principal sponsors are Wellcome and Cancer Research UK, through our core grant and through programme grants to group leaders. In addition, we benefit from several other funding sources including governmental and charitable grants, as shown in the chart below.

The map shows the destinations of our leavers in 2017 and where our group leaders and other researchers gave talks around the world. Nine of our postdoc leavers have taken up research group leader positions including in Götingen, Kharagpur, Okazaki and Vienna; new postdoctoral positions have been taken up across Europe (with many in the UK), the USA and Australia. Leavers not pursuing academic research include new roles as a science teacher, patent examiner, medical student or pharmaceutical scientist. Audiences from South America to Taiwan via much of Europe have heard our scientists describe the full range of our research, from lungs to liver, stem cells to stress, and the germline to genome architecture.

The Institute has an Athena SWAN Bronze Award from the Equality Challenge Unit for promoting equality across the workforce. Examples of successes that led to the renewal of our award in March 2018 include: almost doubling the proportion of female postdoc leavers who took up group leader positions, more than doubling the number of staff taking paternity leave and increasing the representation of women among our high-profile seminar speakers to 50%, compared with the data for our first award in 2014.

The Institute is overseen by a Management Committee, chaired by the Head of the School of the Biological Sciences, Professor Abigail Fowden (Professor of Perinatal Physiology, Dept of Physiology, Development and Neuroscience), and our scientific progress and future research plans are assessed at regular intervals by our International Scientific Advisory Board (see p. 63).

Join us

We welcome enquiries from prospective graduate students. We have a thriving population of graduates who contribute greatly to both the stimulating research environment and the life of the Institute as a whole. Graduates also become members of the biological or medical sciences department to which their group is affiliated. Graduate studentships are supported from a variety of public and private sources. Wellcome finances a number of schemes in the University, including one in developmental mechanisms and one in stem cells. The Cancer Research UK Cambridge Centre also provides studentships. Further details of the schemes available can be found on our website. Applicants should write to the leader of the group they wish to join (by email to: contact@gurdon.cam.ac.uk).

We advertise positions for postdocs through the University’s jobs website at www.jobs.cam.ac.uk and on www.jobs.ac.uk, and group leaders are also happy to be contacted directly by motivated researchers wishing to develop their careers in the relevant field.
Focus on research

Featuring
Group leaders: Julie Ahringer, Andrea Brand, Jenny Gallop, John Gurdon, Meritxell Huch, Steve Jackson, Tony Kouzarides, Rick Livesey, Hansong Ma, Eric Miska, Emma Rawlins, Daniel St John, Azim Surani, Philip Zegerman
Associate group leaders: Martin Hemberg, Ben Simons
Scientific facilities: Alex Sossick
How is chromatin structure regulated to direct correct gene expression programmes? The genome is regulated in the context of chromatin, the organisation of genomic DNA with histones and hundreds of associated proteins and RNAs. Chromatin structure controls gene expression and other nuclear processes, and its regulation is critical for the expression of cell identity, the maintenance of pluripotency, and the transformation to cancer. In addition, aberrations in chromatin architecture underlie developmental defects and disease, underscoring the importance of understanding how chromatin structure is regulated.

Controls occur at many levels, including local interactions between regulatory elements, modification of linear chromatin domains, and larger scale spatial folding and organisation within the nucleus. We use *C. elegans* to study these processes, applying genetics, high-throughput genomics and computational approaches to understand the regulation of gene expression and genome organisation in development.

**Co-workers**
Alex Appert, Francesco Carelli, Chiara Cerrato, Yan Dong, Andrea Frappotti, Tessa Gaarenstroom, Csegne Gal, Ni Huang, Jürgen Jänes, Andrew Katznelson, Florence Leroy, Wei Qian Seow, Jacques Serizay, Garima Sharma, Przemyslaw Stempor

**Selected publications**


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**Top:** Promoters and enhancers are regulatory elements with similar properties. Both bind regulatory factors (TF), initiate bi-directional transcription, and generate long RNAs. How enhancers regulate promoters is poorly understood.

**Bottom:** Circos plot of a 200 MB region of *C. elegans* chromosome X. Inner circle shows individual chromatin interactions, which occur at high frequency at regulatory elements. Outer circle shows genes, and other circles show different histone modifications and regulatory elements.
Stem cell populations in tissues as varied as blood, gut and brain spend much of their time in a mitotically dormant, quiescent state. A key point of regulation is the decision between quiescence and proliferation. The ability to reactivate neural stem cells in situ raises the prospect of potential future therapies for brain repair after damage or neurodegenerative disease. Understanding the molecular basis for stem cell reactivation is an essential first step in this quest. In Drosophila, quiescent neural stem cells are easily identifiable and amenable to genetic manipulation, making them a powerful model with which to study the transition between quiescence and proliferation. These stem cells exit quiescence in response to a nutrition-dependent signal from the fat body, a tissue that plays a key role in the regulation of metabolism and growth. My lab combines cutting-edge genetic and molecular approaches with advanced imaging techniques to study the reactivation of Drosophila neural stem cells in vivo. This enables us to deduce the sequence of events from the level of the organism, to the tissue, the cell, and finally the genome.

Co-workers
Neha Agrawal, Benjamin Badger, Catherine Davidson, Anna Hakes, Robert Krautz, Stephanie Norwood, Leo Otsuki, Takumi Suzuki, Jocelyn Tang, Christine Turner, Jelle van den Ameele, Rebecca Yakob, Mo Zhao

Selected publications

Quiescent neural stem cells extend processes towards neurons in the Drosophila ventral nerve cord. Neural stem cell nuclei are labelled in red; cell membranes in green; neuronal synapses in blue. [Image by Leo Otsuki.]
How do cells generate and maintain their characteristic shapes?

The cell membrane, as the boundary of the cell, is moulded into shape by dynamic remodelling of its links to the actin cytoskeleton during cell division, polarisation, movement, differentiation and for everyday housekeeping. In disease, the actin machinery is hijacked by invading pathogens. Some actin regulators are overexpressed and redeployed during cancer metastasis, and control of the actin cytoskeleton can be disrupted in genetic diseases, causing intellectual disability, kidney dysfunction and other problems.

We are studying how actin filaments polymerise at two types of specialised structures at the cell membrane: filopodia, which are fingerlike protrusions, and endocytic vesicles, which bud inwards to bring in components from the membrane or environment. We have developed model systems using phospholipid bilayers and frog egg extracts that allow us to follow the molecular events of actin self-assembly in different contexts. By focusing on unusual predictions from these in vitro assays, we work out how the actin cytoskeleton is regulated by imaging cells in accessible, native developmental contexts in fruit fly and frog embryos.

Co-workers
- Ulrich Dobramysl, Jonathan Gadsby, Iris Jarsch, Bishara Marzook, Julia Mason, Kathy Oswald, Benjamin Richier, Hanae Shimo

Selected publications

Three-dimensional reconstruction of filopodia-like structures growing from a supported lipid bilayer. The structures were segmented based on fluorescent actin intensity in a stack of microscopy images of size 76.13 x 76.13 x 30 microns. Colours were randomly assigned as a guide for the eye. The segmentation was performed using a custom image-analysis pipeline. [Image generated by Ulrich Dobramysl from microscopy images by Iris Jarsch and an analysis pipeline designed by Richard Butler.]
Can we make cell reprogramming more efficient?

Our group focuses on somatic cell nuclear transfer to amphibian eggs and oocytes from two complementary points of view. One aims to identify the molecules and mechanisms by which the cytoplasm of an egg or oocyte can reprogramme the nucleus of a differentiated somatic cell to behave like that of an embryo. From this state, many different kinds of cells for replacement can be generated. The other aim is to identify the molecules and mechanisms that stabilise the differentiated state of somatic cells, as a result of which they resist reprogramming procedures. For these purposes we use single nuclear transfer to unfertilised eggs or multiple nuclear transfer to ovarian oocytes.

We monitor changes in gene expression induced in nuclei transplanted from cells that have been modified to overexpress chromatin-changing enzymes. This involves the transplantation of single somatic nuclei to enucleated, unfertilised eggs or of multiple mammalian nuclei to the nucleus of a mature oocyte. By this means we identify mechanisms that can convert a specialised somatic cell nucleus to an embryonic state, or that can stabilise the differentiation of normal somatic cells.

**Co-workers**

Can Aztekin, Dilly Bradford, Nigel Garrett, Eva Hörmanseder, Khayam Javed, Jerome Jullien, Magdalena Koziol, Mami Oikawa, Clare Pacini, Angela Simeone, Ming-Hsuan Wen

**Selected publications**


Top: Two types of nuclear transfer experiments, with eggs or oocytes.

Bottom left: H3K4 demethylation of donor nuclei improves development of the resulting nuclear transfer (NT) embryos. Shown is the developmental improvement of gastrula embryos generated by NT using different donor nuclei, including H3K4-methylated and -demethylated neurula stage nuclei (Hörmanseder et al., 2017).

Bottom right: Differential expression of genes after nuclear transfer to oocytes. Mouse embryonic stem cells (ES) or fibroblasts (MEF) contain distinct populations of genes resistant to reprogramming by oocytes, as well as sharing a large proportion of properly reprogrammed genes (Jullien et al., 2017).
How can we repair diseased liver and pancreas?

In adult mammals, many tissues have the capacity to self-renew to maintain healthy function in homeostasis and after damage. But the capacity for cell turnover varies. In the intestine and stomach, adult stem cell populations are constantly replenishing, while in the liver and pancreas cell proliferation is limited. Chronic liver disease and pancreatic cancer are strongly associated with inflammation and tissue damage, which activates stem cells and progenitor cells to repair lost tissue. Our goal is to understand the activation mechanism in order to harness it for therapeutic strategies.

We have established a novel culture system, liver organoids, which allows the massive and infinite expansion of mouse liver cells into three-dimensional structures that resemble functional liver tissue. When transplanted into a mouse model of liver disease (‘FAH –/–’), these cells partially rescued the liver phenotype. We also work with pancreas cells and diseased human liver cells in culture, and are testing how well our models can represent human pathology in a dish.

Co-workers
- Luigi Aloia, Robert Arnes, Laura Broutier, Lucia Cordero Espinoza, Nikitas Georgakopoulos, Mewanthi Flaminia Kaluthantrige Don, Giannarco Mastrogiovanni, Mikel Mckie, Kathy Oswald, Nicole Prior

Selected publications

Liver culture system. Human liver cells can be expanded from human biopsies into 3D structures called organoids, which retain some of the functions of the tissue in vitro. A similar system has been established for pancreas cells.
DNA is constantly damaged by environmental and endogenously arising agents. Cell survival and genome integrity are promoted by the DNA-damage response (DDR), which detects, signals the presence of and repairs DNA damage. DDR defects are associated with developmental disorders, immunodeficiencies, infertility, premature ageing and cancer.

Our research aims to characterise the cell biology and mechanisms of established DDR pathways, and to apply this knowledge to better understand and treat human diseases. We are also developing approaches to identify new DDR components and their functions.

For example, through synthetic viability screening, we recently identified a chromatin post-translational modification that drives sensitivity of cells to the anticancer drug camptothecin, and have shown how similar screens in yeast and mammalian cells can identify functional protein domains that impart drug sensitivity/resistance. With colleagues at the MRC Epidemiology Unit, Cambridge, we identified genetic variants associated with Y chromosome loss, with implications for genome instability and cancer susceptibility. Finally, we are using CRISPR-Cas9 synthetic viability screens to identify novel DDR proteins and define drug-resistance mechanisms.

Selected publications
Nat Rev Drug Discov 17: 57–78.


Do chromatin and RNA modifications offer therapeutic targets?
DNA exists in the cell nucleus wrapped around histone proteins to form chromatin. The DNA and histones are decorated with many types of covalent chemical modifications, which can affect transcription and other cellular processes. In addition, non-coding RNAs that regulate chromatin function can be similarly chemically modified. Our lab is involved in characterising the pathways that mediate and control DNA, RNA and histone modifications. We try to understand the cellular processes they regulate, their mechanism of action and their involvement in cancer.

Our work on histone modifications has led to the identification and characterisation of the enzymes that mediate citrullination of arginines and regulate pluripotency. Our work on RNA identified a novel pathway leading to Acute Myeloid Leukaemia involving an RNA methyltransferase (METTL3) that binds the promoter of leukaemia genes and regulates their translation.

Our interest in defining pathways to intervene in cancer has identified an epigenetic small molecule inhibitor, I-BET, that can be effective against Mixed-Lineage Leukaemia, a disease common in children. This potential drug is currently in clinical trials.

Selected publications

Chemical modifications that regulate different aspects of gene expression have been identified on DNA and histone proteins within chromatin, as well as on non-coding RNAs. We aim to identify and characterise the modifications, the enzymes that catalyse modification reactions and the transcription machinery that recognises the modifications, to yield new strategies for cancer therapies.
FOCUS ON RESEARCH

Studying human brain development and disease in the lab.

The human cerebral cortex, the thinking, decision-making, largest part of our brain, sets us apart from other animals – and poses a challenge for biomedical researchers. Animal models cannot capture the spectrum of characteristics of the human cerebral cortex in development or disease, and so our research uses human cells in tissue culture. Our methods for differentiating human pluripotent stem cells into different types of cells allows us to study neurons and the neural circuits they make, starting from living patients’ cells. We are studying how the human cerebral cortex develops and how that differs from other animals, and how variations in development lead to disease.

We also apply these approaches to study neurodegeneration in dementia, particularly Alzheimer’s disease. Using stem cells from patients with genetic forms of Alzheimer’s disease, we have modelled disease pathogenesis in neurons in culture. We use these systems to understand how Alzheimer’s disease starts and progresses in the brain, and to test the efficacy of potential therapeutic strategies.

Co-workers
Philip Brownjohn, Ashley Campbell, Lewis Evans, Jayne Fisher, Elsa Ghirardini, Moritz Haneklaus, Silvia Hnatova, Qi Ying (Christy) Hung, Federica Marinaro, Ayiba Momoh, Steven Moore, Francesca Paoneessa, James Smith, Ravi Solanki, Frances St George Hyslop, Alessio Strano, Victoria Stubbs, Ellie Tuck

Selected publications

Development, evolution and degeneration of the brain

Human stem cell-derived cortical neurons cultured in vitro. Confocal imaging of microtubules (magenta) and nuclear membrane (cyan). [Image by Jun Ru Zhan.]
How are mitochondrial mutations transmitted? In addition to the nuclear genome, all animals have another genome packed inside the mitochondrion called mtDNA. This maternally inherited genome encodes important proteins for energy production. Pathogenic mitochondrial mutations often arise among the thousands of copies of wild-type genomes in each cell, and beyond a certain threshold the genetic defects will manifest as a disease phenotype. Over 50 such mitochondrial diseases have been described in humans.

Selectivity in the transmission of functional versus pathogenic genomes in somatic cells affects the expression of disease phenotype as we age. Selective transmission in germline cells governs the inheritance of mtDNA mutations from mother to progeny, and in this way its evolution. I have developed genetic tools for mitochondrial studies in Drosophila, which have a mitochondrial genome that is very similar to humans. By artificially mixing different genomes and following their transmission over generations, my lab uses Drosophila to study inheritance of mtDNA mutations to provide insights into human longevity, fertility and disease.

Co-workers
Chieh-Yin (Ason) Chiang, Ying (Ivy) Di, Anna Klucnika, Kathy Oswald

Selected publications

Top: The multi-copied mitochondrial genome (mtDNA) is a circular double-stranded DNA molecule encoding 13 essential polypeptides of the oxidative phosphorylation system. It is usually inherited from only the mother.

Bottom: Artificial mixing of wild-type and mutant mitochondrial genomes resolves two types of selection that influence the competition between co-existing mitochondrial genomes: 1) a purifying selection, where the genome providing more functions always takes over; and 2) a selfish selection, where a ‘bully’ genome takes over a ‘wimpy’ genome if it replicates or transmits better (i.e. independent of function). Occasionally, co-existing mitochondrial genomes recombine to form a new genome. We aim to establish sensitive genetic screens to identify nuclear genes influencing selective transmission of mtDNA.
What does non-coding RNA do in development and disease?
Most of the RNA transcribed from the DNA in our genome is not translated into protein but instead has direct functions in regulating biological processes. This paradigm shift in nucleic acid biology has been supported by technical advances in high-throughput sequencing, molecular genetics and computational biology, which can be combined with more traditional biochemical analyses.

Many species and roles of non-coding RNA have been identified. Our goal is to understand how non-coding RNAs regulate development, physiology and disease. We are exploring microRNA in the pathology of cancer and other diseases, RNA interference in viral immunity, Piwi-interacting RNA in germline development and genome integrity, and endogenous small interfering RNA in epigenetic inheritance – where we predict a big impact in understanding human health. Our model organisms are the nematode worm, the cichlid fishes of the Rift Lakes of East Africa, mouse, and human cell culture. More recently we have developed a technology to assess RNA structure and RNA–RNA interactions in living systems. We used this to uncover unexpected biology for the Zika virus.

Co-workers

Selected publications

Exploring the structure of the Zika virus RNA genome inside human cells in culture. Top: Crosslink Of Matched RNAs And DEep Sequencing (COMRADES) developed by Omer Ziv reveals the pattern of interactions between nucleotides along the RNA molecule, and a close-up shows the precision of this highly reproducible method. Each dot represents an interaction between the corresponding genome coordinates in the x and y axes. Bottom: Open and circular conformations of the viral RNA genome as computed from the resulting data.
How do stem cells build and maintain the lung? The complicated three-dimensional structure of our lungs is essential for respiration and host defence. Building this structure relies on the correct sequence of division and differentiation events by lung progenitor cells, which also maintain the slowly turning-over airway epithelium in the adult. How is the production of different cell types controlled in embryonic development and adult maintenance? We apply mouse genetics, live imaging, single-cell molecular analysis and mathematical modelling to understand lung stem cells, with a longer-term aim of directing endogenous lung cells to repair, or regenerate, diseased tissue.

In the adult lung we focus on the cellular mechanisms that maintain stem cell quiescence at steady-state, but allow a rapid repair response when needed. In the embryonic lung we study a population of multipotent progenitors that undergo steroid-induced changes in competence during development. We have recently established conditions for growing these embryonic progenitors as self-renewing organoids from human tissue samples, and are using them to focus our research on normal human lung development and conditions related to premature birth.

Co-workers
Quitz Jeng, Heleen Kool, Florence Leroy, Kyungtae Lim, Dawei Sun

Selected publications
Nikolić M et al. (2017) Human embryonic lung epithelial tips are multipotent progenitors that can be expanded in vitro as long-term self-renewing organoids. Elife 6: e26575.

We are studying how cell–cell interactions control normal human lung development. In this 16-weeks-gestation human embryonic lung, a close interaction can be seen between the developing epithelial progenitor cells (green) and the endothelial cells (red) that line the developing blood vessels. Cell nuclei in blue. [Image by Valentin Picant.]
How do cells know ‘up’ from ‘down’?
Normal cells in the body are not symmetrical spheres; most take up specialised shapes and perform different functions at opposite ‘ends’. Cell polarity is also essential in development, for example in determining the head-to-tail axis of many animals, for cell migration and for asymmetric stem-cell divisions to supply specialised daughter cells. Furthermore, loss of polarity is a hallmark of tumour cells and is thought to contribute to tissue invasion and metastasis.

We explore polarity in Drosophila and in mouse intestinal organoids. Much of our work focuses on epithelia, the sheets of polarised cells that make up most organs of the body to form barriers between compartments. We study the factors that mark different sides of the cell and how these organise the internal cell architecture. For example, we have determined how cells divide so that both daughters stay in the epithelial layer, and have found a mechanism that pulls cells born outside the monolayer back into place. Now we are using live super-resolution microscopy to visualise polarised transport in epithelial cells.

Co-workers
Edward Allgeyer, Jia Chen, Hélène Doerflinger, Edo Dzafic, Weronika Fic, Jacqueline Hall, Nick Lowe, Dmitry Nashchekin, John Overton, Amandine Palandri, Andrew Plygawko, Jennifer Richens, George Sirinakis, Iola Squires, Vanessa Stefanak, Mihoaka Tame, Vivien Tsang, Helen Zenner-Branco

Selected publications


The lateral surfaces of epithelial cells stick to each other to form epithelial sheets, with their free apical surfaces, which are often covered with microvilli, facing towards the outside or the lumen of an epithelial tube or gland. The lateral junctions (yellow) create an impermeable barrier between cells so that fluids, solutes and pathogens cannot leak across the epithelium. During development, epithelial sheets change shape to form more complex structures, such as tubes, and the individual cells must alter the relative proportions of their apical, lateral and basal domains to facilitate these movements. Most cancers arise from epithelial tissues and one hallmark of the tumour cells is a loss of apical–basal polarity, which often correlates with the malignancy of the tumour.
What makes a germline cell?

We study primordial germ cells (PGCs), precursors to eggs and sperm, in the early embryo. We have established principles of early human development and the mechanisms of cell fate determination with a focus on human PGC specification. A unique epigenetic resetting follows in the germline after hPGC specification. Our work shows that SOX17 is the key regulator of human, but not mouse, germ cell fate. By developing in vitro models, and with authentic hPGCs from human embryos, we have also established how pluripotent stem cells gain competence for germ cell and somatic fates in human. These findings are important for studies on human pluripotent stem cells and regenerative medicine, which are applicable to advances in human development and disease.

Whereas SOX17–BLIMP1 apparently initiate the epigenetic programme in early human germline, BLIMP1–PRDM14 play a similar role in mouse germline, resulting in the comprehensive erasure of DNA methylation (except for some resistant loci), X-reactivation and imprints erasure, followed by re-establishment of sperm- and oocyte-specific imprints. Defects in these gamete-specific imprints lead to a variety of human disease syndromes.

Co-workers

Maud Borensztein, Aracely Castillo Venzor, Elena Drousioti, Lynn Fraggett, Wolfram Gruhn, Nasko Irie, Caroline Lee, Sun Min Lee, Christopher Penfold, Anastasiya Sybirna, Walfred Tang, Frederick Wong

Selected publications


Cycle of human germline development. The zygote develops into the blastocyst, with pluripotent epiblast cells, which give rise to all cell lineages, including the germline. The bilaminar embryonic disc develops after implantation, and primordial germ cells (PGCs) are specified prior to gastrulation to form the ectoderm, mesoderm and endoderm. PGCs then migrate to the developing genital ridges, as they undergo epigenetic reprogramming, including global DNA demethylation. During the formation of sperm and eggs, the genome is remethylated and acquires germline-specific imprints; transmission of this epigenetic information contributes to totipotency at fertilisation.
FOCUS ON RESEARCH

How is DNA replication controlled?

A fundamental requirement for all life on earth is that an exact copy of the entire genome must be made before cell division. DNA replication is therefore tightly regulated because failures in this process cause genomic instability, which is a hallmark of many diseases, most notably cancers. In addition, inhibition of DNA replication is the primary mode of action of many anti-tumour therapies. Therefore investigating DNA replication control is important for finding new ways to diagnose and treat cancers. The evolutionary conservation of DNA replication mechanisms allows us to study this process in multiple systems, facilitating the translation of findings to humans.

We have shown that the levels of several key replication factors are critical to control the rate of genome duplication, not only in the single-celled organism, budding yeast, but also during vertebrate development in frog embryos. Our studies demonstrate that regulation of the levels of these factors is vital not only for normal cell division, but also for regulating the rate of cell proliferation in animal tissue. This has important implications for the deregulation of cell proliferation, which occurs in cancers.

Co-workers

Esther Cabana Morafraile, Geylani Can, Vincent Gaggioli, Fiona Jenkinson, Mark Johnson, Manuela Kieninger, Florence Leroy, Kang Wei Tan

Selected publications


The regulation of DNA replication initiation in eukaryotes

Regulation of replication initiation is critical for normal cell division.
What can sequencing data tell us about disease?

To create the different cell types in an organism, different genes are expressed at different times from the whole genome as transcripts of RNA, which will include both protein-coding and non-coding species. Understanding how, why, when and where genes are expressed is crucial for understanding not just development but also many diseases. High-throughput sequencing of RNA from different tissues can now provide insights into gene expression and related properties, but the experimental datasets are large, high-dimensional and noisy. Computational methods are required to extract maximum information from such data.

Our group uses computational analysis to develop quantitative models of gene expression and gene regulation. In particular, we are exploring single cell RNA sequencing, which can reveal insights that are inaccessible through traditional bulk experiments; for example, to estimate the number of differentiated cell types in the body. Another strand of research aims to further our understanding of noncoding DNA – providing better models of regulatory elements and characterising non-coding RNA.

Co-workers
Tallulah Andrews, Ilias Georgakopoulos-Soares, Louis-François Handfield, Nicholas Keone Lee, Guillermo Parada, Cristian Riccia, Xiaojuan Shen

Selected publications

Starting with single-cell RNA sequence analysis, we apply a computational method that clusters cells showing similar patterns in the sequence data, such that each cluster may represent a different type of cell. Our method, SC3 (unsupervised single-cell consensus clustering), is interactive and provides a robust clustering by combining a large number of outcomes obtained by using different parameters.
How do stem and progenitor cells regulate their fate behaviour to specify and maintain tissues?

In development, tissue precursors must coordinate proliferation and differentiation with collective cell movements to specify organs of the correct size, pattern and composition. In the adult, stem cells must regulate a precise balance between proliferation and differentiation to achieve homeostasis.

To address the mechanisms that regulate stem and progenitor cell fate, we integrate cell lineage-tracing approaches and single-cell expression profiling with concepts and methods from statistical physics. Applied to epithelial tissues, our studies have shown how principles of self-organisation and emergence provide predictive insights into the cellular mechanisms that regulate tissue development and maintenance. As well as questioning stem cell identity and the mechanisms that underpin cell fate stochasticity and state flexibility, these studies establish a quantitative platform to investigate pathways leading to tumour initiation and progression.

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Lemonia Chatzeli, Adrien Hallou, Seungmin Han, Tom Hiscock, David Jorg, Daniel Kunz, Hayden Selvadurai, Min Kyu Yum

Selected publications

Integration of neurons into the dentate gyrus of the mouse hippocampus. The image is obtained using an in vivo live-imaging platform to trace the fate of genetically labelled neural stem cells targeted by an Ascl1 promoter. Neural stem cells are marked in green, progenitors in orange and neurons in red. [Image by Gregor-Alexander Pilz and colleagues (Laboratory of Neural Plasticity, Faculties of Medicine and Science, Brain Research Institute, University of Zurich).]
The primary aim of the Scientific facilities team is to provide support, and access to state-of-the-art equipment, enabling frontline research in cell and developmental biology. Key facilities within this group are: Imaging, Bioinformatics, Scientific computing, Sequencing and Flow cytometry.

One of our major roles is to provide advice and training from basic through to advanced techniques. For example, we support a wide array of microscopy techniques, from wide-field deconvolution microscopes and confocal microscopes to super-resolution microscopy. We also provide cutting-edge analysis solutions for quantitative bio-image informatics and bioinformatics, which are vital components of biological research.

**Imaging**
Alex Sossick, Richard Butler, Nicola Lawrence

**Bioinformatics**
Charles Bradshaw, Keerthi Sannareddy

**Sequencing**
Kevin (Kay) Harnish

A time projection of a Xenopus retinal ganglion cell growth cone electroporated with NeonGreen-ENA and GAP-RFP RNA, and imaged using HILO illumination. The overlay shows automatic segmentation by Filopodyan, a Fiji plug-in developed in a collaboration between the Gallop group and Imaging facility to analyse fluorescence, morphology and dynamics in filopodia. [Image by Richard Butler and Vasja Urbančič.]
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Julie Ahringer
is a member of the Scientific Advisory Boards of the MRC London Institute of Medical Sciences and Wormbase.

Andrea Brand
is a member of the Royal Society Grants Committee and the Royal Society Diversity Committee, a member of the Governance Committee of the Wellcome-MRC Cambridge Stem Cell Institute, a member of the Scientific Advisory Committee of the Center for Integrative Genomics, University of Lausanne and a member of a Site Visit Review panel at the Francis Crick Institute, London. She is a Trustee of the Darwin Trust, Edinburgh and a Manager of the Anne McLaren Trust Fund. She is a Founding Board Member of The Rosalind Franklin Society (USA), a member of the Board of Directors of The Cambridge Science Centre and a Patron of the Cambridge Science Festival.

John Gurdon
is an Honorary Fellow of the Royal College of Physicians, the UK Academy of Medical Sciences and the Cambridge Philosophical Society; an Honorary Fellow (Inaugural Fellow) of the American Association for Cancer Research; and an Honorary Member of the Biochemical Society, the Anatomical Society of Great Britain and the American Anatomical Society.

Meritxell Huch
is an Affiliate Member of the Wellcome-MRC Cambridge Stem Cell Institute.

Steve Jackson
is a Fellow of the European Academy of Cancer Sciences. He is a Member of the Scientific Advisory Boards of the Cancer Research UK/ MRC Oxford Institute for Radiation Oncology, the MRC London Institute of Medical Sciences and the Francis Crick Institute. He is a Member of the Science Committees of the Netherlands Cancer Institute and Cancer Research UK and a Member of the Steering Committee of the Cambridge Cancer Centre. He is Consultant for Mission Therapeutics and Carrick Therapeutics.

Tony Kouzarides
is Co-founder and Director of the Mitner Therapeutics Institute and Founder/Director of Cambridge Gravity. He is a Member of the Executive Board of the Cambridge Cancer Centre and a Member of the Scientific Advisory Board of CABIMER, Seville. He is Founder and Patron of the Spanish Cancer Charity ‘Vencer el Cancer’, Co-founder and Director of Storm Therapeutics and Co-founder of Abcam plc and Chroma Therapeutics.

Rick Livesey
is an Affiliate Member of the Wellcome-MRC Cambridge Stem Cell Institute. He is Director of Gen2 Neuroscience Ltd and Director and Chief Scientific Officer of Talisman Therapeutics.

Eric Miska
is an Associate Faculty Member of the Wellcome Sanger Institute, a Member of the Wellcome Trust’s Interview Committee and of the Fellowship Committee of the European Molecular Biology Organization, and Co-founder and Director of Storm Therapeutics.

Emma Rawlins
is an Affiliate Member of the Wellcome-MRC Cambridge Stem Cell Institute and a member of the Wellcome Postdoctoral Fellowships Interview panel.

Daniel St Johnston
is Director of the Wellcome 4-year PhD Programme in Developmental Mechanisms, University of Cambridge, a Member of the Biochemical Society and a Non-Executive Director of the Company of Biologists. He is a Member of the Scientific Advisory Boards of the RIKEN Center for Developmental Biology, Kobe, Japan and the Developmental Biology Institute of Marseille, France, and Advisory Board Chair of Disease Models and Mechanisms.

Azim Surani
is an Affiliate Member of the Wellcome-MRC Cambridge Stem Cell Institute, a Member of the Royal Society’s Hooke Committee and of the Steering Committee of the UK Stem Cell Bank. He is a Member of the Scientific Advisory Board of the Institute for Stem Cell Biology and Regenerative Medicine, Bangalore, India and an Advisory Group Member of the Cambridge-India Partnership. He is the Founder of CellCentric.

Ben Simons
is a Principal Investigator at the Wellcome-MRC Cambridge Stem Cell Institute.

EDITORIAL BOARDS OF JOURNALS


Andrea Brand: Fly; Neural Development.

Meritxell Huch: Cogent Biology.

Steve Jackson: Biomolecules; Cell Stress; DNA Repair; Genes and Development; The EMBO Journal; The Scientist.

Rick Livesey: BMC Developmental Biology; Molecular Autism.


Ben Simons: Development.

Daniel St Johnston: Development; Faculty of 1000.

Azim Surani: BMC Epigenetics and Chromatin; Cell; Cell Discovery; Cell Research; Cell Stem Cell; Differentiation; Epigenetics; Epigenomics; Faculty of 1000; Nature Communications; Regenerative Medicine; Stem Cell Research and Therapy.
Public engagement, events, and publications in 2017
Public engagement with research (PER) involves exchanges about science and health research between researchers and the public. The Gurdon Institute aims to inspire young people, enable society to value and have confidence in fundamental research and embed PER in its research culture. The public engagement team leads and supports a variety of projects and events, working with researchers, students, children, teachers, artists and community members, and collaborating with other institutions.

ACTIVITIES IN 2017

Members of our staff, from the Core team to group leaders, racked up over 700 hours of project development and interactions with the public in 2017, reaching a variety of audiences numbering dozens up to many hundreds of people. These were some of the activities:

- **Gurdon Institute Public Engagement with Research award**
  - This award was first presented in 2017 to postdoc Weronika Fic at the Institute Retreat by our Director, Daniel St Johnston, for her strong commitment to our PER programme.

- **Gurdon Institute Public Engagement with Research seed fund**
  - In 2017, we created a PER seed fund (up to £1000 to be awarded annually) as an opportunity for our scientists to generate new research-focused PER projects. The public engagement committee selected a photographic proposal from Gianmarco Mastrogiovanni (Huch lab) and Timo Kohler, entitled ‘The Humane Scientist’. This project will be developed during 2018.

- **‘Test Tubes and YouTube’ panel event**
  - In a film theatre at Anglia Ruskin University we gathered a panel of film makers and scientists involved in our own videos and in other projects to explore, with a public audience, the use of scientific content in films created for different platforms. The discussion continued over an informal drinks reception.

- **‘Hands’ on at the Cambridge Science Festival**
  - One of our most popular on-site events featured the build-your-own ZoomBox microscopes, which visitors could use with their mobile phones to analyse prepared slides and then take home to continue using in their own time. A variety of other microscopes were available for use, to stimulate discussion about our research projects in lung development, the cytoskeleton, and cancer.

- **Green Man Festival, Brecon Beacons, Wales**
  - We went to the Green Man Festival with our “One body; trillions of cells” activity to reach festival-goers in ‘Einstein’s Garden’, the area dedicated to science. The public explored the fantastic process of developmental biology by talking with our scientists about what is known about stem cells, cell division and cell differentiation. They created a cell of their choice in coloured felt to help generate the different tissues of a human body on our giant banner.

- **‘Mini-me: how 3D organoids are revolutionising research’ panel discussion**
  - Collaborating with the Wellcome-MRC Cambridge Stem Cell Institute, the Babraham Institute and the Wellcome Sanger Institute, we invited the public to submit questions to and join the debate with a panel of stem cell and organoid researchers, including Emma Rawlins. Speakers and lab members stayed on to continue discussion over drinks afterwards.

- **Big Biology Day at Hills Road Sixth Form College**
  - The ZoomBoxes were in play again at this very busy one-day event for all ages, especially popular with local school students exploring potential topic choices for exams. The event is part of National Biology Week and included a biology careers fair, so our scientists talked with visitors about their careers as well as their research.

- **Mobile lab**
  - We took six scientists and microscopes for up to 30 pupils per two-hour session into several classrooms around the city. Working in small groups, the children learnt what a cell is and how to use microscopes to observe ready-prepared samples. Then they made slides with their own cheek cells for observation. State schools in under-served areas were targeted.

- **Cambridge Academy for Science and Technology**
  - Our microscopy experts created a workshop based on the use of Drosophila as a model organism, as part of the '13-week challenge' for Year 10 students. Talks, technical advice and demonstrations of equipment and image analysis software inspired some of the pupils to return to the Institute to take part in our work experience programme.
The Gurdon Institute Seminar Series brings high-profile international scientists before an audience drawn from across University biological science departments. The speakers we hosted in 2017 are listed below.

24 January
Sharad Ramanathan, Harvard University, USA
Measuring and Modeling the Dynamics of Developmental Decisions in Single Cells

28 February
Irene Miguel-Aliaiga, MRC London Institute of Medical Sciences
Sex differences in organ size and plasticity

10 April
Ross Cagan, Icahn School of Medicine at Mount Sinai, New York, USA
Approach to Cancer Therapies

25 April
James Sharpe, Centro Genomico Regulation, Barcelona, Spain
Multi-scale models of organogenesis: Limb bud development

9 May
Steven McKnight, University of Texas Southwestern Medical Center, USA
A solid state conceptualization of information transfer from gene to message to protein

25 August
Stuart J Forbes, MRC Centre for Regenerative Medicine, University of Edinburgh
Liver Regeneration in the Damaged Liver

26 September
Ruth Lehmann, Skirball Institute, NYU School of Medicine, New York, USA
Protecting Immortality: Germ line Development in Drosophila

10 October 2017 (The Anne McLaren Lecture)
Asifa Akhtar, Max Planck Institute of Immunobiology & Epigenetics, Freiburg, Germany
Epigenetic regulation by histone acetylation

7 November
Geraldine Seydoux, Johns Hopkins University School of Medicine, Baltimore, USA
Are RNA granules liquid organsises? Regulation of P granule dynamics by intrinsically-disordered proteins

27 November
Ron Vale, University of California, San Francisco, USA
RNA Aggregation in Neurdegenerative Disease

“[The Institute] is a great scientific environment and I was very impressed with your students!”

28 November
Sophie Jarriault, Institute of Genetics and Molecular and Cell Biology, Illkirch, France
Robustness and dynamics of natural direct reprogramming

5 December
Pierre Leopold, Institute of Biology Valrose, Nice, France
Growth coordination in Drosophila

Gurdon Institute members organise, co-organise or host several other series of seminars for the local academic community: Developmental Biology Seminars, Cambridge RNA Club, Cambridge Fly Meeting, Cambridge Epigenetics Club, Cambridge 3Rs (repliation, repair and recombination), Postdoc Masterclasses and Worm Club.

We have a long-established Postdoc Association and a newer PhD Student Association, regular social and sports activities, and plenty of special celebrations to bring all staff together. Below is a selection from 2017.

24 March: Special Happy Hour to mark the departure of the Piddini lab to the University of Bristol in June.

15 & 16 June: The Gurdon Institute PhD Student Association began life in 2017 and by June had already organised their first Retreat for 30 students in the Peak District. (see photo on back cover). “Very relaxing and scientifically exciting!” sums up the participant feedback.

6 July: Institute Summer BBQ in the garden.

13 July: The Gurdon Institute Postdoc Association (GIPA) held their retreat at Hughes Hall with the theme ‘Building resilience in research’. Also in 2017 GIPA ran a Career Path Seminar and a Chalk Talk Club along with a special ‘Bench to Business’ workshop, and hosted Magdalena Götze from Helmholtz Zentrum München to speak at a GIPA seminar.

17 September: The Livesey lab fielded the ‘Gurdon Gazelles’ team to run in the local Chariots of Fire race, raising money for Alzheimer’s Research UK.

28 & 29 September: The 2017 Institute Retreat for scientific staff was held at the East Midlands Conference Centre, University of Nottingham. Guest speaker was Tony Green, Director of the Welcome-MRC Cambridge Stem Cell Institute. Talks from group leaders were followed by the ‘Treasure Hunt’ and a dinner and disco. Prizes were announced for best poster (Lucia Cordero Espinoza, Huch lab), best image (Ravi Solanki, Livesey lab), best video (Federica Marinoro, Livesey lab), Public Engagement Champion (Weronika Fic, St Johnston lab) and Martin Evans Award for the greatest contribution to Institute social life (teams led by Navin Ramakrishna, Fabian Braukmann and Grégoire Vernaz, Miska lab).

31 October: Kathy Hilton, our Facilities Manager and employee of 24 years (who as Energy Champion spearheaded our saving of over 5 Million kWh of electricity) retired in September 2017; while Ann Cartwright, our Institute Administrator for 17 years, retired at the end of October. A joint leaving party with a Halloween theme gave them both a big send-off. Two new awards were announced at the event: the Kathy Hilton Energy and Environment Champion is Clive Bennett (Combined Building and Services Group), and the Ann Cartwright Good Citizen is Jackie Hall (St Johnston lab).

19 November: The Institute’s Basketball team finished the term undefeated in the College League Division 1.

12 and 14 December: The children’s and adults’ Christmas parties.
Publications


Turco MY, Gardner L, Hughes J, Cindrova-Davies S, Edmondson J, Loades; p70, Claudia O'Brien with Miranda Landgraf, Suzanne S, Loades; (3) Hélène Doerflinger; p71 (2), Chris Green. Others: p02, p04 (1,2 and 4), Peter Williamson; p06 (1), Wellcome Images; p64, Benjamin McMahan; p68 (1), Peter Williamson; (2), David Powell; p69 (1), brandAnonymous; (2), Chris Loades; (3) Helen Fiddler; p06 (2), Claudia Stocker; p71 (2), Chris Green.
