The Mathematics of Biology and Medicine

School of Mathematics - University of Leeds

Monday 30th September 2019

1 Schedule

TIME	SCHEDULED ACTIVITY
9:30 – 9:45	Registration and welcome
9:45 – 9:50	Opening remarks
9:50 – 10:10	**Godwin Akpeimeh** *Modelling infection risk after exposure to airborne pathogens*
10:10 – 10:30	**Alysha Taylor** *A comparative genomics approach to identify genomic elements implicated in the evolution of placental mammals*
10:30 – 10:50	**Alejandro Frangi** *Large-scale precision imaging: from imaging phenomics to in silico trials*
10:50 – 11:20	Coffee break
11:20 – 12:20	Transferable skills activity
12:20 – 12:50	**Paolo Actis – Keynote speaker** *Injecting single molecules into living cells*
12:50 – 14:00	Lunch
14:00 – 14:20	**Rob Welch** *BioRod and Heretical Abstractions in Mesoscale Modelling*
14:20 – 14:40	**Derek Mitchell** *Thermofluid models for the extended phenotype of the honey bee*
14:40 – 15:10	**Stephan Wilmes – Keynote speaker** *Mapping the Interleukin-27 signalosome – from cell surface receptors to T-cell differentiation*
15:10 – 15:40	Coffee break
15:40 – 16:00	**Ben Hanson** *Hierarchical Biomechanics: Applications to Protein Hydrogels*
16:00 – 16:30	**Catherine Noakes – Keynote speaker** *Modelling airborne infection in hospitals*
16:30 – 16:40	Closing remarks
16:40 – 18:00	Wine reception and poster session

2 Keynote speaker abstracts

Keynote talk: Dr. Paolo Actis
School of Electronic and Electrical Engineering, University of Leeds (UK)
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Injecting single molecules into living cells

Our beauty is in our differences. This is true down to the level of individual molecules that make up our body. I will present research work carried out in collaboration with biologists and physicians to understand the deepest difference between healthy cells and diseased cells.
I will discuss the combination of nanoscale needles with robotic manipulators to deliver biomolecules in and around living cells. I will then discuss the development of mathematical models to precisely evaluate how many molecules are being delivered and show why this is relevant to the pharmaceutical industry. I will end my talk by presenting some of our very recent work enabling the injection of a single molecule inside a living cell.

Keynote talk: Dr. Stephan Wilmes

School of Life Sciences, University of Dundee (UK)

Stephan Wilmes[1], Maximillian Hafer[2], Jonathan Martinez-Fabregas[1], Elizabeth Pohler[1], Paul Fyfe[1], Claire Gorby[1], Jacob Piehler[2] and Ignacio Moraga-Gonzalez[1]

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Mapping the Interleukin-27 signalosome – from cell surface receptors to T-cell differentiation

Cytokines are key regulators of innate and adaptive immunity as well as haematopoiesis. Binding of these soluble messenger proteins to tissue-specific cell surface receptors activates signalling cascades which lead to regulation of target gene expression patterns. A hallmark of cytokine biology is signalling plasticity - the ability to elicit differential cellular responses through the same cell surface receptor. The molecular mechanisms regulating signalling plasticity have so far remained unclear and thus beneficial cytokine bioactivities are frequently accompanied with undesired off-target effects. The lack of understanding how specific cellular responses can be selectively targeted is the main reason why cytokines have found limited access to use in clinical therapy.
The IL-27 system is an excellent model system to study and manipulate functional selectivity of cytokines: the IL-27 receptor (IL-27Rα) is found on all cells of the immune system. IL-27 is a heterodimeric cytokine comprised of the subunits p28 and Ebi3 and is secreted by Dendritic cells, Monocytes and Macrophages. After ligand binding on target cells, IL27Rα will form a heterodimeric receptor complex with gp130, a co-receptor frequently shared in the IL-6/IL-12 receptor superfamily. Receptor assembly leads to activation of the intracellularly associated Janus Kinases (JAK1/JAK2), followed by phosphorylation and activation of members of the Signal Transducer and Activator of Transcription family (STAT1/STAT3). Although many members of the IL-6/IL-12 superfamily share the same set of receptors and JAK/STAT effectors, their downstream effects can even be opposing: while IL-27 is considered be a key regulator for anti-inflammatory effects, IL-6 is a paradigm cytokine for pro-inflammatory responses. So, the differential outcome of these responses must be encoded in the early events of signal activation.
We specifically aim to identify the molecular determinants underlying functional selectivity of IL-27 signalling on T cells: how does a certain cytokine stimulus ultimately lead to cellular decisions? Therefore, we study IL-27 signalling covering multiple layers of signal propagation. We probe diffusion and interaction dynamics of IL-27Rα and gp130 at the cell surface of live cells by dual-colour single-molecule fluorescence imaging. In isolated human T-cells (CD4+, CD8+, naïve vs. activated), we follow the phosphorylation kinetics of key effector molecules and compare it with other members of the IL-6/IL-12 superfamily by high-throughput phospho-flow cytometry. As a readout for long-term effects, we compare IL-27 with IL-6 in their ability to differentiate naïve CD4+ T-cells into specific subsets (e.g. Treg vs. TH17). Finally, we map cytokine-induced changes of the proteome and phospho-proteome by mass spectrometry.
I am convinced that a mechanistic understanding of the cytokine signalosome in the immune system will provide us with critical insights into the large functional pleiotropy exhibited by this family of ligands, which so far represents a major caveat in the implementation of cytokine-based therapies into the clinic.

Keynote talk: Prof Catherine Noakes
School of Civil Engineering, University of Leeds (UK)

Modelling airborne infection in hospitals

Airborne transmission is an infection route for many diseases including communicable infections such as TB and influenza, as well as opportunist pathogens in hospitals. Quantifying risks are necessary to determine appropriate control strategies, both in terms of engineering approaches such as ventilation and management strategies such as locating and scheduling patients. However airborne transmission is complex to evaluate as it requires understanding of the airflows, infection dynamics and human-environment interactions.
This presentation considers how modelling approaches can be applied to assess the influence of airflows on the risk of exposure to infectious microorganisms. Examples from several studies including assessment of naturally ventilated environments and air disinfection technologies are used to discuss how ventilation and health aspects can be quantified and to consider approaches for evaluating the trade-off between risks and energy performance.

3 Contributed talk abstracts

Godwin Akpeimeh - Civil Engineering

Modelling infection risk after exposure to airborne pathogens

Quantitative microbial risk assessment model (QMRA) is a mathematical model used to describe the relationship between the host response and pathogen dose. The dose-responds assessment phase in the QMRA model is the quantitative yardstick for estimating infection risk in the model. In previous studies, most often the average exposure dose were used at the phase to estimate the workers risk of respiratory diseases from exposure to bioaerosols. However, in reality, when bioaerosols considered infectious are inhaled, they are transported to specific regions of the lungs (alveoli) and would have been deposited for an infection to take place (Weir and Haas 2011). Thus, the average exposed dose does not account for the required particle transport through and infection in the respiratory system.
A stochastic (Markov Chain) model was used to model both the transports of the inhaled dose though the human respiratory system whilst accounting for the losses in the process (Figure 1). The model was integrated into the beta Poisson dose response model to estimate workers risks of respiratory and gastrointestinal (GI) infection from exposure to Aspergillus fumigatus and E. coli O157:H7 respectively, especially the immunocompromised. The exposure data was used have been published in previous studies on workers exposure to bioaerosols at open dumpsites during scavenging, waste soring and dumpsite monitoring (Akpeimeh et al., 2019). The result was computed based on initial exposure concentration of E.coli O157:H7 at 10-50% pathogen ingestion rate and Pathogen: Indicator (P:I) of 1:103 and 1:104 , while Aspergillus fumigatus was based solely on the average inhaled concentration.
The results showed that after 11 hrs of workers exposure, workers engaged in scavenging, waste monitoring and site monitoring were at risk of GI infection in the magnitude of 10-1 for P:I = 1:103 and 1: 104. Respiratory infection from Aspergillus fumigatus also showed similar magnitude (10-1). However, GI infection risk estimate associated with specific areas of the dumpsite showed that after 11 hours of exposure (Figure 2), workers at the active area were range of 3.23×10^{-3} - 1.56×10^{-2} and 3.25×10^{-4} - 1.62×10^{-3}, Dormant area: 2.06×10^{-3} - 1.01×10^{-2} and 2.09×10^{-4} - 1.04×10^{-3}, Entrance 1.85×10^{-3} - 9.09×10^{-3} and 1.87×10^{-4} - 9.27×10^{-4} boundary 1.82×10^{-3} - 8.82×10^{-3} and 2.09×10^{-4} - 8.94×10^{-4} for P:I = 1:103 and 1: 104 respectively. Moreover, respiratory infection risk from were Aspergillus fumigatus in the magnitude of 10-1 all sampling locations.
The estimated risk of GI infection and respiratory infections from workers exposure to E. coli O157:H7 and Aspergillus fumigatus especially the immunocompromised workers at Olusosun open dumpsite were high for all activities on the dumpsite. Moreover, due to the possibility of bioaerosols disper-

sion downwind, the residents living near the dumpsite could also be at risk of infection, especially the immunocompromised individuals.

Alysha Taylor - Biological Sciences

A comparative genomics approach to identify genomic elements implicated in the evolution of placental mammals

Alysha S. Taylor[1,2], Thomas A. Walsh[3], Bede Constantinides[1], Haidee Tinning[2], Laura Hume[2], Niamh Forde[2], Mary J. O'Connell[1,4]
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3 Bioinformatics and Molecular Evolution Group, School of Biotechnology, Dublin City University, Glasnevin, Dublin 9, Ireland.
4 Computational and Molecular Evolutionary Biology Group, School of Life Science, University of Nottingham, NG7 2RD, UK.

Placenta emerged on the stem Eutherian mammal lineage 93 million years ago and defines the Eutherian clade. While placentation has arisen multiple times in Animalia, the evolution of mammal placenta is a unique and unreversed event, where a novel organ developed. Unlike other organs, the mammal placenta varies in morphology, influenced by the life history and reproductive methods of the organism. Due to the highly dynamic nature of its development, the evolution of placental tissue was likely facilitated by changes to both protein coding and regulatory regions of the genome. Considering the complex changes in gene expression during the development of mammal placenta, miRNAs are key candidates for investigation as key regulators. We wished to determine if there were particular protein coding gene families that underwent positive selection on the lineage leading to placental mammals and nowhere else in vertebrates. To complement this, we also wished to determine if there were specific miRNAs that emerged on this stem lineage and were never subsequently lost.
Homologous gene families were constructed with representatives from across the vertebrate tree (10 placental mammal species, 2 non-placental mammals – 1 monotreme and 1 marsupial, and 2 non-mammalian vertebrates – 1 bird and 1 fish). Using the VESPA selective pressure pipeline and the CodeML analysis program, 1704 single gene orthologous gene families were investigated for evidence of selective pressure on the lineage leading to placental mammals. Simultaneously, using literature and database searches, we assembled a set of 232 microRNAs that have (i) a putative role in placental function, or (ii) are annotated as specific to placental mammals. Large-scale sequence similarity searches of 20 high quality vertebrate genomes (10 placental mammals, 2 non-placental mammals – 1 monotreme and 1 marsupial, and 8 non-mammalian vertebrates (including birds, reptiles, amphibians and fish) were used to identify the miRNAs specific to the Eutherian and Therian clades. A presence-absence matrix was constructed for these microRNAs and a gain-loss analysis was performed in TNT (Tree analysis in New Technology). Upon confirming the phylogenetic placement of these miRNAs, TargetScan7.0 was used to predict target transcripts.
237 single gene orthologous families were found to have undergone positive selection on the lineage leading to placental mammals. Of these 237 positively selected families, 115 are conserved throughout Placentalia. Of the 232 miRNAs analysed, 112 were found to be specific to placental mammals. 12 of these miRNAs arose on the stem eutherian lineage and were never subsequently lost, with 2 further miRNAs emerging on the stem metatherian lineage and were never subsequently lost. Upon investigating the predicted targets of these miRNAs, 81 of the 115 conserved positively selected gene families were found to be regulated by miRNAs that emerged on the Metatherian and Eutherian lineages.

To further investigate these miRNAs, miRNA arrays were used to test the expression of these 14 stem lineage of miRNAs in uterine tissue from opossum, mouse, cow and pig, confirming their phylogenetic placement. Further miRNA arrays showed an upregulation of a stem lineage miRNA (miR-28) in response to the bovine pregnancy recognition signal (IFNT), along with differential expression of 3 stem lineage miRNAs (miR-340, miR-542 and miR-671) in response to progesterone.
Further investigations will be performed in bovine and human cell lines to observe the expression patterns of these stem lineage miRNAs when treated with early pregnancy recognition signals. In addition, the expression of these stem lineage miRNAs will be investigated in healthy and disease state placental explants. Finally, positively selected genes targeted by the stem lineage miRNAs will be filtered for candidates to manipulate expression on the bench. We present our findings thus far on the original, evolution and function of genomic elements in mammal placenta.

Prof Alejandro Franji - Computing

Large-scale precision imaging: from imaging phenomics to in silico trials

This talk will overview current progress in imaging phenomics and in silico clinical trials that make use of precision imaging, i.e. the use of advanced image-based analysis and modelling rendering more descriptive, integrative and predictive imaging. We illustrate how the convergence of computational image analytics and modelling methods that process large health databases open new challenges. I will introduce various examples from his research lab spanning cerebrovascular, cardiovascular and bone diseases.

Rob Welch - Physics

BioRod and Heretical Abstractions in Mesoscale Modelling

As the quality and quantity of imaging from modalities such as Cryo-EM increases, we need new ways to represent proteins in order to perform simulations at larger time and length scales, from depolymerising microtubules to the movement of motor proteins such as cytoplasmic dynein.
BioRod is one such algorithm, designed to perform dynamical simulations of elongated biological structures such as alpha-helices and coiled-coils, represented as coarse elastic curves that can stretch, bend and twist independently. The curves can have anisotropic and inhomogeneous parameters and arbitrary equilibrium structures, allowing for the highly abstract parameterisation of a wide range of molecules. The speed of the algorithm allows BioRods to access timescales from nanoseconds to seconds with large, complex structures, many millions of atoms in size.
A BioRod was parameterised using data from all-atom simulations of the NDC80c protein complex, and compared against these simulations and negative-stain EM images. Bend angle distributions and principal components were used to validate the BioRod simulation.

Derek Mitchell - Mechanical Engineering

Thermofluid models for the extended phenotype of the honey bee

Honey bees, Apis mellifera, use their nest for the energy intensive processes of: desiccating a low sugar concentration liquid - nectar to a high sugar content liquid – honey (sugar refining), a non-hibernating winter refuge against temperatures as low as -40 C, and close climate regulation of both temperature and humidity of a nursery. They spend over 80% of their lifetime within the nest, and their very complex behaviours in finding, selecting and modifying it are well known, as well as those of heating, cooling and ventilating it, but to what end is all this behaviour exerted? How can metrics

be defined for the success or otherwise of the processes that utilise this part of the super-organism that lies outside of their biological tissues, the extended phenotype? This work addresses the problem with same thermofluid analysis that is applied to an bioenergy sugar refinery with integrated feed stock transportation, evaporation, and active climate control, to determine how the thermofluid characteristics of the honey bee nest bound the energetics of the colony. It discusses the analyses of key parameters: Lumped thermal conductance, thermal efficiency, dew point, condensation rates and forced convection of water vapour, in relation to: colony metabolic rates and metabolites; nectar evaporation, advection and foraging behaviours. It then shows how these parameters determine boundaries for survivable winter temperatures, the maximum range of foraging, viability of nectar sources, honey production rates, and the survival or suppression of varroa parasites. These also provide insight on how the conflicting humidity and temperature requirements for successful reproduction and honey are resolved. By these parameters providing easily accessible metrics for the fundamental differences between man-made and natural nests, they become the criteria for design for improved performance of man-made hives and bee keeping practice in the face of adverse climates and climate change.

Dr. Ben Hanson - Physics

Hierarchical Biomechanics: Applications to Protein Hydrogels

In order to meet the demands of modern medicine, recent years have seen a push towards the rational design of composite biological systems. In the field of hydrogels, and more specifically protein hydrogels, an important factor in the design process is an understanding of how the mechanical properties of the resulting gels depend on the underlying structural components. However, much of the current work in this area is largely qualitative[1][2] whereas for a specific rationally designed system, a more quantitative approach is required.
In this talk I'll show how our group is using theoretical techniques to understand the formation process and resulting structures of chemically cross-linked protein hydrogels. Beginning with a simulation study on globular polyproteins, we show that the persistence length emerges as a function of the geometric properties of the linker domain and protein subunits. By subsequently mimicking the conditions of atomic force microscopy experiments, we show that the persistence length is not an intrinsic property of a polyprotein, but instead is highly dependent upon the imposed boundary conditions.
With this knowledge, I'll present our theoretical methodology for simulating how chemically cross-linked hydrogels form from either single proteins or polyproteins. To characterise these systems, we consider the topological and geometric differences between the two types of protein network and consider how these factors, together with the type of subunit, alter the resulting mechanical properties of the gel. Finally, I'll discuss the future directions of this work, how it complements and is complemented by collaborative experimental work within our group, and the eventual practical applications enabled by this combination.

1. Da Silva, M.A., Lenton, S., Hughes, M., Brockwell, D.J. and Dougan, L,. Biomacromolecules 18 (2), 636-646, 2017
2. Wu, J., Li, P., Dong, C., Jiang, H., Xue, B., Gao, X., Qin, M., Wang, W., Chen, B. and Cao, Y., Nature communications 9(1), 620, 2018

4 Poster abstracts

Haidee Tinning - Medicine

Conceptus-derived proteins, CAPG & P4HB, alter the transcriptome of bovine endometrial cells cultured in vitro to enhance the pregnancy recognition process

Successful early pregnancy in cattle is dependent on the production of sufficient quantities of Interferon Tau (IFNT: the pregnancy recognition signal) by the conceptus to induce successful maternal recognition of pregnancy (MRP). However, the presence of the conceptus alters additional transcripts in the endometrium in addition to those that change in response to IFNT alone. We have shown previously that the bovine conceptus secretes additional proteins coordinate with IFNT production including macrophage capping protein (CAPG) and protein disulphide isomerase (P4HB). We tested the hypothesis that these proteins may act alone or in synergy with IFNT to alter the endometrial transcriptome to facilitate MRP and successful early pregnancy in cattle. Recombinant bovine CAPG and P4HB were produced in E.coli and purified using immobilised metal affinity chromatography. Primary endometrial cells were obtained from abattoir-derived mid-to-late luteal phase bovine uteri (n=3) by enzymatic digestion and purification. Stromal and epithelial-enriched cells were cultured for 24 hours with the following treatments: 1) Control, 2) Vehicle control, 3) IFNT 4) P4HB, 5) CAPG, 6) IFNT+CAPG, and 7) IFNT+P4HB. RNA sequencing was performed to determine transcriptional changes induced by CAPG, P4HB +/- IFNT. Treatment of epithelial cells with IFNT, CAPG or P4HB alone altered 2941, 1020 and 1016 transcripts respectively. Interestingly, 728 transcripts were altered in all three treatment groups, while 32 and 34 differentially expressed genes (DEGs) were detected in CAPG and P4HB treated cells respectively. CAPG & P4HB altered the expression of 131 DEGs that were not altered by IFNT. Comparison of IFNT treatment alone with IFNT plus CAPG or P4HB resulted in 131 and 88 DEGs respectively. Collectively these data indicate that the conceptus-derived proteins P4HB & CAPG have a functional role in enhancing MRP alone and in combination with IFNT.

Chi-Chuan Lin - Cellular and molecular biology

Monomeric Grb2 enhances Shp2 activity

Understanding the molecular basis for the regulation of oncoproteins is key to developing new cancer treatments. Shp2 is a ubiquitously expressed protein tyrosine phosphatase. In the absence of stimuli, Shp2 exists in an autoinhibited conformation which prevents its activation; dysregulated activation of Shp2 has been associated with many cancer types. Our early study demonstrated that the adaptor protein Grb2 exists in a monomer (mGrb2)-dimer (dGrb2) equilibrium and the monomeric form is signalling competent and has been connected to several human cancers. Here we unravelled the molecular basis for the phosphorylation-independent Shp2-mGrb2 interaction and elucidated the effect of mGrb2-binding on Shp2 phosphatase activity in the absence of stimuli. Mechanistically, the Shp2–mGrb2 interaction is mediated via 1) Shp2 nSH2 and mGrb2 SH2 domains, 2) Shp2 PTP and mGrb2 cSH3 domains. Notably, our in vitro study shows that the mGrb2-bound Shp2 exhibits enhanced phosphatase activity independent of Shp2 phosphorylation. This is due to a conformational change upon binding to mGrb2 which leads to the release of Shp2 from its autoinhibited state. Moreover, the mGrb2-bound Shp2 is the preferred substrate to receptor tyrosine kinases (RTKs) in vitro, which further enhances Shp2 activity. Cell-based study also shows knockdown of endogenous Grb2 results in an increase in the phosphorylation level of tyrosine 542 on Shp2, which serves as an activation marker. In addition, we developed a peptide that blocks the mGrb2–Shp2 interaction and down-regulates Shp2 activity both in vitro and in cancer cell lines. Thus, this noncanonical mGrb2–Shp2 interaction represents a mechanism that upregulates Shp2 signal pathways in oncogenesis and our studies suggest a new strategy for targeting Shp2-mediated cancers.

Sijia Li – Mathematics

Jig of Life: An Uncertainty Analysis of Bayesian Network for sc-RNA seq

Single cell RNA sequencing (sc-RNA seq) technique provides us the opportunity to drive insights from data. One of the sc-RNA seq data's application is highly variable gene identification, which link to the exploration of target gene and Embryogenesis. To extract the information of interest, we need to evaluate the influence from different factors to experiment count data. Bayesian Networks are ideal in estimate the contributing factors. Bayesian Networks requires the specification of many input parameters, and outputs highly variable genes to compare with biological results. We investigate the uncertainty in Bayesian Networks for sc-RNA seq data analysis and explore to improve its robustness and accuracy.

Maxx Holmes - Biological Sciences

Sub-Cellular Heterogeneity Determines Spatial Calcium Dynamics in Cardiomyocytes

The intracellular Ca2+ system of cardiomyocytes is responsible for coupling electrical and mechanical function. Regulation of Ca2+ fluxes into and out of the bulk cytoplasm and sarcoplasmic reticulum (SR) is critical to ensure stable cardiac output to meet the body's dynamic physiological demands. The balance of the flux-carrying channels (for example, the SR Ca2+-ATPase (SERCA) and the Na2+/Ca2+ exchanger (NCX)) determines this homeostatic response, and the expression of these channels has been observed through experimental imaging studies to be spatially heterogeneous in the cytoplasm of single cells. However, this sub-cellular spatial non-uniformity has yet to be fully characterised nor its functional impact to be assessed.
Previously attained confocal microscopy data on these targets was re-analysed to quantify spatial variation in channel expression. A variogram analysis was utilised to estimate variation length-scales and anisotropy to describe the cell's spatial non-uniformity (Figure 1A,B). Simulations using computational models of 3D spatial intracellular Ca2+ handling were performed to investigate the effects of sub-cellular heterogeneity and anisotropy on Ca2+ transients (CaTs). Cell geometries (Figure 1C) were created using Gaussian Random Fields (GRFs) with observed variation length-scales.
Analysis reveals estimated length-scales of 0.5–5 μm in control, and 2–11 μm in heart failure (HF) for RyR and SERCA (Figure 1A). Length-scales are generally anisotropic, with greater intercellular variability in length-scale than in relative expression. Simulations reveal that RyR and SERCA heterogeneity results in more spatially disordered CaTs in both upstroke and decay phase, compared to the homogeneous model, while NCX heterogeneity primarily affected the decay phase (Figure 1D). Correlation between these targets was shown to potentially enhance or inhibit local regions of large heterogeneity.
Our results indicate that sub-cellular heterogeneity of the primary Ca2+ flux channels can underlie spatially non-uniform CaTs and even account for inter-cellular variability of CaT properties under maintained homogeneous total channel expression; larger length-scales, as observed in HF, in general led to more variable sub-cellular and cell-average dynamics. Sub-cellular heterogeneity may therefore be an important factor which underlies the changes in cardiac Ca2+ handling associated with conditions such as HF.

5 Acknowledgements

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- Wellcome Trust funded PhD studentship at Leeds

We also thank our three Keynote speakers, six speakers selected from abstract submission and four poster presenters.

We are grateful for the support from our PhD supervisors in organising this event.

Finally, we thank everyone who has attended this conference and we hope it has been an exciting day of science!

Organisers:
Amy Stainthorp (SCMB) and Polly-Anne Jeffrey (Mathematics)