EdMathBio2023 Conference

May 24-25, 2023

Room G.03, Bayes Centre

THE UNIVERSITY of EDINBURGH

EDINBURGH MATHEMATICAL SOCIETY

LONDON MATHEMATICAL SOCIETY EST. 1865

MAXWELL INSTITUTE FOR MATHEMATICAL SCIENCES

Edinburgh Mathematical Biology Conference 2023

Book of Abstracts

Contents

1 Programme

Wednesday, May 24th

9:00-9:45	Registration & Breakfast
9:45-10:00	Opening Ceremony
10:00-11:00	**Plenary talk by Professor Mark Chaplain**
11:00-11:45	**Talk by Dr. Fiona Ruth Macfarlane**
11:45-12:30	**Talk by Dr. Lyndsay Kerr**
12:30-14:00	Lunch Break & Coffee
14:00-14:45	**Talk by Dr. Maximilian Engel**
14:45-15:30	**Talk by Professor Anne Skeldon**
15:30-16:00	Coffee Break
16:00-16:45	**Talk Professor Philip Gerlee**
16:45-17:30	**Talk by Dr. Svitlana Braichenko**
17:30-19:30	Poster Session & Wine Reception

Thursday, May 25th

9:15-10:00	Breakfast
10:00-11:00	**Plenary Talk by Professor Pavol Bokes**
11:00-11:45	**Talk by Dr. Jochen Kursawe**
11:45-12:30	**Talk by Dr Noemi Picco**
12:30-14:00	Lunch Break & Coffee
14:00-14:45	**Talk by Dr. Duncan Sproul**
14:45-15:30	**Talk by Dr. Lucy Martin**
15:30-16:00	Coffee Break
16:00-16:45	**Talk by Professor Sebastian Wieczorek**
16:45-17:30	**Talk by Professor Jonathan Sherratt**

2 Plenary Talks

Talk by **Professor Mark Chaplain**

The University of St. Andrews

"Mathematical Modelling of Cancer Invasion and Metastatic Spread"

We present computational simulations results from a three-dimensional genuinely hybrid atomistic- continuum model that describes the invasive growth dynamics of individual cancer cells in tissue. The framework explicitly accounts for phenotypic variation by distinguishing between cancer cells of an epithelial-like and a mesenchymal-like phenotype. It also describes mutations between these cell phenotypes in the form of epithelial-mesenchymal transition and its reverse process mesenchymal-epithelial transition. The proposed model consists of a hybrid system of partial and stochastic differential equations that describe the evolution of epithelial-like and mesenchymal-like cancer cells, respectively, under the consideration of matrix-degrading enzyme concentrations and the extracellular matrix density.

Talk by **Professor Philip Gerlee**

Chalmers University

"Weak selection and time scale separation in ecological and evolutionary dynamics"

We show that under the assumption of weak frequency-dependent selection a wide class of population dynamical models can be analysed using perturbation theory. The inner solution corresponds to the ecological dynamics, where to zeroth order, the genotype frequencies remain constant. The outer solution provides the evolutionary dynamics and corresponds, to zeroth order, to a generalisation of the replicator equation. We apply this method to a model of public goods dynamics and construct, using matched asymptotic expansions, a composite solution valid for all times. We also analyse a Lotka-Volterra model of predator competition and show that to zeroth order the fraction of wild-type predators follows a replicator equation with a constant selection coefficient given by the predator death rate. For both models we investigate how the error between approximate solutions and the solution to the full model depend on the order of the approximation, and show using numerical comparison, for $k = 1$ and 2, that the error scales according to ε^{k+1}, where ε is the strength of selection and k is the order of the approximation.

Talk by **Professor Sebastian Wieczorek**

University College Cork

"Rate-induced tipping to Zombie fires: Crossing quasi-thresholds in multiple timescale systems"

Surface wildfires are generally believed to be the cause of so-called Zombie fires observed in peatlands, that disappear from the surface, smoulder underground during the winter, and "come back to life" in the spring. Here, we propose rate-induced tipping (R-tipping) to a subsurface hot metastable state in bioactive peat soils as a main cause of Zombie fires. Our hypothesis is based on a conceptual soil-carbon model subjected to realistic changes in weather and climate patterns, including global warming scenarios and summer heatwaves. Mathematically speaking, R-tipping to the hot metastable state is a nonautonomous instability, due to crossing an elusive quasithreshold, in a multiple timescale dynamical system. To explain this instability, we provide a framework that combines a special compactification technique with concepts from geometric singular perturbation theory. This framework allows us to reduce an R-tipping problem

due to crossing a quasithreshold to a heteroclinic orbit problem in a singular limit. Thus, we identify generic cases of such R-tipping via: (i) unfolding of a codimension-two heteroclinic folded saddle-node type-I singularity for global warming, and (ii) analysis of a codimension-one saddle-to-saddle hetroclinic orbit for summer heatwaves, which in turn reveal new types of excitability quasithresholds.

Talk by **Professor Pavol Bokes**

Comenius University in Bratislava

"Effects of large distributed delays on noisy genetic feedback circuits"

Noise in gene expression can be substantively affected by the presence of production delay. Here we consider a mathematical model with bursty production of protein, a one-step production delay (the passage of which activates the protein), and feedback in the frequency of bursts. We specifically focus on examining the steady-state behaviour of the model in the slow-activation (i.e. large-delay) regime. Using a formal asymptotic approach, we derive an autonomous ordinary differential equation for the inactive protein that applies in the slow-activation regime. If the differential equation is monostable, the steady-state distribution of the inactive (active) protein is approximated by a single Gaussian (Poisson) mode located at the globally stable fixed point of the differential equation. If the differential equation is bistable (due to cooperative positive feedback), the steady-state distribution of the inactive (active) protein is approximated by a mixture of Gaussian (Poisson) modes located at the stable fixed points; the weights of the modes are determined from a WKB approximation to the stationary distribution. The asymptotic results are compared to numerical solutions of the chemical master equation. This is a joint work with Alessandro Borri, Pasquale Palumbo, and Abhyudai Singh.

Talk by **Professor Anne Skeldon**

The University of Surrey

"The fast-slow dynamics of sleep regulation"

The regulation of sleep and wake occurs through the interaction of specific neuronal populations in the brain with macroscale internal rhythms and external environmental cues, such as the light-dark cycle. Mathematical models of sleep-wake regulation have been very successful at providing a conceptual framework for understanding the timing and duration of sleep, with implications for public policy on design of our living environments, the timing of our clocks (e.g. daylight saving time versus standard time) and the timing of our work schedules. Not surprisingly, since we remain in the states of sleep or wake for some time, but switch between states rapidly, models typically have multiple time scales. Here I'll give a brief overview of the fast-slow dynamics of some recent models of sleep-wake regulation and the insights that understanding that structure brings.

Talk by **Dr. Maximilian Engel**

Freie Universität Berlin

"A geometric singular perturbation approach to the Brusselator"

The Brusselator is a chemical reaction network which has become a pototypical model for self-sustained chemical oscillations. Under suitable assumptions, this chemical system can be modelled by a system of ODEs with two parameters a and b. It is well known that the system exhibits a Hopf bifurcation at a critical value of b for given a, yielding a globally attracting limit cycle. For b sufficiently large in comparison to a, numerical explorations show that the system exhibits a time scale separation with mixed-mode oscillations. However, the limit cycle as a whole diverges to infinity when this time scale separation is understood via a singular limit. We analyse the multiscale system via a singular perturbation approach, compactifying the state space and identifying the critical cycle, using the geometric blow-up method to deal with its non-hyperbolic parts. We are then able to identify and quantify the slow and fast dynamics along the actual limit cycle, also demonstrating the relevance for a stochastic version of the Brusselator. This is joint work with Guillermo Olicon Mendez (also FU Berlin).

Talk by **Dr. Lucy Martin**

The University of Edinburgh

"Modelling the spread of senescence"

Cellular senescence is a cell surveillance mechanism that arrests the cell cycle in damaged cells. The senescent phenotype can spread from cell to cell through paracrine (secreted protein) and juxtacrine (cell contact) signalling, but the dynamics of this process are not well understood. Although senescent cells are important in ageing, wound healing, and cancer, it is unclear how the spread of senescence is contained in senescent lesions. In the absence of the immune system, senescence could theoretically spread infinitely from one cell to another, but this contradicts experimental evidence. To investigate this issue, we developed both a minimal mathematical model and a stochastic simulation of senescence spread. Our results suggest that differences in the number of signalling molecules secreted between subtypes of senescent cells can limit the spread of senescence. We found that dynamic, time-dependent paracrine signalling prevents the uncontrolled spread of senescence and we demonstrate how model parameters can be determined using Bayesian inference in a proposed experiment.

Talk by **Dr. Noemi Picco**

Swansea University

"Protective niches and the emergence of drug resistance in cancer"

Cancer drugs that target key mutations often follow a pattern of initial significant response, followed by relapse, indicating the emergence of resistance to the treatment. The evolutionary nature or cancer can explain this response where from within the heterogeneous population of tumour cells driving the tumour there is a population of cells that evolve resistance to the treatment drugs. In addition to this mechanism, the environment in which the tumour lives is known to modulate drug resistance, so called microenvironment-mediated drug resistance (EMDR). In this talk I will describe a mathematical framework that can capture the interactions between the tumour and the microenvironment in the presence of treatment drugs, across experimental, spatial, and temporal scales. The aim is to understand the contributions of the tumour microenvironment and tumour heterogeneity in regulating treatment response and the emergence of drug resistance. I will use a range of modelling approaches that can explicitly

capture the spatio-temporal dynamics of tumour growth, invasion and resistance observed clinically and experimentally. Preliminary results hint at the emergence of transient protective niches that facilitate EMDR, and offer insight into therapeutic strategies that can successfully control the emergence of drug resistance.

Talk by **Dr. Svitlana Braichenko**

The University of St. Andrews

"PoMo: Inferring Phylogenies and Disentangling Modes of Natural Selection"

The interplay between mutation, genetic drift, directional, and balancing selection in shaping populations' diversity is highly convoluted and difficult to disentangle. This requires sophisticated phylogenetic models that have a high degree of flexibility and can handle multi-individual data. For these purposes, our group has developed a polymorphism-aware phylogenetic set of models called PoMos. These models are based on the Moran model and have recently been proven effective in inferring species trees as well as mutational effects, fixation biases and GC-bias rates in great apes and grasshoppers. To make these models more accessible, we implemented them in the open-source Bayesian inference framework RevBayes. The advantage of the framework is its implementation in a graphical model environment and the possibility to compute coverage frequencies for the validation analysis. In this study, we further developed PoMos to study neutral, directional and, for the first time, balancing selection. The key advantage of our novel approach for studying the balancing selection is that PoMos allow for ancestral polymorphisms that can be maintained, and parameters that can measure frequency-dependent selection. We have tested our new method on a set of simulated data with a popular evolutionary framework Slim and a custom Moran model simulator implemented in RevBayes. Furthermore, we investigated real sequences of African human populations to understand the evolutionary history of genomic regions that are known to be under balancing selection driven by malarial parasites.

Talk by **Dr. Lyndsay Kerr**

The University of Edinburgh

"Cluster mean-field theory accurately predicts statistical properties of large-scale DNA methylation patterns"

Many diseases are associated with changes in the genome and epigenome. DNA methylation is an epigenetic mark and large-scale alterations in methylation patterns are seen in development, ageing and disease, including megabase-scale methylation loss in cancer. Previous mathematical modelling studies have aided the understanding of methylation patterns, but the computational expense associated with these studies have limited their use to the study of small-scale patterns. During this talk, I will discuss how to construct a mean-field model that can be used to quickly, and accurately, predict statistical properties associated with large-scale DNA methylation patterns. This model should be a valuable tool in future investigations into the mechanisms underlying the large-scale changes to methylation patterns observed in development and disease. The material discussed in this talk is joint work with Ramon Grima (University of Edinburgh, School of Biological Sciences) and Duncan Sproul (University of Edinburgh, Institute of Genetics and Cancer) and is based on the publication https://royalsocietypublishing.org/doi/10.1098/rsif.2021.0707.

Talk by **Dr. Jochen Kursawe**

The University of St. Andrews

"Emergence and consequences of transcription factor dynamics during development"

The dynamics of transcription factor concentrations influence cell state in many contexts. For example, oscillations of bHLH transcription factors have been shown to maintain the progenitor state during neurogenesis in multiple organisms. However, the mechanisms regulating such dynamics, as well as the mechanistic link between transcription factor dynamics and cell fate are often unclear. Here, we present theoretical tools that can help illuminate transcription factor dynamics. By comparing mathematical models to transcription factor time series data from single-cell live-imaging microscopy data in zebrafish and mouse models, we identify differences between cell populations by quantifying kinetic rates of transcription, translation, and degradation without further experiments. We further model how differences in transcription factor dynamics can lead to differential gene expression of down-stream targets, and thus changes in cell fate. We use our methods to explain observations in zebrafish hindbrain morphogenesis and mouse spinal cord development.

Talk by **Dr. Duncan Sproul**

The University of Edinburgh

"Genome-wide single-molecule analysis of long-read DNA methylation reveals heterogeneous patterns at heterochromatin"

High-throughput sequencing technology is central to our current understanding of the human methylome. The vast majority of studies use chemical conversion to analyse bulk-level patterns of DNA methylation across the genome from a population of cells. While this technology has been used to probe single-molecule methylation patterns, such analyses are limited to short reads of a few hundred basepairs. DNA methylation can also be directly detected using Nanopore sequencing which can generate reads measuring megabases in length. However, thus far these analyses have largely focused on bulk-level assessment of DNA methylation. Here, we analyse DNA methylation in single Nanopore reads with a mean length of 24.6kb, to show that bulk-level metrics underestimate large-scale heterogeneity in the methylome. We use the correlation in methylation state between neighbouring sites to quantify single-molecule heterogeneity and find that heterogeneity varies significantly across the human genome, with some regions having heterogeneous methylation patterns at the single-molecule level and others possessing more homogeneous methylation patterns. By comparing the genomic distribution of the correlation to epigenomic annotations, we find that the greatest heterogeneity in single-molecule patterns is observed within heterochromatic partially methylated domains (PMDs). In contrast, reads originating from euchromatic regions and gene bodies have more ordered DNA methylation patterns. By analysing the patterns of single molecules in more detail, we show the existence of a 185bp periodicity in DNA methylation that accounts for some of the heterogeneity we uncover in long single-molecule DNA methylation patterns. We find that this periodic structure is partially masked in bulk data in a manner that is consistent with imperfect phasing of nucleosomes between molecules. Our findings demonstrate the power of single-molecule analysis of long-read data to understand the structure of the human methylome.

Talk by **Dr. Fiona Ruth Macfarlane**

The University of St. Andrews

"Bridging the gap between individual-based and continuum models of growing cell populations"

Stochastic individual-based modelling approaches allow for the description of single cells in a biological system. These models generally include rules that each cell follows independently of other cells in the population and allow for heterogeneity of population to be considered. However, these models cannot be analysed mathematically. Therefore, it can be beneficial to derive the corresponding deterministic model from the underlying random walk of the stochastic model. The resulting deterministic models, usually partial differential equations (PDEs), can then be analysed to provide further information about the biological systems studied. We have developed a range of simple IB models that describe biological systems with various properties of interest, such as chemotaxis and pressure-dependent growth and proliferation. Ultimately, the results illustrate how the simple rules governing the dynamics of single cells in our individual-based model can lead to the emergence of complex spatial patterns of population growth observed in continuum models. These models can be applied to a variety of biological situations such as bacterial population growth and tumour invasion processes.

Talk by **Professor Jonathan Adam Sherratt**

The Heriot-Watt University

"An Impulsive PDE Model for Semi-Arid Vegetation"

Patterns of vegetation are a characteristic feature of many semi-arid regions. A large body of modelling work argues that these patterns are self-organised, arising from plants competing for water but at the same time benefitting one other via shading and changes in soil structure. Most mathematical models for this phenomenon assume that rainfall is constant, or at best seasonally varying. But in reality, many semi-arid regions experience short and intense rainfall events that cause a pulse of biological processes such as plant growth and seed dispersal. We propose an impulsive partial differential equation model to investigate the effects of this rainfall intermittency. Our model is based on the established Klausmeier reaction-advection-diffusion system, and our investigation focuses on the parameter region in which a transition between uniform and patterned vegetation occurs. Our results show that decay-type processes associated with a low frequency of precipitation pulses inhibit the onset of patterns and that under intermittent rainfall regimes, a spatially uniform solution is sustained at lower total precipitation volumes than under continuous rainfall, if plant species are unable to efficiently use low soil moisture levels. Unlike in the classical setting of a reaction-diffusion model, patterns are not caused by a diffusion-driven instability but by a combination of sufficiently long periods of droughts between precipitation pulses and water diffusion. This work was done in collaboration with Lukas Eigentler.

3 Posters

Poster by **Viktoria Freingruber**

Heriot-Watt University

"Modelling the collective migration of Neural crest cells/ How cells work together to migrate more efficiently"

Collective migration is a multicellular phenomenon that arises in various cell populations, including cancer cells and various developmental cells. A contributing factor for the "collectiveness" are cell-cell interactions such as co-attraction and contact inhibition of locomotion. These mechanisms act on cell polarity, pivotal for directed cell motility, through influencing the intracellular dynamics of small GTPases such as Rac1. To model these dynamics, we introduce a biased random walk model, where the bias depends on the internal state of Rac1, and the Rac1 state is influenced by cell-cell interactions and chemoattractive cues. In an extensive simulation study we demonstrate and explain the scope and applicability of the introduced model in various scenarios. The usage of a biased random walk model allows for the derivation of a corresponding partial differential equation for the cell density while still maintaining a certain level of intracellular detail in the individual based setting.

Poster by **Toyo Vignal**

Heriot-Watt University & University of Edinburgh

"Vegetation stripes in Scotland: self-organisation of heather (Calluna vulgaris) in regular bands"

Regular stripes of heather (Calluna vulgaris) can be observed in some windy areas of Scotland. The stripes consist of both rippling in the soil profile and alternation between vegetated and non-vegetated bands; all moving in the same direction as the prevailing winds. A description of the dynamics of these peculiar formations is given in a 1984 ecological paper by N.Bayfield. We build a system of partial differential equations to mathematically model the phenomenon.

Poster by **Elizabeth Howell**

Heriot-Watt University

"Using model systems to understand infection dynamics in a reservoir host: a case study of squirrelpox in red and grey squirrels"

Pathogen that are capable of infecting multiple hosts are ubiquitous and often persist in key reservoir hosts outside the species they most impact. An important case-study example is the transmission of squirrelpox virus (SQPV), from its reservoir host, the invasive grey squirrel (Sciurus carolinensis) to the endangered red squirrel (Sciurus vulgaris) population in the British Isles. This poster demonstrates and discusses the results of developing mathematical models to explore the effect of re-infection or recrudescence on the epidemiological dynamics of SQPV in grey squirrels and understand the impact of this upon their invasion of red squirrel strongholds

Poster by **Iain Souttar**

Heriot-Watt University

"Piecewise deterministic models for collective navigation: a multiscale analysis"

Motivated by models of the collective movement of animals, we study the large switching rate limit of a system of piecewise deterministic Markov processes. That is, the velocities are a continuous time Markov chain and the position of each animal is simply the integral of its velocity over time. We study the limit in which the animals switch their velocity often in comparison to the speed of movement. An analytical result is presented alongside numerics and preliminary study into the limit of large numbers of animals.

Poster by **Sofie Verhees**

Heriot-Watt University & University of Edinburgh

"Modelling intracellular signalling processes and elasticity"

Communication and interactions between cells happen mostly through intercellular signalling processes. These signalling pathways are important in all physiological activities of the cell, such as cell division, cell movement, immune response, and tissue development. In many of these signalling pathways, the chemical processes and mechanics of the cell work together [1]. However, how exactly these two phenomena communicate is not well known. A common way to model the chemical processes of cell signalling pathways are reaction-diffusion equations [2]. Regarding the chemical process, our model includes the diffusion of signalling molecules and membrane receptors, and the reactions between the molecules and receptors. The mechanical properties of the cell are modelled assuming elastic constitutive relationships. Simulation results, benchmarking and a comparison to literature will be presented.

[1] P. Romani, L. Valcarcel-Jimenez, C. Frezza, and S. Dupont. Crosstalk between mechanotransduction and metabolism. Nature Reviews Molecular Cell Biology, 22:22–38, 2021.

[2] M. Ptashnyk, C. Venkataraman. Multiscale analysis and simultion of signalling process with surface diffusion. MMS, SIAM Journal, 18:851-886, 2020.

Poster by **Alex Richardson**

The University of Edinburgh

"Neural Cellular Automata: a modelling framework for emergence"

Neural Cellular Automata (NCA) are a fascinating new modelling technique. They are simply cellular automata on a discrete lattice of continuous cell states, equipped with a neural network update rule mapping local neighbourhood configurations at one timestep to a cell state at the next. The most significant consequence of this construction is that the local update rule is differentiable with respect to global behaviour, meaning that local dynamics responsible for globally emergent behaviours can be efficiently learned with gradient based methods. Our work is based on that of Mordvintsev et al 2020, who demonstrate NCA and their ability to 'grow' images from a single pixel. We extend the NCA to learn the dynamics of a time series of images, and apply it in the context of modelling early morphogenesis in human embryonic stem cell cultures. By considering spatial symmetries, the neural network of the NCA can be kept small and interpretable, which allows for actually understanding the local dynamics that result in observed emergent behaviour. NCA are a promising modelling framework for exploring complex systems with emergent behaviours arising from purely local interactions, and they are general enough to

be applicable in a wide range of contexts. They are conceptually similar to recurrent neural networks and finite difference methods for PDEs. In this poster we will define what NCA are, demonstrate their training and show how to interpret underlying local dynamics.

Poster by **Xiaoyuan Liu**

University of York

"Adaptive Dynamics in Switching Environments: Modelling the Evolution of Sex"

"The evolutionary origins of the sperm-egg system are rooted in the quality-quantity tradeoff. More numerous sperm cells compete for fertilisation success while larger eggs improve survival probability of the resulting zygote. Traditional models assume obligate sexual reproduction [1] with self-incompatible mating types (x and y) and a fixed environment. In our adaptive dynamics model [2], we release these assumptions and observe a wider range of evolutionary outcomes. In a fixed environment, we evolve obligate sex from an initially asexual population. We find that the evolution of oogamy (motile microgametes, immotile macrogametes) can arise from evolutionary branching, an effect that occurs on the evolutionary timescale. In stochastically switching environments, we observe environmentally-triggered facultative sex both when the system undergoes bet-hedging and phenotypic plasticity. When the system undergoes bet-hedging, the timescale of the environmental switching can have a significant impact on the evolutionary dynamics. Since the conditions that favour multicellularity broadly coincide with those that favour sex (harsher environment) [3], our model may be potentially applicable for studying the evolutionary origins of multicellularity.

[1] Parker, G.A.; Baker, R.; Smith, V.G. The origin and evolution of gamete dimorphism and the male-female phenomenon. J. Theor. Biol. 1972, 36, 529–553.

[2] Liu, X.; Pitchford, J.W.; Constable,G.W.A The evolution of parthenogen fertilization rates in switching environments: from facultative cell-fusion to oogamy bioRxiv 2023

[3] JT Bonner, The origins of multicellularity. Integr. Biol. Issues, News, Rev. Publ. Assoc. with 923 The Soc. for Integr. Comp. Biol. 1, 27–36 (1998)"

Poster by **Dr. Giuseppe Torrisi**

The University of Edinburgh

"Gene interaction networks inferred from activation and inhibition perturbations"

Gene regulatory network (GRN) inference remains a challenging problem, particularly when using transcriptomic data. To address this challenge, we present a novel framework for inferring regulatory interactions from perturbation experiments involving both gene activation and inhibition, i.e., Crispr-a and Crispr-i experiments. Our approach is based on Bayesian inference and employs message-passing and dynamic programming techniques. We evaluate our method on synthetic networks and quantify its effectiveness in inferring gene interactions. Specifically, we applied our approach to the K562 cell line and analyzed the inferred regulatory interactions between genes. Overall, our work provides a new tool for GRN inference and informs the design of future experiments by highlighting the importance of including both inhibition and activation perturbations for a more comprehensive understanding of gene interactions.

Poster by **Dr. Teresita Suarez Noguez**

Roslin Institute

"Data-driven predictions of disease outbreaks in the UK pig industry"

Incursions of new animal diseases in the UK have previously caused high impacts into animal health, welfare and productivity of farms. They may also produce secondary effects on human health and wellbeing. In this work, we study the probability of an African Swine Fever (ASF) outbreak in the UK as it is currently circulating in several EU countries. ASF is a disease with a very high mortality rate in the host animals and there is not an effective vaccine available. For this purpose, we use mathematical models of disease spread to assess the potential impact of ASF into the British pig network. We use the records of the movements of pigs and haulage companies employed for their transport to understand the most at-risk areas for introduction of pig pathogens in the UK, to understand the connection between industries in the UK and affected countries abroad, and, to simulate the spread of diseases across these networks.

Poster by **Andrew Mair**

The Maxwell Institute for Mathematical Sciences

"Modelling the influence of preferential flow and root architectural traits on drought resistance in plants"

Plants combine a diverse range of morphological and physiological mechanisms to adapt to water deficit and drought. As an additional mechanism for plant drought resistance, this work considers how root-induced preferential flow redistributes soil water in a way that depends upon root system architecture. We developed a model for water transport through vegetated soil that incorporates root-induced preferential flow, then used Bayesian optimisation to calibrate the model against experimental data. A finite element scheme was used to simulate the model and assess how the fate of soil water is impacted by root system architecture. Root systems with a decreased gravitropic response were found to retain the most water in the rhizosphere following precipitation, which meant they most effectively delayed the onset of a water deficit. This work provides new insight into the role of root system traits in plant drought resistance and identifies root system architectures that could improve water use efficiency within cropping systems.