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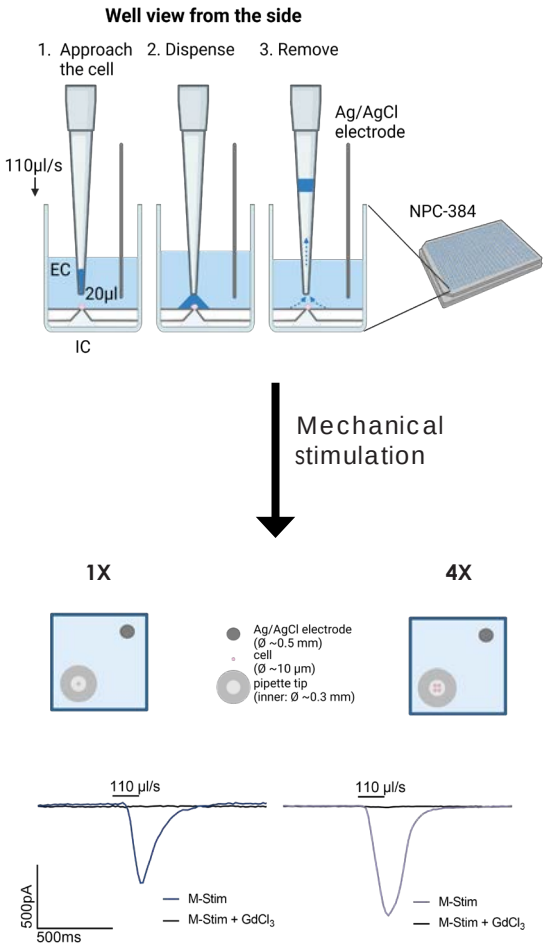

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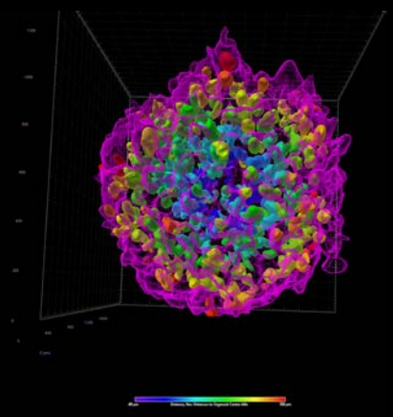

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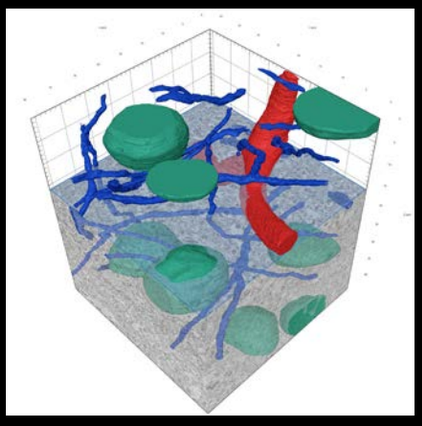

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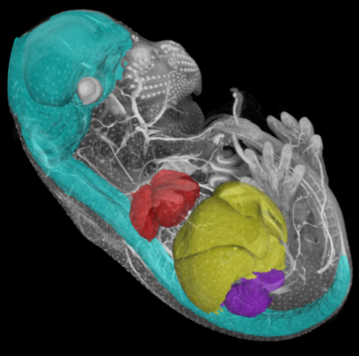

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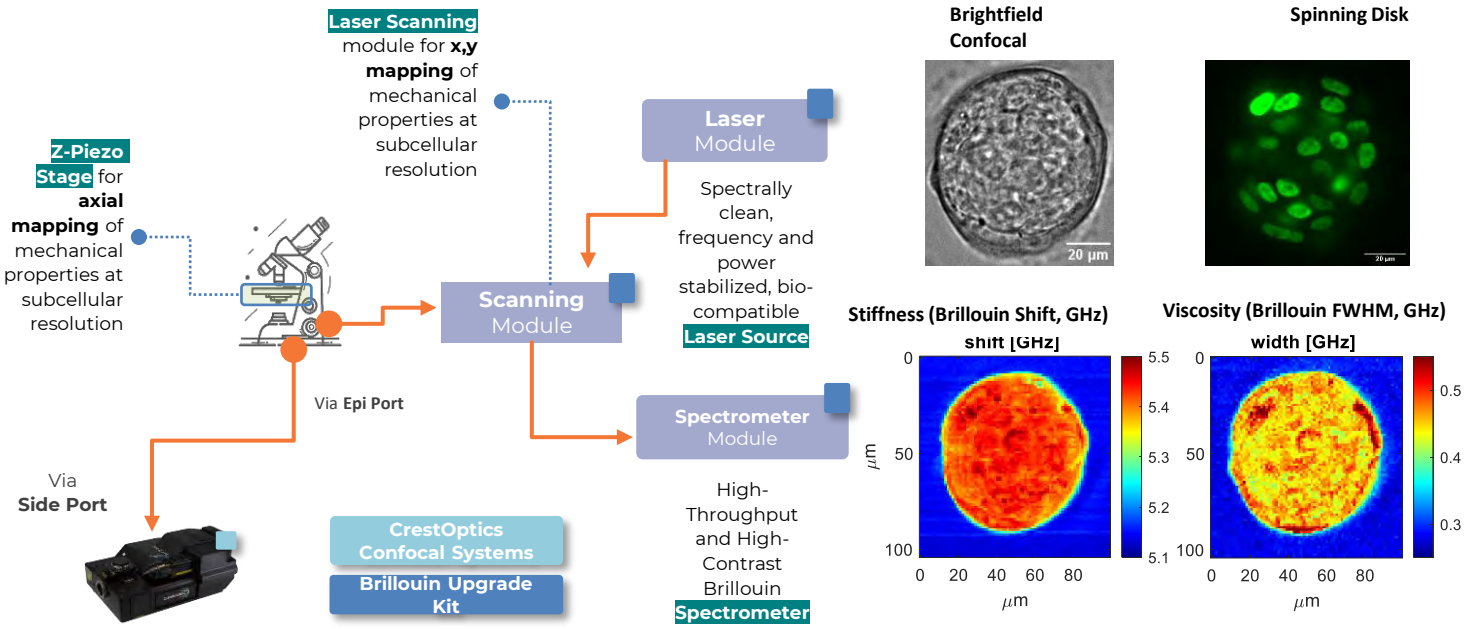

Schematic representation of the system architecture. Representative data acquired on the CrestOptics Brillouin System.

Automated calibration routine

Automated, sample-free, programmable calibration, a **patented** CrestOptics technology delivering real-time stability over days.

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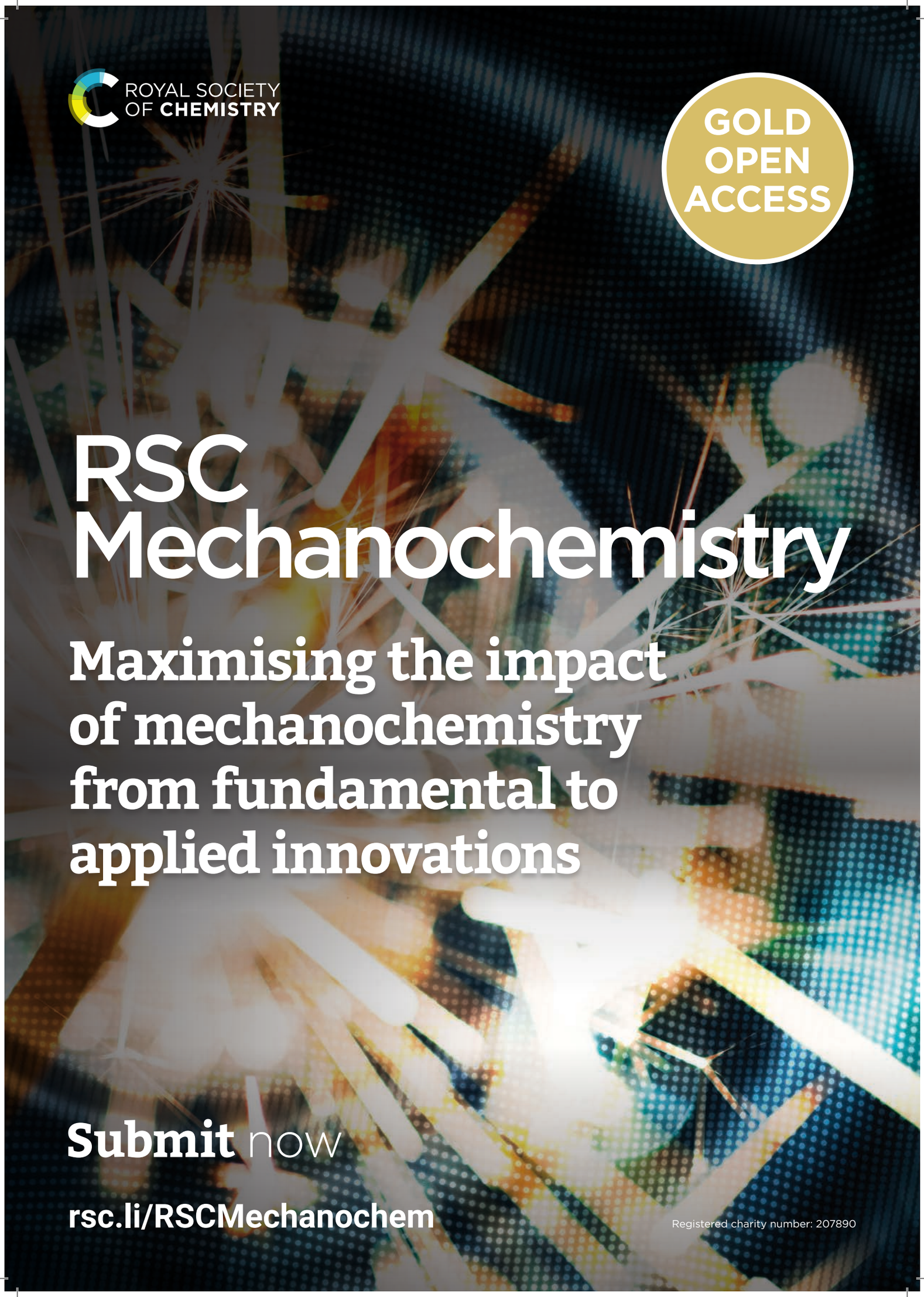
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Add mechanical contrast to tissue imaging for fibrosis, cardiovascular disease, ageing, regeneration and mechanomedicine studies.

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Plenary Speakers

Innovation for global justice

Patricia Osseweijer[1]
[1]Delft University of Technology, Netherlands

Plenary Session: Patricia Osseweijer, July 13, 2026, 11:00 - 11:45

The transition from a fossil- to a bioeconomy has long been promoted as a way to mitigate climate change by reducing carbon emissions. However, progress has been slow. At the same time, competing uses of biomass - for food, feed, plastics, fuels and chemicals - became the subject of intense public and political debate. The sustainability of biomass production was increasingly questioned, feedstock availability became a major concern, and the renewable products proved often more costly than the fossil products they aimed to replace. These criticisms weakened support for the bioeconomy and shifted attention towards alternative technological pathways. While science and innovation in the bioeconomy domain continued to improve biomass conversion technologies, logistics and market development, transition agendas became increasingly dominated by wind and solar energy and renewed interest in nuclear power with large enthusiasm for hydrogen as novel energy storage system.
This shift risks overlooking the broader societal contributions of research and innovation for the bioeconomy. Sustainable agriculture, including soil and water management and biodiversity conservation, is essential for health, for food and water security, while the responsible use of biomass and waste streams can simultaneously strengthen energy security, rural development and social resilience. Wind, solar and nuclear energy based transitions cannot provide these wider benefits, while hydrogen remains only an energy carrier.
The bioeconomy can therefore be considered to be much more than a substitute for fossil resources. It spans the entire innovation chain, from fundamental research in biological processes and biotechnology to sustainable production systems and circular value chains. Providing valuable insight in life itself and the complex ecological system on our globe. Importantly, biomass utilisation uniquely provides opportunities for improving livelihood and social development for rural communities. However, reaching such objectives requires other design principles and integration with social sciences.
This places an important responsibility on scientists and innovators. Rather than focusing solely on technological performance or carbon reduction, research should be designed from the outset with societal objectives such as human wellbeing, resilience and global justice in mind.
In this lecture, I will examine the assumptions underlying the bioeconomy debate and reflect on lessons from public communication and stakeholder engagement around genetic modification (GM). I argue that scientists have an essential role not only in developing new technologies, but also in considering the societal directions in which they evolve. This requires integrating moral questions ex ante into research and innovation, while engaging more proactively with society, policy and industry. Drawing on several interdisciplinary projects, I will show how scientific research can be organised to maximise technological performance while simultaneously advancing sustainability, social development and global justice.

May the Force be with you! Piezo channels in mechanosensory biology

Ardem Patapoutian[1]
[1]Howard Hughes Medical Institute, Scripps Research, United States

Plenary Session: Ardem Patapoutian, July 13, 2026, 09:00 - 09:45

Mechanotransduction was perhaps the last major sensory modality not understood at the molecular level. Proteins/ion channels that sense mechanical force are postulated to play critical roles in sensing touch/pain (somatosensation), sound (hearing), shear stress (cardiovascular function), etc.; however, the identity of ion channels involved in sensing mechanical force had remained elusive. The Patapoutian lab identified PIEZO1 and PIEZO2, mechanically activated cation channels that are expressed in many mechanosensitive cell types. Genetic studies established that PIEZO2 is the principal mechanical transducer for touch, proprioception, baroreception, bladder, and lung stretch, and that PIEZO1 mediates blood-flow sensing, which impacts vascular development and iron homeostasis. Clinical investigations have confirmed the importance of these channels in human physiology. Current work in Patapoutian continues to explore the role of mechanosensation and interoception in physiology and disease.

The human organ atlas as a resource for open biomedical research

Claire Walsh[1]
[1]University College London, United Kingdom

Plenary Session: Claire Walsh, July 13, 2026, 09:45 - 10:30

Understanding the human body across scales remains one of the central challenges in biomedical research. Clinical imaging can visualise whole organs but lacks microscopic detail, while histology reveals cellular structure but only within small, physically sectioned samples. The Human Organ Atlas was formed to bridge this gap: to create an open, multiscale, three-dimensional reference of intact human organs in health, ageing and disease.
The Atlas grew from the development of Hierarchical Phase-Contrast Tomography, a synchrotron-based imaging approach established through an international collaboration between clinicians, imaging scientists, engineers and data specialists. Initially driven by the urgent need to understand organ-level pathology during the COVID-19 pandemic, the project rapidly evolved into a broader open-science effort. By imaging intact organs non-destructively, while retaining the ability to zoom into selected regions at near-cellular resolution, the Atlas preserves the spatial context that is often lost when organs are reduced to small samples or two-dimensional sections.
This talk will describe the history of how the Human Organ Atlas was formed: from method development and early biological discoveries, to the creation of an open-access data and visualisation platform, and the building of a wider community around this technique. I will discuss how technical work in large-scale data formats, metadata, web-based visualisation and reusable analysis pipelines has made these complex datasets accessible beyond the original imaging teams. I will also highlight how clinical and biological collaborations are turning the Atlas into a resource for health impact, supporting anatomical discovery, disease research, validation of clinical imaging, education, and the development of AI models trained on high-quality, spatially contextualised human data.

Cardiac mechanics to cure cancer

Serena Zacchigna[1]
[1]International Centre for Genetic Engineering and Biotechnology, Italy

Plenary Session: Serena Zacchigna, July 13, 2026, 11:45 - 12:30

The talk will focus on the mechanisms that prevent the spontaneous regeneration of the heart after damage and discuss mechanistic link with the rarity of cardiac cancers, to eventually move to therapeutic solutions to both promote cardiac repair and treat cancer.

Satellite Sessions Invited Speakers

Nanopipettes for Single-Cell Surgery

Paolo Actis[1]
[1]University of Leeds, United Kingdom

BioSPM: Session 1, July 14, 2026, 09:00 - 11:00

Physiological and pathological processes within the human body are controlled by complex cell-cell interactions within the context of a dynamic microenvironment. The ability to dynamically measure phenotypes (i.e. gene expression, protein activities, ion fluctuations, signalling) at the single cell level is key to understanding cellular behaviour in a complex environment. My research aims at developing nanoscale tools to dissect the dynamic contributions of single cells to disease processes.
I will present the development of nanoprobes based on nanopipettes for manipulating living cells. The integration of these nanoprobes with scanning probe microscopy techniques allows the delivery of biomolecules with single molecule resolution and the extraction of genetic material and organelles from living cells.

Mechanics and Mechanosensing of Platelet Migration

Prof. Tilman Schäffer[1]
[1]University of Tuebingen, Germany

BioSPM: Session 2, July 14, 2026, 11:30 - 13:00

Platelets migrate by haptotaxis as part of the innate immune response, yet the underlying physical mechanisms remain poorly understood. Using scanning ion conductance microscopy (SICM) and complementary imaging approaches, we investigated the interplay between platelet mechanics and mechanosensing. SICM revealed that migrating platelets exhibit a polarized morphology and a characteristic subcellular stiffness distribution with a dynamically remodeling leading edge associated with increased local F-actin density. Substrate stiffness strongly influenced migration, with stiff substrates promoting platelet polarization and efficient migration, while soft substrates suppressed both processes. Together, these findings establish a direct link between platelet mechanics and mechanosensing and demonstrate how SICM can uncover the nanoscale mechanics underlying cellular motility.

Learn it from ZEISS featuring VR technology

Katja Jacob[1], Jens Jehle[1]

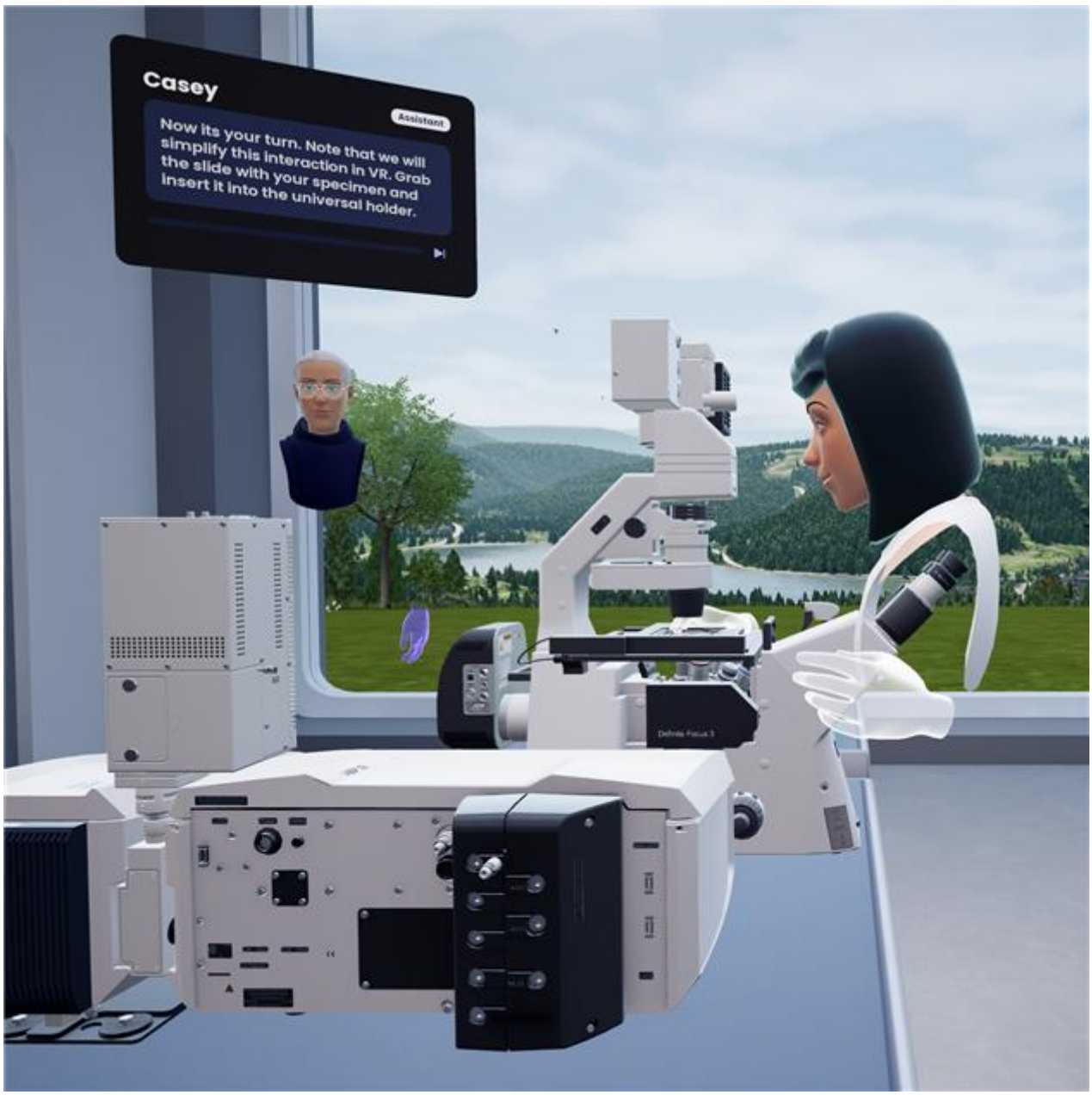
[1]ZEISS Microscopy, Germany

Extended Reality for Life Science Skills Training: Session 1, July 15, 2026, 11:00 - 13:00

Extended reality is becoming a practical solution for scaling advanced microscopy training in life sciences, especially where instrument access, trainer capacity, and facility time are limited. This talk presents how ZEISS has developed and deployed immersive learning programs that use virtual reality as part of blended training pathways for service teams, customers, and academic partners.

Over the past eight years, these programs have grown from internal training initiatives into customer-facing certified professional courses delivered in collaboration with universities and imaging facilities. The presentation will highlight how VR modules are integrated with talks, eLearning, recommended hands-on sessions, and assessment to create consistent, scalable learner journeys in confocal and scanning electron microscopy. Examples include VR training for confocal principles such as pinhole optimization, SEM workflows covering instrument operation and imaging controls, and field service scenarios such as column adjustment on a virtual GeminiSEM 460.

The talk will also outline a practical framework for developing immersive training, from needs assessment and learning objective definition through storyboarding, testing, validation, and rollout. Drawing on deployment experience since 2019, it will show how extended reality can improve accessibility, standardization, and learner engagement while reducing training bottlenecks and creating new opportunities for institutions and industry providers to expand high-quality skills development.


Casey
Assistant
Now its your turn. Note that we will simplify this interaction in VR. Grab the slide with your specimen and insert it into the universal holder.

RESILIENCE: Building an industry-ready life science talent pipeline through XR training

Dr. Laura Porcza[1]
[1]Heriot-Watt University, United Kingdom

Extended Reality for Life Science Skills Training: Session 2, July 15, 2026, 14:00 - 16:00

Extended Reality (XR) technologies are rapidly transforming life science education and workforce development by enabling immersive, experiential learning environments that bridge theory and practice. Within RESILIENCE, XR is used to deliver a comprehensive training approach that integrates both practical laboratory skills and soft/meta skills development. Practical skills training is delivered through immersive simulation software developed by FourPlus, while communication, teamwork and reflective practice are supported through immersive role-play modules developed by Bodyswaps.

Together, these tools enable deployment of XR-based training modules across laboratory, manufacturing and industrial life science sectors. By simulating complex procedures in virtual and mixed reality environments, RESILIENCE reduces laboratory waste while improving accessibility, consistency and learner engagement in a safe, risk-free environment. Learners can safely practise high-stakes techniques, repeat procedures as often as needed and build confidence through structured, hands-on practice.

XR training supports scalable delivery of practical experience, reduces dependence on physical resources and consumables and helps ensure more equitable access to high-quality training opportunities across geographically dispersed regions. Early deployments demonstrate improved learner confidence, faster acquisition of procedural skills and stronger retention of key steps, alongside measurable improvements in assessment performance and readiness for real-world application.

This session will outline the digital components underpinning the RESILIENCE ecosystem, the implementation approaches used across both education and industry settings, key outcomes from pilot assessments, and provide a short demonstration of the practical skills XR software developed by FourPlus and deployed within RESILIENCE. Overall, RESILIENCE positions XR as a practical, evidence-based approach to strengthening life science workforce readiness and modernising training across the sector.

mechanobiology-based medicine in the bone marrow

Prof Manuel Salmeron-sanchez[1]
[1]Institute for Bioengineering Of Catalonia, Spain, [2]University of Glasgow, United Kingdom
In-silico Mechanomedicine: Session 1, July 14, 2026, 11:30 - 12:30

The bone marrow is a key organ that houses hematopoietic stem cells (HSCs) and serves as the origin of all blood cells in the body. It is both a regenerative organ and a site of disease, as it is where blood cancers arise. Physically, bone marrow can be described as a viscoelastic hydrogel containing a diverse population of cells, including mesenchymal stromal cells (MSCs), which provide critical support for HSCs.
We have developed hydrogels with controlled viscoelastic properties that underpin a simplified model of the bone marrow niche, incorporating MSC spheroids and HSCs. To understand the response of MSCs to viscoelastic environments, we first designed viscoelastic 2D surfaces and analysed cellular responses using a variation of the molecular clutch model. In this framework, integrins transduce the dynamic mechanical properties of the extracellular matrix (ECM) and connect to the actin cytoskeleton via focal adhesions. We also introduce the role of Piezo1 in enabling mechanotransduction in soft viscoelastic environments.
We then extend this analysis to 3D systems, examining the behaviour of MSC spheroids within viscoelastic hydrogels. This includes tracking the evolution of mechanical properties over time using Brillouin microscopy, as well as quantifying cellular forces exerted on the hydrogel through traction force microscopy.
Finally, we incorporate either healthy HSCs or diseased leukemic cells into hydrogels containing MSC spheroids. We interrogate these systems under mechanical stimulation using a nanovibrational bioreactor and assess their mechanical response via Brillouin microscopy. This approach allows us to explore whether mechanical properties can be used to predict the evolution of the system in both health and disease.

Spectral stochastic finite-element approach for in silico population studies

Dr Pinaki Bhattacharya[1], Jacob Wilkinson[1], Saleh Pouresmaeeli[1], Claire Brockett[1]
[1]University Of Sheffield, United Kingdom

In-silico Mechanomedicine: Session 2, July 14, 2026, 14:00 - 16:00

In silico approaches are increasingly informing medical decision making. A key information produced is statistical inference of some kind, e.g. the likelihood of an outcome, which is typically based on a virtual population (VP) study. VP studies commonly employ sampling, thus generating numerous plausible but as-yet-unobserved patients, and then passing these to a predictive individualised (i.e. patient-specific) in silico model. The model predictions for disease / treatment outcomes are obtained for the VP and analysed statistically.

This approach works well when the unit cost of the patient-specific model is negligible. Mechanics-based predictive models, common in musculoskeletal (MSK) and cardiovascular (CV) diseases, do not fall in this category, as these often require a partial differential equation to be solved using finite-element (FE) or finite-volume analysis. With MSK and CV counting among the top five categories of morbidity ranked by their worldwide socioeconomic burden, this presents a significant challenge to the potential advancement of in silico approaches in informing medical decision making.

Spectral stochastic FE (ssFE) analysis is a sampling-free alternative. Here, the stochasticity in the population of the FE input and output variables are expressed in the spectral domain. Instead of predicting many individual outcomes, ssFE predicts (using one simulation) the spectral stochastic coefficients of the output variable in the whole population.

Although well-established, ssFE is not commonly used in in silico medicine. A challenge is to modify the FE algorithm to account for stochastic and spatial degrees of freedom, which obviates commercially available solvers. A new FE solver was developed to meet this challenge. It was used to analyse a femur neck model with a focal defect region with uncertain elastic modulus. The ssFE method was ~200x faster than the most efficient sampling-based approach for the same level of accuracy. Ongoing work and potential future directions will be discussed.

Multiscale Tissue Mechanics for Early Disease Detection and Tissue Health Assessment

Ciara Durcan[1], Izabela Nowakowska[1], Mahmood Saleh[1], Calum Anderson[1], Kathrine Kirkwood[2], Vidya Rajasekaran[3], Hugh Paterson[2], Bill MacPherson[1], Susan Farrington[3], Duncan Hand[1], Yuhang Chen[1]
[1]Heriot-Watt University, United Kingdom, [2]Western General Hospital, NHS Lothian, United Kingdom, [3]University of Edinburgh, United Kingdom

In-silico Mechanomedicine: Session 2, July 14, 2026, 14:00 - 16:00

Multiscale tissue mechanics provides a framework for understanding how biological tissue behaviour emerges from interactions across hierarchical spatial and temporal scales, spanning cellular architecture to whole-organ function. Critically, microscale alterations in structural organisation and material composition can propagate to influence macroscale mechanical responses. Establishing quantitative links between microstructural features and bulk mechanical properties therefore offers a powerful approach to detect and interpret early pathological changes through measurable physical signatures.

In this talk, we present a series of case studies demonstrating the application of multiscale tissue mechanics to clinically relevant problems. First, we examine structure–property relationships in human prostate tissues, highlighting differences between normal and cancerous states. We show how variations in elasticity and viscoelasticity correlate with microstructural remodelling and may enable improved identification of clinically significant tumours. Second, we investigate the use of mechanical properties as diagnostic “signatures” for tumour margin detection, using both human and animal colorectal cancer models. Our findings emphasise the importance of accounting for micromechanical heterogeneity and interspecies differences when translating biomechanical markers into clinical practice. Finally, we introduce ongoing work employing a three-dimensional digital image correlation (3D-DIC) approach for intraoperative assessment of tissue condition. This technique enables full-field measurement of deformation and mechanical response, with potential to provide real-time, non-invasive evaluation during surgical procedures.

These studies demonstrate how integrating multiscale mechanical insights with experimental measurement can advance diagnostic capabilities, improve surgical precision, and support the development of biomechanical biomarkers for disease detection and monitoring.

Mechanics of the Brain Across Scales: Cells - Tissue - Organ

Paul Steinmann[1]
[1]FAU/GCEC, Erlangen-nürnberg, Germany

In-silico Mechanomedicine: Session 2, July 14, 2026, 14:00 - 16:00

At the cellular scale, we attempt describing by a continuum formulation how cells interact with the extracellular matrix to form neuronal networks. Because the resulting PDEs are of the convection-diffusion/advection type, standard simulation methods are challenged. We are breaking through these barriers by developing novel computational approaches specifically designed to handle these complex equations.

At the tissue scale, we seek to unlock the secrets of spinal cord regeneration—a remarkable feat for zebrafish, but a missing capability in humans. To get there, we are endeavouring to map the precise mechanics of the spinal cord using cutting-edge indentation and rheometry, allowing us to identify and calibrate robust continuum models.

At the organ scale, we tackle a long-standing puzzle: why do ex vivo and in vivo brain mechanics tests often contradict each other? We hypothesise that vascularisation and blood pressure pulsations drive these discrepancies. By establishing a computationally-assisted protocol for virtual magnetic resonance elastography (MRE), we aim to conquer a major hurdle—inverting the wave equation for elastic and viscoelastic soft materials.

PIEZO1-dependent Mechanopathology- From single channel biophysics to genetic disorders.

Ruhma Syeda[1]
[1]UT Southwestern Medical Center, United States

In-silico Mechanomedicine: Session 3, July 14, 2026, 16:30 - 17:15

Mechanical forces are fundamental regulators of cellular behavior, and understanding how cells sense (mechanosensing) and respond (mechanotransduction) to these forces is essential for uncovering the mechanisms underlying normal physiology and human disease. Mechanical stimuli, such as pressure and membrane stretch, activate the mechanosensitive ion channel PIEZO1, allowing the influx of cations, Ca^{2+} and Na^{+}, which initiate downstream signaling pathways essential for numerous physiological processes. This intricate biophysical transition of PIEZO1 between closed and open conformational states (gating), together with the resulting cation flow (permeation), underlies PIEZO1-mediated mechanotransduction in diverse cell types, including endothelial, epithelial, and glial cells, where it plays a critical role in maintaining cellular and tissue homeostasis.

Importantly, genetic mutations that alter the biophysical properties of PIEZO1 channels can disrupt normal cellular functions, and are implicated in a wide range of pathological conditions. Recently, our lab identified the molecular basis of human PIEZO1 channelopathies by functionally characterizing disease-associated variants identified in patients with rare genetic bladder disorder and inherited osteoarthritis. Functional analysis revealed that these mutations significantly reduce channel gating in response to pressure (decreased open probability), consistent with a loss-of function phenotype. Importantly, the small-molecule agonist Yoda1 restored channel activity by increasing open probability, demonstrating that disease associated variants remain pharmacologically responsive.
The single-channel electrophysiology current traces were further utilized to develop a kinetic scheme that closely reproduces the gating of Wild-Type PIEZO1 and mutant channel, including the recently identified subconductance states. This scheme was incorporated into biophysical models of red blood cells, smooth muscle cells, and astrocytes to determine how the different variants of PIEZO1 affect the ion and volume homeostasis across various cell types.

These studies bridge the gap between single-channel biophysics and cellular and tissue physiology, providing mechanistic insights into PIEZO1-dependent diseases and establishing a multiscale framework for understanding disease mechanisms, and highlight the therapeutic potential of targeting PIEZO1 in the treatment of genetic disorders.

Guard cell form and function: a combined experimental and modelling approach

Andrew Fleming[1]
[1]The University of Sheffield, United Kingdom

Plant Mechanobiology 1, July 15, 2026, 09:00 - 10:30

The extent and rate of guard cell (GC) swelling/deflation determines gas exchange into and out of the leaf- the core function of stomata. This is a mechanical process, influenced not only by ion transport activity and consequent water flux, but also the material properties and shape of the GCs- the endpoint of stomatal development. Combining experimental and modelling approaches, our research is dissecting the biomechanics underpinning stomatal function, exploring the role of cell geometry and the cell wall in stomatal function.

Using live imaging of Arabidopsis stomata, our recent data show that GCs undergo complex changes in 3D form to drive pore movement. This response is dependent on initial GC shape (defined by the wall), differential wall thickness and by the material properties of the wall itself, leading to an interplay of geometry and wall structure that ultimately determines stomatal response to environmental triggers. Moreover, our results indicate that cells neighbouring the GCs play an active role in pore movement, suggesting that our views on stomatal function need to integrate the wider response of the epidermis.

In conclusion, our results highlight the importance of GC shape in stomatal function. They set the scene for future work understanding how shape develops, how it has altered over evolutionary time, and the consequences for plant and crop gas exchange.

Light control of plant movements

John Christie[1]
[1]Plant Science, University of Glasgow, United Kingdom

Plant Mechanobiology 2, July 15, 2026, 11:00 - 12:30

How light activates processes like phototropism, organelle movement (chloroplasts) and stomatal opening and how to fine tune these movements using optogenetics approaches.

Eversion of bacterial colonies: how oxygen gradients trigger large-scale cellular fluxes

Stephan Wimmi[1], Isabelle Wielert[1], Kai Zhou[2], Marc Hennes[1], Benedikt Sabass[3], **Berenike Maier**[1]

[1]Institute for Biological Physics, and Center for Molecular Medicine Cologne, University of Cologne, Germany, [2]School of General Education, Wenzhou Business College, China, [3]Technical University of Dortmund, Germany

The Physics of Microorganisms: Session 1, July 14, 2026, 09:30 - 11:00

Colonies and biofilms are a predominant form of bacterial life. Surrounded by an extracellular matrix, bacteria form structured communities with emergent properties that protect them from external stresses and confer tolerance to antimicrobial treatments. In this talk, I will focus on how the attractive forces between cells control these emergent properties. We explore these questions using the human pathogen Neisseria gonorrhoeae as a model system. In colonies, the mechanical properties are determined by type 4 pili (T4P). We have established biophysical and microbiological methods related to these molecular motors and developed them into a toolbox that allows us to control the mechanical properties of colonies formed by N. gonorrhoeae, including colony shape, viscosity, surface tension, local order, and sorting. A characteristic feature of this system is that the cellular network formed by T4P is inherently active and consumes energy. We recently discovered that this energy consumption creates an oxygen gradient within the colonies which causes depletion of the electrical membrane potential and reduction of T4P-mediated attractive forces at the centre of the colony. The resulting gradient renders the large-scale colony morphology metastable and susceptible to a non-linear shape instability. This instability generates large-scale cellular fluxes that redistribute bacteria toward environments more favourable for their growth. By combining experimental approaches with an idealized computational model, we identify how energy consumption, active forces, and material instabilities interact to control morphology at the colony level, providing a physical understanding of shape dynamics in living, active matter systems.

The orchestration of collective movement in biofilms

William Durham[1]
[1]The University of Sheffield, United Kingdom

The Physics of Microorganisms: Session 3, July 14, 2026, 14:00 - 16:00

Bacterial motility drives the spread of infections and shapes the structure of microbial communities, yet we still know surprisingly little about how bacteria attached to surfaces regulate their movement. In this talk I will present recent work uncovering how the pathogen Pseudomonas aeruginosa coordinates its collective movement across surfaces using grappling hook-like appendages called pili.
Combining engineered strains, high-throughput cell tracking, and mathematical modelling, we reveal how individual bacteria regulate their behaviour within densely packed collectives to generate highly polarised movement that propels the colony into new territory. We then show that this polarised movement can drive the spontaneous unmixing of strains that move at different rates, sorting genetically distinct cells into separate patches. Finally, we find that the flow patterns produced by these collectives exhibit conformally invariant behaviour at the macroscopic level that is nearly identical to that generated by collectively moving human breast cancer and dog kidney cells, demonstrating the emergence of universal patterns of collective movement that transcend cell type and mechanism of propulsion.
Together, these findings reveal how bacteria coordinate their movement to colonise surfaces and form the biofilms that underlie many difficult-to-treat infections.

Embodied AI for HRI: From Multimodal Social Cue Integration to Human-Robot Cooperation

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Understanding and Engineering Human Behaviour with Social AI: Session 1, July 13, 2026, 14:00 - 15:00

Humanoid robots are increasingly expected to operate in complex social environments, where successful interaction depends not only on recognising individual signals, but also on integrating gaze, facial expression, gesture, attention, and action over time. In this talk, I will present a series of studies on embodied AI for multimodal human-robot interaction, moving from social cue integration to human-robot cooperation. I will first introduce work on how human-like attentional mechanisms can be modelled and implemented in humanoid robots. I will then discuss how robot gaze, facial expressions, mirroring behaviour, and personality cues shape human perception, trust, and collaborative behaviour. Building on our recent framework for robot gaze control in multimodal social scenarios, I will consider how embodied agents can move beyond isolated cue responses towards more adaptive, context-sensitive social behaviour. Together, these studies show how cognitive principles can inform the design of socially responsive robots, while also using robots as controllable platforms for studying human social behaviours.

Enhancing human interactions with Augmented Reality—cognitive consequences and ethical considerations

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Understanding and Engineering Human Behaviour with Social AI: Session 2, July 13, 2026, 15:30 - 16:30

Recent advances in Artificial Intelligence techniques enable users to transform the voice and face of individuals in real time (e.g clone a person's face or change their emotional expressions) . These so called Augmented Reality (AR) filters have been massively deployed to the smartphones of billions of individuals. While no regulation exists to date, recent research suggests these filters may influence psychological processes. In parallel, AR filters also enable scientists to control the social signals produced by participants in free social interactions, a critical feature for the study of human social interactions. Therefore, AR filters can provide scientists a crucial tool to study social interactions, but also represent an increasingly important ethical phenomenon to study. During this talk, I will present the experimental platform DuckSoup, which we developed to investigate these questions. DuckSoup is an open-source videoconference experimental platform which enables researchers to perform human interaction experiments, while transforming the voice and face of participants in real time with AR filters. I will present three studies where we used DuckSoup to investigate social cognition mechanisms. In two studies we aligned the smiles of interacting participants with face transformations to see how smile alignment influenced liking. In another study, we transformed the vocal masculinity of participants during decision making tasks to quantify how vocal masculinity biases negotiations. I will conclude highlighting that AR filters can enable us to uncover social cognition mechanisms, but also that we should study their impact on psychology to support ethical regulations.

ECR and Satellite Session Oral Presentations

Probing Molecular Structure-Mechanics Relationships in Protein-based Materials with Single-Molecule Force Spectroscopy

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BioSPM: Session 1, July 14, 2026, 09:00 - 11:00

The mechanical functionality of biological materials arises from hierarchical organization, where molecular-scale interactions govern macroscopic properties. A central challenge is to understand how specific structural motifs and interfacial interactions translate into defined mechanical responses, and how these relationships can be harnessed for the rational design of bioinspired mechanoresponsive materials. Atomic force microscope-based single-molecule force spectroscopy (SMFS) provides a powerful approach to directly quantify these structure-mechanics relationships at the molecular level.

This talk introduces protein-based building blocks as tunable mechanical elements in biological and bioinspired materials. As a first example, α-helical coiled coils (CCs) are highlighted, as they are both ubiquitous in biological materials and particularly well suited for rational design. SMFS measurements have revealed sequence-encoded local helix stability and force-loading geometry as key parameters that determine rupture forces. These insights allowed us to establish a library of mechanically calibrated CC motifs that serve as modular building blocks with predictable force-response characteristics, applicable as smart hydrogel crosslinks or molecular force sensors in cell culture. The second system focuses on spider-derived β-sheet proteins that mediate protein-chitin interactions in arthropod cuticle. Here, SMFS directly quantifies binding strengths at the protein-chitin interface. This approach opens pathways for engineering protein variants with programmable binding characteristics for chitin-based composite materials. Together, these examples demonstrate how SMFS bridges molecular structure and material mechanics, offering a general framework for studying and engineering functional materials from the bottom up.

Combining Scanning Particle Induced X-ray Emission (PIXE), the anomalous diffraction signal and XRF measurements to enhance metalloprotein biochemistry

Professor Elspeth Garman[1], Geoff Grime[2], Edward Snell[3]
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BioSPM: Session 1, July 14, 2026, 09:00 - 11:00

The Protein Data Bank (PDB) contains information on over 200,000 proteins. Of these ≥60,000 are metalloproteins, being either proteins in which a small number of metal atoms define the three-dimensional shape (and hence function), or those involved in metal transport or metabolism. For most PDB metalloprotein entries, the metal atom identity has been inferred indirectly from the X-ray diffraction data or by other methods, including the presence of an anomalous signal.
In the 1990s, we developed a method for unambiguously identifying and quantifying protein metal atom stoichiometry (+/-10%) using scanning microbeam Particle Induced X-ray Emission (PIXE) and proton Rutherford Backscattering [1, 2, 3]. μPIXE has since informed many metalloproteins studies, but although allowing identification of different metals present, it does not give any positional information.
We have now automated the PIXE pipeline, permitting analysis/day of up to 100 aqueous protein samples deposited on a prolene film as a spot array using a non-contact inkjet printer, allowing substantially faster throughput. We have analysed 32 [4], and subsequently another 70 PDB deposited metalloproteins, with the surprising result that >30% of the PDB deposited models had misidentified metals and another 30% contained additional metals. In one of the former cases, we used the experimental and theoretical anomalous signal ratios to position the 3 different metals detected by PIXE and re-refined the deposited structure, revealing a previously missed active site ligand and thus new biological information. We have cross checked our PIXE analyses using the identical samples for XRF (X-ray fluorescence) measurements, with good agreement [5]
This contribution will review our latest results: using the correct metal in the metalloprotein structure allows improvement of the PDB models, so revealing previously unknown biological functions of the proteins studied.

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Intracellular Magneto-Mechanical Profiling: Measuring and Manipulating Forces Inside Living Cells

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BioSPM: Session 1, July 14, 2026, 09:00 - 11:00

Mechanical forces are fundamental regulators of cellular organisation, yet our ability to measure and manipulate them directly inside living cells has remained severely limited. Existing force spectroscopy tools such as optical tweezers cannot operate intracellularly without causing laser-induced damage, and conventional magnetic tweezers have required particles larger than one micron to generate sufficient force — particles too large to target specific intracellular structures without disrupting cell function.

To address this challenge, we engineered electromagnetic tips with optimised geometry that achieve theoretically maximal force generation, applying up to 10 pN to 100 nm magnetic nanoparticles — approximately ten times greater than previously achieved. We developed robust and reliable particle delivery methods that ensure high cell survival and the ability to target particles specifically to intracellular structures. This enabled recruitment of biocompatible 50–100 nm nanoparticles to microtubules in both interphase and mitotic cells, allowing targeted force application to the microtubule cytoskeleton and mitotic spindle.

Using this platform, we have demonstrated directed manipulation of nanoparticles inside living cells, targeted force application to microtubules in cancer cell lines, and mechanical perturbation of individual cells within human organoids. The system is compatible with standard fluorescence microscopy and does not require specialist physical training to operate.

This technology opens new experimental possibilities for mechanobiology, cancer research, and developmental biology, and represents a significant step towards a commercially deployable instrument for the broader life sciences community.

Multimodal mechano-SICM and FRET with stretch for probing cardiomyocyte function

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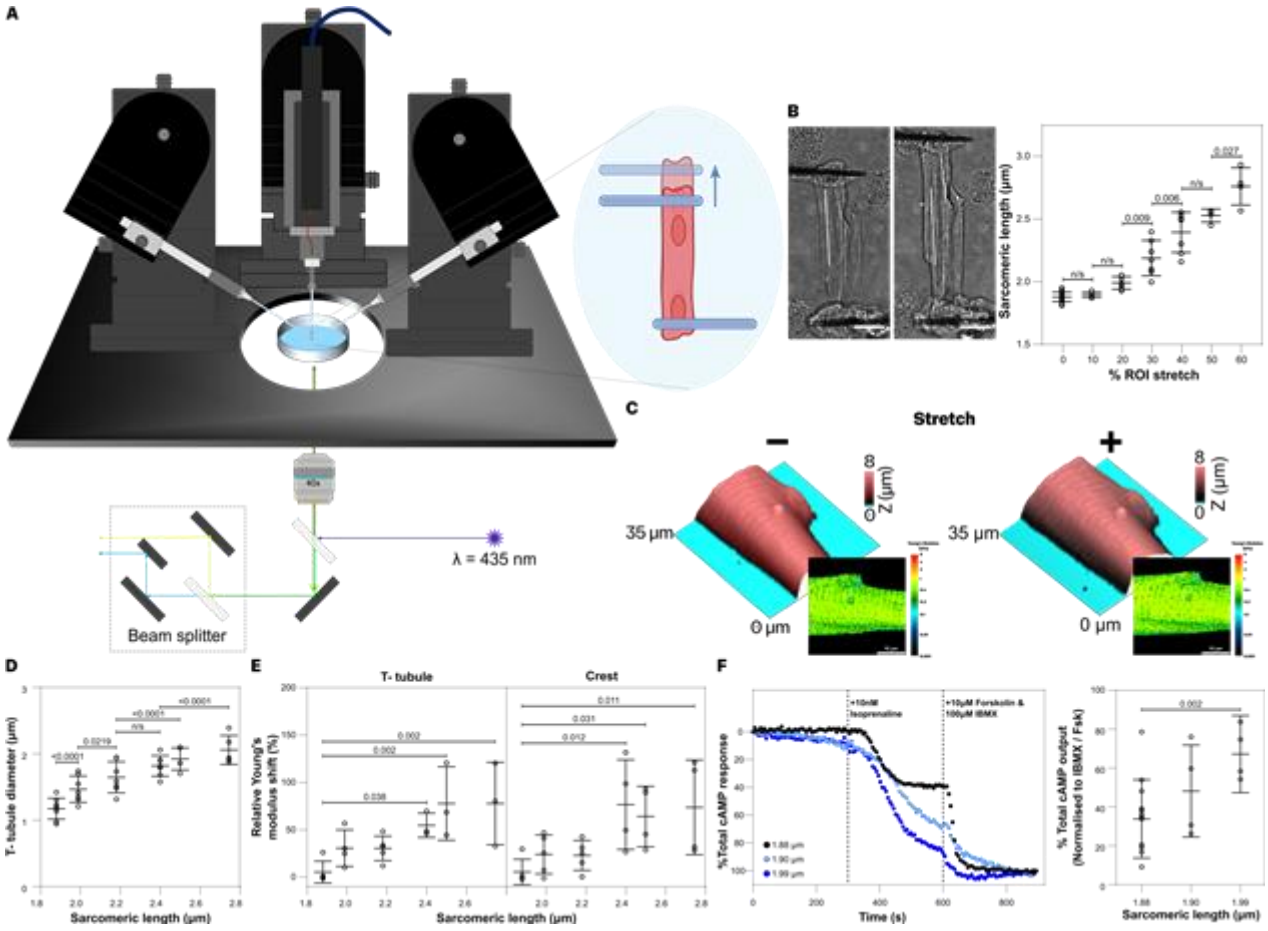
BioSPM: Session 1, July 14, 2026, 09:00 - 11:00

We present a novel multimodal system combining a Scanning Ion Conductance Microscope (SICM) with micro-controlled stretch apparatus and FRET microscope (figure 1A). This approach allows for high-resolution topographical and Young's modulus mapping of live cells under mechanical load simultaneously with optical measurement of sub-cellular signalling.
This technique is particularly advantageous for characterisation of the surface of isolated adult cardiomyocytes. In situ, cardiomyocytes are subjected to mechanical load associated with the contraction cycle, this load can be chronically elevated in diseases involving pressure overload such as hypertension. How the surface topography of the cardiomyocyte changes under different loads, and how this may modulate cell function, has not yet been identified.
It is our hypothesis that increased cardiomyocyte load alters the surface topography of the cell. We also hypothesise that these changes in the cell membrane will alter key signalling nanodomains associated with the membrane.
Isolated rat ventricular cardiomyocytes were progressively stretched using Matrigel-coated micro-rods. A single rod was positioned to achieve the desired load/stretch (figure 1B) which was verified by measurement of the sarcomeric length. The modality of SICM is particularly advantageous, as no sample movement is required (X, Y and Z movement is performed by the nanopipette). SICM topographical and Young's modulus maps were generated from the same cell area before and after stretch was applied (figure 1C).
Under increased diastolic load, we observe a significant increase of T-tubule opening diameter (figure 1D) and transverse Young's modulus of the cell (figure 1E). We also identify that the total cAMP response to 10 nM Isoprenaline is increased in cells undergoing 25% stretch (figure 1F).
These data give the first evidence that this multimodal stretch-SICM-FRET system can be used to interrogate the effect of load upon both the cell surface and sub-cellular signalling of adult cardiomyocytes.


A
λ = 435 nm
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B
Sarcomeric length (µm)
% ROI stretch
C
Stretch
35 µm
0 µm
Z (µm)
D
T- tubule diameter (µm)
Sarcomeric length (µm)
E
T- tubule
Crest
Relative Young's modulus shift (%)
Sarcomeric length (µm)
F
%Total cAMP response
Time (s)
% Total cAMP output (Normalised to IBMX / Fsk)
Sarcomeric length (µm)

Large-Area AFM Mapping for Mechanobiology Studies in Cells and Tissues

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BioSPM: Session 2, July 14, 2026, 11:30 - 13:00

Mechanical properties regulate mechanotransduction and transport processes, shaping signaling, molecular trafficking, and tissue organization. Atomic force microscopy (AFM) enables nanoscale characterization of stiffness, adhesion, and viscoelasticity, and is also widely used for high-resolution structural analysis – critical for understanding force-driven transport in complex biological systems. However, conventional AFM is limited by sample roughness and restricted lateral range, constraining large-scale analysis of heterogeneous specimens.
We present an advanced AFM imaging approach using SmartMapping technology, which synchronizes AFM head motors with XYZ-piezo stage movement. This innovation enables automated, high-resolution mapping across extended areas without user intervention, improving reproducibility and throughput for multi-region mechanobiological studies.
Using this technology, we analyzed Cytochalasin D-treated 3T3 fibroblasts versus controls, revealing time-dependent changes in mechanics and cytoskeletal architecture that influence intracellular transport. Highly corrugated 3D SKOV-3 spheroids (>100 µm) exceeding 100 µm in height, focusing on their structural and mechanical features. By mapping mouse brain tissue along the anterior-posterior axis we draw functional correlations between the regional structural and mechanical differences and the underlying anatomical composition and architecture
This integrated approach establishes a new standard for large-area mechanobiological imaging, enabling spatially resolved analysis of mechanical cues that influence transport in cells and tissues.

High-resolution AFM reveals changes in the orientation and porosity of the E. coli peptidoglycan network when exposed to mecillinam

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BioSPM: Session 2, July 14, 2026, 11:30 - 13:00

E. coli is a rod-shaped, Gram-negative bacterium whose morphology is maintained by a peptidoglycan (PG) biopolymer (1). While the chemical composition of PG is well known (2), its detailed molecular organization and the structural changes it undergoes during antibiotic-induced death remain unclear. To address this, we used high-resolution tapping-mode atomic force microscopy (AFM) with a 0.25 nm pixel size to image extracted PG immobilized on a flat substrate. We tracked location-dependent nanoscale changes in PG architecture across different regions as a function of exposure time to the antibiotic mecillinam.

Qualitatively, the cylindrical body of untreated PG features glycan chains oriented perpendicular to the cell long axis. In contrast, mecillinam-treated PG is filled with short, disordered chains lacking any predominant orientation. To quantify these architectural changes, fiber orientation was measured using fiberFinder (3), a custom MATLAB GUI that utilizes a ridge-detection algorithm and linear regression to determine local fiber alignment. The analysis confirmed that untreated fibers maintain a principal orientation close to 90° relative to the cell long axis, while mecillinam-exposed PG exhibits random orientation and shorter average chain lengths.

Additionally, we developed in-house hole analysis software to measure grain size distribution after thresholding the AFM images. The data revealed that antibiotic treatment caused the average hole size within the PG mesh to increase from 23 nm^2 to 43 nm^2. Ultimately, these findings demonstrate the power of high-resolution AFM in deciphering the bacterial cell wall. This methodology provides critical insights into how antibiotics disrupt PG molecular organization, which subsequently regulates PG function and structural stability.

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The role of PIEZO1 in Purkinje Fibre-mediated arrhythmia development: A study from the whole heart to the nanoscale level

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ECR Session 1, July 12, 2026, 11:15 - 12:15

Premature ventricular contractions originate from the Purkinje fibre (PF) network; however, their underlying mechanism and propagation is unknown. Such answers would be revolutionary for targeting arrhythmia development within ablation therapy. This study looks at the role that PIEZO1, a mechano-sensitive ion channel, has within PF-mediated arrhythmia development.

In accordance with local ethical regulations: (1) Langendorff-perfused, male Wistar rat hearts (N=8) underwent mechanical stimulation by a left ventricular (LV) fluid-filled balloon (100 µL inflation over 2s; maintained for 38s). The LV was perfused with Tyrode then 50 µM Yoda1, a PIEZO1 activator, for 5 minutes, followed by Lugol's. (2) Isolated female Ovine PFs (N=3) were loaded with 10 µM Fura-2 to obtain calcium recordings in the presence and absence of Yoda1 whilst the PF underwent electrical and mechanical stimulation. (3) Isolated male Porcine PFs (N=2) were sectioned (350 µm) and incubated with 10 µM Rhod2; 10 µM blebbistatin and 7.5 µM RH237 for optical mapping recordings (0.5 Hz field stimulation; 35°C). All tissue underwent immunofluorescence staining and 4x expansion microscopy for PIEZO1. Data presented as mean ± SEM; One-Way ANOVA ($P<0.05$).

When the LV was dilated, (1) in the presence of Yoda1, ectopic activations increased 32% (12.21 ± 1.10) compared to Tyrode alone (9.25 ± 0.82), decreasing 75% to 3.09 ± 0.43 after Lugol's. Isolated PFs (2) had an altered calcium transient wavefront upon stretch, which was exacerbated in the presence of Yoda1 (denoted in brackets). Amplitude increased 65% (80%), with a 9% (14%) slower time to peak and 6% (27%) slower time to fall. (3) Voltage and calcium activation was mapped across the PF, with PIEZO1 localised to the PF membrane to correlate function with structure.

We propose PIEZO1 to have a key role in the PF network, its structural heterogeneity being fundamental to the mechanism of arrhythmia development.

Decoding vascular aging: Substrate stiffness and shear stress orchestrate endothelial inflammation and remodelling via mechanosensitive pathways

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ECR Session 1, July 12, 2026, 11:15 - 12:15

Vascular aging is a major factor which underlies many cardiovascular diseases including atherosclerosis and myocardial infarctions. Characteristics include stiffening of the vascular wall which disrupts endothelial homeostasis and promotes chronic inflammation, yet the underlying mechanisms remain poorly understood due to technological limitations. Here, we have developed an in vitro microfluidic model to investigate how biomechanical forces, specifically substrate stiffness and shear stress, interact to regulate endothelial cell functions. Using RNA sequencing and functional assays, we found that endothelial cells exposed to both physiologically high levels of shear stress were more sensitive to increases in substrate stiffness in which the cells exhibited a greater number of differentially expressed genes and enhanced activation of inflammatory signalling pathways.

We identified enriched pathways involved in inflammatory signalling and extracellular matrix remodelling due to substrate stiffening. Endothelial cells exposed to high shear stress and increased stiffness showed up-regulation of genes such as ICAM1 and VCAM1 which mediate leukocyte adhesion, and LAMB3 and MMP28 which mediate extracellular matrix remodelling. Importantly, these findings were benchmarked using human aortic tissue which confirmed concordant molecular and histological changes between stiff (aged) and soft (healthy) aortas.

We have also explored the role of mechanosensitive ion channel Piezo1 under the combined effect of shear stress and substrate stiffness in endothelial morphology, senescence, proliferation, and inflammation. Piezo1 facilitated endothelial adaptation to flow and regulation of inflammation under both atheroprotective and atherogenic shear stress levels. In endothelial senescence, Piezo1 contribution was limited to the soft substrate and atheroprotective shear stress.

Ultimately, this validated microfluidic platform provides proof of concept that biomechanical forces characteristic of vascular aging drive endothelial inflammation and vascular remodelling, offering new mechanistic insight and a powerful experimental framework to advance therapeutic strategies targeting vascular stiffness and age-related vascular disease in which the process may involve Piezo1 mechanotransduction.

Seeing Forces in Flow: Label-Free 3D TFM of Endothelial Remodeling in Perfused Microvessels

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ECR Session 1, July 12, 2026, 11:15 - 12:15

Endothelial cells integrate hemodynamic and matrix cues to regulate vascular stability and remodeling. While responses to wall shear stress are well characterized, how they transmit forces into a three-dimensional extracellular matrix under continuous perfusion remains unclear, particularly during remodeling and disease. Here, we present a novel 3D traction force microscopy-integrated vessel-on-chip (3DTFM-VOC) platform that enables direct, label-free quantification of endothelial force transmission in perfused microvessels. The system consists of parallel, perfusable cylindrical microchannels (120 μm diameter) embedded within collagen hydrogels and endothelialized with human brain microvascular endothelial cells, including CCM2-silenced cells to model cerebral cavernous malformations (CCM) on chip. Importantly, matrix deformations are captured using confocal reflectance microscopy, allowing direct visualization of collagen fiber displacement without the need for embedded tracer particles. Continuous perfusion at low and high wall shear stress (0.1–1 Pa) was maintained for up to 72 hours to replicate physiological flow conditions. Confocal μPIV confirmed stable flow distribution across acellular channels, while matrix stability analysis (over the perfusion period) verified that force measurements were not influenced by bulk hydrogel motion under flow. We further mapped 3D matrix displacements generated by endothelial cells over time. Endothelial forces produced stable, radially inward displacement fields around the vessel lumen that decayed with distance from the wall. In contrast, CCM2-deficient vessels exhibited enhanced angiogenic sprouting and vessel dilation, which are important hallmarks of CCM lesions. Sprouting was accompanied by localized amplification of collagen displacements at sprout tips, indicating spatially heterogeneous force transmission during pathological remodeling. Together, this work establishes 3DTFM-VOC as a powerful platform to quantify endothelial mechanobiology in real time and interrogate the interplay between fluid flow and cell-matrix forces in perfused microvessels. Our findings provide new insight into how force transmission governs vascular remodeling in health and disease and highlight its relevance to cerebrovascular pathologies such as CCM.

Investigating heart-generated mechanical stimuli as an innovative cancer therapy

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ECR Session 1, July 12, 2026, 11:15 - 12:15

Cancer is the second leading cause of death worldwide, accounting for approximately 1500 cancer-related deaths every day. Despite significant therapeutic advances, current treatments are often associated with severe side effects and rely on systemic approaches for metastatic disease. There are no effective technologies to control local cancer recurrence.

While most organs are frequently affected by cancer, primary cardiac malignancies are sporadic. This observation is intuitive for cardiomyocyte-derived tumors, given cardiomyocyte limited proliferative capacity, but remains unexplained for other cardiac cell types that retain replicative potential. Moreover, cardiac metastases are uncommon, despite constant exposure of the heart to circulating tumor cells.

We hypothesize that the unique mechanical forces generated by the beating heart act as a natural barrier to cell proliferation, including that of cancer cells. Building on this concept, we aim to develop an innovative technology capable of mimicking cardiac contraction and applying physiologically relevant mechanical forces to cancer cells.

By recreating heart-like mechanical stimulation in vitro, we investigated how dynamic mechanical cues regulate cancer cell proliferation and survival in a mouse model of cutaneous melanoma. We found that a specific combination of pressure intensity and frequency significantly reduced cancer cell proliferation and tumor growth. No metastatic spread was detected upon mechanical stimulation.

These data introduce a new paradigm in cancer research, leveraging mechanical systems to control tumor growth, and pave the way to a mechanical therapy for human cancer.

Mechanosensitive Ion Channel Piezo1 Regulates Osteoclast Differentiation by Activating the Metalloproteinases ADAM10 and ADAM17

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ECR Session 2, July 12, 2026, 12:45 - 13:30

Bone remodeling is a continuous process throughout life and serves to adjust bone architecture to meet changing mechanical needs. The mechanosensitive ion channel Piezo1 is known to sense mechanical forces in osteoblasts leading to increased bone formation by these cells. By contrast, the role of Piezo1 in osteoclasts, is less understood.
We have recently shown that the constitutive activity of the metalloproteinases ADAM10 and ADAM17 on osteoclast precursor cells limits osteoclast development from precursor cells that differentiate in response to macrophage colony-stimulating factor (M-CSF) and receptor activator of NF-κB ligand (RANKL). This is associated with the proteolytic shedding of cell surface expressed receptors for RANKL and M-CSF (RANK and CSF1R, respectively), thereby reducing cellular responsiveness of the cells. In the present study, we investigated whether this regulatory process can be modulated by mechanical forces and signaling through Piezo1. Stimulation of Piezo1 by Yoda1 led to a considerable reduction of osteoclast development in response to M-CSF and RANKL. This was mediated in part by increased activity of ADAM10 and ADAM17 as demonstrated by selective ADAM10 inhibition and ADAM17 knockout. Furthermore, Piezo1 activation reduced RANK and CSF1R surface expression on osteoclast precursor cells again depending on both proteases. Finally, also mechanical compression of the precursor cells diminished RANK and CSF1R surface expression partly involving ADAM10 and ADAM17 activity. These data indicate that ADAM10 and ADAM17 activity is upregulated via the mechanosensitive ion channel Piezo1 leading to enhanced receptor shedding thereby preventing induction of osteoclast differentiation. Also on osteoblasts, we found that stimulation of Piezo1 activates ADAM10 mediated release of RANKL. This implies that less transmembrane RANKL is available on osteoblasts for cell-to-cell stimulation of osteoclasts. Thus, Piezo1 may be locally relevant to limit bone resorption through activation of ADAM10 and ADAM17 which would complement its already well described pro-osteogenic role in osteoblasts.

Actomyosin-dependent PIEZO1-mediated mechanotransduction amplifies aldosterone production

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ECR Session 2, July 12, 2026, 12:45 - 13:30

Adherens junctions (AJs) couple intercellular cadherins to the actomyosin network via αCatenin and are central to sensing mechanical force at cell–cell contacts. The adrenal zona glomerulosa (zG) produces aldosterone in response to increases in intracellular Ca2+ in a highly coordinated manner. In adrenal slices, acute disruption of cadherin binding decreased synchronized intercellular Ca2+ bursts and decreased aldosterone production, suggesting intact AJs support coordinated zG activity. However, how tissue mechanics and intercellular coupling shape zG Ca2+ dynamics and steroidogenesis remains unclear. We hypothesized that AJs transduce mechanical signals through actomyosin to activate mechanosensitive ion channels, thereby amplifying Ca2+ entry and aldosterone production.

To define the downstream mechanotransduction pathway, we combined confocal imaging, atomic force microscopy (AFM), and patch clamping in control, αCatenin-knockout and PIEZO1-knockout human adrenocortical cells (NCI-H295R), and in primary mouse zG cells, and zG-specific mouse models. Upon K+ stimulation (a major physiological secretagogue of aldosterone), cells showed increased actin polymerization (via confocal imaging) and elevated membrane stiffness (via AFM) at cell–cell contact regions, which were blunted when the AJ–actomyosin linkage was disrupted in αCatenin-knockout cells. Pharmacological inhibition of actomyosin (via cytochalasin D, ML7, or blebbistatin) or mechanosensitive channels (via GsMTx4) attenuated K+-evoked Ca2+ influx and aldosterone production in NCI-H295R cells. Conversely, activation of PIEZO1 by Yoda1 induced membrane depolarization, Ca2+ influx, CYP11B2 expression and aldosterone production. Piezo1 deletion in mouse zG cells and NCI-H295R cells reduced K+-stimulated aldosterone production and rendered aldosterone production insensitive to the effects of actomyosin inhibition, consistent with PIEZO1 acting downstream of K+-induced actomyosin contractility.

We propose a mechanotransduction model by which K+-evoked actomyosin-dependent changes in cell–cell junction mechanics activate PIEZO1 to drive Ca2+ signaling and aldosterone production. Our findings establish the zG as a mechanosensitive endocrine tissue and identify an AJ–actomyosin-PIEZO1 axis as a potent regulator of aldosterone production.

Force Sensing via Ion Channels in Pancreatic Cancer

Mr Riley Mumberson[1]
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ECR Session 2, July 12, 2026, 12:45 - 13:30

The tumour microenvironment (TME) of pancreatic cancer is comparatively denser and more dynamic than other cancerous TME's. The progression of pancreatic cancer has been associated with significant changes in the mechanics of its tumour microenvironment, which requires cells to be able to sense and distinguish the physical forces they are exposed too. Force sensors such as mechanically activated (MA) ion channels have been shown to regulate cancer cell behaviour in response to changes within the TME. We found that there are multiple force sensing MA ion channels linked with migratory, invasive and/or poor prognostic outcomes in other cancers that are expressed in several pancreatic cancer cell lines. These were shown to exhibit mechanically evoked currents in response to membrane-stretch, indentation and deflections at the cell-substrate interface.
When either ELKIN1 or PIEZO1 were knocked-down (KD), cells displayed alterations in their mechanically driven behaviours depending upon the force imposed, rather than an ablation of their response. ELKIN1-KD's for example, increased currents in response to cellular indentation and membrane tension, however currents were reduced when deflected at the cell-substrate interface.
To understand these effects and investigate the influence multiple MA ion channel expression has on cellular force sensing, a series of inducible heterologous cell lines were created that express either one or two MA ion channels at similar concentrations. The distinct combination of channels produced diverging impacts on the magnitude of mechanically evoked currents, aligning with and expanding upon what was observed in the pancreatic cancer cell lines. This suggests a complex crosstalk between these force sensors when different MA ion channels are co-expressed in cells that can lead to changes in cellular mechanosensitivity. The data implies that it is essential to understand the molecular interconnectivity between these molecules to predict changes in cell mechanosensitivity of disease states such as pancreatic cancer.

Role of epigenetic modifications in the establishment of mechanical memory

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ECR Session 3, July 12, 2026, 14:45 - 15:45

Mechanical memory is defined by the persistent effect of past environment rigidity on cell adaptation to a new environment. Different mechano-sensitive signaling pathways have been implicated in mechanical memory. However, it is currently unknown how past information about substrate stiffness is stored inside the cell. Here, we report that the histone demethylase KDM7a controls mechanical memory of cell division by regulating H3K27 demethylation in a nuclear membrane tension-dependent manner. We observed that KDM7a was overexpressed in cells cultured on a soft substrate and was required to encode memory of a past soft substrate. On soft environments, reduced nuclear membrane tension controlled KDM7a recruitment at the nuclear envelop and H3K27 demethylation. Conversely, KDM7A localized to the nucleoplasm on stiff environments and H3K27 was methylated. These differences of histone methylation was maintained once cells primed on a soft substrate were transferred to a stiff secondary environment. Manipulation of nuclear membrane tension revealed that KDM7A localization was controlled by tension. We propose that information about substrate rigidity is encoded and stored in the epigenome through nuclear membrane tension-regulated H3K27 methylation dynamics.

Mechanical force unlocks cryptic phosphorylation in FAK to sever cooperative paxillin binding

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ECR Session 3, July 12, 2026, 14:45 - 15:45

Mechanical forces at cell-matrix adhesions drive biochemical signals that regulate cell behavior; yet, the molecular mechanisms linking force to specific enzymatic events remain largely unresolved. Here, we demonstrate that mechanical unfolding of the focal adhesion targeting (FAT) domain of FAK gates Y925 phosphorylation by the Src kinase, linking force to a key signaling modification that regulates focal adhesion turnover and cell motility. Using single-molecule magnetic tweezers, we show that the FAT domain unfolds under low physiological forces and that paxillin engages its LD2 and LD4 motifs through a mechanically cooperative interaction disrupted at forces preceding FAT unfolding. Exploiting the long-term stability of our magnetic tweezers, we capture phosphorylation of Y925 in real time, demonstrating that it occurs exclusively when FAT is mechanically unfolded. Immediate readout of the modified protein exposes a marked mechanical destabilization and an altered folding pathway, which we rationalize with molecular dynamics simulations. Phosphorylation further abolishes cooperative paxillin binding, linking force to a defined sequence of molecular events that collectively reshape the mechanical and biochemical properties of the domain. Our findings establish a molecular mechanism by which force drives a specific phosphorylation event with direct roles in adhesion dynamics, suggesting that mechanically gated enzymatic activity may be a broader principle underlying adhesion complex signaling.

Investigating the catalytic mechanism of a membrane-associated enzyme by EPR spectroscopy and MD simulations

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ECR Session 3, July 12, 2026, 14:45 - 15:45

Phosphatidylserine decarboxylase (PSD) is a membrane-associated enzyme conserved across all kingdoms of life.1 PSD catalyses the biosynthesis of phosphatidylethanolamine (PE) from phosphatidylserine (PS) and exists as a homodimer, with each protomer comprising one α-chain and one β-chain. Previously reported X-ray crystal structures of PSD in the apo and PE-bound states have advanced our understanding of PE biosynthesis, but the transitions PSD undertakes during enzymatic activity remains unclear.1,2,3 Here, we employ Pulsed Electron Electron Double Resonance (PELDOR) also known as Double Electron Electron Resonance (DEER) spectroscopy to investigate the conformational ensemble of the lipid bound enzyme. Using atomistic molecular dynamics (MD) simulations, we explore the conformational space of the enzyme and derive minimal conformational ensembles of PSD consistent with EPR, under different conditions. Finally, we apply single channel electrophysiology to monitor the activity of mechanosensitive (MS) ion channels modulated by the presence of PSD, providing a link between lipid biosynthesis enzymes and MS ion channel modulation. This work is supported by the School of Biological Sciences, Faculty of Biology, Medicine and Health, The University of Manchester and the Biotechnology and Biological Sciences Research Council (BBSRC).
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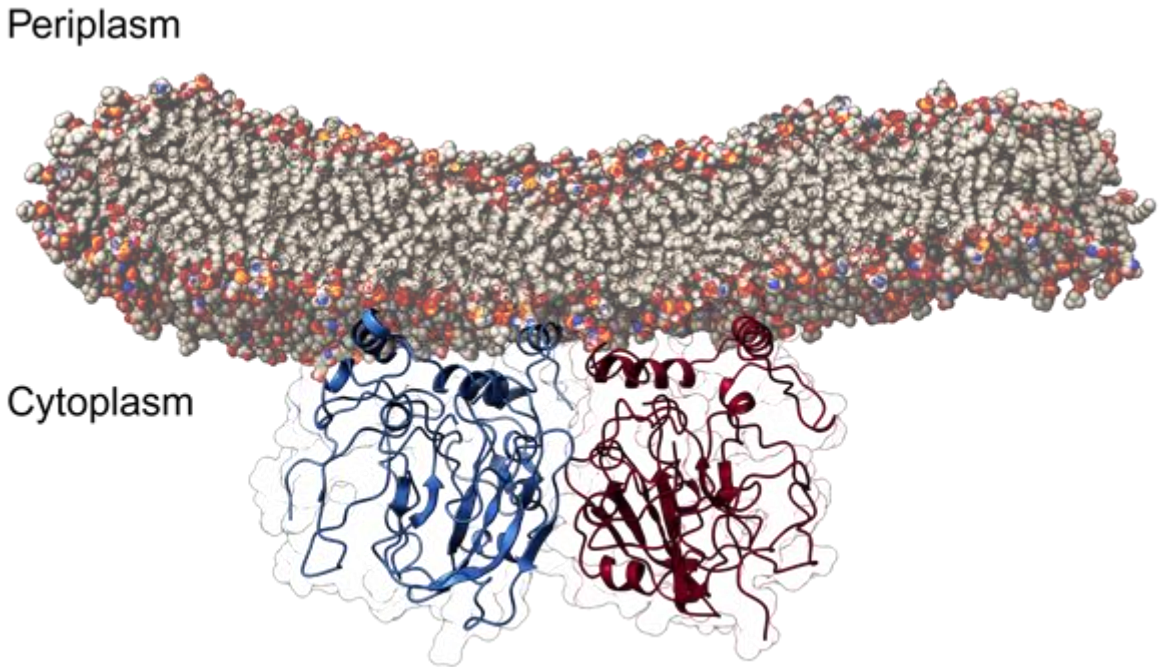

Intricate Folding Pathways of the Bacterial Conjugation Enzyme TrwC Uncovered by Force Spectroscopy

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ECR Session 3, July 12, 2026, 14:45 - 15:45

Bacterial conjugation, the contact-dependent secretion of DNA from a donor cell to a recipient cell, is one of the main roads for horizontal gene transfer and spreading of virulence factors and antibiotic resistance genes. One crucial model enzyme for bacterial conjugation is the relaxase enzyme TrwC, a large 966 amino acids long multidomain protein with various activities. TrwC recognizes the origin of transfer within its conjugative plasmid, primes and covalently binds DNA. Likely, a second TrwC molecule unwinds the conjugative plasmid, and the covalently bound TrwC is secreted together with the single-stranded DNA towards the recipient cell. Successful secretion of the TrwC-DNA complex is a tightly controlled mechano-chemical process powered by molecular motors in the Type IV Secretion System (T4SS), which requires the unfolding of TrwC due to the constrictive nature of the secretion channel. Here, we studied the mechanical unfolding of TrwC using high resolution magnetic tweezers. We discover multiple folds with stabilities ranging from 5 pN up to 100 pN, large energy dissipation, and intricate folding pathways. Split and truncated variants of TrwC show stable folds with enriched pathways by inter-domain interactions. Refolding is surprisingly fast for the size of TrwC, which is likely enabled by independent subdomain folding. Finally, we observed how DNA regulates mechanical stability of TrwC, suggesting that mechanical stability would allow the T4SS to discriminate between TrwC and TrwC-DNA. Our findings provide novel insights on TrwC mechanobiology, which we anticipate will allow a better mechanistic understanding of bacterial conjugation crucial for our race against antimicrobial resistance.

The Interaction of Permeation Enhancers and the Skin Barrier

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ECR Session 4, July 12, 2026, 16:00 - 16:45

The stratum corneum (SC), the outermost layer of the skin, acts as an effective barrier to permeation of chemicals both into and out of the skin. Topical drug formulations often include permeation enhancers such as propylene glycol (PG) and isopropanol (IPA) to improve delivery of the active ingredient through the skin barrier. To understand how PEs interact with and influence the structure and organisation of the lipid lamella observed in the SC, molecular dynamics simulations have been conducted to examine the effects of PG and IPA.
Model SC bilayers containing Ceramide Non-hydroxy fatty acid/Sphingosine (Cer[NS]24), lignoceric acid (C24), and cholesterol in a 1:1:0.5 molar ratio have been studied in contact with water containing different concentrations of PG (0–100 wt%) and IPA (0–20 wt%). PG/water mixtures caused measurable structural changes, including increased area per lipid and reduced bilayer thickness, indicating disruption of lipid packing but limited broadening of the interfacial region. In contrast, IPA was found to have a limited effect on lipid packing but significantly disrupted interfacial order, broadening the interface and increasing lipid mobility. Solvents containing mixtures of PG and IPA exhibited non-additive effects: the significant interfacial disruption seen with IPA alone was diminished in the presence of PG, while the systems became more structurally heterogeneous. These findings demonstrate that permeation enhancers operate through distinct yet interconnected mechanisms, emphasising how their combined presence modifies bilayer organisation beyond the effects of each component individually. This work adds to a mechanistic framework for understanding enhancer-lipid interactions in the skin barrier.

LILACs: Looking Inside Living Algal Cell Walls

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ECR Session 4, July 12, 2026, 16:00 - 16:45

The macroalgae, or seaweeds, are unusually resilient organisms that deal with extreme environmental changes on a tidal cycle. The biopolymers that make up their outer coverings - their cell walls - have evolved to cope with these extreme changes. However, in comparison to land plants, we know relatively little about the composition and mechanics of marine-adapted algal cell wall polymers. To address this gap, we are using a combination of biophysical approaches to study the marine biopolymers found in the common green 'sea lettuce', Ulva. Firstly, we are extracting and chemically characterising the protein and polysaccharide components of the Ulva cell wall using chemical analytical methods (mass spectrometry and NMR). Secondly, the biophysical properties of the extracted structural polysaccharides are being studied using novel microrheological methods to provide insight into polymer gelation across length and time scales. Further work will focus on how extracted proteins can alter these properties. Third, and finally, direct imaging of cell autofluorescence is being used to observe the impact of changing environments on polymer behaviour in vivo and we are using the extracted protein sequences to develop fluorescent molecular rotors that will allow us to image changes in local viscosity. Insights will shed light on the mechanical properties of the marine-adapted structures that support life in climate change-sensitive coastal ecosystems, while also exploring novel biopolymers with a range of tuneable structural properties that may help to develop sustainable materials, for instance in bio packaging.

Double-Strand Break Stabilisation by PARP Proteins

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ECR Session 4, July 12, 2026, 16:00 - 16:45

Poly ADP-ribosylation polymerases (PARPs) are DNA-strand-break-sensing proteins frequently targeted in BRCA mutation-associated cancer treatment. PARP inhibitors (PARPi) bind PARP–DNA break complexes, trapping PARPs at breaks and inhibiting the site repair otherwise triggered. BRCA-mutant cells, with dysfunctional homologous recombination pathways, accumulate unrepaired double-strand breaks (DSBs) leading to cell death. Off-target effects are common for PARPi and may stem from lesser-understood PARP2-DSB interactions as PARP1, being the more well-understood paralogue, is often used to characterise PARPi affinities and effects. Previous research found that PARP2-DSB affinity is highly dependent on structural characteristics of DSBs.

In this project we used magnetic tweezers to investigate how PARP2-DNA binding varies with DSB structure. We found that reducing the overhang length of DNA flanking the DSB by only one base (4 to 3 bases) decreased the possible configurations of PARP2 on the DSB from two to one. This suggests that PARP2 stabilisation of DSBs can happen via multiple configurations and that not all PARP2 binding modes are possible at all DSB structures. PARP2 affinity for DSBs therefore appears more sensitive to DSB structure than PARP1, potentially due to PARP1's much more extensive DNA-binding domains, highlighting the complexity of effective PARPi characterisation.

For this work, I also developed a more stable surface-tethering technique for DNA. Replacing the typically used digoxigenin-antidigoxigenin interaction, this system uses covalent HaloTag protein-ligand chemistry to increase tether durability and lifetime. This high stability enabled repeated force ramps reaching 120 pN, within which we observe the overstretching of torsionally constrained B-DNA (at ~110 pN).

Innovative teaching from microscope to extended reality

Professor Craig Daly[1]
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Extended Reality for Life Science Skills Training: Session 1, July 15, 2026, 11:00 - 13:00

Virtual reality (VR) and related extended reality (XR) technologies have matured rapidly over the past decade. Head-mounted displays have become lighter, more capable and, in many cases, more affordable, making multi-station XR teaching laboratories increasingly feasible within higher education. However, one of the major barriers to wider curricular adoption remains the limited availability of high-quality, discipline-specific teaching content. Recent advances in AI-assisted code generation may help lower the technical barriers to XR content creation.

The University of Glasgow has invested substantially in XR facilities for both research and teaching, and many thousands of students have now experienced learning in VR. Current physiology-related applications include molecular methods, gut physiology, infection biology, hypertension and cardiac myocyte function. In addition, a new collaboration with Zeiss has enabled the development of virtual training environments for confocal and electron microscopy.

This presentation will describe a workflow for converting microscope-based scientific content into VR experiences for head-mounted displays and will reflect on lessons learned from nine years of developing immersive teaching materials for the life sciences. The work highlights both the opportunities and the practical challenges of embedding XR into life science education, with particular emphasis on creating scientifically rigorous, reusable content that supports higher-education teaching.

XR-TIE Summary - Towards a Comprehensive Framework for XR Learning

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Extended Reality for Life Science Skills Training: Session 1, July 15, 2026, 11:00 - 13:00

Since 2016 the University of Glasgow has invested into XR provisions for teaching and research. The existing XR infrastructure is soon to be expanded and improved under a new organisation called RIG (Realities and Immersion Glasgow). This organisation will coordinate facilities for XR teaching and learning, while adding a development team. Over 1200 students every year make use of the teaching facility with an equivalent number of researchers using ARC-XR. With increasing focus on XR in learning there is a requirement for a comprehensive means of assessing the efficacy teaching interventions

The XR Teaching Intervention Efficacy (XR-TIE) project is currently with the aim of developing a validation framework for doing that. To date, there is no single standardised approach to evaluating XR systems for educational ends. The value of XR for education is based on an increasing number of heterogenous studies with markedly ontological assumptions. What defines XR and what are its affordances? Studies take different methodological approaches. How do you measure the success of a XR teaching intervention? The practical context of each XR teaching intervention is different. What are the pedagogical considerations?

We will establish a methodological framework for the assessment of XR teaching interventions. This will provide a unified and consistent ontological approach. It will provide guidance on aligning the affordances of XR with theories of learning. And finally, it will provide practical guidance on the design and deployment of XR teaching interventions. This framework draws on existing research on the validation of teaching interventions and adapt it for the specialised context of immersive technology. The second strand of the project is designed to apply the XR-TIE framework to three specific XR interventions at the University of Glasgow.

Virtual Reality peer-to-peer learning: a pilot study investigating peer-to-peer VR supported lessons in Higher Education through a staff-student partnership

Dr Claire Donald[1], Nicola Veitch[1], Sevinj Rustamova[1], Lavinia Hirsu[1], Gabriella Rodolico[1], Imants Latkovskis[1], Neil McDonnell[1]
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Extended Reality for Life Science Skills Training: Session 2, July 15, 2026, 14:00 - 16:00

Virtual reality is a digital learning tool in Higher Education where users benefit from immersion within interactive environments. Peer-to-peer learning is a well-established pedagogical approach known to improve academic outcomes, social skills, and cost-saving. While both VR and peer learning have demonstrated benefits individually, limited research has examined how these approaches can be meaningfully integrated. This study implemented an evidence-based, peer-to-peer learning strategy within a VR lesson and proposes a supportive framework for its broad implementation within HE teaching. A staff-student partnership was funded by an SEB Education and Research development grant to use a bespoke Disease Diagnostic VR simulation which focuses on quantitative Polymerase Chain Reaction to evaluate the research. Student participants were randomly assigned into two groups: those who worked individually through the VR lesson and those who worked in pairs and shared a single VR headset. We investigated the effects each approach had on learning, engagement and student confidence through pre- and post- activity questionnaires. The results of the pilot study will be presented during the session. This pilot study highlights the needs for a structured learning framework to scaffold student learning while participating in VR lessons. These insights inform the development of a pedagogical framework for embedding peer learning in VR lessons which can be evaluated in the future.

When Cells Run Out of Space: A Minimal In-Silico Model of Crowding-Regulated Cell-Cycle Progression and Migration

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[1]University of Glasgow, United Kingdom, [2]University of Coimbra, Portugal
In-silico Mechanomedicine: Session 1, July 14, 2026, 11:30 - 12:30

Cell proliferation and migration underpin numerous physiological and pathological processes, including tissue development, wound healing and cancer progression. Increasing evidence demonstrates that cell-cycle progression is regulated by crowding-induced contact inhibition, whereby cells arrest in G0/G1 as available space for division becomes limited. However, many minimal mathematical models incorporating cell-cycle dynamics do not explicitly account for this phenomenon and can predict biologically unrealistic long-term distributions of cells across cell-cycle phases.

We present a new continuum model of cell proliferation that incorporates crowding-regulated cell-cycle progression. The model considers two cell populations corresponding to the G0/G1 and S/G2/M phases and introduces a novel density-dependent transition term that acts as a mathematical representation of contact inhibition. Unlike existing minimal formulations, the proposed transition law explicitly prevents cells from entering proliferative phases when local crowding indicates insufficient space for successful division.

Mathematical analysis demonstrates that the model reproduces the experimentally observed accumulation of cells in G0/G1 as carrying capacity is approached, whereas alternative minimal models predict unrealistic phase distributions at long times. Parameters are inferred from published pancreatic cancer cell-line data, and model results are compared against measurements of both total cell number and cell-cycle phase composition.

The framework is further extended to incorporate cell migration through a continuum transport formulation, and comparisons are made with tissue expansion data.

Despite containing only three fitted parameters, the model provides improved biological fidelity and strong agreement with experimental observations, establishing a computationally efficient in-silico framework for studying crowding-regulated tissue growth and a foundation for future predictive tools in mechanomedicine.

Simulation-augmented atomic force microscopy of cells

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[1]University of Glasgow, United Kingdom

In-silico Mechanomedicine: Session 1, July 14, 2026, 11:30 - 12:30

Mechanobiology studies how physical forces influence pathophysiological processes across scales [1]. Cell and extracellular mechanical properties are central: substrate elasticity guides stem-cell differentiation, while single-cell deformability indicates physiological state and supports diagnostic and therapeutic applications [2].

Cell elasticity is commonly identified by atomic force microscopy (AFM) nanoindentation. However, standard analysis fits analytical models treating cells as homogeneous, isotropic, linear elastic solids. Combining nanoindentation with finite-element analysis (FEA) captures heterogeneity and nonlinear behaviour, improving interpretation of experimental data [3].

Our objective is to improve the reliability of mechanical parameters extracted from AFM experiments by developing an experimentally informed numerical model. MoFEM, an open-source parallel finite element library [4], is used as the FEA engine, enabling accurate contact simulations with arbitrary indenter shapes. Hyperelastic and viscoelastic materials undergoing large deformations are included through MFront [5].

We demonstrate the model using two examples. The first extracts viscoelastic properties of a hydrogel. For such materials, the analytical contact model can be extended to viscoelasticity only for instantaneous indentation followed by long relaxation [3], requiring experiments lasting minutes. In contrast, FEA can extract parameters from only indentation, reducing experiments to seconds or less.

The second example concerns mechanical characterisation of post-infection changes in the fly intestinal tract. The numerical model represents organ geometry, multilayered structure and heterogeneous properties, enabling more accurate parameter identification than analytical approximations.

Therefore, finite element simulations can augment the AFM nanoindentation pipeline and increase the precision of extracted viscoelastic properties. Future work will incorporate real surface topography.

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Trends and variations in human aortic biomechanical properties: a UK biobank analysis

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[1]Glasgow Computational Engineering Centre, University Of Glasgow, United Kingdom
In-silico Mechanomedicine: Session 2, July 14, 2026, 14:00 - 16:00

Introduction

Biomechanics of the aorta plays a key role in development and long-term outcome post treatment of serveral cardiovascular diseases. However, it is currently not fully understood and not taken into account in clinical decision making. One challenge in incorporating biomechanics as a clinical tool is its variation: spatial and across the population. The properties of the aortic root, ascending aorta, and descending aorta are known to be different. Moreover, these properties vary substantially from person to person, even in the healthy state. In this study, we present an analysis based on a large dataset with the aim to quantify the trends and variations in mechanical properties of the general population's aorta.

Materials and Methods

We used 80,000 MR images from the UK Biobank and used a pre-trained U-Net model to segment the ascending and descending aorta, from which their in-vivo lumen areas and diameters were measured. From the diameters, the aortic distensibility was derived. Next, we fit a Gaussian-mixture model to determine the probability distribution of aortic area, distensibility, and demographical factors. We used information theoretic metrics to quantify dependence between various factors. Lastly, we combined the in-vivo dataset is combined with ex-vivo dataset of aortic tissue properties to infer the distribution of aortic constitutive parameters and stresses in the different aortic tissue.

Results

We find distensibility and aortic size are negatively correlated. With age aorta dilates and becomes stiffer. However, with larger body surface area (height or weight), only the aortic diameter increases. Aortic distensibility decreased with systolic blood pressure. Aortic wall stresses and associated uncertainty were calculated for any given aortic size and distensibility.

Discussion

These results provide first insight into the aortic properties, and will form the basis of future investigations into their role in medicine.

Integrating MD simulations with PELDOR/DEER spectroscopy and cryoEM reveals the dynamics and energetics underlying mechanosensation

Qaiser Waheed[1], Joshua Wort[1], Yu Ma[1], Xinyu Liu[1], Christos Pliotas[1]
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In-silico Mechanomedicine: Session 2, July 14, 2026, 14:00 - 16:00

Mechanosensitive ion channels respond to membrane physical properties through large-scale conformational transitions that couple protein dynamics to lipid bilayer mechanics. However, the energetic and structural mechanisms governing these transitions remain poorly understood. Here, we combine molecular dynamics (MD) simulations with PELDOR/DEER spectroscopy and cryoEM to investigate the membrane-dependent conformational landscapes of two mechanosensitive ion channels: the bacterial mechanosensitive channel of large conductance (MscL) and the mammalian K2P channel TRAAK.

For EcMscL, cryoEM structures resolved closed and expanded conformations in lipid nanodiscs (1), while MD simulations in bilayers and nanodiscs revealed that membrane thickness, leaflet interdigitation, and hydrophobic mismatch strongly influence channel expansion. Simulations showed that nanodiscs facilitate MscL opening by thinning the bilayer and relieving hydrophobic mismatch, thereby stabilising expanded conformational states.

For TRAAK, PELDOR/DEER spectroscopy revealed coexisting swapped and non-swapped extracellular cap conformations with temperature-dependent population shifts (2). MD simulations demonstrated that the associated energetic differences arise from both intrinsic conformational rearrangements and membrane deformations induced by the channel. Analysis of effective protein–membrane non-bonded interaction energies, including Lennard-Jones and electrostatic contributions, revealed how membrane mechanics and lipid-dependent interactions govern the conformational equilibrium.

Together, these studies demonstrate how multiscale MD simulations integrated with structural and spectroscopic techniques can resolve the energetic basis of mechanosensation by linking membrane physical properties to conformational dynamics and functional state transitions in ion channels.

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Prokaryotic origins of mechanosensitive channels in plants

Boris Martinac[1]
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Plant Mechanobiology 1, July 15, 2026, 09:00 - 10:30

Mechanosensitive channels are membrane-embedded protein complexes that function as primary mechanoreceptors, converting mechanical stimuli into electrochemical signals that influence cellular processes, including gene expression. Through this function, mechanosensitive channels enable cells to detect and adapt to mechanical stimuli such as touch, sound, stretch, or osmotic pressure. In bacteria, osmotic pressure imposes substantial mechanical stress affecting the cell membrane. To survive hypo-osmotic shock, bacteria have evolved mechanosensitive channels that relieve excessive turgor pressure by opening and releasing osmolytes and water, thereby restoring osmotic balance and preventing cell lysis. Several types of mechanosensitive channels have been found in Escherichia coli, a key model organism for studying cellular mechanotransduction. Based on their primary structure, mechanosensitive channels of E. coli are classified into two major subfamilies: the MscL (large-conductance) and MscS (small-conductance) family. MscL homologues are found across a wide range of organisms, including both cell-walled and wall-less bacteria, Archaea, fungi, and eukaryotic oomycetes. In contrast, MscS homologues are more broadly distributed, extending to higher plants and unicellular green algae. Multiple MscS-like channels often coexist within a single organism. For instance, E. coli has six MscS-like proteins, while Arabidopsis thaliana encodes ten MscS-like (MSL) channel proteins.

In plants, MSL channels have diversified to perform a wide range of functions. These include osmoregulation (as in bacteria and Archaea), mechanosensory roles in touch-sensitive species such as the Venus flytrap, root mechanosensing, maintenance of chloroplast and mitochondrial integrity, gravity perception, and defence against pathogens. This evolutionary “tinkering” has thus repurposed the ancestral bacterial MscS channel from its primordial role in osmoregulation to a variety of mechanotransduction functions in plants. Hence, the MscS-like superfamily is a remarkable example of molecular evolution and serves as an invaluable system for studying membrane protein adaptation in mechanobiology of plants, as well as for developing novel biotechnological applications such as biomolecular nanodevices.

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Structured Transport and Accessibility Dynamics in the Oxygen-Evolving Complex of Photosystem II

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Plant Mechanobiology 1, July 15, 2026, 09:00 - 10:30

Structured Transport and Accessibility Dynamics in the Oxygen-Evolving Complex of Photosystem II

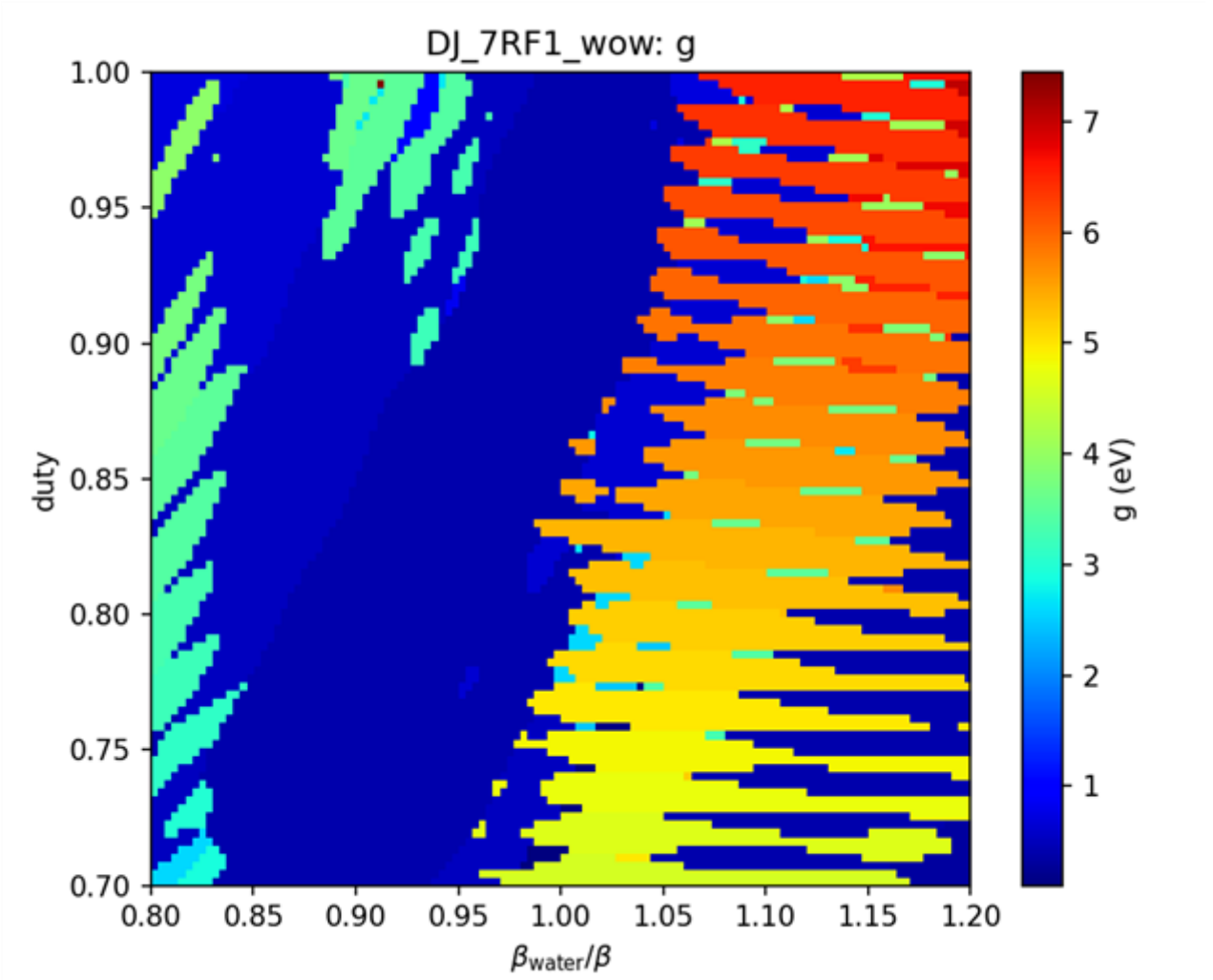
The oxygen-evolving complex (OEC) of photosystem II represents one of the most highly coordinated catalytic systems in biology, integrating multi-centre redox chemistry, proton transport, structural reorganisation, and ultrafast environmental coupling within the CaMn4O5 cubane-cluster. Despite major advances in XFEL structural analysis and catalytic cycle reconstruction, important questions remain concerning how organised transport behaviour and perturbation-sensitive transitions emerge across the catalytic manifold.

Here we present an exploratory geometry-guided accessibility framework originating from systems-oriented pharmaceutical and catalytic transport modelling. Rather than treating catalytic progression solely as a sequence of local state transitions, the approach models the OEC as a constrained accessibility landscape in which transport persistence, recurrence structure, and environmental ordering collectively influence catalytic evolution.

Applied to published OEC structural and perturbation regimes, the framework identifies unexpectedly organised bifurcation behaviour associated with hydrogen isotope substitution and selected chalcogenide-related perturbations. These transitions exhibit non-random segmentation patterns suggestive of coordinated accessibility restructuring rather than purely stochastic catalytic degradation. Accessibility surfaces reconstructed from the framework display curvature-sensitive transition zones and quasi-stable transport channels across specific energetic configurations.

Comparative analysis with recently reported organised transport and shielding-like persistence phenomena in structured condensed-matter systems reveals notable geometric parallels in recurrence stability and perturbation tolerance despite substantial physical differences between the systems. These observations motivate the possibility that constrained accessibility organisation may provide a useful descriptive layer linking catalytic coherence, transport stability, and emergent ordering behaviour across diverse complex systems.

The framework is not intended as a replacement for established quantum chemical or biochemical models, but as a complementary organisational formalism for identifying latent regime structure within highly coordinated catalytic environments. More broadly, the work aims to contribute to emerging interdisciplinary dialogue between photosynthetic catalysis, systems biophysics, geometry-informed modelling, and coherent transport theory.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Walls biomechanics underpin plasmodesmata biological function

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Plant Mechanobiology 2, July 15, 2026, 11:00 - 12:30

Plant cell walls exist as a complex and varied blend of polysaccharides and proteins; the combination of which has evolved over millions of years. Research on how these components interact is key to understanding a plant's mechanical, structural, communicative, and biological traits. However, knowledge on cell wall components, its biophysical properties and cellular functions remains sparse. Particularly challenging is the analysis of cell wall microdomains such as plasmodesmata. Plasmodesmata are membranous bridges embedded in cell walls facilitating cytoplasm-to-cytoplasm (i.e., symplasmic) transport of diverse factors, including proteins and signalling molecules that control plant development. Here, we review recent research on plasmodesmata cell walls connecting structural and mechanical properties of their components and evidence of their function at plasmodesmata. Most work in this area focuses on callose (a beta-1,3 glucan that accumulates at plasmodesmata), but compositional and proteomic analysis indicate interplay with wall pectins, xyloglucans and cellulose structures that remains under-investigated. We discuss the importance of understanding polymer interactions at the molecular and biophysical level and their relevance for plasmodesmata biomechanics. We also highlight new techniques and outstanding questions and reflect on the opportunities for translation of knowledge in the improvement of plant traits and in biomaterial design.

Negative Capacitance in Microbial Biofilms

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The Physics of Microorganisms: Session 1, July 14, 2026, 09:30 - 11:00

We investigated electrical impedance spectroscopy of biofilms formed by Gram-negative bacteria, Gram-positive bacteria, and a fungus, including Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Enterococcus faecalis, Staphylococcus epidermidis, and Candida albicans. Across all tested species, we observed low-frequency negative capacitance under small applied DC bias voltages. This response was not organism-specific, but instead appeared to be a general feature of biofilm–electrode systems.

The impedance spectra were reproduced using an equivalent circuit containing a constant phase element and an inductive branch. The constant phase element captures the heterogeneous biofilm–electrode interface, while the inductive branch represents delayed ionic or electrochemical dynamics responsible for the negative-capacitance response. Although electrode–electrolyte coupling strongly shapes the spectra, the results are also consistent with biologically regulated ion transport, including ion-channel-mediated electrical activity previously reported in E. coli biofilms.

Overall, these findings suggest that negative capacitance is a robust electrical signature of microbial biofilms, arising from coupled biological, ionic, and interfacial processes.

Using solid-state NMR to probe intact fungal cell wall glucans in different motion regimes

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The Physics of Microorganisms: Session 1, July 14, 2026, 09:30 - 11:00

Understanding how microorganisms build and remodel their cell surfaces remains a major challenge, particularly for fungi, which have an insoluble, heterogeneous cell wall that is difficult to probe in intact conditions. Solid-state NMR spectroscopy offers unique atomic-scale insight into such complex biological materials, but the cost and complexity of isotopic labelling strategies have limited widespread application. By developing cost-effective sample preparation approaches, we acquired two-dimensional ^{13}C-^{13}C solid-state NMR spectra directly from intact fungal cells. We verified the degree of biological and experimental variability using this approach.

Using 2D NMR on intact cells, we investigated the molecular organisation and dynamics of fungal cell walls in multiple organisms. In the fission yeast Schizosaccharomyces pombe, we studied mutants associated with specific glucan synthases, including Ags1, Bgs1, and Bgs4, enabling direct comparison of altered cell wall architectures arising from perturbation of glucan biosynthesis pathways. We additionally examined Aureobasidium pullulans, whose morphology changes substantially depending on growth medium, allowing correlation of macroscopic phenotype with molecular-scale structural adaptations.

We showed that the intact cell wall glucans exist in at least two distinct motional populations. One population is relatively rigid, with motions slower than approximately microseconds, while another is considerably more mobile, with motions faster than nanoseconds. These populations can be selectively observed using dynamics-edited solid-state NMR experiments, introducing a powerful additional dimension of contrast beyond chemical composition alone. Our work highlights the importance of probing intact fungal cell wall components across different timescales.

Why Do Bacteria Build Biomolecular Condensates? Physics, Function and Synthetic Aggresomes

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The Physics of Microorganisms: Session 2, July 14, 2026, 11:30 - 12:30

Biomolecular condensates are emerging as ubiquitous organising principles across all domains of life, yet the physical mechanisms governing their assembly, dynamics and biological function remain incompletely understood. In this talk I will present bacterial aggresomes as a powerful quantitative model system for uncovering these principles. Combining single-molecule microscopy, quantitative biophysics, computational modelling and theory, we have investigated how aggresomes self-assemble in response to diverse cellular stresses and characterised their physical properties across molecular and cellular length scales. Our work reveals that aggresomes comprise both proteins and RNA, and identifies a key biological function: the protection of messenger RNA from enzymatic degradation during stress, thereby promoting cellular survival. These findings establish bacterial aggresomes as an experimentally tractable platform for linking physical mechanisms of biomolecular condensation with biological function. Finally, I will discuss how these insights are beginning to enable the rational engineering of synthetic aggresomes capable of storing RNA cargo, opening opportunities for stabilising RNA-based therapeutics and illustrating how fundamental biological physics can inspire new biotechnology.

Title: dSTORM characterisation of membrane distribution in colistin-persistent bacteria

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The Physics of Microorganisms: Session 2, July 14, 2026, 11:30 - 12:30

Antimicrobial resistance (AMR) is an increasing threat to global health, capable of undermining antibiotic efficacy and complicating treatment of infections. Gram-negative bacterial pathogens, including nine of the fifteen WHO-designated 'bacterial priority pathogens', cause a wide range of infections and are prone to multidrug resistance. Colistin is an antibiotic of last resort against multidrug-resistant Gram-negative bacteria, targeting lipopolysaccharides (LPS) within the outer membrane (OM). The threat of antibiotic persistence, where a heterogeneous subpopulation within a susceptible clonal population survives antibiotic killing without acquiring resistance genes or undergoing mutagenesis, remains largely invisible as standard diagnostics fail to detect it. In this study, we aimed to characterise the distinct features of colistin-persistent bacterial cells at the nanoscale.
We utilised direct stochastic optical reconstruction microscopy (dSTORM) to study the nanoscale distributional changes of the outer membrane and LPS in individual bacterial cells with a resolution of 20–30 nm. The OM was labelled using a lipophilic styryl dye FM4-64 and the rhodamine B-labelled polymyxin B LPS probe respectively. Escherichia coli and Pseudomonas aeruginosa persisters were isolated following exposure to high doses (16×MIC) of colistin and displayed heterogenous OM organisation and strong LPS clustering when compared to their susceptible counterparts. The shared morphology between E. coli and P. aeruginosa persister cells indicates that LPS clustering may be an effective defence against antibiotics, and this work identifies a rare morphological indicator of persistence.
To advance this work in a cost-effective manner, we have migrated our dSTORM data analysis from a costly commercial software package to a workflow consisting of open-source software and in-house-built Python scripts. Ultimately, this work expands our characterisation of clustering phenotypes and provides a framework to aid future diagnostic development.

Labyrinthula: An underwater microbial railway network

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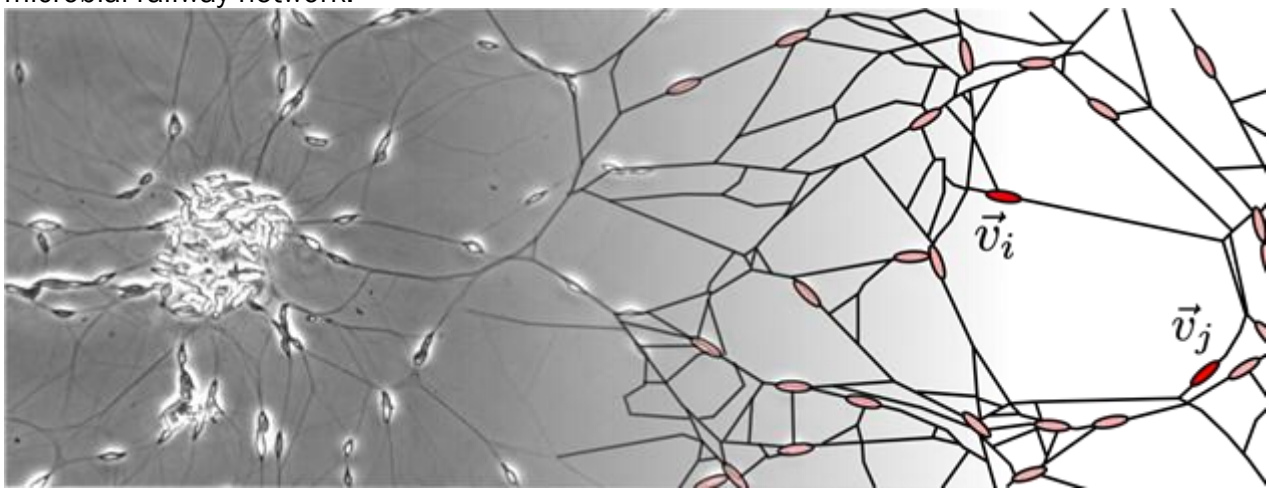
The Physics of Microorganisms: Session 3, July 14, 2026, 14:00 - 16:00

Labyrinthula species are protistan organisms found predominantly in coastal marine environments, notably as residents on seagrass leaves. A fascinating characteristic of this order, observed over a century ago but little studied since, is the ability for cells to secrete an extracellular ectoplasmic net. This allows colonies to form a spatial network of interconnected extracellular filaments across a substrate. Individual Labyrinthula cells are confined within these filaments and move independently about this network. We introduce a novel pipeline for tracking the development and organisation of this colony morphology that we describe as a microbial railway network.



Origins and Consequences of Variability in the Life History Parameters of a Lytic Bacteriophage

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The Physics of Microorganisms: Session 3, July 14, 2026, 14:00 - 16:00

Bacteriophages (phages) play a critical role in controlling bacterial populations, both in natural ecosystems and as potential therapeutic agents. Their success in eliminating target cells and proliferating depends on key 'life history parameters', including burst size (the number of virions produced per infected cell) and lysis time (the time taken to lyse the host cell). Life history parameters are constrained by biology, which imposes
both absolute and relative limits on the values these parameters may take, leading to an environment-dependent system of evolutionary trade-offs.
Historical study of bacteriophage life history parameters has focused on populationaverage values for bacteria infected while growing in ideal laboratory conditions. While illuminating, this approach fails to account for the fact that phage-bacteria interactions are both intrinsically stochastic in nature and highly environmentally-dependent.

In the studies discussed, we combine agent-based stochastic simulations with single-cell laboratory work to determine how LHPs vary both intrinsically, and in response to external factors such as the availability of nutrients to the host cell, as well as the evolutionary consequences of such variation. In simulation, we uncover unexpected and rich competitive dynamics, while in laboratory studies we find both expected and surprising correlations.

Keywords: evolution, bacteriophage, agent-based modelling, simulation, microfluidics, stochastic, resource-limitation.

Pole positions for porins and polymyxins: mapping outer membrane organisation from single trimers to whole cells in living Escherichia coli

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The Physics of Microorganisms: Session 3, July 14, 2026, 14:00 - 16:00

Infections associated with antimicrobial resistance represent an increasing risk to global health and are projected to surpass cancer as a cause of mortality by 2050[1]. Gram-negative bacteria dominate the World Health Organisation's list of critical strains to monitor[2] due partly to their cell envelope, which contains an additional "outer" membrane. This structure, an asymmetric mix of phospholipids, outer membrane proteins (OMPs) and lipopolysaccharides, acts as a molecular sieve[3] and structural support[4], protecting the cell from many antibiotics[5], while permitting rapid cell growth and division.

Recent data have shown the multi-scale complexity of the outer membrane: in Escherichia coli, OMP trimers form phase-separated, ordered networks across the cell[6]. However, we are yet to discover how these originate and evolve and how such dynamic heterogeneity is linked to cell viability or membrane integrity.

Here, we use high-resolution atomic force microscopy (AFM) to directly map the organisation of the outer membrane on the scale of single OMP trimers (~nm), up to entire cells (~μm) in living E. coli. We find that the structure of the proteinaceous network depends on the position relative to the poles, as well as cells' metabolic state. Finally, we link this variation in outer membrane composition to susceptibility to polymyxin B[5], a last-resort antibiotic. Our results begin to unveil the interplay between outer membrane biophysics, cell cycle and energetics, and subsequent response to antibiotics, illustrating the need for such multi-scale characterisation.

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How Do Simple Stochastic Switches Help Biofilms Survive Antibiotics?

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The Physics of Microorganisms: Session 4, July 14, 2026, 16:30 - 17:30

Biofilms are the most common form of communal bacterial growth, which contains clusters of bacterial cells embedded within a matrix of extracellular polymeric substances. They exhibit diverse phenotypic heterogeneity, often resulting in spatially organized gene-expression patterns even within clonal populations. While this organization is traditionally considered to environment-responsive phenotypic switching, the underlying mechanistic origins and evolutionary advantages remain poorly understood. Using the matrix-flagellated phenotypic switch in Bacillus subtilis as a model system, we investigated how these spatial patterns emerge and how they drive overall biofilm resilience against environmental stress.
By integrating experimental observations with physical modeling, we compared wild-type biofilms against engineered mixtures of flagella- and matrix-null mutants. Surprisingly, our results reveal that the native, spatially organized pattern does not require an environment-responsive switch to develop. Instead, the architecture can be explained by a stochastic switch coupled to a fitness landscape that selects specific phenotypes at the population level.
This local coexistence between flagellated and matrix-producing cells allows the biofilm to actively manipulate its own morphology and internal cell transport properties, functioning similarly to a primitive vascular system. Crucially, when we introduced sudden antibiotic perturbations, the biofilm utilized this structural network to release resistant mutants into the surroundings, which had previously appeared trapped.
Ultimately, our findings demonstrate that simple stochastic phenotypic switches can drive collective, emergent behaviors that enhance a population's adaptability far beyond traditional bet-hedging strategies. This study highlights how the biology and ecology of biofilms are intrinsically tied to their physical properties, offering new insights into how phenotypic diversity shapes communal bacterial survival.

Nanoplasmonic Surface-Enhanced Raman Spectroscopy (SERS) for Signalling Molecule Detection Directly in E. coli Cultures

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The Physics of Microorganisms: Session 4, July 14, 2026, 16:30 - 17:30

Tryptophanase (TnaA) is a promiscuous enzyme which regulates the production of amino acid-derived metabolites, essential for bacterial communication. TnaA is known to convert tryptophan into indole, an important signalling molecule influencing a range of behaviours - from bacterial quorum sensing to urinary tract infections (UTI) - but other conversion routes remain obscure. Here, we show that nanoplasmonic surface-enhanced Raman spectroscopy (SERS) combined with targeted genetic subtraction enables the label-free, cellular characterisation and structural identification of TnaA-derived metabolites directly from Escherichia coli culture solutions, without isolation or purification. We systematically profile the SERS spectra of wild-type and tnaA gene knockout strains (E. coli BW25113 and the uropathogenic 536) supplemented with each of the twenty amino acids, uncovering a previously unreported TnaA-dependent metabolic signature, I*, whose Raman fingerprint does not match free indole or any common indole derivative characterised by mass spectrometry. These findings demonstrate that nanoplasmonic SERS provides a powerful framework for probing enzyme activity and bioactive metabolite production directly from living bacterial cultures with nanomolar sensitivity and structural specificity.