IndiaBioscience

DEPARTMENT OF BIOTECHNOLOGY
Ministry of Science & Technology

Academic Partner
SYMBIOSIS INTERNATIONAL (DEEMED UNIVERSITY)

18th Young Investigators' Meeting 2026

2-6 March 2026

Pune

ABSTRACT BOOK

Warli art is a traditional tribal art form from Maharashtra, characterised by simple geometric shapes to portray everyday rural life and nature. In this artwork, Warli-inspired motifs are adapted to illustrate the dynamic ecosystem of the life science community. The graphic is designed by Moumita Mazumdar.

IndiaBioscience

IndiaBioscience fills a unique niche in the ecosystem of the life sciences in India by being a catalyst to promote the change that affects the culture and practice of the field through engagement with academia, government, and industry at various levels.

www.indiabioscience.org

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IndiaBioscience is thankful for the support they received from:

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जैवप्रौद्योगिकी विभाग
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सत्यमेव जयते

The YIM 2026 Abstract Book was developed by **Moumita Mazumdar** and **Siuli Mitra** with inputs from **Manjula Harikrishna**.

The graphic design of the book was done by **Moumita Mazumdar**.

About IndiaBioscience

IndiaBioscience

IndiaBioscience is an organisation that fills a unique niche in India's life science ecosystem by being a catalyst for change that shapes the culture and practice of the field, through engagement with academia, government, and industry at various levels. IndiaBioscience aims to increase the visibility of science in society by serving as a hub for policy discussions and science communication, and as an aggregator of information.

IndiaBioscience plays an administrative and organisational role in each year's Young Investigators' Meeting, but its engagement with the participants extends beyond the meeting. IndiaBioscience aims to forge a long-standing bond with YIM alumni to support their career development and foster the growth of their research groups. Through this sustained ripple effect, it aims to create a meaningful and lasting impact on the life sciences research ecosystem in India.

www.indiabioscience.org

Engage with us

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The Young Investigators' Meeting Series

Building a community of young Indian biologists.

The YIM series aims to build a vibrant community of biologists by providing a platform for young Indian scientists from different regions and institutions to establish friendships, initiate collaborations, and learn through peer mentorship.

The annual Young Investigators' Meeting (YIM) brings together young investigators and senior scientists, heads of institutes, and representatives from funding agencies for **four and a half days** of discussions and interactions focused on science and careers across a broad range of biological disciplines, as well as mentorship and networking.

Since its inception in 2009, YIM has provided a vibrant space for networking and exchanging ideas to catalyse collaborations among life scientists in India. The YIM series also pairs Postdoctoral Fellows (PDFs) from India and abroad with Young Investigators (YIs) and senior scientists to foster new connections and facilitate a first-hand exchange on building and establishing research groups in India.

YIM 2026 will provide participants with a flavour of all the components of this flagship meeting of IndiaBioscience.

The meeting is divided into two parts: The first part is the three-day Young Investigators' Meeting, which will be attended by Young Investigators from all over India and Postdoctoral Fellows from across India and the world. The second part is the one-and-a-half-day Postdoctoral Satellite Meeting for the Postdoctoral Fellows at YIM 2026.

YIM 2026 will feature mentor talks by renowned scientists, special talks on a range of science topics, poster sessions, interactive workshops, icebreaker sessions, round table discussions and networking events, that focus on a wide range of subjects and issues, including starting and building an independent research career in India, contemporary conversations on funding for science in India, international funding opportunities for scientists in India, academia-industry relationships, and research management and science engagement at institutions.

The **Postdoctoral Fellows Satellite Meeting** at YIM 2026 will enable participating PDFs to learn about job opportunities in India and meet Directors/Vice-Chancellors from across India. Although YIM makes an effort to facilitate job searches for Postdoctoral fellows, it is not a job fair. The meeting focuses on conducting science in India and mentoring Young Investigators and Postdoctoral Fellows.

Please write to us at ***yim2026[at]indiabioscience[dot]org*** for queries.

Programme Schedule

The Programme Schedule may change. Scan the QR code for updates.

02 March 2026

DAY 1: Young Investigators' Meeting 2026, Pune

Moderator: Siuli Mitra, IndiaBioscience

12:00 - 14:00	**Check into the hotel | Lunch & registration** Sandipani Hometel, Symbiosis International (Deemed University), Pune | Registration: Convention Centre, Symbiosis International (Deemed University), Pune	
India's life science ecosystem		
14:00 - 14:10	**Engaging communities, Enabling change** Siuli Mitra, IndiaBioscience	
14:10 - 14:20	**Welcome note** L S Shashidhara, NCBS-TIFR & Adviser, IndiaBioscience	
14:20 - 14:40	**Welcome note** Vidya Yeravdekar, Symbiosis International (Deemed University), Pune	
14:40 - 15:10	Keynote 1	**Address by Secretary, Department of Biotechnology, Government of India** Rajesh Gokhale, Secretary, Department of Biotechnology, Govt. of India
15:10 – 15:20	**Welcome note** S B Mujumdar, Chancellor, Symbiosis International (Deemed University)	
15:20 - 15:50	Networking break + Group photo	**Tea | Coffee | Light refreshments**
Building a Research Group		
15:50 - 16:20	Mentor talk 1	**Cooperation and conflict: Insights and lessons from a social bird** Manjari Jain, Indian Institute of Science Education and Research, Mohali
16:20 -16:50	Mentor talk 2	**Mentorship: From guidance to growth** Pallavi Kshetrapal, BRIC-Translational Health Science and Technology Institute, Faridabad
17:00 - 17:30	Keynote 2 (Online)	**Address by CEO, Anusandhan National Research Foundation, Government of India** Shivkumar Kalyanaraman, ANRF (Govt. of India)
17:30 - 18:00	Mentor talk 3	**Curiosity over metrics: Great questions drive scientific progress** Mohan Balasubramanian, University of Warwick, UK
18:00 - 19:00	Breakout session	**Getting started as a PI (Starting Strong: Building your lab, your questions, and the research culture)**
19:00	**Shuttle leaves the Convention Centre**	
19:30 - 20:45	**Dinner** Sandipani Hometel, Symbiosis International (Deemed University), Pune	
20:45 - 21:30	Open interactions	**Networking mixer (YIs + PDFs only)**

03 March 2026

DAY 2: Young Investigators' Meeting 2026, Pune

Moderator: Harinath Doodhi, GITAM Deemed to be University

8:00 - 9:00	**Breakfast** Sandipani Hometel, Symbiosis International (Deemed University), Pune	
9.00	**Shuttle leaves from Sandipani Hometel**	
9:30 - 10:00	Mentor talk 4	**From molecules to morphogenesis: Deciphering the interface of genome stability, tissue geometry, and chemical biology** Mayurika Lahiri, Indian Institute of Science Education and Research, Pune
	Funding for life science research in India	
10:00 - 10:15	Spotlight Talk	**Funding opportunities at DBT/WT India Alliance** Apurva Sarin, DBT/WT India Alliance
10:15 - 10:30	Spotlight Talk	**Funding medical research in India: Navigating ICMR priorities and opportunities for Young Investigators** Kriti Sikri, Indian Council Medical Research
10.30-10.45	Spotlight Talk	**Unlocking India's blue transformation of aquaculture in *Amrit Kaal*; Opportunities through research, innovations and entrepreneurship** Kuldeep K Lal, ICAR-Central Institute of Brackishwater Aquaculture, Chennai
10:45 - 11:15	Networking break	**Tea | Coffee | Light refreshments**
11.15-11.30	Spotlight Talk	**The European Molecular Biology Organisation (EMBO) programmes and activities for life science researchers in India** Gerlind Wallon, European Molecular Biology Organization, Germany
11:30 - 11:45	Spotlight Talk	**HFSP funding schemes for life science researchers in India** Rashna Bhandari, BRIC-Centre for DNA Fingerprinting and Diagnostics, Hyderabad
11:45 - 12:00	Spotlight Talk	**Funding initiatives by Ignite for life scientists in India** Shravanti Rampalli, Ignite Life Science Foundation
12:00- 12:15	Spotlight Talk	**Funding opportunities at Murty Trust** Neha Pankow, Murty Trust
12:15-13:00	Panel	**Ask Us Anything: Funding for life science and biotechnology research in India** Moderator: Harinath Doodhi, GITAM Deemed to be University, Bengaluru Panellists: Apurva Sarin, DBT/WT India Alliance; A.V. Balachandar, Anusandhan National Research Foundation; Gerlind Wallon, EMBO; Kriti Sikri, ICMR; Kuldeep K Lal, ICAR-CIBA; Neha Pankow, Murty Trust; Rashna Bhandari, BRIC-CDFD; Shravanti Rampalli, Ignite Life Science Foundation
13:00- 14:00	**Group photo & lunch**	
14:00 - 14:30	EMBO GLS talk 1	**Energy, inflammation, and curiosity: A journey through brain metabolism and discovery** Anna Barron, Nanyang Technological University, Singapore
	Bridging the academia-industry gap in life sciences	
14:30 - 15:00	Mentor talk 5	**Translational research from Indian academia - path to create societal impact!** Praveen Kumar Vemula, BRIC-Institute for Stem Cell Science and Regenerative Medicine, Bengaluru
15:00 - 15:15	Special talk 1	**Bridging the gap: Redefining academia–industry collaboration in India's life sciences ecosystem** Monika Puri, Roche Pharmaceuticals (India)
15:15 - 15:45	Networking break	**Tea | Coffee | Light refreshments**
15:45 - 16:00	Special talk 2	**From idea to impact: Why ecosystems matter in science** Priya Nagaraj, Council on Energy Environment and Water (CEEW), Delhi
16:00 - 16:45	Panel	**Academia-industry relationship dynamics** Moderator: Sudarshan Gadadhar, BRIC- InStem, Bengaluru Panelists: Monika Puri, Roche Pharmaceuticals; Praveen Kumar Vemula, BRIC-inStem; Premnath V, CSIR-National Chemical Laboratory, Pune; Priya Nagaraj, CEEW, Delhi
17:00 - 17:45	Panel	**Research integrity in the life sciences** Taylor and Francis Team
17:45-19:00	Poster session	**PDF Poster session**
19:00	**Shuttle leaves from Convention Centre**	
19:30 onwards	**Dinner** Sandipani Hometel, Symbiosis International (Deemed University), Pune	

04 March 2026

DAY 3: Young Investigators' Meeting 2026, Pune

Moderator: Neha Jain, Indian Institute of Technology Jodhpur

8:00 - 9:00	**Breakfast** Sandipani Hometel, Symbiosis International (Deemed University), Pune	
9.00	**Shuttle leaves from Sandipani Hometel**	
9:30 - 10:00	Mentor talk 6	**Starting an independent research lab: Strategic choices and lessons** Dimple Notani, National Centre for Biological Sciences -TIFR, Bengaluru
10: 00 - 10:30	EMBO GLS talk 2	**Building a synthetic cell (community)** Marileen Dogterom, Delft University of Technology, Netherlands
10:30 - 11:00	Mentor talk 7	**Ecology and evolution in the lab, and of the lab: Challenges for a young faculty member** Bodhisatta Nandy, Indian Institute of Science Education and Research Berhampur
11:00 - 11:45	Networking break	**Tea | Coffee | Light refreshments**
11:45 - 12:00	Special talk 4	**Science policy in India: Why it matters and how can one contribute?** Suryesh K Namdeo, Indian Institute of Science, Bengaluru
12:00 - 13:00	Panel	**Strengthening science engagement in institutions** Moderator: Sarah Iqbal Neha Jain, IIT Jodhpur; Samir Dhurde, IUCAA, Pune; Shalini Sharma, IISER Pune; Suryesh K Namdeo, IISc, Bengaluru
13:00 - 14:00	**Group photo & lunch**	
14:00 - 14:15	Special talk 5	**Genes, regulations, and resilience: Building a patient-centric research ecosystem in India** Arka Subhra Ghosh, Narayana Nethralaya Foundation, Bengaluru
14:15 - 14:30	Special talk 6	**Bridging funding and discovery: Improving research through grants management** Ajay Pillai, BRIC-National Centre for Cell Science, Pune
14:30 - 14:45	Special talk 7	**Best practices in research and project management** Vandana Gambhir, Indian Institute of Science Education and Research, Pune
14:45 - 16:00	Breakout session	**Beyond the bench: Mentorship and building meaningful research careers**
16:00 - 16:30	Networking break	**Tea | Coffee | Light refreshments**
16:30 - 16:45	**Summary of YIM 2026** Siuli Mitra, IndiaBioscience	
16:45 - 17:00	**Closing remarks** Saman Habib, CSIR- Central Drug Research Institute, Lucknow & Adviser, IndiaBioscience	
17:00 - 18:00	Poster session	**YI Poster session**
18:30 - 18:40	**Shuttle leaves convention centre**	
19:30 onwards	**Banquet Dinner** Lawn, Opposite Sandipani Hometel	

05 March 2026

DAY 4: PDF Satellite Meeting

Moderator: Sudarshan Gadadhar, BRIC-Institute for Stem Cell Science and Regenerative Medicine, Bengaluru

8:00 - 9:00	**Breakfast** Sandipani Hometel, Symbiosis International (Deemed University), Pune	
9.00	**Shuttle leaves from Sandipani Hometel**	
9:30 - 9:40	Talk	**Introduction to the PDF Satellite Meeting** Rashna Bhandari, BRIC-Centre for DNA Fingerprinting and Diagnostics, Hyderabad & Adviser, IndiaBioscience
9:40 - 10:40	Talks	**Institutional talks session 1** Rajiv Yeravdekar, Symbiosis International Deemed University, Pune; Shamik Sen, Indian Institute of Technology Bombay, Mumbai; Sanjeev Galande, Shiv Nadar University, Greater Noida; Ullas Kolthur Seethara, BRIC-CDFD & TIFR-Hyderabad
10:40 - 11:10	Networking break	**Tea | Coffee | Light refreshments**
11:10 - 12:00	Lightning talks	**PDF talks session 1** 10 PDF Participants
12:00 - 13:00	Talks	**Institutional talks session 2** Anjan Banerjee, IISER Pune; Bodhisatta Nandy, IISER-Berhampur; Maddika Subba Reddy, BRIC-CDFD, Hyderabad; Maneesha Inamdar, BRIC-inStem, Bengaluru; Sandeep Ameta, Ashoka University, Sonipat
13:00 - 14:00	**Group photo & lunch**	
14:00 - 14:50	Lightning talks	**PDF talks session 2** 10 PDF Participants
14:50 - 15:50	Talks	**Institutional talks session 3** Neeraj Kumar Mishra, GITAM Deemed to be University, Bengaluru; Rajiv Janardhanan, SRM Institute of Science & Technology, Kattankulathur; Rakesh Mishra, Tata Institute for Genetics and Society, Bengaluru; Uma Ramakrishnan, National Centre for Biological Sciences-TIFR, Bengaluru; Vivek Tanavde, Ahmedabad University
15:50 - 16:20	Networking break	**Tea | Coffee | Light refreshments**
16:20- 17:10	Lightning talks	**PDF talks session 3** 10 PDF Participants
17:10 - 18:00	Breakout session	**Mentorship circles by Institutional representatives: Administrative and infrastructural challenges** PDFs and Institutional representatives
18:00 - 19:00	Poster session	**PDF Poster session**
19.00	**Shuttle leaves Convention Centre**	
19:30 onwards	**Dinner** Sandipani Hometel, Symbiosis International (Deemed University), Pune	

06 March 2026

DAY 5: PDF Satellite Meeting

Moderator: Moumita Mazumdar, IndiaBioscience

8:00 - 9:00	**Breakfast** Sandipani Hometel, Symbiosis International (Deemed University), Pune	
9.00	**Shuttle leaves from Sandipani Hometel**	
9:30 - 10:30	**Talks**	**Institutional talks session 4** Narottam Acharya, BRIC-Institute of Life Sciences, Bhubaneswar; Neha Jain, IIT Jodhpur; Sagar Sengupta BRIC-NIBMG, Kalyani; Swasti Raychaudhuri, CSIR-Centre for Cellular and Molecular Biology, Hyderabad
10:30 - 11:00	**Networking break**	**Tea | Coffee | Light refreshments**
11:00 - 11:50	**Lightning talks**	**PDF talks session 4** 10 PDF Participants
11:50 - 12:45	**Breakout session**	**Mentorship circles by Institutional representatives: Hiring policies & practices at Indian institutions** PDFs and Institutional representatives
12:45 - 13:00	**Talks**	**Closing remarks for the PDF Satellite Meeting** Roop Mallik, IIT Bombay & Adviser, IndiaBioscience
13:00 - 14:00	**Open interactions**	**Group photo & lunch | Departure**
14:00	**End of PDF Satellite Meeting**	

YIM 2026 Organising Committee

Harinath Doodhi

Harinath is a cell biologist working at GITAM (Deemed to be University), Bengaluru. He received his PhD in Molecular Biology from TIFR, Mumbai and worked as postdoctoral fellow at Utrecht University, The Netherlands and as Senior Research Associate at the University of Dundee, UK. His broad research interests include microtubules and microtubule associated cell biological processes. His current research involves studying mitosis with focus on error correction mechanisms, involving microtubule and kinetochore attachments, which has wider implications in aneuploidy, genome instability and cancers.

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Manjula Harikrishna

Manjula holds a Master's degree in Microbiology and an MBA in Human Resource Management. She has been an integral part of the IndiaBioscience team since 2016, working closely with the life science community, optimising processes, and contributing to numerous projects. Currently, she leads the 'Community Building' vertical striving to find innovative ways to engage and expand the community. She is also actively involved in the outreach activities, workshops, conferences, building resource materials and organising the Young Investigators' Meeting (YIM).

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Neha Jain

Neha is an Associate Professor and Acting Head of the Department of Bioscience and Bioengineering at IIT Jodhpur. Her lab utilises various tools to answer some of the fascinating questions pertaining to amyloid fibrils of different origins and their association with disease progression. Neha is passionate about scientific outreach and sharing knowledge beyond institutional boundaries.

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Siuli Mitra

Siuli transitioned from a career in research to science communication. As a communications specialist, she has experience crafting impactful narratives for science organisations in government and academia. Her work has ranged from content development across diverse media to formulating external and internal communication strategies and advising senior management on public communication of science and technology.

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Sudarshan Gadadhar

Sudarshan is a trained cell biologist and biochemist, with a PhD in Biochemistry from the Indian Institute of Science. He has extensive expertise in cilia biology and the microtubule cytoskeleton from his postdoctoral tenure at the Institut Curie in Paris. He started his independent research group at BRIC-inStem in 2022, where he continues to explore the impact of the regulation of the core microtubule cytoskeleton of cilia and flagella in mammals. Apart from mentoring, supervising students and teaching, he also has Institutional engagements, both in scientific as well as administrative roles.

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IndiaBioscience Team

Moumita comes from a background in microbiology and science communication, with expertise in science writing and audio-visual content creation. In her role as Program Manager (Science Communication) at IndiaBioscience, she is eager to drive the organisation's science communication initiatives forward. She also serves as the editor of the articles published by IndiaBioscience. She is also pursuing a PhD in Science Communication at CSIR-NIScPR, Delhi, where her research focuses on strategies to combat misinformation in India.

Moumita Mazumdar

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Sreshtha comes from a background of zoology and biotechnology, where she likes to explore human physiology and proteomics. In her role as Program Manager (Digital Initiatives) at IndiaBioscience, she works at the intersection of technology, communication, growth and community. Her learning path to the Master's degree went through the institutes of DU, IIT-D, JMI, and CSIR-IGIB. Her tryst with science journalism and science communication continued alongside, and she finally took the plunge into research in science communication and human cognition at NIAS, on the IISc campus, Bengaluru. When not behind the laptop, you'll find the spotlight shining on her as a Dancer (BA in Kathak) and hosting quizzes.

Sreshtha Mondal

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Shwetha graduated with an M.Com and joined IndiaBioscience in 2018. She manages day-to-day administrative and finance-related operations at IndiaBioscience. She curates the jobs, grants and events sections for the IndiaBioscience website and the IndiaBioscience Jobs and Internships monthly newsletter.

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Supporters of YIM 2026

Primary supporter

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DBT is the largest supporter of both YIM and IndiaBioscience. This Department, established in 1986, gave a new impetus to the development of modern biology and biotechnology in India. Over the course of nearly four decades, the department has promoted and accelerated the development of biotechnology in the country through diverse programmes and has played a critical role in shaping several policies impacting biological sciences research in India.

Partner

SYMBIOSIS INTERNATIONAL (DEEMED UNIVERSITY)

It is a multi-disciplinary university offering its students and faculty a vibrant learning ecosystem designed around its multi-cultural and innovative ethos. Established in 1971, the University has campuses at six cities in India and one international campus.

Other supporters

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Ashoka University, a pioneering institution in India, offers a multidisciplinary education focusing on liberal arts and sciences. It is renowned for fostering critical thinking and leadership while emphasising holistic learning. By encouraging diverse perspectives and societal contributions, Ashoka cultivates innovative thinkers poised to tackle global challenges through academic excellence and a commitment to social impact, creating a new generation of thoughtful leaders adept at addressing complex issues.

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BRIC — INSTITUTE FOR STEM CELL BIOLOGY AND REGENERATIVE MEDICINE, BENGALURU

BRIC-inStem is a state-of-the-art research institute in Bangalore, India, dedicated to the study of stem cell and regenerative biology. An autonomous institute funded by the Dept of Biotechnology, Govt. of India, inStem emphasizes collaborative research in stem cell biology. inStem's mandate to allow this cross-disciplinary, multi-pronged approach to research, straddles the divide between clinical and laboratory research in stem cell biology. In trying to answer intractable and challenging questions that face the field, inStem seeks to rewrite the paradigm of the research institute: without barriers and across disciplines.

BRIC — NATIONAL INSTITUTE OF BIOMEDICAL GENOMICS, KALYANI

BRIC-NIBMG is a research institute dedicated to genomics and human health. It advances genome science, disease biology, and precision medicine through research, training, and collaborations. Based in Kalyani, it supports national genomic initiatives and translates genomic knowledge into healthcare and public health impact nationwide.

Supporters of YIM 2026

EUROPEAN MOLECULAR BIOLOGY ORGANIZATION (EMBO), GERMANY

EMBO is an organisation of more than 1,900 leading researchers that promotes excellence in the life sciences in Europe and beyond. The major goals of the organisation are to support talented researchers at all stages of their careers, stimulate the exchange of scientific information, and help build a research environment where scientists can achieve their best work. The EMBO communities are global networks of top-level scientists at various stages of their careers. They give privileged access, particularly to young researchers, to cutting-edge science and the opportunity to build international contacts.

GITAM DEEMED TO BE UNIVERSITY, BENGALURU

Founded in 1980, GITAM Deemed to be University stands at the intersection of multidisciplinary applied education and translational research. With 12 schools across Bengaluru, Hyderabad, and Visakhapatnam, GITAM empowers students to discover their ikigai through global perspectives, industry collaborations, and holistic development. Guided by integrity and the pursuit of knowledge and moral values, GITAM shapes future-ready citizens who drive entrepreneurship, innovation, and create meaningful societal impact.

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IISER Pune is a research-intensive teaching institute. Our faculty and students investigate questions in science that lie beyond the boundaries of conventional thinking. The whole ambience is very academic with high energy levels to pursue high quality research.

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IIT Bombay is a premier public research university in Mumbai, established in 1958. Known for excellence in engineering, science, design, and management, it drives innovation, entrepreneurship, and high-impact research, and has produced leading scientists, technologists, and startup founders in India and globally.

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Anna Barron
Nanyang Technological University, Singapore
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BRIC - Institute for Stem Cell Science and Regenerative Medicine, Bengaluru
Translational research from Indian academia - path to create societal impact!

EMBO GLS Talk

Anna Barron

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Energy, inflammation, and curiosity: A journey through brain metabolism and discovery

Abstract

Academic success is often judged by external metrics that are unpredictable and frequently disappointing, particularly when shaped by comparison with peers. My early encounters with failure and rejection in science taught me the value of defining success internally—by understanding my motivations and what gives my work meaning. This internal compass has been essential for sustaining energy and resilience across a non-linear scientific journey. Driven by curiosity, and a necessary sense of humour, my path has crossed disciplines and research cultures, from doctoral training in Australia to postdoctoral work in the United States and Japan, before joining Nanyang Technological University, Singapore, to establish an independent research programme. These transitions sharpened my interest in how metabolic state and inflammation shape brain function and vulnerability across the lifespan. My laboratory now investigates how mitochondria regulate brain energy metabolism and inflammatory responses during ageing and disease using advanced neuroimaging approaches, including PET and multiphoton fluorescence microscopy, alongside cellular and molecular techniques, with the aim of identifying novel biomarkers and therapeutic avenues to promote healthy brain ageing. Along the way, I have learned that curiosity is a renewable source of energy; that inflammation—scientific or professional—can be informative rather than purely destructive; and that embracing non-linear paths while anchoring success internally are foundations for creative, sustainable science and meaningful mentorship.

Bodhisatta Nandy

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Ecology and evolution in the lab, and of the lab: Challenges for a young faculty member

Abstract

After completing PhD, every young scientist passes through a transition phase, which is very dynamic as well as unnerving at times. While most get trained to do science, develop problems, write papers and grant applications, few are mentored for the role of a faculty. Yet, many aspire to become a faculty member in a university or an academic institute. I will discuss my personal journey through this turbulent phase as I was trying to make my own niche as an independent scientist – an Evolutionary Biologist. I will discuss a few research problems that I had developed, and talk about the balance I had to achieve in planning short term projects and long term bigger problems. While many young faculty aspirants would know how to address this aspect of the job, many do not realise all the expectations, which is usually far more than only teaching a few classes and carrying out independent research. One needs to build a research team and lead it, mentor students, take up administrative roles, serve in committee, process purchase, manage grants, conduct conferences and outreach activities, and so on. It is extremely important to be mentally and pragmatically prepared for this. I will talk about how the expected role of faculty members as student mentors has changed considerably over the last ten years. By working at a Central University, and having spent ten years building a new institution of national importance, I will share some of my experiences highlighting the various challenges that are typical to the Indian academic ecosystem, which is patient enough, but has become increasingly competitive over the years.

Dimple Notani

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Starting an independent research lab: Strategic choices and lessons

Abstract

My scientific journey did not begin with strong academic performance. Yet, over time, I built an independent biology laboratory addressing mechanistic questions in genome regulation, work that is technically demanding and funding-intensive. In this mentoring talk, I will discuss how the unique Indian research ecosystem shapes the way we do science, highlighting both its advantages and challenges. I will emphasise the freedom in India to pursue original, out-of-the-box questions that may not be fashionable elsewhere, alongside the realities of funding timelines and infrastructure. I will share practical dos and don'ts of running a PhD-driven lab, writing grants, and sustaining high-quality, independent research in India.

Manjari Jain

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Cooperation and conflict: Insights and lessons from a social bird

Abstract

Cooperatively breeding species provide a window to examine the evolutionary balance between helping and competition in social groups. In such species, individuals often delay independent breeding and instead assist others to raise their offspring. This altruistic behaviour challenges the notion of animals being in a constant state of conflict and opens avenues to examine the factors that drive cooperation. While one must refrain from drawing moral lessons from animal behaviour, I will highlight the obvious parallels in terms of balancing cooperation and competition in animal societies and navigating academia. Drawing from the ongoing long-term research on social birds along with my own journey in academia, I will highlight the challenges of decision-making about when to 'disperse from natal groups', navigating hurdles faced when establishing independent research groups and handling situations of conflict. I will also discuss how communication is key for humans and non-human animals alike. Lastly, I will highlight the value of paying forward and the importance of being an ally.

EMBO GLS Talk

Marileen Dogterom

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Building a synthetic cell (community)

Abstract

In my group we are interested in understanding how dynamic and force-generating properties of the cytoskeleton contribute to the spatial organisation of cells. I will talk about our long-term journey and highlight recent advances (and challenges) in our efforts to reconstitute minimal, functional cytoskeletal systems in artificial confinement. Examples are the reconstitution of basic eukaryotic mitotic spindles in microfluidic droplets and the design of a minimal DNA segregation system for synthetic cells. These efforts fit in a long-term ambition to build, in collaboration with others, an autonomously growing and dividing synthetic cell from scratch. Part of my talk will be dedicated to our efforts to build a collaborative global research community around this goal.

Mayurika Lahiri

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From molecules to morphogenesis: Deciphering the interface of genome stability, tissue geometry, and chemical biology

Abstract

My research journey reflects a progressive evolution from investigating the fundamental molecular mechanics of cancer biology to understanding these processes within the physiological complexity of tissue organisation. After completing my PhD in the UK and postdoctoral training at Tufts University and MGH Cancer Center, Harvard Medical School, Boston, USA, I established my laboratory at IISER Pune in 2008. My work has since pivoted to address how DNA repair pathways function not just in isolated cells, but within the context of three-dimensional (3D) tissue geometry.

My laboratory utilises 3D culture models mimicking breast epithelial morphogenesis to study the bidirectional relationship between DNA Damage Response (DDR) signalling and cell polarity. A major focus is dissecting the roles of specific checkpoint regulators and anti-apoptotic proteins, such as TopBP1 and Api5, in maintaining glandular architecture. Crucially, the laboratory has expanded its scope through interdisciplinary collaborations with the Department of Chemistry at IISER Pune. This work involves the biological validation of novel synthetic ion transporters. By investigating how these synthetic molecules disrupt ion homeostasis to induce targeted apoptosis in cancer cells, the group bridges the gap between chemical synthesis and cancer therapeutics, offering a multi-scale approach to understanding and targeting breast cancer.

Mohan Balasubramanian

University of Warwick, UK
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Curiosity over metrics: Great questions drive scientific progress

Abstract

In 1988, I began investigating cytokinesis in fission yeast *Schizosaccharomyces pombe*, a question that has shaped my research for 38 years. My group was the first to identify the core parts list of proteins involved, which, together with other labs, led to the full description of the cytokinetic actomyosin ring, possibly the best-characterised cytokinetic actomyosin ring in all eukaryotic biology. We isolated defective mutants, cloned the genes, tracked protein dynamics, reconstituted key processes, initiated structural studies, and explored how cells achieve robust division despite variability. This work expanded into synthetic biology approaches for bacterial and archaeal cytoskeletons, revealing shared evolutionary roots, and developing practical tools for the community, including methods for recombinant actin production, fluorescent protein reporters, and banks of forward/reverse genetic mutants.

Along the way, I trained about 70 researchers, a quarter of whom now lead independent labs across three continents. In this talk, I will describe how focusing on curiosity, rather than metrics like h-index, impact factors, or grant totals, sustained this long-term effort through challenges. In today's environment, it is easy to get pulled toward numbers, but for a decades-long career in science, questions that genuinely interest you are what keep the work going. For young scientists searching for their next position, I recommend prioritising who your colleagues will be over the size of a famed "start-up" package—the people around you shape your daily science far more than initial funding. I will also reflect on opportunities I missed, what they taught me about maintaining a "prepared mind", why getting involved in administration can strengthen the scientific ecosystem, and the value of going all out, boldly seeking opportunities, jobs, or positions, with examples from my own path of when I acted decisively and when I wish I had.

Pallavi Kshetrapal

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Mentorship: From guidance to growth

Abstract

An inquisitive mind shapes a scientist, moving intentionally between disciplines, institutions, and, most importantly, between the laboratory and the clinic, shaped my scientific journey. Doctoral training in developmental genetics, being the start point, followed by postdoctoral work, extensively deepened my exposure to rigorous, hypothesis-driven science and advanced experimental systems. The initial days of immersion at the grassroots level in a district hospital highlighted the critical gap between biomedical discovery and real-world healthcare.

This defined the shift in my journey and helped build my independent laboratory, designed not as a standalone academic unit but as a translational extension of clinical practice, "Lab of Perinatal Research". Indulging in the research questions, molecular technologies, and multi-omics platforms was driven by unmet needs observed in hospital settings, with a focus on feasibility, scalability, and the integration of my research into existing clinical workflows. Adopting this approach has assisted in developing population-relevant biomarkers, standardised biobanking frameworks, and clinically actionable molecular insights.

Another important facet of this journey has been team building. I have, by design, built a team of diverse PhD students and postdoctoral fellows with complementary expertise in molecular biology, omics, biobanking, data science, and clinical translation. Moreover, meticulous mentorship has been an integral part of bolstering this model, fostering scientists who are as comfortable engaging with clinicians as with datasets.

The journey is ongoing, where my work strives for continuous evolution in embedding robust scientific aptitude within clinical systems and in ensuring that research at all points remains responsive to societal needs while nurturing the next generation of independent investigators.

Praveen Kumar Vemula

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Translational research from Indian academia - path to create societal impact!

Abstract

Translational research provides a path to generate innovative technologies and convert them into viable solutions to solve unmet clinical needs. This journey cannot be completed by travelling through a conventional path. During this talk, we can discuss how some fundamental principles of translational science could be used to address nationally relevant unmet needs and navigate the Indian academic ecosystem. We can also discuss how efficiently one can build a bridge between academia and Industry by practising science entrepreneurship while establishing world-class research programmes in academic labs.

Keynote Talks

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Welcome Talks

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Special Talks

Bridging funding and discovery: Improving research through grants management

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Genes, regulations, and resilience: Building a patient-centric research ecosystem in India

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Bridging the gap: Redefining academia–industry collaboration in India's life sciences ecosystem

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From idea to impact: Why ecosystems matter in Science

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Science policy in India: Why it matters and how can one contribute?

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Best practices in research and project management

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Spotlight Talks

Funding opportunities at DBT/WT India Alliance

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Funding medical research in India: Navigating ICMR priorities and opportunities for young investigators

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Funding opportunities at Murty Trust

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Funding initiatives by Ignite for life scientists in India

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The European Molecular Biology Organisation (EMBO) programmes and activities for life science researchers in India

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Unlocking India's blue transformation of aquaculture in Amrit Kaal; Opportunities through research, innovations and entrepreneurship

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Human Frontier Science Programme (HFSP) funding schemes for life science researchers in India

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Panel Discussion

Funding for life science and biotechnology research in India

Ask-Me-Anything session with funding agencies' representatives

Moderator

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Panel Discussion

Academia-industry relationship dynamics

Moderator

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Strengthening science engagement in institutions

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Institutional Representatives

Each year, representatives from various institutions across India attend the YIM, particularly the Postdoctoral Fellows' Satellite Meeting and give talks about their institutes. Listed below are the institutions and representatives at YIM 2026.

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Young Investigators' Abstracts

The abstracts have been reproduced as submitted by the participants, with only minimal edits to improve readability.

YI 01 **Adarsh Kumar**
Macroalgae abundance, collection, heavy metal digestion and preparation of bioplastic using seaweeds

YI 02 **Aditya Kurdekar**
Integrating surface plasmon-coupled emission with real-time PCR for high-sensitivity DNA detection

YI 03 **Akhilesh Mishra**
Deciphering the genome's dynamics: From bioinformatics tools to clinical innovations

YI 04 **Ambuja Navalkar**
Mechanistic roles of biomolecular condensates

YI 05 **Amrita Saxena**
Interlayer signalling facilitated by TCP4 for robust organ shape morphogenesis

YI 06 **Ankit Mahajan**
A 261 kb deletion spanning three genes is causing Rubinstein-Taybi syndrome type 1 in a 6-year-old boy belonging to Kashmir valley, India

YI 07 **Ashitha SNM**
Investigating differential immunomodulatory effects of lithium in bipolar disorder responders and non-responders under inflammatory stress

YI 08 **Ashish Goyal**
Epigenetic Therapy-Induced Antigens—A New Class of Immunotherapy Targets

YI 09 **Chintan Bhavsar**
Liver evading nanoparticles for precise delivery to tumours: Learning from naturally occurring extracellular vesicles (EVs)

YI 10 **Devivasha Bordoloi**
Targeted ovarian cancer immunotherapy through engineering of follicle-stimulating hormone receptor (FSHR) antibody to engage T cells

YI 11 **Gundeep Kaur**
Structural basis for autoinhibition and activation of HELLS

YI 12 **Harshiny M**
Eco Water Pods: Biopolymer-based hydrogel capsules for sustainable water detoxification

YI 13 **Kamakshi D**
Molecular insights into obesity-associated neurodegeneration: A quantitative proteomic approach

YI 14 **Kavita Agarwal**
Vaginal sialidase activity and the microbiome: evaluating bacterial vaginosis diagnosis in Indian women

YI 15 **Mousumi Khatun**
Identification of novel early detection markers for chronic hepatitis virus infection-associated hepatocellular carcinoma

YI 16 **Mrinmoy Das**
S. aureus exposure during cutaneous antigen sensitisation causes basophil- and IL-4-dependent exaggerated food anaphylaxis

YI 17 **Neha Bokey**
From microbes to metabolism: Exploring the microbiome's role in lifestyle disorders

YI 18 **Nidhi Gupta**
Identification of a putative toxin-antitoxin gene in the human pathogen Helicobacter pylori

YI 19 **Nishana Mayilaadumveettil**
Chromatin architecture in crisis: How altered organisation drives disease

YI 20 **Parth Sharma**
Pharmacological evaluation of tributyrin and vitamin D in the management of glucocorticoid induced osteoporosis in rats

Young Investigators' Abstracts

The abstracts have been reproduced as submitted by the participants, with only minimal edits to improve readability.

YI 21 **Piyush Agarwal**
Cracking cancer codes: Network biology meets machine learning in multi-omics

YI 22 **Pooja Choudhary**
Unlocking the potential of small millets: Stress-responsive omics and starch-based innovation

YI 23 **Pranav Tiwari**
One-step simultaneous liquid phase exfoliation-induced chirality in graphene and their chirality-mediated microRNA delivery

YI 24 **Pratik Kumar**
Organic dyes as molecular tools beyond imaging

YI 25 **Rajalakshmi Kalaivanan**
Impact of novel feed ingredients on growth, digestive function, biochemical responses, immunity and gene expression of Penaeus vannamei

YI 26 **Rakesh Ganji**
Organelle Biology lab: Inter-organellar communications in cellular health and disease

YI 27 **Ravindra Phatake**
Natural product-inspired small molecules: A platform for drug discovery and biological modulation

YI 28 **Rohil Jain**
Spectroscopic and microfluidic diagnostic platforms in medicine and surgery

YI 29 **R Vignayanandam Muddapu**
From data to dynamics: Multiscale integration for Atlas-based modelling of mouse striatal networks

YI 30 **Santosh Chaudhary**
Engineering proximity-mediated protein function: A scalable platform using group-transfer chimeras and ultrasmall chemogenetic tags

YI 31 **Sarita Puri**
Dimer dissociation and exposure of hot-spot residues synergistically accelerate light-chain variable-domain aggregation associated with AL amyloidosis.

YI 32 **Shikha Singh**
The emergence of ABCFs (ATP Binding Cassette F) as novel translational regulators: their role in cellular homeostasis and antibiotic resistance

YI 33 **Sojit Tomo**
Assessment of Sphingosine-1-phosphate pathway in connective tissue disease patients and its association with interstitial lung disease

YI 34 **Sree Sankar D**
Microbiomes in various environments: A unified one health perspective on animals, humans, and environment

YI 35 **Sugitharini V**
Role of immune receptors and their epigenetic regulation in ischemic stroke in a sex-specific manner by membrane proteomics

YI 36 **Surjit Singh**
Histone H3 lysine K4 tri-methylation regulation of iron deficiency response in rice

YI 37 **Tahsin Bennur**
Herbal-Embedded Nanocomposites: Characterisation and efficacy-assessment

YI 38 **Upma Singh**
Uncovering the genomic landscape of Stenotrophomonas maltophilia to understand its drug resistance, invasive potential, and host adaptation

YI 39 **Vikash Yadav**
LncRNA: An untapped source of micro peptides in flowering plants

YI 40 **Vinay Sharma**
Molecular profiling using nanopore technology: single-cell and single-molecule discrimination for next-generation biosensing

Adarsh Kumar

YI 01

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Macroalgae abundance, collection, heavy metal digestion and preparation of bioplastic using seaweeds

Abstract

Plastic pollution has been a well-recognised environmental pollutant, rising every day globally. Recently, it has been evidenced that the breakdown of plastics into microplastics introduces serious health risks to both wildlife and humans through contamination of food and water sources. These microplastics are recently found in human blood, lungs and placenta (United Nation Environmental Protection, WHO). Recent reports from Italy (2024) and China (2025) evidenced deposition and accumulation of microplastics in human arteries, leading to heart attacks and in 50 species of birds, respectively. India itself generates 3.4 million tonnes of plastic waste annually and the majority of them remain persistent in the soil (less than 60% recycled). Despite new methods to produce eco-friendly plastics, alternatives to plastic are still lacking in the market because of the high cost of raw materials and industrial setup.

Present work explored the potential of seaweed as a sustainable and biodegradable raw material for bioplastic film production, with a focus on both ecological availability and material characterisation. Abundance studies conducted along the Visakhapatnam coastline during 2024-2025 revealed a notable presence of *Padina tetrastromatica*, *Ulva lactuca*, and *Gracilaria corticata* species. Further, analytical techniques such as Muffle Furnace analysis and Microwave Plasma-Atomic Emission Spectroscopy (MP-AES) were employed to evaluate the ash and elemental content of the selected macroalgae. These analyses confirmed the presence of essential constituents that contribute to the functional properties of macroalgae, reinforcing their suitability for industrial and biotechnological applications, including bioplastic production. Among the different formulations tested, bioplastic films developed using a composite of Green-Red algae in varying compositions demonstrated promising physical characteristics, indicating their efficiency and potential for further development in biodegradable film technology.

Macroalgae abundance, collection, heavy metal digestion, and preparation of bioplastic using seaweeds.

Aditya Kurdekar

YI 02

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Integrating surface plasmon-coupled emission with real-time PCR for high-sensitivity DNA detection

Abstract

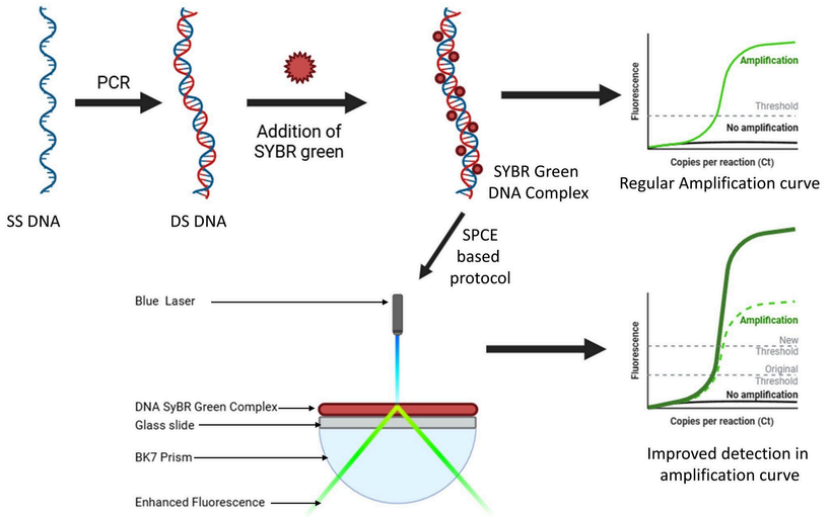
Surface plasmon coupled emission (SPCE) offers a powerful approach to enhance fluorescence detection sensitivity by exploiting the interaction between excited fluorophores and surface plasmon modes on metallic films (1).

We demonstrate the integration of SPCE with a conventional polymerase chain reaction (PCR) platform to enhance the signal of SYBR Green I, a widely used intercalating dye for real-time DNA quantification. A thin-film architecture comprising a 45 nm silver layer coated with a 10 nm SiO_2 spacer was optimised to achieve directional fluorescence emission and maximal coupling efficiency.

Experimental results revealed a 7.8-fold enhancement in emission intensity compared to conventional fluorescence detection under identical PCR conditions. Moreover, the SPCE-based detection reduced the threshold cycle (Ct) value by an average of 2.3 cycles, corresponding to a ~6× improvement in detection sensitivity for low-copy-number DNA templates. The emission exhibited strong polarisation dependence and a well-defined angular distribution centred at 52°, consistent with theoretical SPCE predictions. The system maintained excellent linearity ($R^2 = 0.996$) across five orders of magnitude of DNA concentration, with no measurable photobleaching-induced distortion. These findings confirm that SPCE can substantially boost fluorescence readout efficiency in real-time PCR without altering reaction kinetics, offering a pathway toward miniaturised, highly sensitive bioanalytical platforms. Future optimisation may include integration with nanostructured plasmonic arrays and multiplexed dye systems to improve performance.

References

1. Lakowicz JR. (2005). Radiative decay engineering 5: Metal-enhanced fluorescence and plasmon emission. Analytical Biochemistry 337(2):171–194.



The double-stranded DNA/RNA is introduced into the SPCE platform, which enhances the fluorescence signal from SYBR green, thus improving the threshold for classification, leading to higher sensitivity.

YI 03

Akhilesh Mishra

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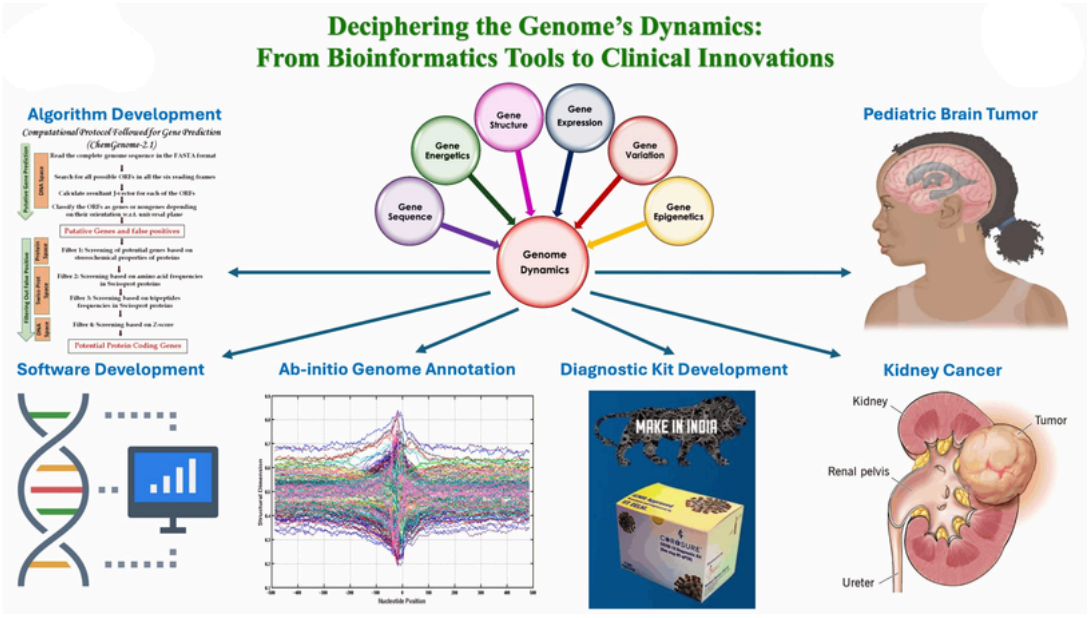
Deciphering the genome's dynamics: From bioinformatics tools to clinical innovations

Abstract

My research focused on deciphering the parameters involved in genome dynamics through exploring sequence, energy, structure, expression, variation and epigenetic parameters of DNA. Initially, the focus was aimed at analysing DNA sequences and identifying potential drug targets for cancer and pathogens. This led to the development of two software - Onco-Regulon and Pathogen-Specific DNA Drug Finder. Subsequently, the research shifted to exploring the energetic aspects of DNA, highlighting the roles of solvation energy, stacking energy, and hydrogen bonding energy in distinguishing functional units within the genome. The investigation then expanded to the structural analysis of DNA, explicitly predicting transcription start sites (TSS) in prokaryotes and intron-exon boundaries in eukaryotes. Notably, a genome annotation software, ChemGenome2.1, was developed based on these physicochemical properties.

I made significant contributions to the development of India's most cost-effective COVID-19 Diagnostic Kit, which received ICMR approval. In my postdoctoral research positions, I studied the interplay between DNA sequence variants, structural alterations, and transcriptional programs. In metastatic renal cell carcinoma (RCC), somatic single-nucleotide variants in TP53 and SMARCA4 delineate aggressive trajectories, shortening time under active surveillance. In MiT/TFE translocation RCC, fusion-driven rewiring produces convergent expression modules between mouse and human, with a low background mutation burden, highlighting genome structure over SNV-driven evolution. Investigating rapalogs resistance reveals that fibroblasts within the tumour microenvironment govern adaptation to rapalogs, shaping therapeutic evolution. HIF2a inhibition by RNA-targeting drugs in ccRCC provides proof of principle and establishes a paradigm for tumour-directed RNA-based therapeutics in cancer.

Currently, my Computational Oncology lab at NIT Rourkela focuses on studying the genomic and molecular profiles of Adamantinomatous Craniopharyngioma (ACP), to investigate its intratumor heterogeneity and intercellular networks, and to dissect the mechanism of Tumour Grade Progression in Renal Cell Carcinoma.



Deciphering the genome's dynamics: From bioinformatics tools to clinical innovations

YI 04

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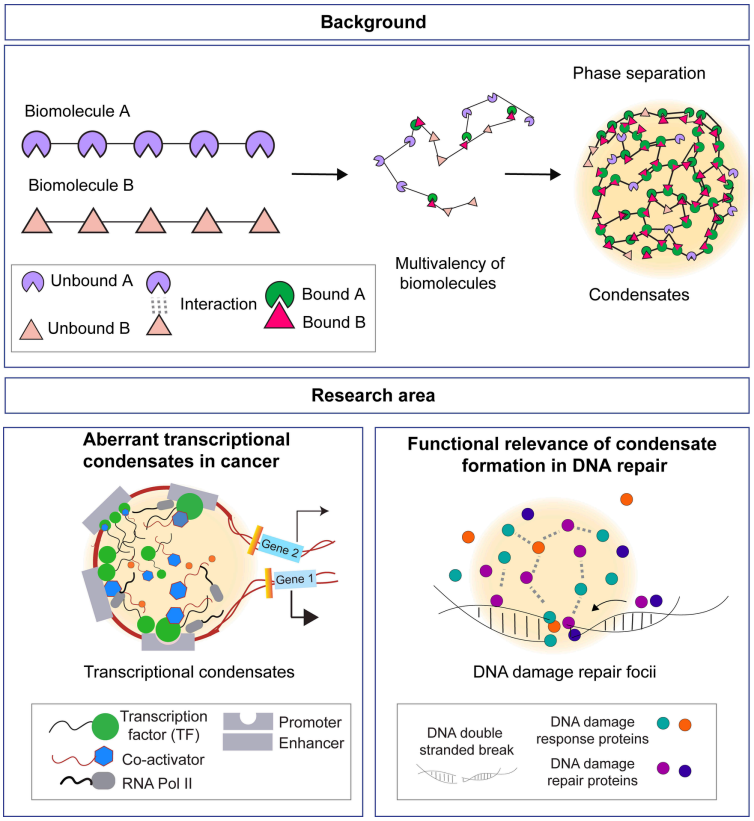
Mechanistic roles of biomolecular condensates

Abstract

Protein conformational states and assemblies have been studied for decades due to their relevance to physiological cellular processes and diseases. Dynamic compartmentalisation of biomolecules into phase-separated condensates has emerged as a central organising principle in cellular regulation. The current research examines how phase separation governs two essential processes, transcription and DNA-damage repair, to reveal mechanisms by which intrinsically disordered proteins create responsive regulatory environments.

Transcription involves proteins such as RNA polymerase II, transcription factors, and mediator proteins, which are intrinsically disordered and drive condensation assemblies. The condensates locally concentrate transcriptional machinery and can contribute to transcriptional activity and oncogenic gene activation. In our work, using a combination of *in vitro*, cytomimetic, and cell biological approaches, we explore the relevance of transcription factor condensate formation.

In a parallel study, we examine proteins implicated in DNA-damage response, which form dynamic repair foci at double-strand breaks. These structures exhibit hallmarks of condensates, suggesting organisation as transient repair hubs. We use cell-based perturbations and mouse models to define how loss or alteration of foci can affect genome stability and repair kinetics.
Overall, our research aims to establish a mechanistic framework in which phase separation, which underlies condensate formation, can be important in physiological processes like transcriptional activation and DNA repair fidelity. Understanding phase separation offers new avenues to therapeutically modulate aberrant condensate or assembly behaviour in cancer, genome-repair and neurodegeneration.



Phase separation of biomolecules leads to condensate formation, implicated in cellular processes like transcription and DNA damage repair

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YI 05

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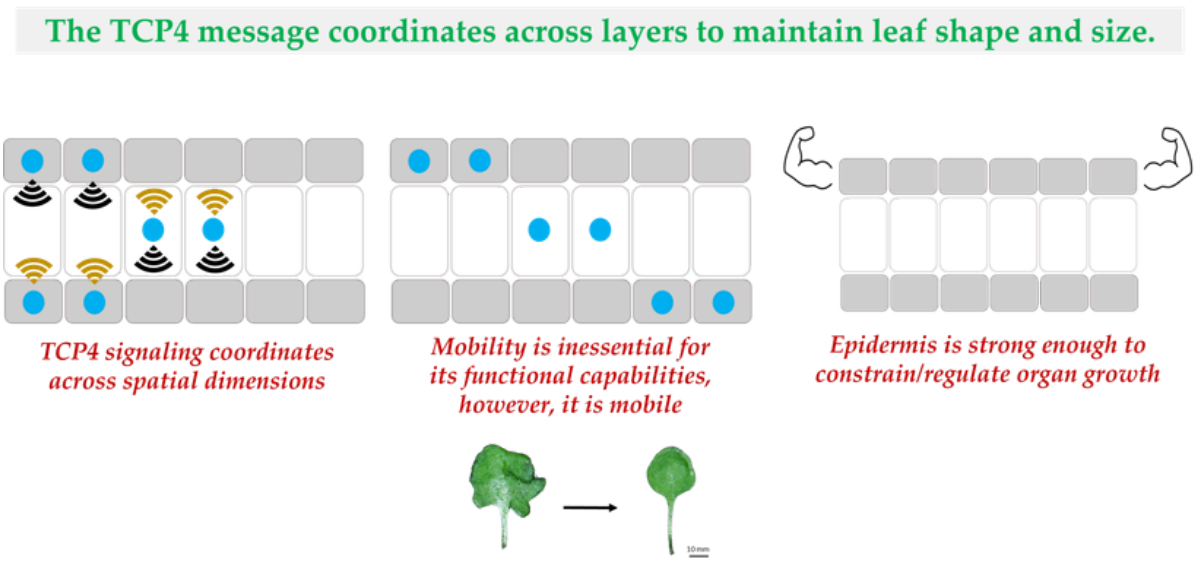
Interlayer signalling facilitated by TCP4 for robust organ shape morphogenesis

Abstract

The developmental programming of an organism ensures the robust reproduction of organ shape and size. Yet, how this developmental blueprint is orchestrated in 3-dimensional space and time via genetic factors remains insufficiently explored. In the context of leaves, a cluster of five redundant genes, CINCINNATA-like TCP genes (CIN-TCPs), has been identified as regulators of leaf shape. The microRNA miR319 negatively regulates the transcripts of these 5 TCPs. Over-expression of MIR319A in the jaw-D line results in crinkly and lobed-leaf morphology. Remarkably, inducing just one TCP protein (TCP4) in the loss-of-function background can restore the leaf phenotype to the wild-type level. However, TCP4 protein activity varies across different cell layers of leaf primordia, indicating a coordination of CIN-TCP messages among these layers.

To explore this, miR319-resistant TCP4 lines were created, enabling specific layer activation. Activating TCP4 in the epidermal and subepidermal layers rescued CIN-TCP mutants, demonstrating bidirectional layer communication. Furthermore, expression of fluorescent TCP4 was seen in the sub-epidermis despite being expressed constitutively in the epidermis.

This highlights the mobility and functional capabilities of TCP4, as the mutants portray rescue of phenotype. Introducing bulky TCP4 fused to 3X-GFP in the first layer completely restored abnormal leaves, implying an unknown downstream CIN-TCP signal coordinates intercellular communication. Tracking the bulky fluorescent TCP4 led us to conclude that there is no inter-layer protein movement, emphasising the cell-autonomous functional capabilities of the TCP4 protein. This research illuminates the intricate interplay of genetic factors, underscoring the challenges in comprehending the 3D spatial and temporal orchestration in biological systems.



TCP4 message coordinates across layers to maintain leaf shape and size

YI 06

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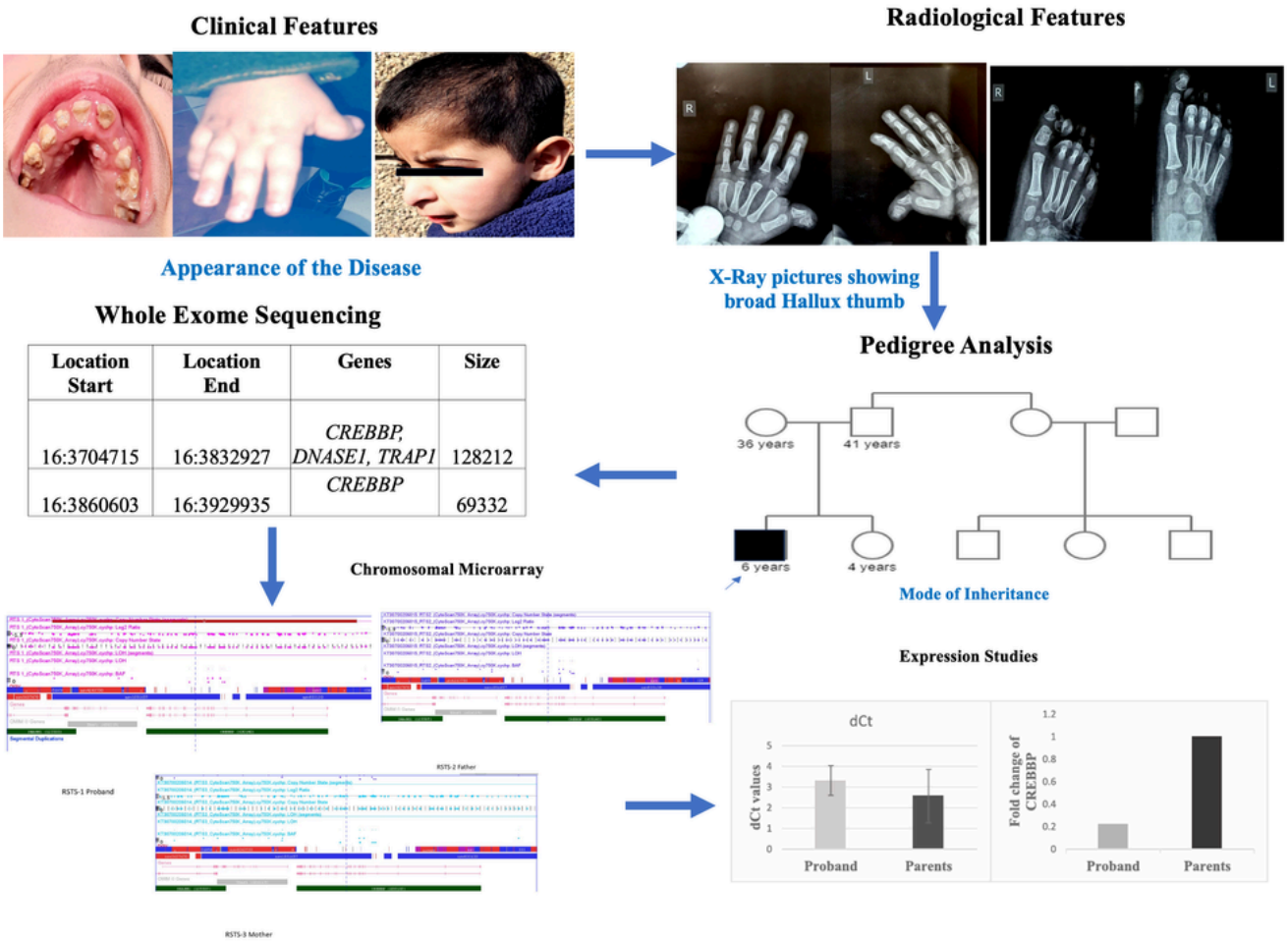
A 261 kb deletion spanning three genes is causing Rubinstein-Taybi syndrome type 1 in a 6-year-old boy belonging to Kashmir valley, India

Abstract

Rubinstein–Taybi syndrome (RSTS) is a rare genetic disorder affecting about one in 300,000 births, marked by intellectual disability, delayed growth and motor development, distinctive facial features, and an increased risk of tumours. Changes in the CREBBP gene cause RSTS type 1, while type 2 is linked to EP300. Most cases involve CREBBP variants.

We report a six-year-old boy from Srinagar, Jammu and Kashmir, India, diagnosed with RSTS type 1 based on clinical examination and radiological findings. Using whole exome sequencing and array comparative genomic hybridisation, we identified a novel, *de novo* 261 kb microdeletion on chromosome 16p13.3 (Chr16:3,694,760–3,955,374) affecting three genes: DNASE1, TRAP1, and CREBBP. This deletion was absent in both parents. Gene expression analysis showed a significant reduction of CREBBP in the patient, indicating that loss of gene function drives the condition. This is the first report of this specific microdeletion causing RSTS type 1.

Our findings highlight the importance of combining detailed clinical evaluation with advanced genomic tools to uncover the causes of rare diseases and expand the known spectrum of genetic variants across diverse populations.



A 261 kb deletion spanning three genes is causing Rubinstein-Taybi syndrome type 1 in a 6-year-old boy belonging to Kashmir valley, India

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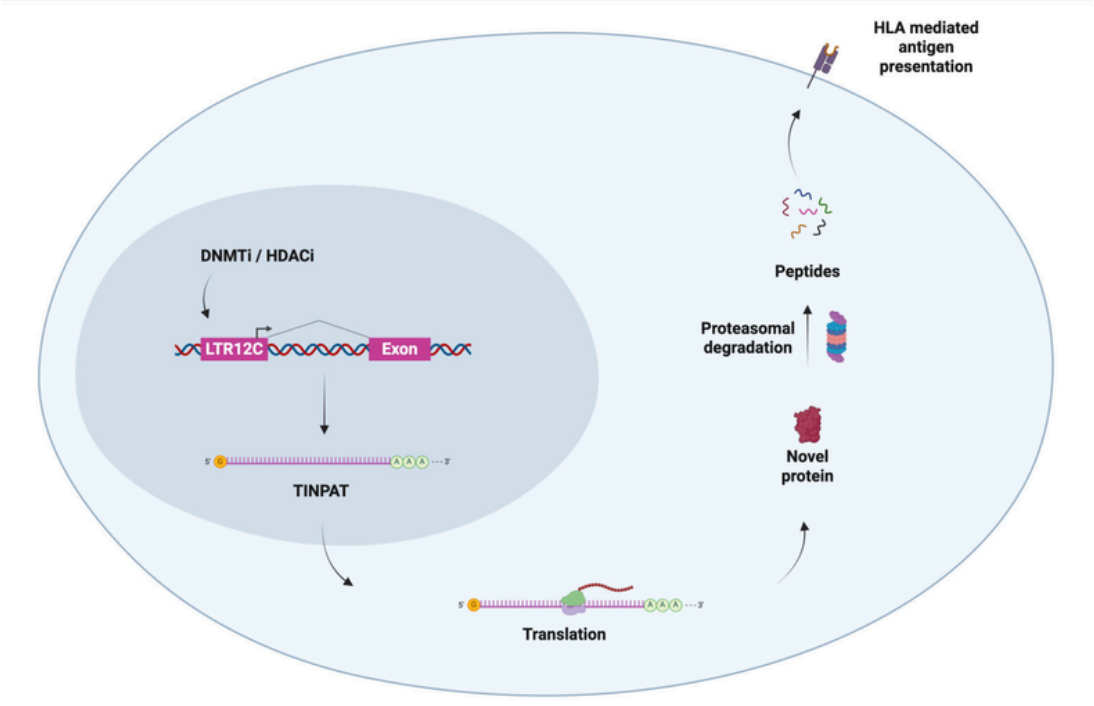
Epigenetic Therapy-Induced Antigens – A New Class of Immunotherapy Targets

Abstract

Immunotherapies targeting cancer-specific neoantigens have revolutionised the treatment of cancer patients. Recent evidence from our lab suggests that epigenetic therapies synergise with immunotherapies, mediated by the de-repression of endogenous retroviral element (ERV)-encoded promoters (Brocks et al, Nature Genetics, 2017).

Now, using deep RNA sequencing from cancer cell lines treated with DNA methyltransferase inhibitor (DNMTi) and/or Histone deacetylase inhibitor (HDACi), we assemble a *de novo* transcriptome and identify several thousand ERV-derived, treatment-induced novel polyadenylated transcripts (TINPATs). Using immunopeptidomics, we demonstrate the human leukocyte antigen (HLA) presentation of several treatment-induced neopeptides (t-neopeptides) arising from TINPATs. We illustrate the potential of the identified t-neopeptides to elicit a T-cell response to target cancer cells effectively. We further verify the presence of t-neopeptides in Acute myeloid leukaemia (AML) patient samples after *in vivo* treatment with the DNMT inhibitor Decitabine.

Our findings highlight the potential of ERV-derived antigens in epigenetic and immune therapies directed against cancer (Goyal et al., Nature Communications, 2023). These antigens hold the promise to treat any type of cancer and offer the possibility of generating off-the-shelf immunotherapeutic approaches.



DNMT and HDAC inhibition induce immunogenic neoantigens from human endogenous retroviral element-derived transcripts

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YI 08

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Investigating differential immunomodulatory effects of lithium in bipolar disorder responders and non-responders under inflammatory stress

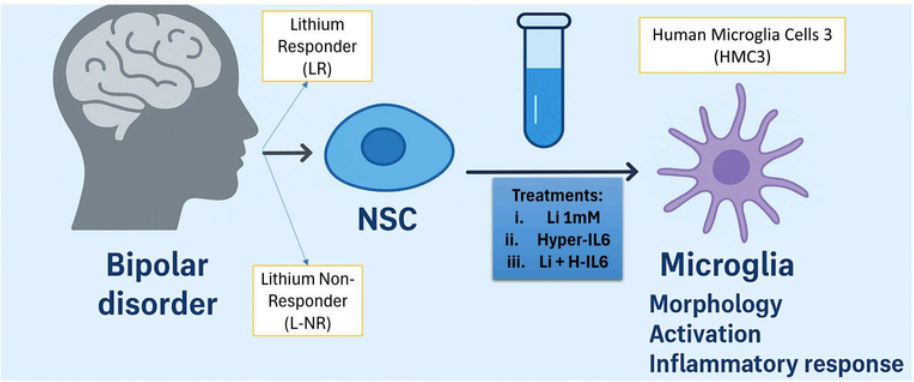
Abstract

Bipolar disorder (BD) is a chronic psychiatric illness characterised by recurrent episodes of mania and depression. Lithium (Li) remains the gold-standard treatment for BD; however, only 30–50% of patients respond adequately. The biological basis of this differential response remains poorly understood. Emerging evidence suggests that a subset of BD patients exhibits elevated inflammatory profiles, raising the possibility that neuroinflammation may influence Li responsiveness. Given lithium's known immunomodulatory effects, we hypothesise that Li attenuates inflammatory signalling in BD patients with heightened immune activation, and that underlying neuroinflammation may modulate this effect.

To test this, we use patient-derived neural stem cells (NSCs) from Li responders and non-responders, exposed to hyper-IL6 to induce a pro-inflammatory state, with or without Li treatment. The conditioned media (secretome) from these NSCs will be applied to human microglial cells (HMC3) to evaluate how Li-mediated changes in NSC signalling influence microglial activation. Morphological and functional alterations in microglia will be assessed, focusing on cytoskeletal remodelling, activation markers, and inflammatory gene expression.

We expect that (i) baseline immune responses will differ between NSCs derived from Li responders and non-responders, and (ii) Li treatment will differentially modulate their inflammatory secretome. Consequently, microglial activation and morphology are anticipated to reflect these differences, indicating a neuro-immune interplay that may underlie Li responsiveness.

This study aims to elucidate how neuroinflammatory states modulate lithium's immunoregulatory effects, offering novel insights into cellular mechanisms governing treatment variability in BD. Understanding these interactions may help identify biomarkers predictive of Li response and inform personalised therapeutic strategies.



Differential inflammatory response of patient-derived NSCs in relation to their diagnosis

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YI 09

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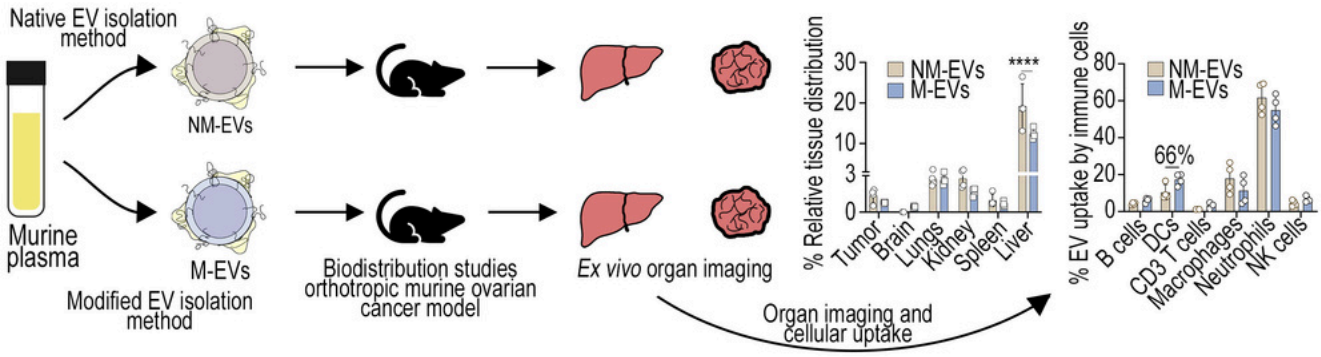
Liver evading nanoparticles for precise delivery to tumours: Learning from naturally occurring extracellular vesicles (EVs)

Abstract

BDrug/RNA therapeutic-loaded nano-sized particles (e.g., Doxorubicin liposomes) have improved treatment response. However, a majority of nanoparticles are sequestered by the liver within the first hour of administration. This liver sequestration reduces tumour localisation, alleviating their anti-tumour activity. To achieve an optimum tumour localisation and overcome liver sequestration, a study by Ouyang et al. (Nature Materials, 2020) predicts the injection of 1.5 quadrillion nanoparticles. Achieving this number could be challenging and could lead to nanoparticle-mediated toxicities. To circumvent this challenge, efforts to develop lipid-based nanoparticles with liver-evading properties are underway.

During my PhD and postdoctoral training, I developed a methodology to modify extracellular vesicles (M-EVs) with liver evasion ability. Using bioluminescence studies, I observed that intravenous injection of M-EVs reduced their liver sequestration significantly (~40%) compared to non-modified EVs (NM-EVs). Molecular profiling of these M-EVs and NM-EVs highlighted that M-EVs lacked a protein corona that promotes liver sequestration. Our results were in corroboration with previous studies, which highlighted the role of protein corona in modulating tissue biodistribution. However, more interestingly, I observed a 2.5-fold higher endocytosis of M-EVs by dendritic cells compared to NM-EVs. More importantly, as these EVs were endogenous (i.e., obtained from the plasma of non-tumour-bearing mice), I observed a consequent reduction in CD3+ T cell numbers. These comprehensive immune profiles between mice injected with M-EVs and NM-EVs suggest an immune-specific feedback loop. Therefore, future research employing an inflammatory source (e.g., macrophages) to obtain M-EVs encapsulating RNA therapeutics that prime dendritic cells to educate T cells with anti-tumour potential is warranted.



Injected synthetic nanoparticles and extracellular vesicles (EVs) are rapidly cleared by liver leading to a decrease in tumour cell accumulation of injected carriers

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YI 10

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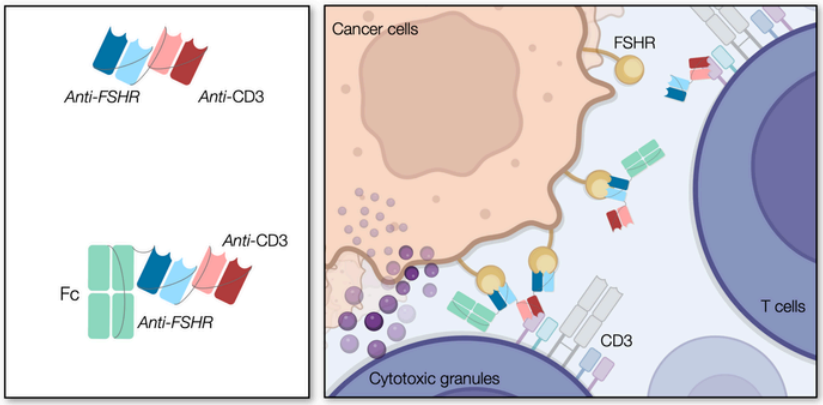
Targeted ovarian cancer immunotherapy through engineering of follicle-stimulating hormone receptor (FSHR) antibody to engage T cells

Abstract

Ovarian cancer (OC) is the deadliest gynecologic malignancy and is a high need area for novel therapeutic interventions; development of treatment approaches with anti-tumour function in the tumour microenvironment is of particular importance. Follicle-stimulating hormone receptor (FSHR) is one such important target, with selective expression in ovarian granulosa cells and high expression in 50-70% of serous ovarian cancer cells.

We developed several monoclonal antibodies which targeted FSHR and focused our studies on a potent cell-binding clone for detailed characterisation. We further utilised this clone to develop a bispecific T cell engager (TCE) targeting FSHR and evaluated for binding to FSHR and CD3 as well as its *in vitro* and *in vivo* killing efficacy in human OC model systems.

We observed that D2AP11 bound specifically to FSHR-positive cells and tissues, with no nonspecific cell or tissue binding. As D2APP induced modest ADCC, we sought to improve its potential by designing D2AP11-TCE (T cell engager). Besides exhibiting strong bidirectional binding, this TCE induced potent *in vitro* killing of multiple human ovarian cancer cells, including CaOV3, OVISE, Kuramochi, OVCAR3-FSHR, OVCAR4, and PEO-4, which are resistant to various drugs without off-target toxicities. IC50 values of D2AP11-TCE killing were obtained at 24.7 and 15.9 ng/ml in OVISE-FSHR and OVCAR3 cells, respectively. Further, this TCE significantly attenuated tumour burden and enhanced survival in OC-challenged mice, indicating its *in vivo* efficacy. These studies illustrate the utility of targeting FSHR for different human OC subsets. Additionally, the study demonstrates the utility of a potent bispecific human T cell engager through engineering of FSHR to impact ovarian tumour growth in the pre-clinical settings.



Schematic of FSHR targeted T cell engager working mechanism

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YI 11

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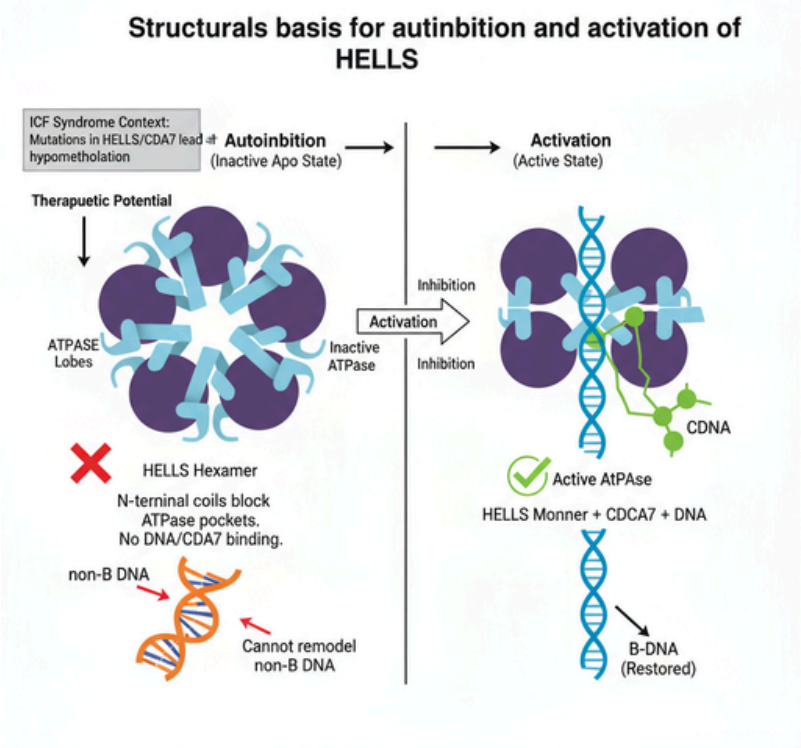
Structural basis for autoinhibition and activation of HELLS

Abstract

HELLS (lymphoid-specific helicase) is an SNF2-family ATP-dependent chromatin remodeler abundantly expressed in proliferating lymphoid cells and embryonic stem cells. It plays a specialised role in DNA methylation and is uniquely inactive in its apo form. Mutations in HELLS cause the rare ICF syndrome (immunodeficiency–centromeric instability–facial anomalies), characterised by centromeric hypomethylation. Another gene implicated in ICF, CDCA7, binds non-B-form DNA exclusively at centromeres and recruits HELLS, activating its ATPase and nucleosome remodelling activities.

We aim to understand the molecular mechanisms of HELLS autoinhibition and activation, including its role in remodelling non-B-form DNA into B-form DNA. Using high-resolution cryo-EM, we determined structures of HELLS in apo form and in complex with CDCA7 and DNA. In the apo state, HELLS exists as a dynamic hexamer; its N-terminal coiled coils interact with ATPase lobe 1, preventing DNA and CDCA7 binding and maintaining autoinhibition. The ATPase lobes are open and do not form a functional ATP-binding pocket. Deletion of ATPase lobe 2 does not alter oligomerisation, but DNA binding disrupts the hexamer, and HELLS binds as a monomer when interacting with CDCA7 and DNA.

Our study provides a mechanistic framework for understanding HELLS regulation and activation, highlighting how protein–protein and protein–DNA interactions relieve autoinhibition to enable chromatin remodelling, with implications for ICF syndrome and epigenetic regulation.



HELLS is autoinhibited in its hexameric apo state by N-terminal coil interactions that block DNA and CDCA7 binding, inactivating its ATPase. Binding of CDCA7 and non-B DNA disrupts the hexamer, relieves autoinhibition, and activates HELLS for chromatin remodelling, offering insight into ICF syndrome pathogenesis and epigenetic regulation

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YI 12

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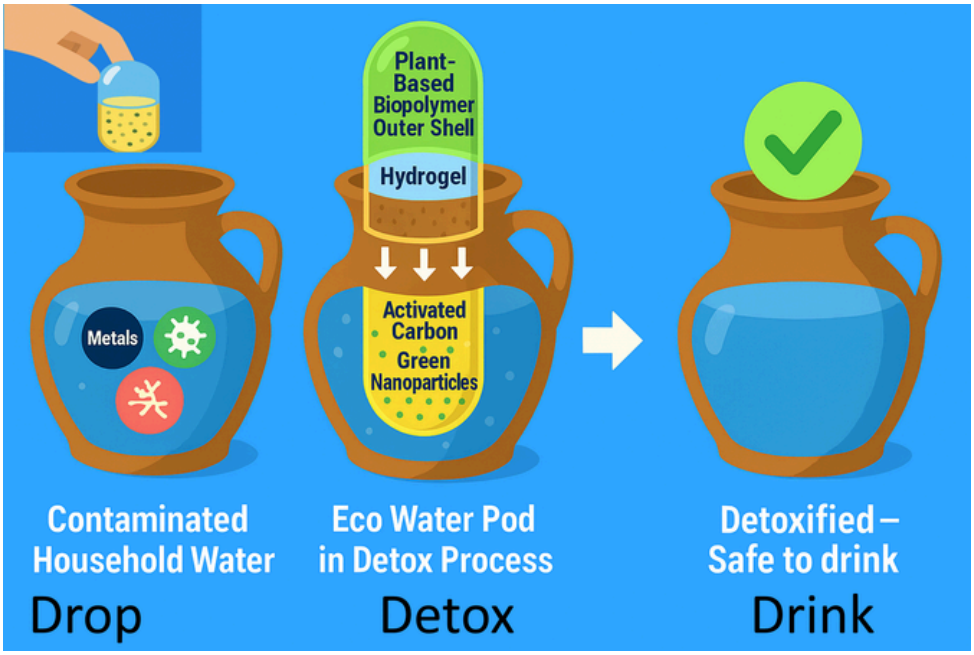
Eco Water Pods: Biopolymer-based hydrogel capsules for sustainable water detoxification

Abstract

Access to clean, safe drinking water is one of the most urgent challenges communities across the world face, especially in rural and semi-urban areas. My research introduces Eco Water Pods, which are small, biodegradable biopolymer hydrogel capsules that purify contaminated water in a simple, sustainable way. Each capsule is made from plant-based biopolymers, such as pectin and cellulose, combined with activated carbon and green-synthesised nanoparticles. When the pod is dropped into polluted water, it begins an eco-friendly detox process, adsorbing heavy metals, organic pollutants, and harmful microbes, and leaving the water clear and safe to drink. The pods are biodegradable, low-cost, and energy-free, making them suitable for households, disaster relief zones, and areas with limited access to clean water. In laboratory studies, they showed excellent efficiency in removing iron, lead, and copper, while improving water quality within minutes. This project combines green nanotechnology and biotechnology to create a practical, scalable solution that bridges science and sustainability. In the future, I plan to enhance the design with colour-based or sensor-integrated indicators for real-time water quality monitoring.



A simple "Drop–Detox–Drink" process showing how Eco Water Pods made of plant-based biopolymer hydrogels remove metals and microbes from contaminated water, making it safe to drink.

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YI 13

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Molecular insights into obesity-associated neurodegeneration: A quantitative proteomic approach

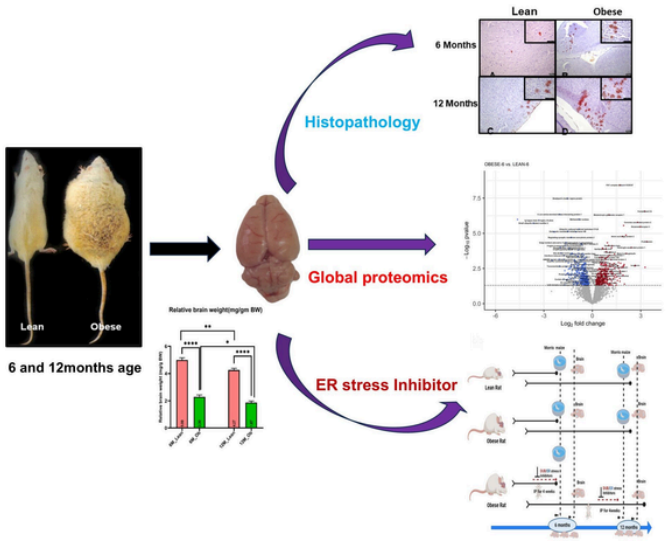
Abstract

Obesity is a global public health challenge affecting millions of people, significantly increasing the risk of neurodegenerative diseases, especially with advancing age. The treatment and management of these diseases remain clinically challenging. Therefore, a deeper understanding of the molecular mechanisms linking metabolic dysregulation to neurodegeneration is crucial, as the interplay among obesity, ageing, and neurodegeneration remains poorly elucidated.

In this study, we investigated proteomic alterations in the cortex and hippocampus of 6 and 12-month-old WNIN Ob/Ob obese rats, a spontaneous obesity model, compared with lean rats using label-free quantitative proteomics by mass spectrometry. We also carried out histopathological assessments using haematoxylin and eosin, Nissl, and Congo red staining to evaluate neuronal integrity and amyloid deposition.

Obese rats exhibited significant differences in body weight, with a greater than 50% reduction in relative brain weight compared to age-matched lean rats. Histological analysis demonstrated increased pyknotic nuclei, reduced cell numbers in the hippocampal CA3 region, and extensive amyloid aggregation in the brains of obese rats, indicating neurodegeneration. Furthermore, quantitative proteomics identified over 3000 proteins, revealing significant alterations in cholesterol metabolism, Rho GTPase signalling, mitochondrial metabolism, synaptic vesicle trafficking, synaptic plasticity, and oxidative stress in aged obese rats. Notably, synaptic and proteostasis-related proteins, including neuritin, N-cadherin, UCHL1, and GSK3β, were consistently downregulated in obese rats at both ages. Westernblot analysis confirmed increased ubiquitinated proteins in obese individuals, suggesting impaired protein homeostasis.

Together, these results indicate that obesity exacerbates neurodegeneration by disrupting synaptic integrity and proteostasis, providing a molecular link between metabolic stress and age-associated brain dysfunction. These findings laid the foundation for ongoing ubiquitinome profiling and pharmacological intervention studies using the ER stress inhibitor 4-PBA to identify key molecular players driving obesity-associated neurodegeneration. Ultimately, our goal is to identify novel biomarkers and key therapeutic targets for neurodegenerative diseases.



Global proteomic profiling unveils ER stress-related mechanisms in obesity-induced neurodegeneration

YI 14

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Vaginal sialidase activity and the microbiome: evaluating bacterial vaginosis diagnosis in Indian women

Abstract

Bacterial vaginosis (BV), a common vaginal dysbiosis affecting reproductive-age women, is characterised by low abundance of protective *Lactobacillus* species and high abundance of Gram-variable and Gram-negative anaerobes (1). In low-resource settings such as India, BV remains underdiagnosed due to the lack of molecular diagnostic tests and reliance on subjective microscopic methods. Conventional approaches, Amsel's criteria and Nugent scoring require skilled personnel, are time-consuming, and prone to variability, often resulting in misdiagnosis and inappropriate empirical antibiotic use. This contributes to antimicrobial resistance, recurrent infections, and unresolved dysbiosis. Previous research shows that sialidase, produced by BV-associated bacteria such as *Gardnerella* and *Prevotella*, is a prominent marker of BV in Caucasian women (2). Presence of sialidase activity has also been linked to increased risk of preterm birth, stillbirth, and miscarriage (3).

In India, however, the status of vaginal sialidase activity in women with BV has not been investigated. Hence, the diagnostic relevance of sialidase remains unvalidated, despite reported BV prevalence rates of 14–33% in major cities (4). This represents a critical knowledge gap, as microbial community structure and enzyme expression may differ across populations. Our preliminary findings confirm measurable sialidase activity among Indian women with BV, underscoring its potential universality as a biomarker.

This study aims to systematically evaluate sialidase activity in BV-positive Indian women to determine its diagnostic and prognostic value, thereby bridging a translational gap and advancing biomarker-driven approaches to improve women's reproductive health globally.

References

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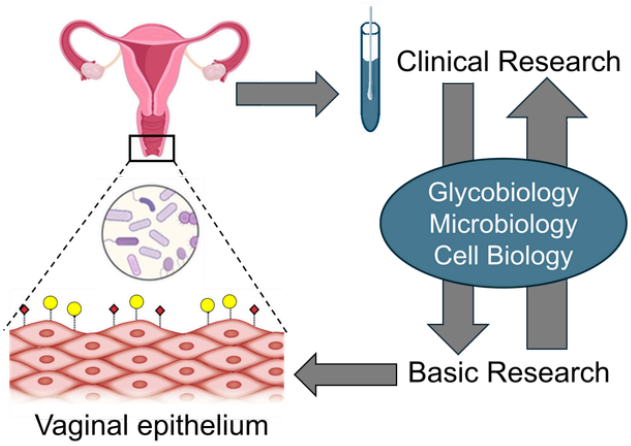

From clinical glycobiology to basic research: Unraveling host–microbiota interactions in vaginal dysbiosis

YI 15

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Identification of novel early detection markers for chronic hepatitis virus infection-associated hepatocellular carcinoma

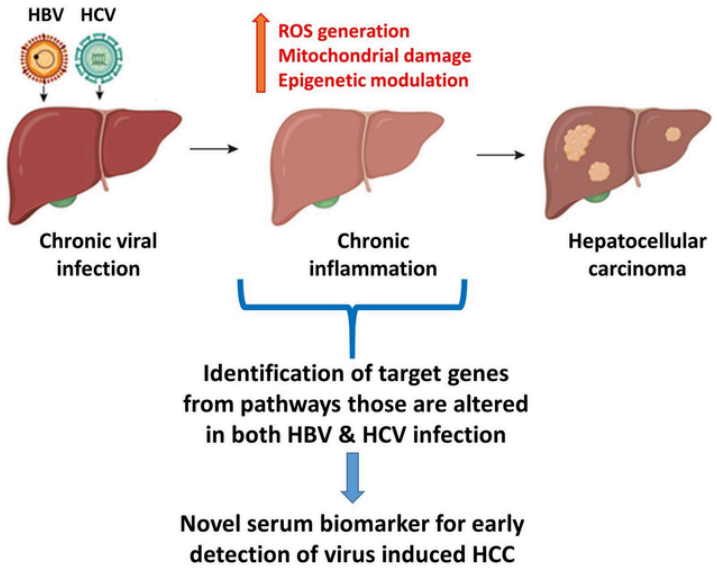
Abstract

Chronic hepatitis B and C virus (HBV and HCV) infection leads to the development of a wide range of liver pathogenesis, including hepatocellular carcinoma (HCC). HCC is a very devastating disease and remains challenging because of poor diagnosis at the early stage and a lack of proper treatment. For these reasons, in most cases, invasion, migration, and resistance of cancer cells occur, resulting in a high cancer recurrence rate in patients.

We aimed to identify specific targets for detecting HCC progression at very early stages. We re-analysed multiple RNA-sequence datasets from HBV- and HCV-infected cell lines and patient samples available in public databases and identified specific genes or pathways commonly altered in both HBV and HCV infection. We further validated the expression of those genes in the HCV-infected cells from our laboratory.

Gene ontology analysis showed that the most altered pathways in both HBV and HCV infection are responses to hypoxia, mitochondrial alteration and epigenetic regulations. We selected six genes among them, CITED2, LIF, EGR1 were downregulated, and EGLN3, SOX4 and BCAN-AS1 were upregulated in both HBV- and HCV-infected samples. Expression of these genes showed similar results in the HCV-infected cells from our laboratory.

Our preliminary data indicated the commonly altered pathways and genes involved in virus-mediated HCC. We are currently focusing on determining the role of these genes as an early detection marker for virus-induced HCC. We are also interested in investigating the molecular mechanisms behind these altered pathways. This study may lead to the identification of specific targets that can be used in combination with conventional markers to improve the management of HCC at an early stage.



Identification of early detection markers for virus-induced HCC

YI 16

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S. aureus exposure during cutaneous antigen sensitisation causes basophil- and IL-4-dependent exaggerated food anaphylaxis

Abstract

The skin of patients with atopic dermatitis (AD) is frequently colonised by *Staphylococcus aureus* that produces superantigens, particularly staphylococcal enterotoxin B (SEB), which has been associated with food allergy. However, the mechanisms underlying these associations are unknown. Interleukin-4 (IL-4) plays an important role in food allergy.

We found serum IL-4 levels were elevated in AD patients with *S. aureus* skin colonisation and correlated with the presence of food allergy. Using a mouse model of AD elicited by epicutaneous application of antigen to tape-stripped skin, we demonstrate that application of antigen together with superantigen-producing *S. aureus*, but not superantigen-negative *S. aureus*, caused a heightened systemic antigen-specific T helper-2 (Th2) response, elevated serum IL-4 levels and exaggerated anaphylaxis to oral, but not intraperitoneal (i.p.), OVA challenge compared with application of antigen alone. T cell-derived IL-4 acted on intestinal epithelial cells to enhance intestinal permeability and exaggerate anaphylaxis to an enteral antigen challenge. Furthermore, we demonstrate that SEB bound to keratinocytes via CD40 and triggered IL-33 release, which, in turn, led to T cells producing IL-3, thereby eliciting a basophil influx in skin-draining lymph nodes (dLNs). Basophil-derived IL-4 augmented Th2 polarisation by dendritic cells that captured cutaneously applied antigen and emigrated to dLNs.

These results suggest novel therapeutic interventions that might attenuate food allergy in these patients.

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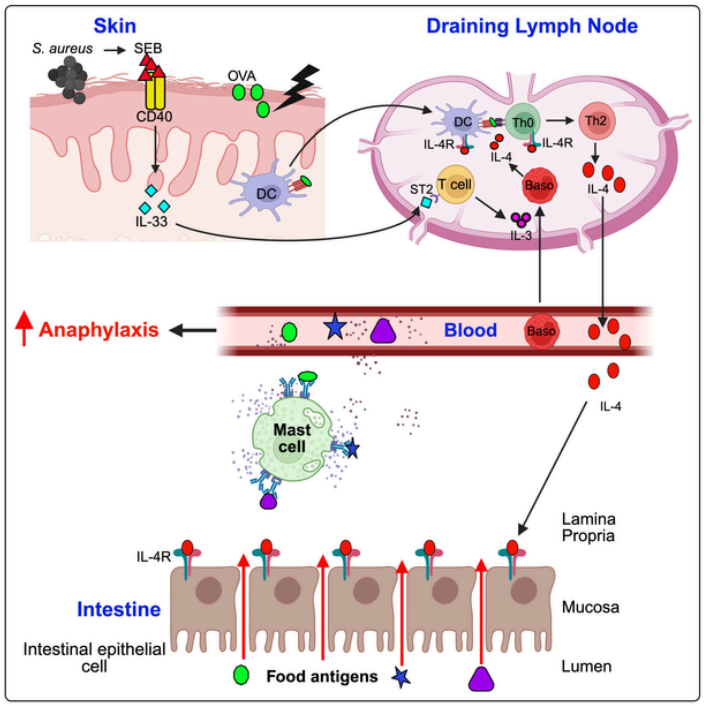

S. aureus infection on atopic skin amplifies food-induced anaphylaxis

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YI 17

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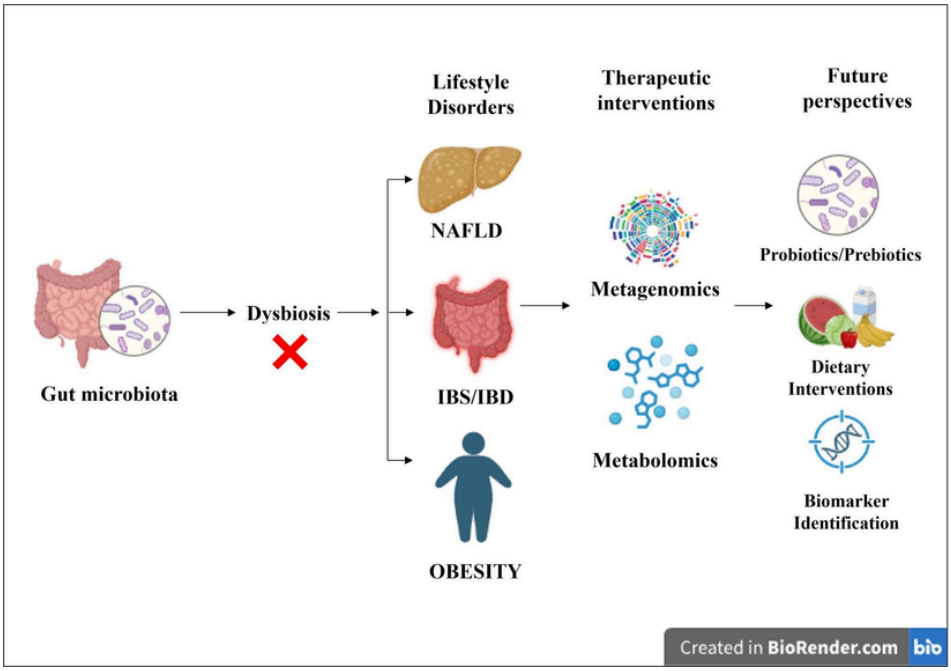
From microbes to metabolism: Exploring the microbiome's role in lifestyle disorders

Abstract

Non-alcoholic fatty liver disease (NAFLD) has emerged as a major global health concern and is characterised by excessive fat accumulation in the liver, independent of alcohol consumption. Increasing evidence suggests that gut microbiota dysbiosis plays a key role in NAFLD pathogenesis by disrupting host metabolic and immune pathways. Around 5-10% of the world population suffers from the Irritable Bowel Syndrome (IBS), a common disorder related to the stomach and intestines. Persistent and untreated IBS in patients may lead to complications such as Inflammatory Bowel Disease (IBD). The current diagnostic approach relies on symptom-based criteria. IBD patients are diagnosed with a biopsy or colonoscopy, which is an invasive method. Short-chain fatty acids (SCFAs) play an important role in the pathophysiology of IBS. Considerations of IBS and NAFLD as lifestyle disorders.

We collected samples from healthy individuals and NAFLD and IBS patients, followed by DNA extraction. Extracted DNA was quantified, followed by library preparation and sequencing using Oxford Nanopore technology. Sequencing data were analysed for taxonomic profiling, functional annotation, and diversity indices. Healthy individuals exhibited higher alpha diversity and a more balanced microbial composition compared to NAFLD patients, who showed reduced diversity and dysbiotic shifts. While dominant phyla such as *Pseudomonadota*, *Bacteriota*, and *Bacillota* were detected in both groups, their relative abundance varied significantly in NAFLD samples, reflecting a disrupted gut-liver axis.

The study demonstrates a clear relationship between gut microbiota composition and NAFLD progression. Metagenomic insights highlight microbial imbalances that contribute to disease pathogenesis, supporting the potential of microbiome-based personalised therapeutic interventions.



Gut microbiome and lifestyle diseases - future perspective

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YI 18

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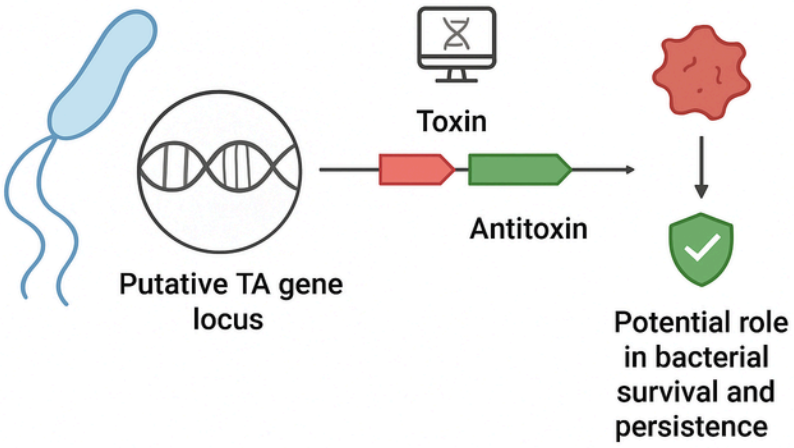
Identification of a putative toxin-antitoxin gene in the human pathogen Helicobacter pylori

Abstract

Helicobacter pylori is a Gram-negative bacterium implicated in chronic gastritis, peptic ulcers, and gastric cancer. The persistence of this pathogen under host-induced stress conditions is critical for its virulence, yet the underlying molecular mechanisms remain incompletely understood. Toxin–antitoxin (TA) systems are small genetic modules known to regulate bacterial stress responses, dormancy, and survival. In this study, we performed *in silico* analysis and genome mining of the *H. pylori* strain 26695 to identify putative TA genes. One candidate locus was further characterised through molecular cloning and transcriptional analysis. Preliminary results indicate that the identified TA gene may modulate bacterial survival, suggesting a role in persistence and pathogenesis. These findings provide a foundation for exploring TA systems as potential therapeutic targets against *H. pylori* infections.



Identification of a putative toxin-antitoxin gene in the human pathogen Helicobacter pylori

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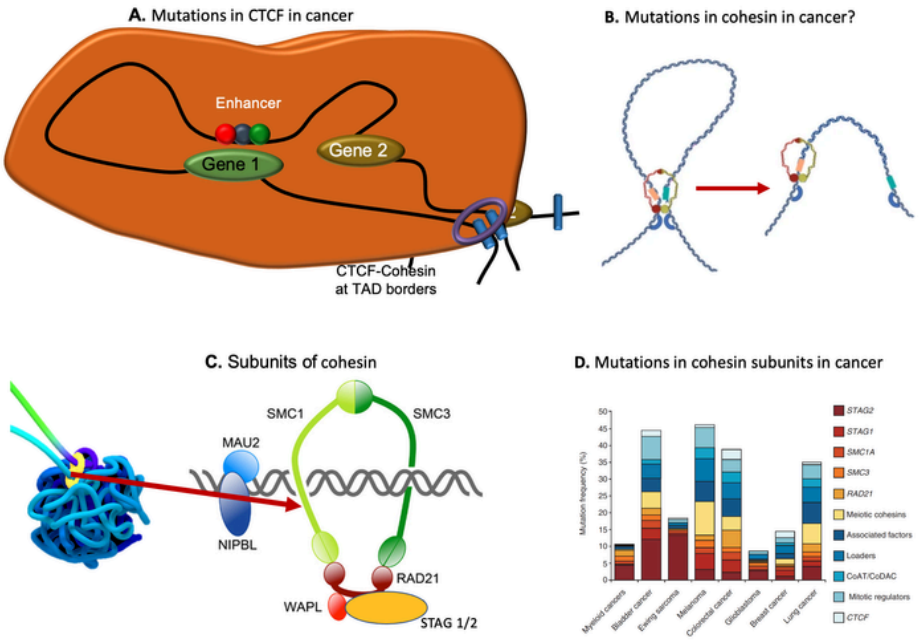
Chromatin architecture in crisis: How altered organisation drives disease

Abstract

Our research aims to investigate how CTCF and cohesin coordinate the process of loop extrusion to organize the genome in three dimensions and facilitate proper gene regulation. We are particularly interested in understanding how errors in this mechanism; such as altered binding of CTCF, mutations in cohesin components, or defects in the extrusion process disrupt chromatin architecture, leading to misregulated gene expression, compromised genome stability, and disease. By dissecting these processes, we hope to reveal how precise spatial genome organisation is maintained under normal conditions and how its perturbation contributes to cellular dysfunction.

To address these questions, we employ molecular biology techniques to manipulate CTCF and cohesin, and genomic methods to map three-dimensional interactions and their functional consequences. Through this integrated strategy, we aim not only to chart the dynamics of loop formation and maintenance but also to identify key regulatory nodes that are sensitive to perturbations.

Ultimately, our goal is to uncover the fundamental principles governing chromatin folding, provide insights into the relationship between 3D genome organization and gene regulation, and illuminate how errors in this system contribute to disease.



Chromatin architecture in crisis: How altered organisation drives disease

YI 20

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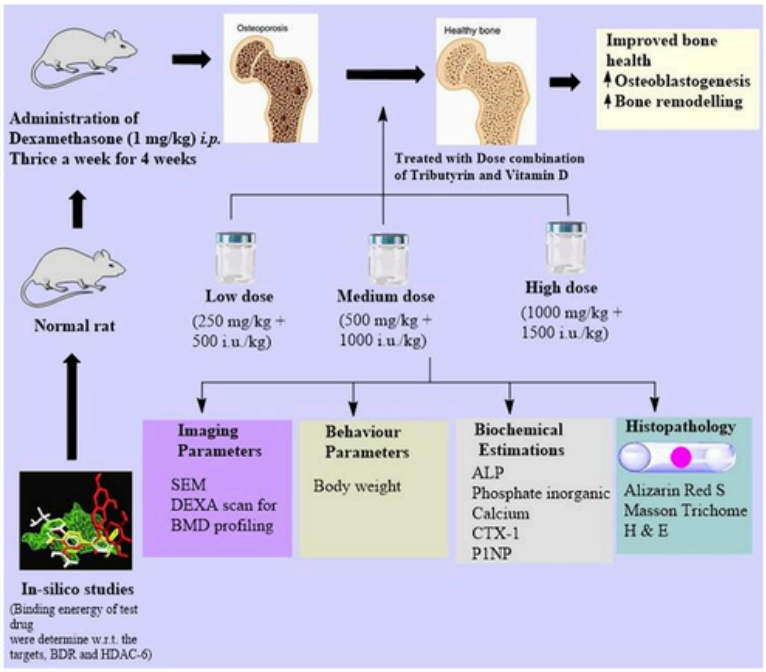
Pharmacological evaluation of tributyrin and vitamin D in the management of glucocorticoid induced osteoporosis in rats

Abstract

Osteoporosis is acknowledged as a bone predisposing disorder involving persistent breakdown of bone tissue, leading to a negative bone balance. It eventuates due to poor osteoblastic function (bone-forming cells) and hyperactivity of osteoclasts (bone-resorbing cells), directly contributing to impairment in bone remodelling and risk of sudden fractures. The International Osteoporosis Foundation (IOF) reports that 1 in 3 women and 1 in 5 men aged 50–55 years suffer from osteoporosis, with nearly 300 million people in the nation experiencing these painful bone deformities. The underlying mechanisms include decreased serum calcium and phosphate levels, chronic steroidal drug intake, altered vitamin D levels, and other pathological causes. It is a serious, chronic, and initially asymptomatic disorder requiring long-term pharmacotherapy of about 2–3 years.

The current preclinical study is designed to mitigate the complications associated with disease progression through an effective treatment regimen. The investigation employs a rat model for 28 days, wherein osteoporosis is induced by chronic exposure to dexamethasone for 28 consecutive days. The treatment, administered during the last 14 days, involves preventive therapy using tributyrin and vitamin D in specific dose combinations. Vitamin D and tributyrin stimulate calcium ion transporters and phosphate absorption, thereby improving bone health. This combination is aimed at elevating Ca^{2+} influx, enhancing systemic calcium availability by targeting catalysts like HDAC, and promoting bone cell differentiation and remodelling. The study primarily aims to determine the submaximal effective therapeutic dose with a short treatment duration and explore potential synergistic effects in managing osteoporosis.

Conclusively, it seeks to establish the bone restorative potential of this treatment approach to counteract the detrimental effects of the progressive bone disorder.



Pharmacological evaluation of the combination dose of tributyrin and vitamin D for the management of osteoporosis induced by the administration of corticosteroid dose

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Cracking cancer codes: Network biology meets machine learning in multi-omics

Abstract

Differentially expressed genes (DEGs) do not accurately reflect disease aetiology and are not context-specific. Additionally, DEGs-based approaches often fail to capture systems-level perturbations underlying disease states. To address these limitations, we developed a novel network-based approach that identifies differentially expressed paths and creates a functional subnetwork.
A novel node-weighting scheme was developed in which we computed expected fold change by regressing the absolute log fold change using a Loess fit with the control as the base expression, and the difference between the expected and observed log fold change was used as the node weight. This new node weighting scheme was incorporated into a tool called PathExt, which uses a knowledge-based directed network and omics data as inputs, then creates sub-networks to identify shortest paths, followed by functional sub-networks and key central genes.

PathExt was implemented in several studies where it outperformed DEGs-based methods. PathExt characterises subtype-specific genes in breast cancer, and machine learning models classified patients as responders/non-responders to a treatment with an AUROC of 0.80. FOXA1, CTNNB1, JUN, FOS, and ALB were found to be associated with chemoresistance. Likewise, in head and neck cancer, PathExt identified genes specific to HPV-positive (TP53, UBC) and HPV-negative (TGFB1, EGFR) tumours.
We proposed a generalisable and interpretable tool that can be implemented for any diseased condition to address disease heterogeneity and identify novel therapeutic targets.

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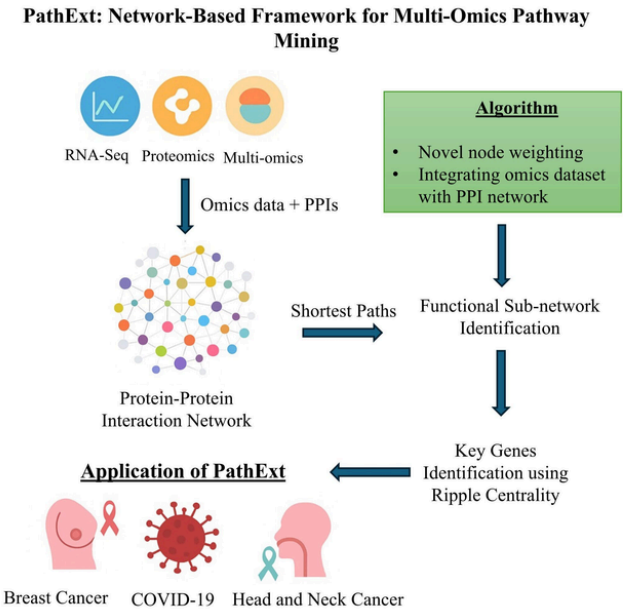

Framework of our novel network-based approach, which integrates omics data with protein-protein interaction networks to characterise disease-specific functional subnetworks and mechanistic insights for precision medicine applications.

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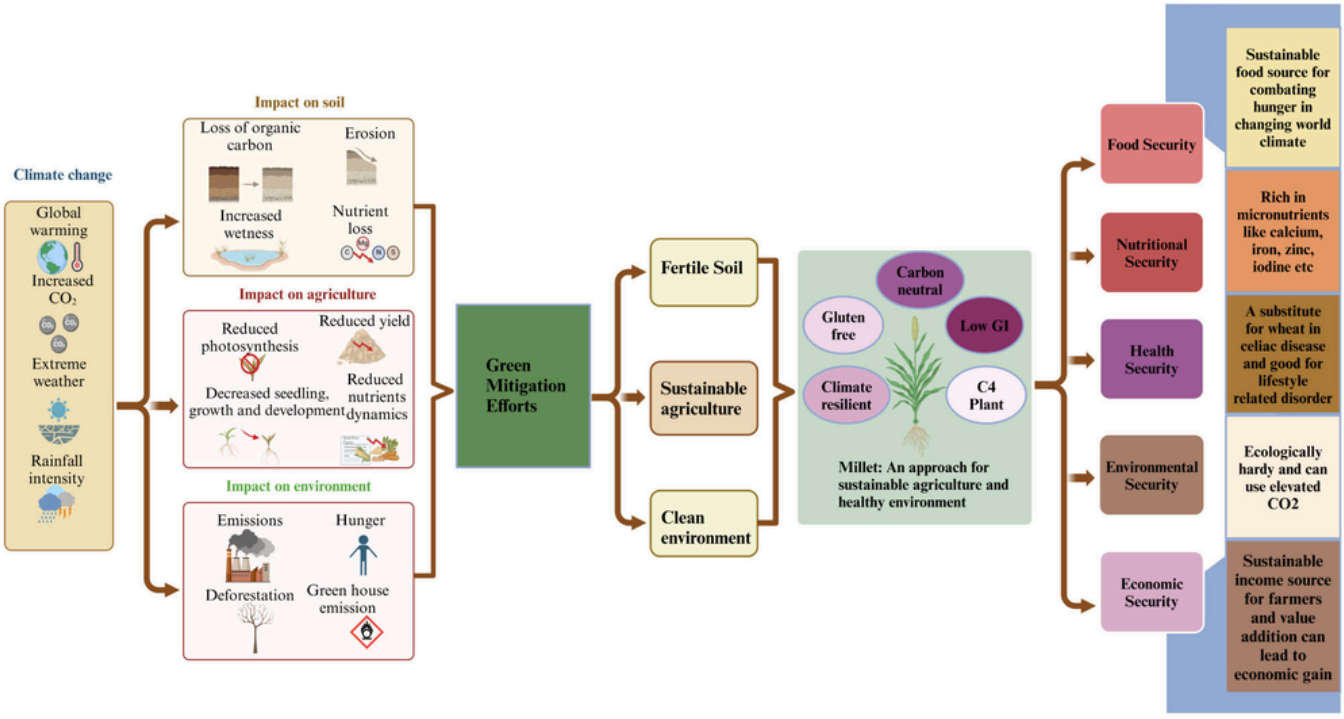
Unlocking the potential of small millets: Stress-responsive omics and starch-based innovation

Abstract

Feeding a projected ten billion people by 2050 is a major challenge, made more complex by climate change that threatens yields and nutritional security in staple cereals like rice, wheat, and maize. Small millets, however, remain underutilised despite their resilience and rich nutrient profiles. Capable of thriving in marginal, arid, and semi-arid regions with low inputs, they hold great promise for food security and ecological sustainability while aligning with Sustainable Development Goals such as zero hunger, climate action, and biodiversity.

Recent omics-based studies in foxtail, finger, proso, and barnyard millets have uncovered the molecular basis of stress tolerance. Transcriptomic analyses under drought, salinity, and heat reveal key genes, transcription factors, ion transporters, antioxidant pathways, osmolytes, and non-coding RNAs that drive resilience. Beyond climate adaptation, millet starch exhibits unique traits, including gluten-free status, high amylose content, and favourable swelling and pasting behaviour, making it valuable for functional foods and sustainable packaging. Structural and functional diversity across varieties includes differences in crystallinity, granule morphology, solubility, and gelatinisation. Physical modification methods further improve starch stability, enabling applications as thickening agents, stabilisers, edible films, and biodegradable bioplastics.

By combining omics-driven breeding with starch functionalisation, small millets can be advanced as both climate-smart crops and sources of industrially useful starches. Greater research and investment in this neglected group of nutri-cereals will be key to meeting future food and nutrition needs under changing climates.



Small millet: An approach for sustainable agriculture and healthy environment

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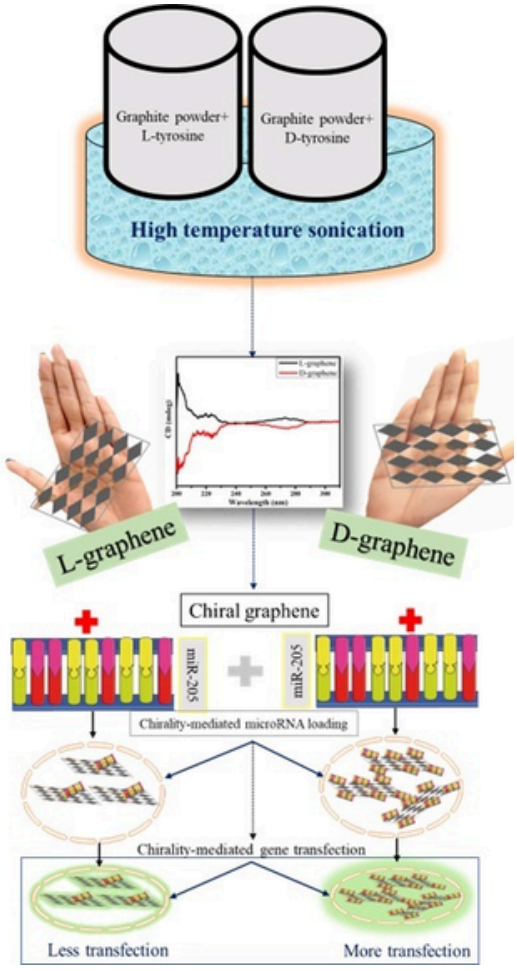
One-step simultaneous liquid phase exfoliation-induced chirality in graphene and their chirality-mediated microRNA delivery

Abstract

Graphene (G) has established itself as an exciting prospect for a broad range of applications owing to its remarkable properties. Recent innovations in chiral nanosystems have enabled sensors, drug delivery, catalysis, and other applications, owing to stereospecific interactions between nanosystems and enantiomers. As the molecular structure of G itself is achiral, introducing chirality in G by the simple attachment of a functional group (a chiral ligand) on the G nanosheet may result in more diverse applications.

We demonstrate direct liquid-phase exfoliation and chiral induction in G nanosheets, abbreviated as L-graphene and D-graphene, in the presence of chiral L-tyrosine and D-tyrosine, respectively, and by applying high-temperature sonication. The obtained exfoliated nanosheets demonstrated stable chirality, confirmed by circular dichroism. Fourier transform infrared (FTIR) spectra, Raman spectroscopy, transmission electron microscopy (TEM), X-ray photoelectron spectroscopy (XPS), and differential scanning calorimetry (DSC) showed functional, structural, morphological, surface, and thermal characteristics of L-graphene and D-graphene.

The hemocompatibility of these chiral graphenes was evaluated for the first time using human red blood cells. Lastly, this was the first attempt to explore enantiomeric binding between chiral L-graphene and D-graphene with microRNA (miR-205) and their potential for chirality-mediated gene delivery in prostate cancer cells.



Chirality-mediated microRNA loading and its delivery inside cells

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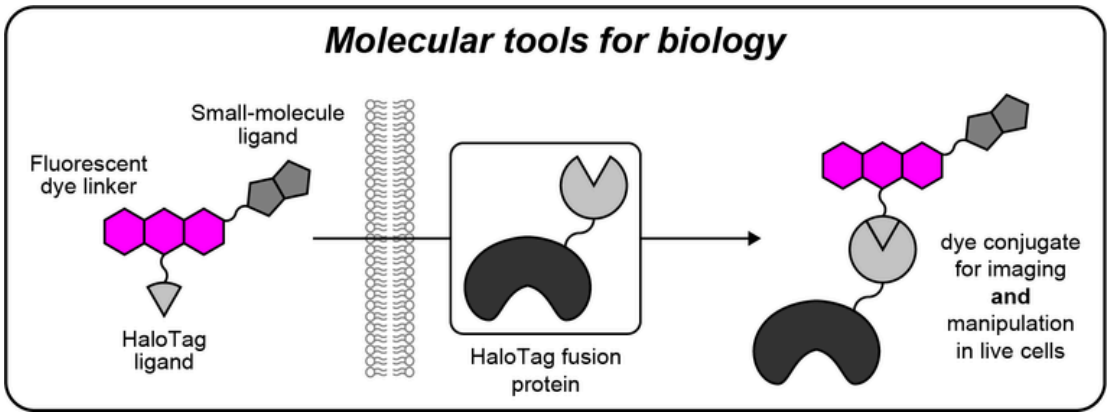
Organic dyes as molecular tools beyond imaging

Abstract

Small molecules are vital to biology and medicine. They are routinely employed to visualise, purify, and manipulate cellular components, and 90% of all drugs sold are small molecules. However, getting them across the cellular membrane and restricting their delivery to the target protein is challenging, hindering performance and mechanistic evaluation.

I will present our approach for utilising rhodamine dyes to enhance the cellular permeability of small-molecule ligands and deliver them to HaloTag fusions of target proteins rapidly and covalently. These multifunctional dyes combine the best of two worlds: 1. acute onset and dosage control of small molecules, and 2. the specificity of genetics. I will demonstrate the potential of multifunctional dyes by using biotin-rhodamine-HaloTag ligand for affinity capture of intracellular organelles/proteins and JQ1-rhodamine-HaloTag ligand for altering chromatin dynamics. This strategy is also extendable to visualise and manipulate other biomolecules (e.g., DNA, lipids). The multifunctional dyes enable both fluorescence visualisation and biochemical manipulation of the labelled proteins, thereby representing a new avenue in designing dye-based tools for biology. This general concept will apply to thousands of available small-molecule ligands, enabling new live-cell experiments.



Pharmacological evaluation of the combination dose of tributyrin and vitamin D for the management of osteoporosis induced by the administration of corticosteroid dose.

YI 25

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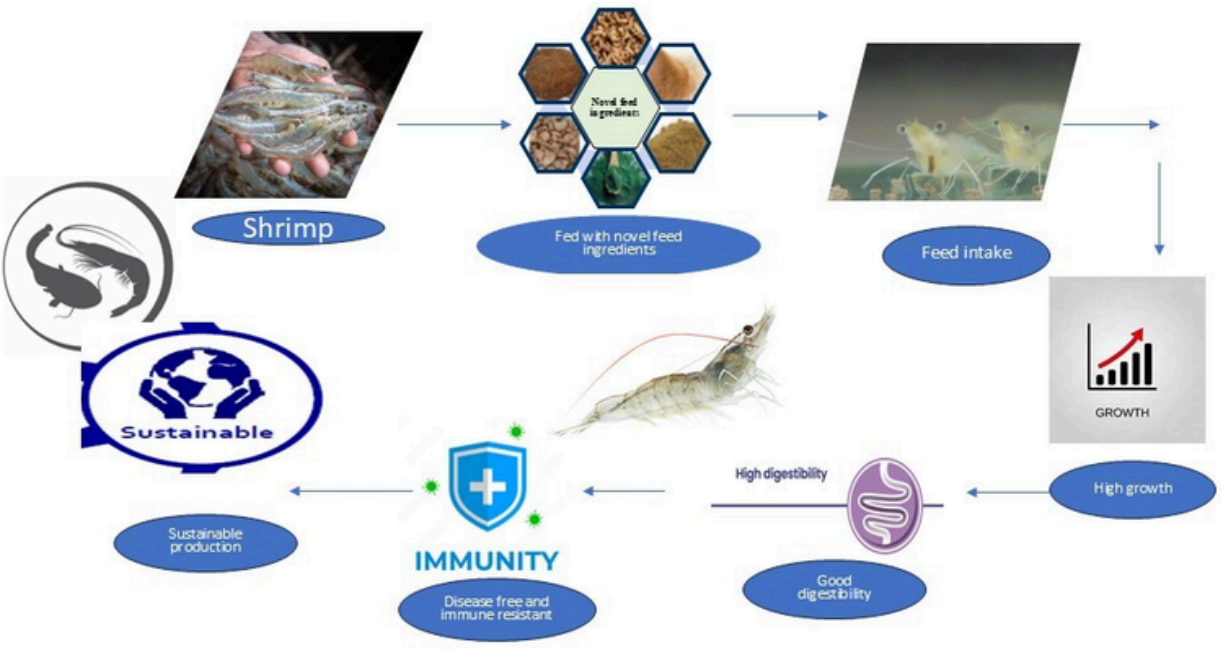
Impact of novel feed ingredients in growth, digestive function, biochemical responses, immunity and gene expression of Penaeus vannamei

Abstract

The transition towards a circular economy is driving interest in novel feed solutions that convert waste and underutilised biomass into valuable resources. These feeds enhance sustainability by reducing waste, lowering environmental impact, and improving resource efficiency, while supporting resilient and circular food and feed systems. An eight-week feeding trial was conducted to evaluate the potential of combining various novel feed ingredients as replacements for fish meal (FM) and to assess their effects on growth performance, nutrient digestibility, enzymatic activity, immune responses, tissue histology and gene expression in Pacific white shrimp (*Penaeus vannamei*). Five isonitrogenous (36% crude protein) and isolipidic (6% crude lipid) diets were formulated. In the control diet (Diet 1), FM was the primary protein source. In Diet 2, FM was replaced by a 1:1 mixture of poultry by-product meal (PBM) and single-cell protein (SCP). Diet 3 comprised insect meal (IM), rapeseed meal (RM), and SCP in equal proportions (1:1:1). Diet 4 contained fish waste (FW), peanut meal (PM), and SCP (1:1:1). Diet 5 was formulated with equal proportions of PBM, SCP, IM, FW, PM, and RM (1:1:1:1:1:1). Juvenile shrimp with an initial mean body weight of approximately 1 g were randomly distributed into experimental tanks following a completely randomised design (CRD), with triplicate groups for each treatment. Shrimp fed Diet 5 exhibited significantly higher ($p<0.05$) weight gain and specific growth rate (SGR), comparable to those fed Diet 1 and 3. Feed conversion ratio (FCR) was significantly improved ($p<0.05$) in shrimp fed Diet 1 and 5. Prophenoloxidase activity was significantly elevated ($p<0.05$) in shrimp fed Diet 1, with values comparable to Diet 5. Shrimp fed with Diet 1 and 5 showed significantly higher ($p<0.05$) expression of growth and immune genes compared to other treatments. Therefore, a balanced combination of PBM, SCP, IM, RM, PM and FW at equal inclusion levels (1:1:1:1:1:1) can effectively replace fishmeal without compromising growth performance, nutrient utilisation and health in *P. vannamei*.



Drive towards circular economy and sustainability through novel feed

YI 26

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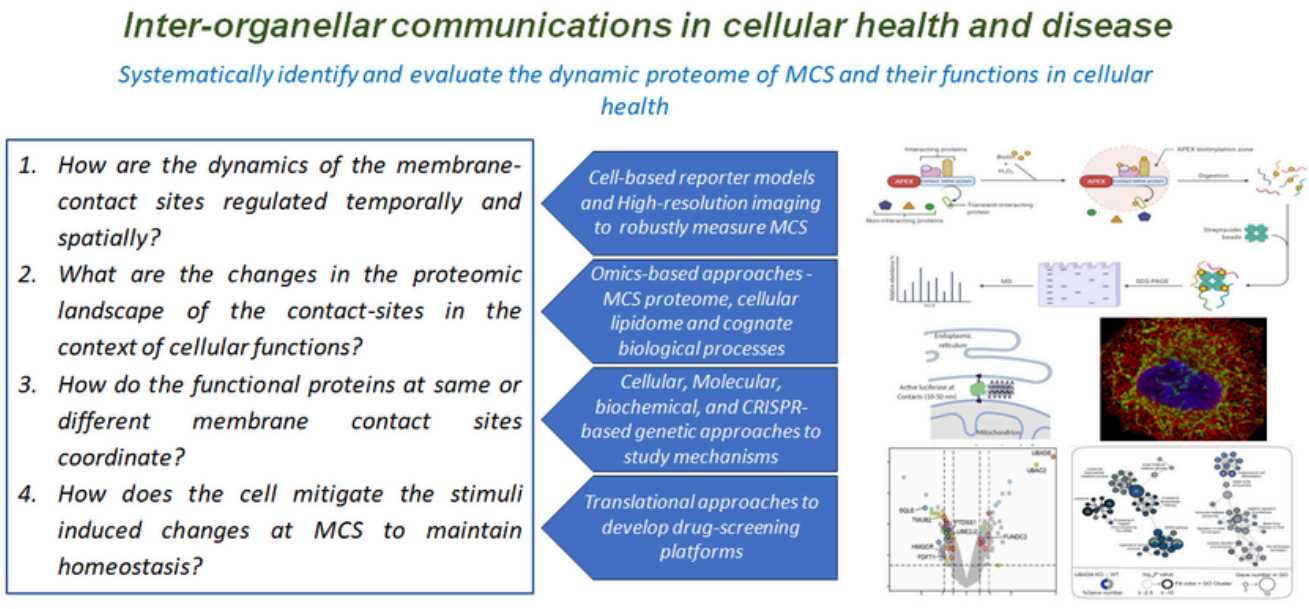
Organelle Biology lab: Inter-organellar communications in cellular health and disease

Abstract

Organelle communications via membrane contact sites (MCS) carry out essential biological processes ranging from innate immune signalling to cellular metabolism, organelle dynamics, apoptosis, and autophagy. MCS are highly dynamic, and the major organelles involved include mitochondria, ER, endosomes, lysosomes, peroxisomes, and lipid droplets, which perform critical metabolic and signalling functions in a coordinated manner. MCS are often found disrupted in various diseases. In the past five years, several metabolic, neurodegenerative, and ageing-associated disorders have been shown to dysregulate or circumvent the inter-organellar communications, specifically MCS, leading to disease pathology. Diseases that alter MCS add another layer of complexity to the elucidation of disease-specific interaction networks and are an active area of research in cell biology. Moreover, the molecular mechanisms by which dysregulation of MCS contributes to disease pathology and overall cellular health are unknown. Although there has been significant advancement in understanding how MCS affects cellular function, many questions about membrane contact sites remain unanswered. Understanding the biology of inter-organellar communications via MCS and their role in disease biology will be the focus of my laboratory.

My laboratory will use the cell-based models and employ proteomics, lipidomics, precision-guided gene editing tools, and molecular and biochemical approaches to address the following questions in the coming years: 1. How are the dynamics of the membrane-contact sites regulated temporally and spatially? 2. What are the changes in the proteome landscape of the contact-sites during external or internal stimuli? 3. What are cellular mechanisms to recuperate with the disease-induced changes in MCS to alleviate the pathology?

While answering these questions will help uncover the exact mechanistic roles of MCS in cellular homeostasis, it will also provide insights into complex disease biology.



Membrane contact sites in health and disease

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Natural product-inspired small molecules: A platform for drug discovery and biological modulation

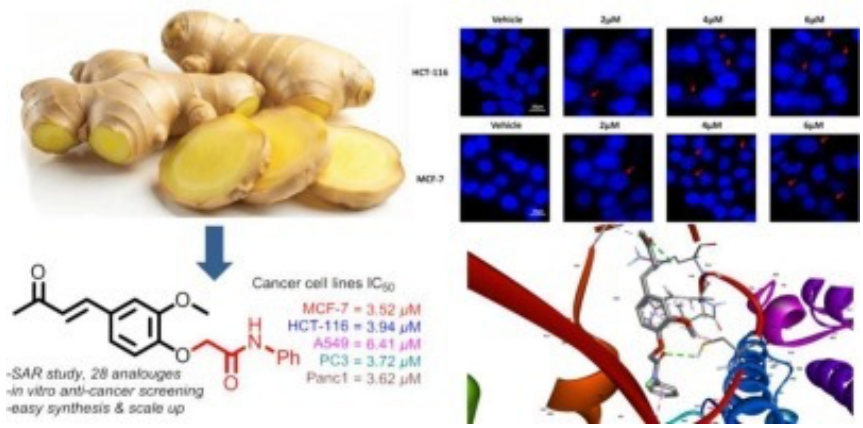
Abstract

Natural products have historically served as a rich source of therapeutically relevant molecules, offering structural diversity and biological specificity unmatched by synthetic scaffolds. However, direct application of these compounds is often limited by issues such as low bioavailability, poor selectivity, or complex synthetic accessibility. Our research addresses these challenges by designing, synthesising, and biologically evaluating derivatives of bioactive natural products, creating a versatile platform for drug discovery.

We have focused on phenoxy-acetamide derivatives of dehydrozingerone, thia-Michael derivatives of dehydrocostuslactone, scoparone analogues, and cycloartane-type triterpenes. These scaffolds were selected based on their inherent biological activity and structural features conducive to chemical modification. Using chemoselective, sustainable synthetic methods, we generated libraries of hybrid molecules and evaluated their anti-proliferative, anti-metastatic, and anti-inflammatory activities. *In vitro* and *in silico* studies guided structure-activity relationship (SAR) optimisation, leading to the identification of promising lead compounds with enhanced potency and selectivity.

In addition to their biological activity, some derivatives were designed as fluorescent probes, enabling cellular imaging and mechanistic studies and bridging the interface between medicinal chemistry and chemical biology. This integrative approach demonstrates how natural product scaffolds can be chemically diversified to generate molecules with dual therapeutic and diagnostic utility.

Looking forward, we aim to expand this platform by incorporating AI-assisted molecular design, predictive bioactivity modelling, and high-throughput screening, accelerating the discovery of new chemical entities for infectious, inflammatory, and cancer-related targets. Our work highlights the continued relevance of natural product-inspired chemistry in modern drug discovery, emphasising sustainable synthesis, rational design, and translational potential.



Natural product-inspired small molecules: A platform for drug discovery and biological modulation

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Spectroscopic and microfluidic diagnostic platforms in medicine and surgery

Abstract

Approximately 80% of organ transplantations in India rely on living donors. Expanding the use of marginal organs from deceased donors, especially Donations after Circulatory Death (DCD), could reduce this dependence. Ex vivo machine perfusion helps achieve DCD transplantation by optimising organ function before transplantation. Central to its success is an accurate, real-time viability assessment to minimise adverse recipient outcomes and guide perfusion strategies.

Burn injuries affect an estimated seven million people and cause 140,000 fatalities annually in India. Effective treatment begins with a precise diagnosis of burn depth, yet current methods rely heavily on subjective clinical evaluation.

To address these diagnostic gaps, we developed a portable spectroscopic device based on Resonance Raman Spectroscopy (RRS) to non-invasively and in real-time assess mitochondrial and haemoglobin redox states. In ex vivo rat and swine liver perfusion models that mimic clinical perfusions, mitochondrial function, as measured by RRS, accurately predicted organ viability [1]. In a swine model of multi-depth burns, the same technology achieved >85% accuracy (AUC) in predicting burn depth, markedly outperforming clinical assessments (50–70%) [2]. These results underscore the translational potential of RRS-based diagnostics for a wide range of complex medical conditions.

In parallel, I will also briefly showcase my doctoral research on a) a heterogeneity-on-a-chip model of cancer [3], and b) a microfluidic platform for the isolation of circulating tumour cells from peripheral blood circulation.

References:
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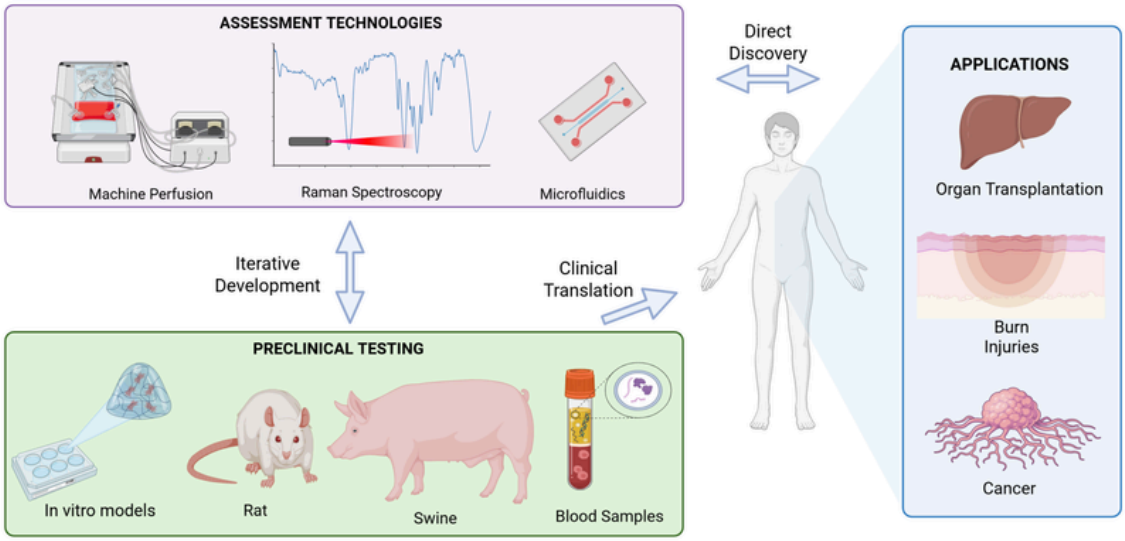

The scope of previous projects is illustrated through an interplay of three key factors: the type of assessment technology used, the level of preclinical validation, and the application areas. The arrows connecting the buckets indicate the stage of research or technology translation.

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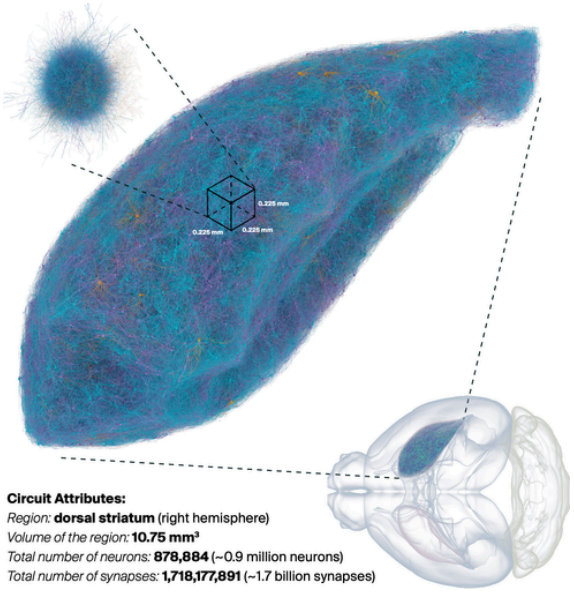
From data to dynamics: Multiscale integration for Atlas-based modelling of mouse striatal networks

Abstract

The striatum is a critical component of the basal ganglia and plays a significant role in controlling motor behaviour and reward processing. Dysfunctions in the striatum have been associated with several neurological disorders, including Parkinson's disease, Huntington's disease, addiction, etc. Dopamine loss is a hallmark of Parkinson's disease and has been implicated in the pathogenesis of striatal dysfunction. However, the precise mechanisms by which dopamine loss leads to dysregulated striatal outputs, dopamine-acetylcholine chemical imbalances, pathological local field potential oscillations, and the interplay between these changes must be fully comprehended. By understanding these mechanisms, we can gain insight into the neural dynamic pathology in the basal ganglia.

To study these phenomena, we developed an atlas-based, anatomically constrained model of the mouse dorsal striatum. The model integrates various layers of data, including cellular, synaptic, electrophysiological, and morphological information. Expanding upon the prior work on the striatal microcircuit, the model integrates data-driven enhancements to the Blue Brain Cell Atlas, specifically addressing cell density and composition. The structural and functional intrastriatal connectivity was constrained and validated using experimental data while populating the dorsal striatum with synthesised morphologies. Through this approach, the study provides a comprehensive computational tool for exploring differences in striatal phenomena between normal and pathological states, including dopamine modulation, rhythmic oscillations, excitation-inhibition balance, and multisensory integration.

The model represents a significant advancement in understanding the nuanced function of the striatum in neural processes. Through iterative validation, it lays the groundwork for an in-depth exploration of striatal phenomena and their interaction with other brain regions, in both normal and pathological conditions. This research contributes to our understanding of neurological disorders associated with striatal dysfunction and provides a framework for future investigations into therapeutic interventions targeting the basal ganglia.



Overview of Atlas-based dorsal striatum circuit

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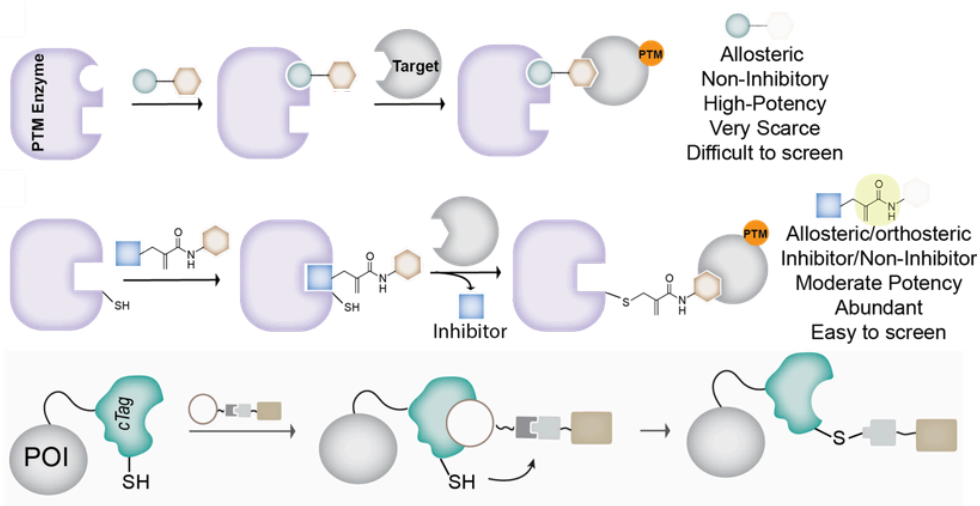
Engineering proximity-mediated protein function: A scalable platform using group-transfer chimeras and ultrasmall chemogenetic tags

Abstract

Small molecules have been classically used to inhibit enzyme function (i.e., loss-of-function). But several new classes of small molecules that endow new functions to enzymes via proximity-mediated effects are emerging. We recently reported Phosphorylation-Inducing Chimeric Small molecules (PHICS) that selectively induce phosphorylation of the protein of interest by bringing the kinase and target protein into proximity. PHICS rewired kinase specificity, leading to the phosphorylation of non-native substrates (i.e., neo-substrates) using first-generation PHICS, which were formed by joining the target binder via a linker to an allosteric, non-inhibitory kinase binder, which is scantily available. Conversely, kinase inhibitors (allosteric/ATP competitive) are abundant and used as drugs for hyperactive/overexpressed kinases in several diseases.

We have developed new group-transfer chimeras for inducing proximity (GRIPs) that leverage readily available kinase inhibitors using cysteine-based group transfer chemistry. Here, the kinase inhibitors equipped with a cleavable linker on GRIPs are strategically positioned proximal to the cysteine or lysine for nucleophilic attack, appending the target protein binder onto the kinase. Subsequently, the non-covalent inhibitor is released from the kinase, allowing for the phosphorylation of the target protein. Additionally, chemogenetic tags were designed to facilitate exploration of the activities of a protein-of-interest (POI) lacking small-molecule ligands; however, most tags are too large for several POIs. Two ultrasmall chemogenetic tags (mgTag and cTag) of 36 and 50 amino acids (aa) respectively, that, to the best of our knowledge, are the smallest. These tags exhibit transferase-like reactivity with their ligands, enabling attachment of any moiety of interest to the tag.

GRIPs and tags enable a programmable, scalable, and selective modulation of protein function across diverse biological systems, with applications in basic research, biomedicine, and biotechnology.



Scalable platforms for proximity-mediated protein engineering

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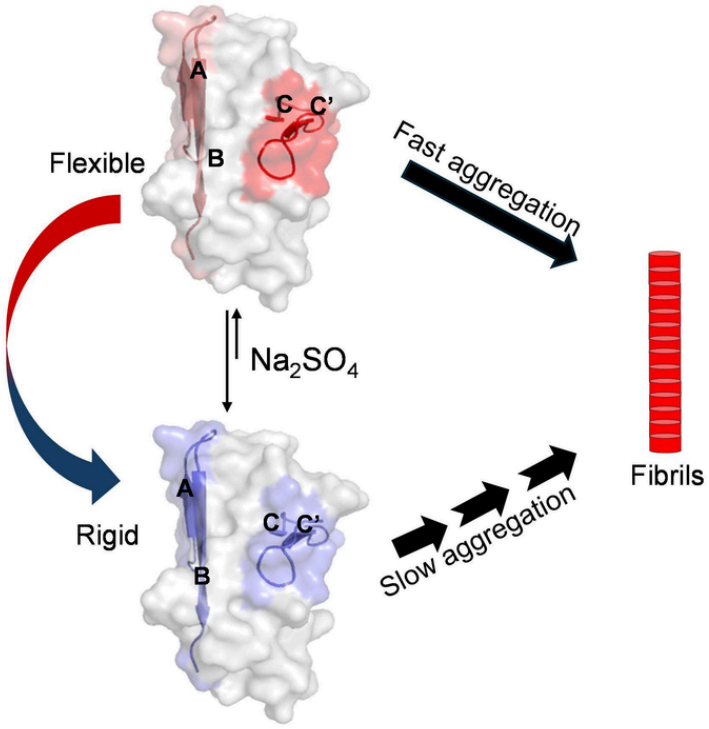
Dimer dissociation and exposure of hot-spot residues synergistically accelerate light-chain variable-domain aggregation associated with AL amyloidosis.

Abstract

Light chain (AL) amyloidosis is a life-threatening systemic disorder caused by the aggregation and deposition of antibody light chain (LC) fragments in multiple organs, including the heart and kidneys.

In this study, we investigated the early events of aggregation of the highly unstable variable domain (VL) from AL55, a known amyloidogenic and cardiotoxic light chain. Our results show that dimer disruption and exposure of aggregation hot spots synergistically accelerate aggregation. At neutral pH, concentration-dependent dimerisation reduces aggregation by limiting aggregation-competent monomers. Dilution or lowering the pH disrupts dimerisation, exposes aggregation-prone regions (APRs), and accelerates aggregation. In contrast, when APRs are chemically stabilised, the aggregation rate decreases despite high monomer availability. Together, this study establishes that AL55 VL domain aggregation is regulated by dimer dissociation, electrostatic modulation, and formation of an aggregation-competent conformation involving a dynamic N-terminal (residues 5-26) and dimeric interface (residues 38-56) regions, ultimately yielding structurally compact and highly stable fibrils.



Schematic showing aggregation of AL55 VL domain

YI 32

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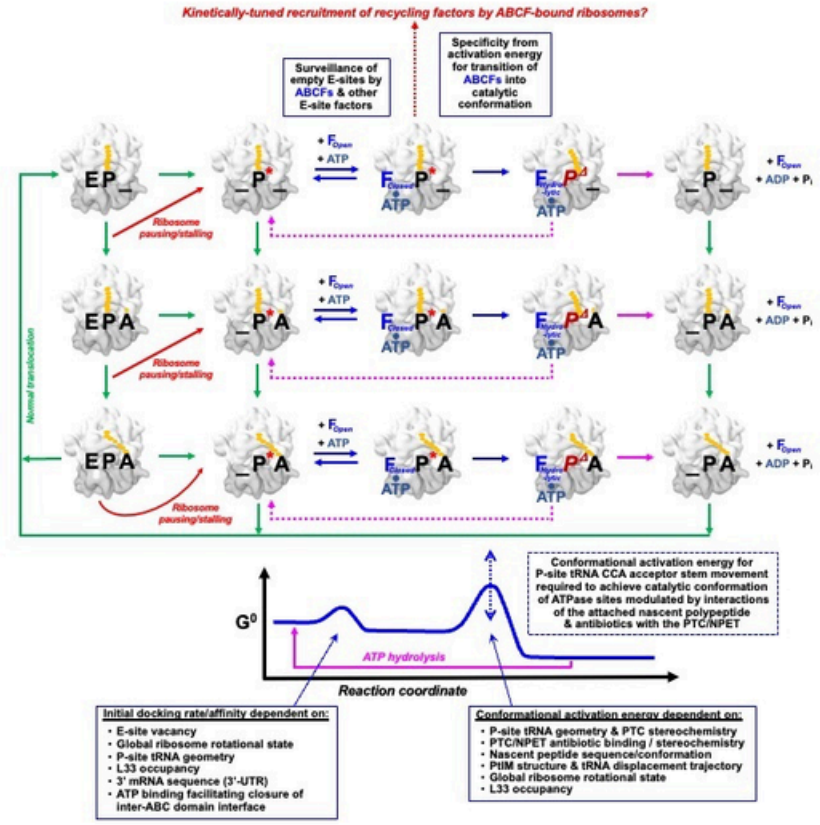
The emergence of ABCFs (ATP Binding Cassette F) as novel translational regulators: their role in cellular homeostasis and antibiotic resistance

Abstract

The ATP-binding cassette superfamily mostly comprises transmembrane receptors, except for ABCE and ABCF. Multiple paralogous ABCF ATPases are encoded in most genomes, but the physiological functions remain unknown for most of them. An *Escherichia coli* ABCF, EttA, that was the first one to be structurally and biochemically characterised, was shown to gate the first step of polypeptide elongation on the ribosome, dependent on the ATP/ADP ratio.

In this study, we compared the four *E. coli* K12 ABCFs – EttA, Uup, YbiT, and YheS using biochemical and structural tools. When we knocked each of the *E. coli* ABCFs individually, the strains with Δ*uup* and Δ*ettA* knocked out exhibited strongly reduced fitness when growth was restarted from long-term stationary phase. But neither ΔybiT nor ΔyheS exhibited this phenotype. *In vitro* studies showed that YbiT and EttA had higher binding affinity for the ribosome than Uup and YheS under our experimental conditions. Several other groups have shown that a subclass of ABCFs known as the Antibiotic Resistance factors (AREs), which are paralogs in other organisms, both regulate and directly mediate resistance to ribosome-targeted antibiotics. We employed single particle cryogenic electron microscopy to decipher the structures of ribosome complexes of all four *E. coli* ABCF paralogs (EttA, Uup, YbiT, and YheS), which, together with previously determined ARE structures, show that ABCFs control the binding geometry of the tRNA in the peptidyl-tRNA-binding (P) site on the ribosome. They modulate the position of its acceptor stem relative to the peptidyl transferase centre (PTC) in a manner that can either promote (in case of EttA and Uup) or disrupt (YbiT, YheS, and the AREs) proper catalytic geometry.



ABCFs bind to the E-site on the ribosome and utilise the energy generated upon ATP hydrolysis to position the P-site tRNA, via its P-site tRNA Interaction Motif, thereby regulating translation elongation.

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Assessment of Sphingosine-1-phosphate pathway in connective tissue disease patients and its association with interstitial lung disease

Abstract

Sphingolipids are major components of the plasma membrane which are acted upon by various enzymes to generate signalling molecules. The metabolites of the sphingolipid pathway have been identified to play a significant role in various cellular processes. Sphingosine-1-phosphate (S1P) is one of the major intermediary molecules of the sphingolipid pathway. S1P is formed by the phosphorylation of sphingosine by sphingosine kinase 1 and 2 (SphK1 and SphK2) and degraded by S1P lyase. Sphingosine-1-phosphate acts via a family of cell surface receptors and is crucial in the control of cell trafficking. The balance between sphingosine-1-phosphate and ceramide is necessary for cell and tissue homeostasis. Both sphingosine-1-phosphate and ceramide have paradoxical actions and the predominance of any one will lead to disease phenotypes. The dysregulation of S1P metabolism has been associated with the pathogenesis of rheumatoid arthritis (RA). However, few studies have explored the plasma levels of S1P and ceramide in RA and SLE patients.

We recruited 16 patients with Systemic Lupus Erythematosus (SLE) for the study, of which 45 patients have Rheumatoid arthritis (RA), and 8 patients have Connective Tissue Disease-associated Interstitial Lung Disease. Among the 8 Connective Tissue Disease-associated Interstitial Lung Disease cases, 5 patients have Anti-Synthetase Syndrome (ASS)-ILD, 1 patient has Mixed connective tissue disease (MCTD)-ILD, and 2 patients have Rheumatoid arthritis-ILD. The ELISA for Sphingosine-1-phosphate has been standardised. Sphingosine-1-phosphate was estimated in 45 patients.

The Sphingosine-1-phosphate concentration was higher in CTD-ILD than in CTD. Similarly, Sphingosine-1-phosphate concentration was observed to be higher in RA when compared with SLE. However, neither result is statistically significant. Comparing with controls will yield further insights into the current data.

Table 1: Sphingosine-1-phosphate concentration in CTD patients with and without ILD

Group	N	p50	p25	p75	P value
CTD	39	37.335	24.963	51.521	0.6366
CTD-ILD	6	41.708	32.31	54.478	

Table 2: Sphingosine-1-phosphate concentration in patients with various CTD

CTD	N	p50	p25	p75	P value
RA	35	38.19	26.338	51.521	0.5166
SLE	4	26.794	22.3635	74.5765	

Sphingosine-1-phosphate concentration in CTD patients

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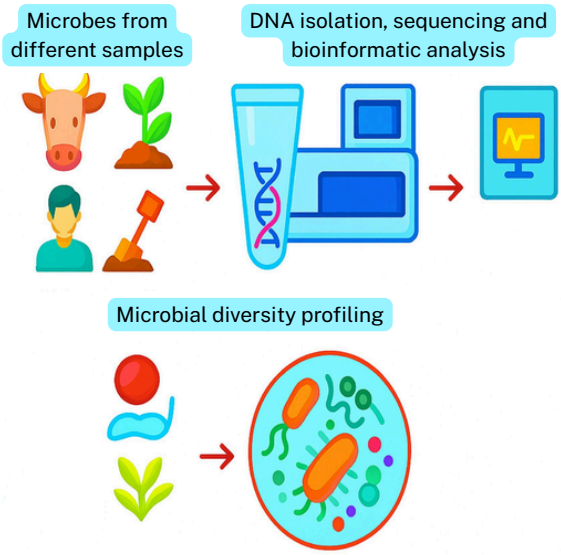
Microbiomes in various environments: A unified one health perspective on animals, humans, and environment

Abstract

Microbiomes, dynamic communities of microorganisms residing in various environmental niches and in animals and humans, are crucial for maintaining ecological balance and promoting host health. Environmental microbiomes facilitate nutrient cycling, enhance soil fertility, promote the degradation of pollutants, and contribute to ecosystem resilience. However, disturbances from climate change, land-use alterations, and pollution can disrupt these microbial networks, adversely affecting ecosystem functioning. In animals, host-associated microbiota play a significant role in regulating digestion, immunity, growth, behaviour, and disease susceptibility, thereby greatly influencing livestock productivity, wildlife conservation, and veterinary health. Similarly, in humans, the gut, skin, and oral microbiomes are pivotal in maintaining metabolic homeostasis, facilitating neuroimmune interactions, and determining susceptibility to both chronic and infectious diseases.

Growing evidence highlights the strong interconnections among environmental, animal, and human microbiomes, underscoring the necessity for a One Health approach to comprehend cross-domain microbial influences. To investigate these relationships, we employed an integrated methodology that involved collecting soil, water, animal, and human microbiome samples using standardised aseptic protocols. Following this, we performed DNA extraction tailored to diverse sample types. We utilised amplicon sequencing (16S rRNA/ITS) and whole metagenome shotgun sequencing to characterise microbial diversity and functional capacities. Bioinformatic analyses using open-source tools and network-based approaches enabled taxonomic profiling, diversity assessment, functional annotation, and identification of shared microbial signatures across domains. Preliminary analysis revealed higher abundances of key genera, including *Pseudomonas*, *Bacillus*, *Lactobacillus*, and *Bacteroides*, across multiple sample types, suggesting potential microbial transmission routes and shared ecological drivers.

We expect to expand this analysis using machine learning–based integrative models to identify predictive microbial signatures associated with environmental conditions, animal health indicators, and human physiological states. These findings demonstrate the interconnected nature of microbiomes across ecosystem and host boundaries and highlight the potential for microbiome-informed strategies to promote environmental sustainability, enhance animal welfare, and support human health within a unified One Health framework.



Metagenomic workflow

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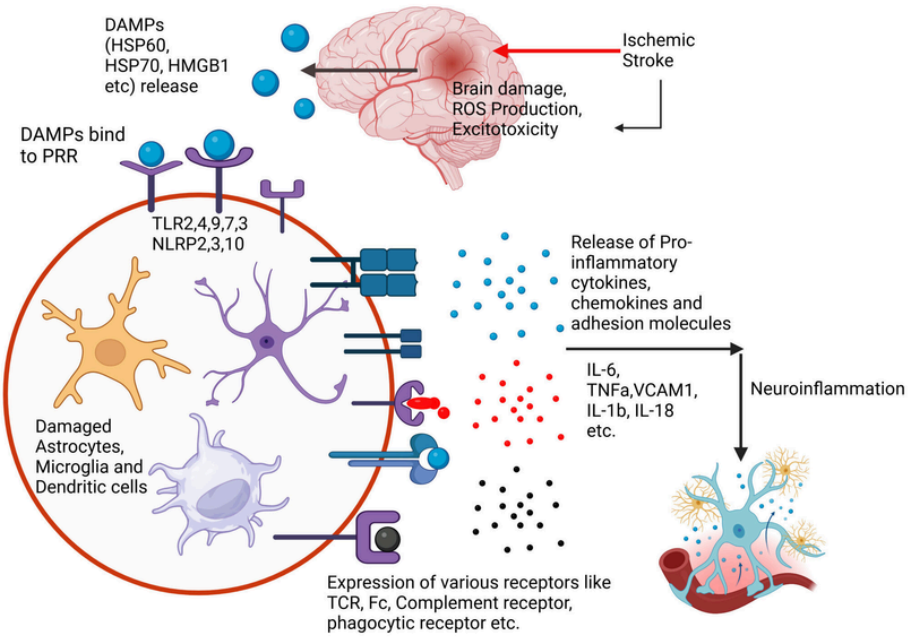
Role of immune receptors and their epigenetic regulation in ischemic stroke in a sex-specific manner by membrane proteomics

Abstract

Ischemic stroke is caused by the disruption of blood supply to the brain due to cerebral arterial embolism and is the second leading cause of death globally. Utilising membrane proteomics may help identify key immune receptors at a given time. Epigenetic studies, including novel prenylation mechanisms of potential proteins in the membrane proteome, may uncover our understanding at the genetic level. Moreover, crucial proteins involved in hypoxia may be regulated differently in males and females, as their physiological conditions differ.

We aim to profile the gender-specific membrane proteome to unravel the role of immune functionality in ischemic stroke in animal models. Hypoxia will be induced in male and female mice and zebrafish. Brain sections will be removed, membrane proteins will be extracted, samples will be purified to remove detergents, trypsin-digested, and analysed by LC-MS/Proteomics software. We also reanalysed our existing zebrafish and mouse proteomic data in our lab to identify significant immune proteins that play an important role in stroke.

We have standardised our membrane protein isolation protocol for hippocampus from the mouse brain and whole brains from zebrafish. For membrane protein isolation, we used 4% CHAPS for solubilisation. Plasma membrane markers such as occludin and conventional housekeeping proteins were used as cytosolic markers to confirm the purity of our isolated membrane fraction. Upon re-analysis of the whole proteome of zebrafish, most of the proteins are upregulated in females as compared to males. In the male mouse proteome, 153 out of 173 proteins were upregulated. Moreover, only 44 proteins were similar in both animal models of ischemic stroke. Interestingly, MHC Class–1 was downregulated in both. The new leads obtained, upon further confirmation, may help to understand the immune and novel epigenetic mechanisms in ischemic stroke, leading to the development of new therapeutic targets.



Role of immune receptors in ischemic stroke

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Histone H3 lysine K4 tri-methylation regulation of iron deficiency response in rice

Abstract

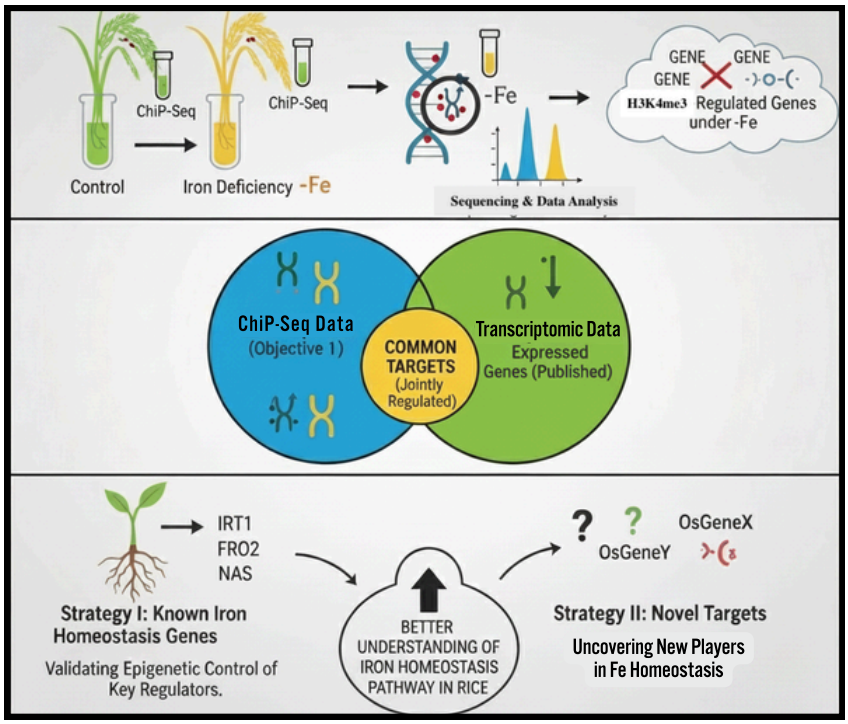
Iron (Fe) deficiency is a major constraint on rice productivity and grain quality, especially in regions with calcareous or alkaline soils. While Fe uptake and transport pathways have been extensively studied, the epigenetic regulation of these responses remains largely unexplored in crops.

My research program focuses on deciphering how Histone H3 Lysine-4 trimethylation (H3K4me3), a chromatin modification that promotes gene activation, regulates Fe-deficiency response pathways in rice. We hypothesise that H3K4me3 fine-tunes the expression of Fe-responsive genes, thereby shaping the plant's adaptive strategies under low-iron conditions.

As part of my ongoing DST-SERB Start-up Research Grant (2024–2026), we will employ Chromatin Immunoprecipitation Sequencing (ChIP-Seq) to generate genome-wide profiles of H3K4me3 enrichment in rice roots and shoots under Fe-deficient and Fe-sufficient conditions. These datasets will be integrated with publicly available transcriptome data from Fe-deficient rice to identify candidate genes jointly regulated by H3K4me3 and Fe availability.

Although the sequencing experiments are planned and not yet completed, this approach will enable the discovery of key regulatory targets linking epigenetic control to canonical iron-acquisition strategies (Strategies I and II). Downstream functional analyses will elucidate their roles in Fe uptake, transport, and homeostasis.

Unravelling these mechanisms is expected to provide novel molecular targets for breeding or engineering Fe-efficient, nutrient-dense, and climate-resilient rice varieties, thereby addressing critical challenges in crop nutrition and food security.



H3K4me3 mediated control of iron homeostasis in rice

YI 37

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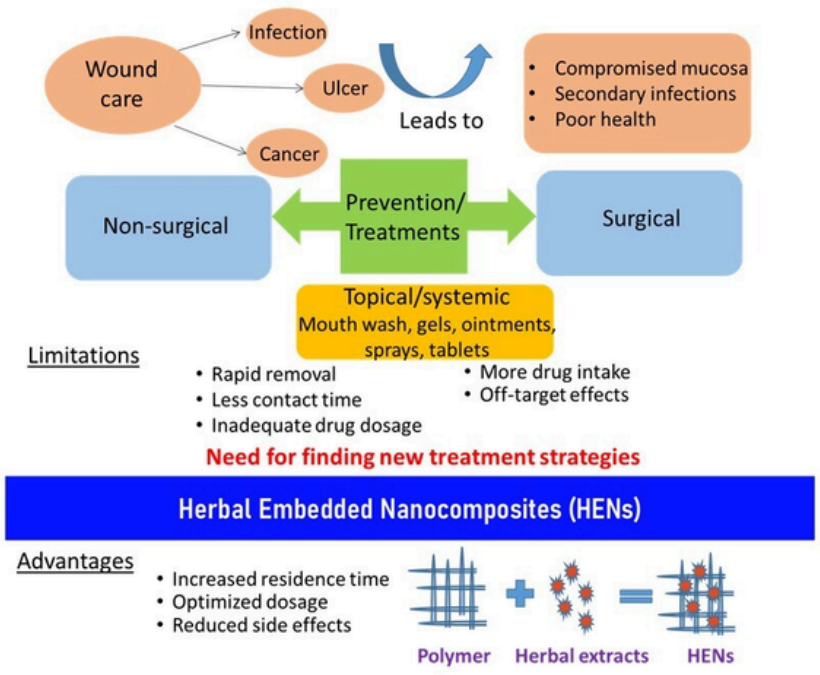
Herbal-Embedded Nanocomposites: Characterisation and efficacy-assessment

Abstract

Traditional herbs, while effective, face hurdles such as variable year-round availability and poor patient compliance due to the need for repeated, real-time preparation. This study addresses these challenges by developing Herbal-Embedded Nanocomposites (HENs) based on the natural polymers chitosan and alginate, leveraging the synergistic potential of nanotechnology and herbal bioactives. Biomaterials play a pivotal role in facilitating wound healing by providing a conducive surface for cell attachment, ensuring sustained release of bioactives, and maintaining a favourable microenvironment. Prior studies have successfully synthesised composites using natural polymers such as gelatin, chitosan, and cellulose in combination with aloe vera extracts. Li and their group synthesised a Sodium Alginate/Carrageenan/Cellulose composite hydrogel by incorporating extracts of *Amaranthus spinosus* and *Rubia cordifolia*, and studied its wound-healing properties. Additionally, synthetic polymers like polyethylene oxide, polyacrylonitrile, and poly(lactic-co-glycolic acid) have been investigated for their compatibility with herbal extracts, showcasing the potential of such interdisciplinary approaches for overcoming challenges in herbal wound care.

Our approach utilises a sustainable, eco-friendly method inspired by the biosynthesis of silver nanoparticles (AgNPs) using *Moringa oleifera* leaf extract, which exhibits potent inhibitory effects against common pathogens such as *Escherichia coli* and *Staphylococcus aureus*. The nanoparticles were characterised by UV visible spectroscopy, FTIR, TEM and found to be spherical in shape. Various parameters, including temperature, salt concentration, and leaf extract, were optimised. This principle of integrating potent herbal bioactives and nanoparticles is central to the HENs strategy. The successful assessment of herbal embedded nanocomposites could be a significant step toward developing alternative wound care products. They could offer a non-antibiotic approach, potentially reducing the risk of resistance development.



HEN-based wound-care solutions

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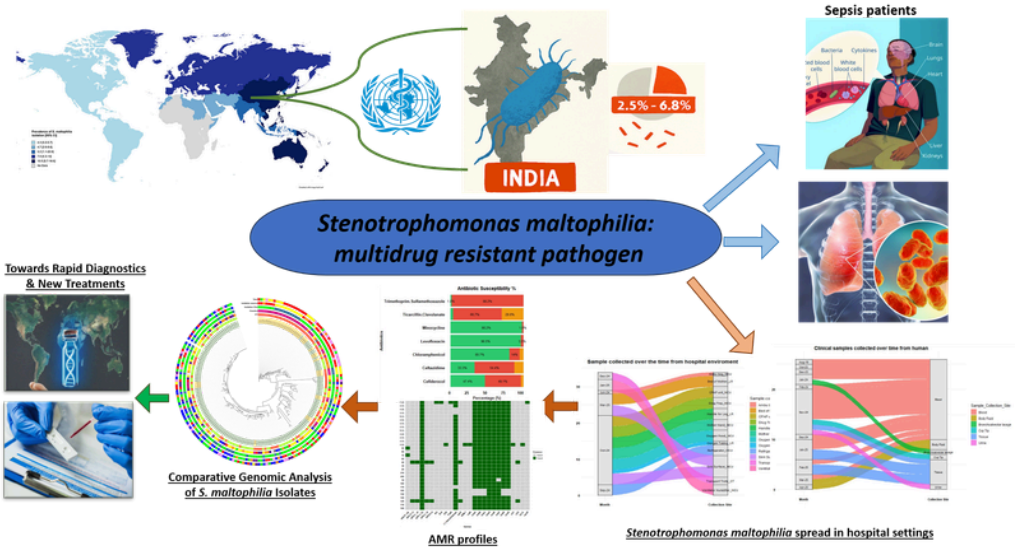
Uncovering the genomic landscape of Stenotrophomonas maltophilia to understand its drug resistance, invasive potential, and host adaptation

Abstract

Healthcare systems worldwide serve as reservoirs and transmission hubs for multidrug-resistant (MDR) pathogens, which pose a significant threat through nosocomial infections. Among these, *Stenotrophomonas maltophilia*, the fourth most prevalent non-fermenting Gram-negative bacillus (NFGNB) in sepsis patients, has emerged as a critical concern. Known for causing pneumonia and bloodstream infections, this opportunistic pathogen is notoriously difficult to treat with conventional antibiotics due to its intrinsic and acquired resistance mechanisms.

Our objectives were (1) Isolation and cultivation of *S. maltophilia* from clinical and non-clinical sources from Indian hospitals, and (2) Comprehensive genomic analysis of *S. maltophilia* to identify genes responsible for invasiveness, inflammation and drug resistance to develop new strategies to combat this pathogen. Hospital samples were collected from different Indian hospitals. *S. maltophilia* strains were isolated on Stenotrophomonas selective agar and confirmed via 16S rRNA sequencing. AST was tested using the broth microdilution method. Whole genome sequencing of the genomic DNA of respective bacterial strains was done using the Illumina HiSeq, followed by genome assembly, gene prediction, and annotation using bioinformatics tools. A meta-analysis indicated that *S. maltophilia* exhibits high resistance to trimethoprim-sulfamethoxazole, ceftazidime, and cefiderocol. WGS data analysis revealed that *S. maltophilia* strains also possess various AMR genes and virulence factors.

This study on the genomic basis of *S. maltophilia* invasiveness, inflammation, and drug resistance provides valuable insights into the mechanisms by which these pathogens colonise different tissues and their potential sources of inflammation. Notably, some genetic signatures were found to be similar in *S. maltophilia* strains isolated from human clinical samples and hospital biospecimens. This is one of the first studies from an Indian perspective examining the genetic basis of AMR and the virulence of the opportunistic bacterium *S. maltophilia* in hospital settings.



Uncovering the genomic landscape of Stenotrophomonas maltophilia to understand its drug resistance, invasive potential, and host adaptation

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LncRNA: An untapped source of micro peptides in flowering plants

Abstract

Long non-coding RNAs (lncRNAs) typically lack a conventional annotated open reading frame (ORF), and thus have long been regarded as non-coding regulatory molecules. Emerging evidence indicates that some lncRNAs harbour small ORFs (sORFs) that encode micropeptides with diverse biological functions [1]. Despite these advances, the prevalence of translation from lncRNAs in plants and the physiological significance of the resulting peptides remain poorly understood.

Our earlier work demonstrated that plant lncRNA expression corresponds to the diel light cycle, implicating them in circadian regulation [2]. We also showed that lncRNAs possess conserved genomic and epigenomic characteristics with potential roles in growth and development [3]. Building on this foundation, the present study applied multiple computational pipelines to systematically identify sORFs within lncRNAs and evaluate their potential to encode micropeptides in flowering plants (Unpublished). Sequence similarity–based clustering combined with phylogenetic analyses revealed that sORFs across different species within the same family exhibit strong conservation. In contrast, those from more distantly related taxa show greater divergence (Unpublished). These findings expand the functional repertoire of lncRNAs beyond their established regulatory roles, underscoring their dual capacity as modulators of gene expression and potential sources of bioactive peptides.

Our study provides a conceptual advance that may open new avenues for exploring plant adaptation, development, and evolutionary innovation.

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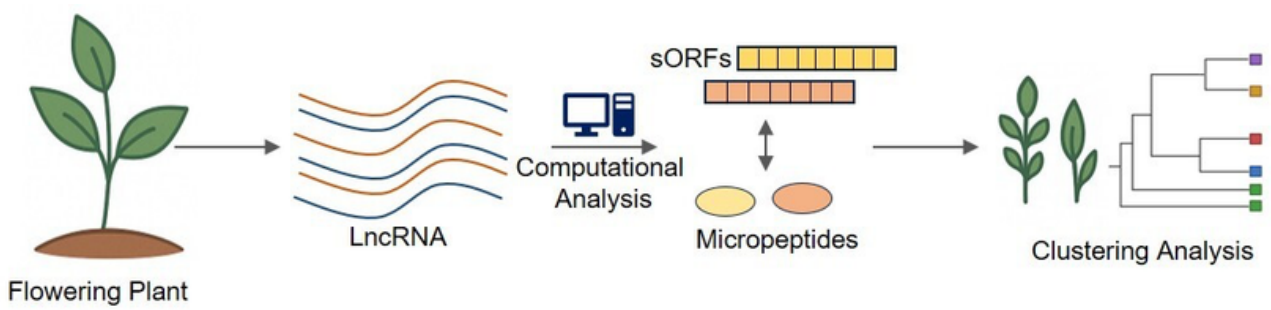

From lncRNAs to conserved micropeptides

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Molecular profiling using nanopore technology: single-cell and single-molecule discrimination for next-generation biosensing

Abstract

Nanopore technology provides an unprecedented window into the nanoscale world of biomolecules and cells, enabling real-time, label-free analysis of their structural and dynamic properties. We utilise solid-state nanopores to develop innovative methods for molecular profiling and single-cell discrimination, integrating physics, nanofabrication, and bioengineering to advance diagnostic science.

We engineered pressure-biased, constricted nanopores capable of differentiating single biomolecules and even entire cells based on size and charge. These systems exploit controlled ionic flow and pressure gradients to achieve recapture-based identification, allowing discrimination between cells. Building on these insights, we developed single-cell mass discrimination technology and a single-molecule recapture method to quantitatively profile cells and biomolecules at sub-nanometer resolution.

References:
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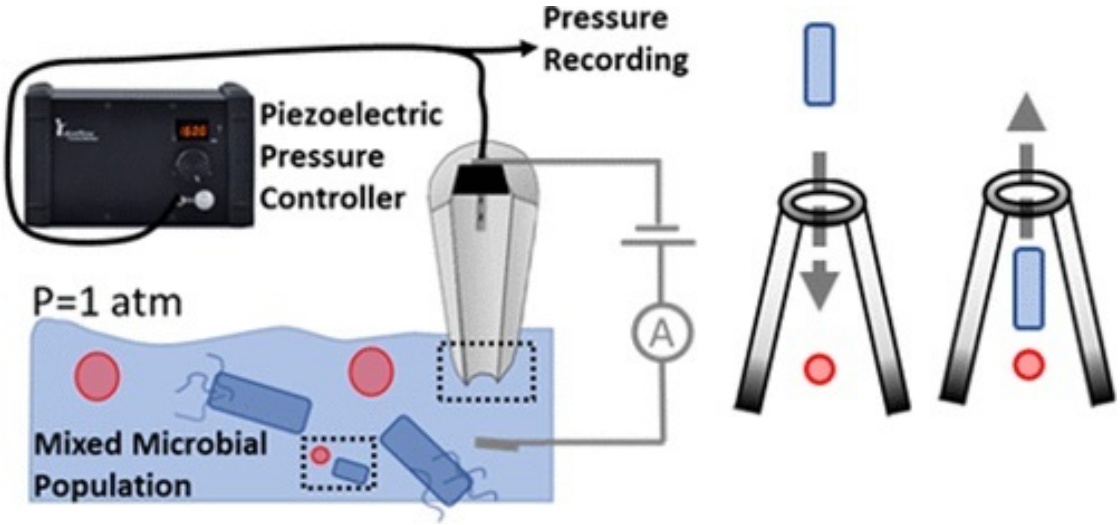

Detection through analyte transit in a narrow aperture is a versatile and robust approach, widely applied from DNA gating to single-cell analysis.

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Ajay Nair

PDF 01

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scRegacti identifies core regulators of liver librosis, a pre-tumour microenvironment for liver cancer

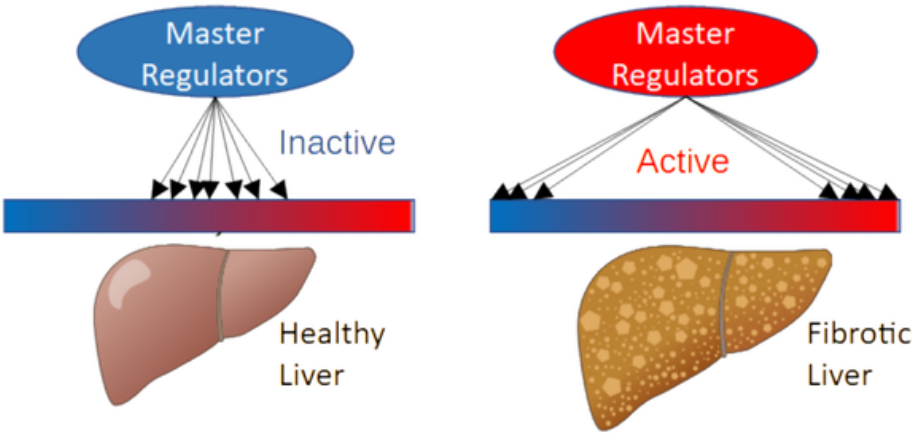
Abstract

Liver fibrosis affects 3.6-13% of the global population and shows an increasing trend. Liver fibrosis is the excessive scarring of liver during chronic injury. Fibrosis usually progresses to cirrhosis, loss of liver function, and tumour development. Hepatic stellate cells (HepSC) are the main source of fibroblasts in the liver and the primary contributor of liver scarring. The key regulators of HepSC activation are still unknown and there are no currently approved drugs for liver fibrosis. Therefore, identifying the master-regulators of HepSC activation can help to understand the biology of liver fibrosis and identify effective drugs.

We generated large single-nucleus/single-cell RNA-sequencing (sn/scRNA-seq) datasets of mouse and human liver fibrosis. We also developed a single-cell regulator activity inference pipeline (scRegacti) to predict master-regulators of disease states from sn/scRNA-seq data. ARACNe and VIPER are well-known tools for regulatory-network and master-regulator inference from bulk RNA-seq data. Here, we optimised both the tools to work with sn/scRNA-seq data. Specifically, we developed procedures to de-noise and filter the sn/scRNA-seq data using statistical and biological principles to remove noise and drop-outs issues. This pipeline integrates easily with the popular single-cell analysis package Seurat.

scRegacti was used to infer master-regulators from both human and mouse datasets. Knockdown of these regulators showed reduction in HepSC activation or fibrosis. Further, drug screening experiments identified drugs that reduce fibrosis and drugs that revert an activated HepSC to a quiescent state.

In summary, liver fibrosis has high prevalence but no approved drugs. Activated HepSC are the primary cause of liver fibrosis. Using large sn/scRNA-seq datasets and scRegacti we inferred core regulators of HepSC activation. We also identified drugs that reduce or revert HepSC activation, a central event in the development of liver fibrosis.



Identifying the master regulators of liver fibrosis

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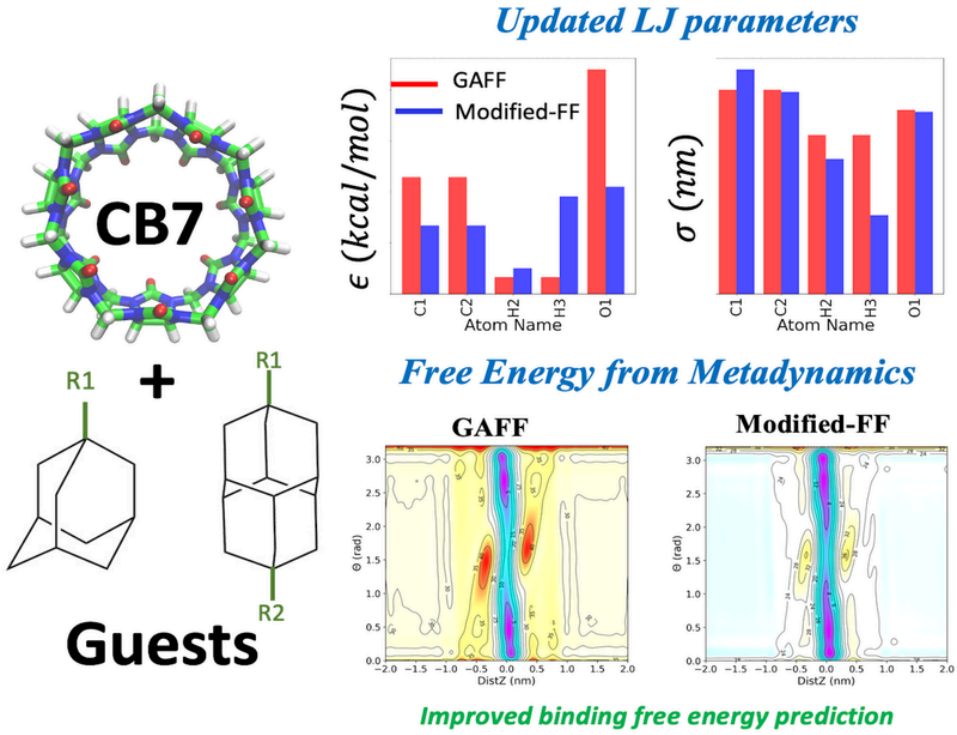
From computation to application: Understanding and engineering metabolite synthetic receptor–analyte complexes for biomedical innovation

Abstract

High-affinity host–guest systems such as Cucurbit[n]uril (CBn) macrocycles are central to diverse applications, including targeted drug delivery, self-healing materials, biosensing, and molecular diagnostics, owing to their remarkable molecular recognition capabilities. Molecular simulations (MS) can, in principle, predict binding poses and affinities of ligands with these macrocycles and thereby guide the design of host–guest systems. However, the limited accuracy of conventional force fields (FFs) for synthetic receptors has hindered their widespread use.

Here, we show that incorporating electron-density–derived Lennard-Jones parameters and charges into FFs significantly improves the accuracy of free-energy calculations. As a case study, we examine five adamantane derivatives and two diamantane derivatives binding to the CB7 macrocycle. Our multiple-walker, well-tempered funnel metadynamics simulations yield binding free energies in reasonable agreement with experiment for all adamantane ligands. For the larger diamantane ligands, however, discrepancies remain, indicating the need for further refinement.



Representation of workflow

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PDF 03

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Nanoengineered MXene-DNA hybrid hydrogel for precise detection of coagulation biomarker thrombin

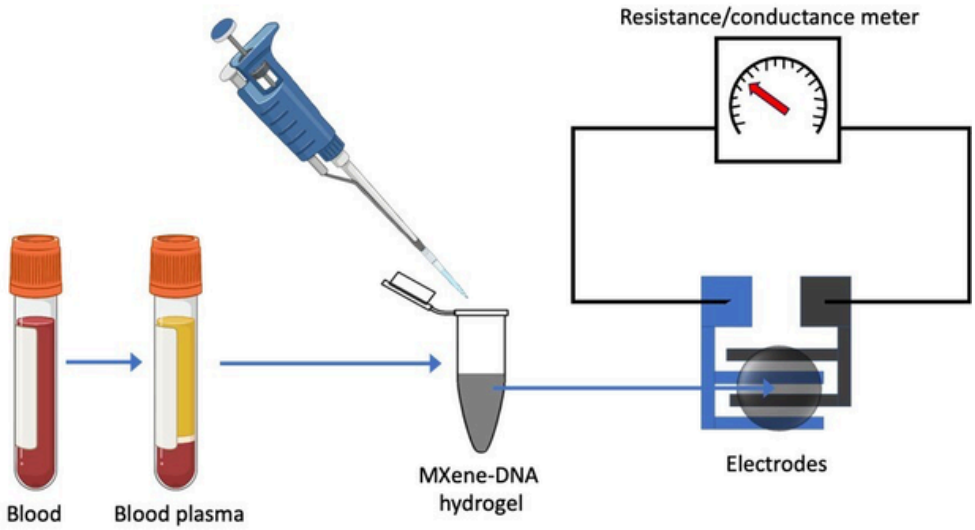
Abstract

Two-dimensional (2D) MXenes and DNA-based hydrogels have emerged as promising materials for advanced biosensing due to their complementary properties and tunability. While MXenes exhibit high surface area, electrical conductivity, and chemical versatility, DNA offers molecular recognition, biocompatibility, and dynamic responsiveness. The integration of these materials creates a multifunctional platform suitable for precise biochemical detection.

In this study, a novel 2D MXene-DNA hybrid hydrogel was developed for the sensitive detection of thrombin, a key serine protease involved in the blood coagulation cascade. Thrombin catalyses the conversion of fibrinogen into fibrin, and its abnormal levels are associated with coagulation-related disorders, such as haemophilia and Von Willebrand disease. The hydrogel system was constructed using delaminated MXene sheets functionalized with thiol-modified thrombin-binding aptamers (TBA) that serve as molecular crosslinkers. The TBA strands hybridised with their complementary oligonucleotides, forming a compact, conductive hydrogel network. Upon exposure to thrombin, the aptamer selectively binds to the enzyme, releasing its complementary strands and partially disassembling the hydrogel matrix. This structural change results in a measurable variation in electrical resistance, providing a direct signal for thrombin detection. The developed biosensor exhibited outstanding analytical performance. The system demonstrated high reproducibility, with a relative standard deviation of 8-10% in artificial sample analyses.

This innovative approach provides a versatile, robust sensing platform, enabling the design of customisable biosensors for diverse biomedical targets via aptamer substitution. The MXene-DNA hybrid hydrogel concept holds strong potential for integration into future smart and adaptive diagnostic devices [1].

Reference:
Morya, V., Bhatia, D., Ghoroi, C. and Yadav, A.K. (2025). A functional 2D MXene–DNA hybrid hydrogel for portable detection of blood disorder biomarker thrombin in human plasma. Journal of Materials Chemistry B, 13(23), pp.6742-6754.



Depiction of the proposed thrombin detection set-up with MXene-DNA hybrid hydrogel

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PDF 04

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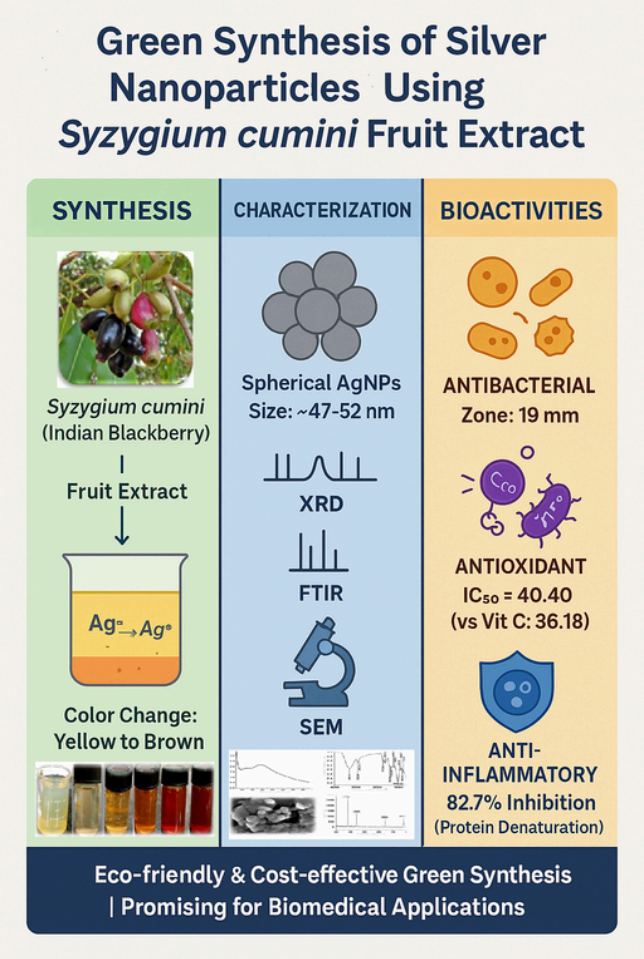
Harnessing Syzygium cumini for green synthesis of silver nanoparticles with biomedical potential

Abstract

The biosynthesis of silver nanoparticles has attracted significant attention in nanotechnology due to their antimicrobial and biomedical applications. In the current research scenario, the green synthesis of metal nanoparticles is expected to be a cost-effective and environmentally benign alternative.

In this effort, fruit extracts from *Syzygium cumini* were used as the reducing agent to synthesise silver nanoparticles (AgNPs), and the antioxidant, antibacterial, and anti-inflammatory properties of the synthesised nanoparticles were evaluated. The synthesised Ag nanoparticles were characterised by UV-Visible spectroscopy (UV), Fourier transform infrared spectroscopy (FTIR), Powder X-ray Diffraction (XRD), and scanning electron microscopy (SEM). The formation of silver nanoparticles was confirmed by optical performance (i.e., colour change) using UV-Visible spectroscopy, which showed a characteristic peak at 443 nm. The presence of OH and C=O groups in the plant secondary metabolites of *S. cumini* was confirmed by FTIR spectroscopy, which is liable for capping and reducing the surface of metal nanoparticles. Powder XRD analysis was used to analyse the crystal structure. This medicinal plant extract-mediated green biosynthesised silver nanoparticles exhibited good *in vitro* antioxidant, antibacterial, and anti-inflammatory activities.

Therefore, results of the present study highlights the synthesis of cost-effective, eco-friendly drugs with less side effects, which showed that there is the need of *in vivo* studies at a molecular level to develop silver nanoparticles through greener approach using the medicinal plant (*S. cumin*) extract as antibacterial, antioxidant, and anti-inflammatory agent for the treatment of bacterial infection, inflammation and many free radical oriented diseases.



Green-synthesised silver nanoparticles using S. cumini fruit extract exhibit potent antibacterial, antioxidant, and anti-inflammatory activities

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PDF 05

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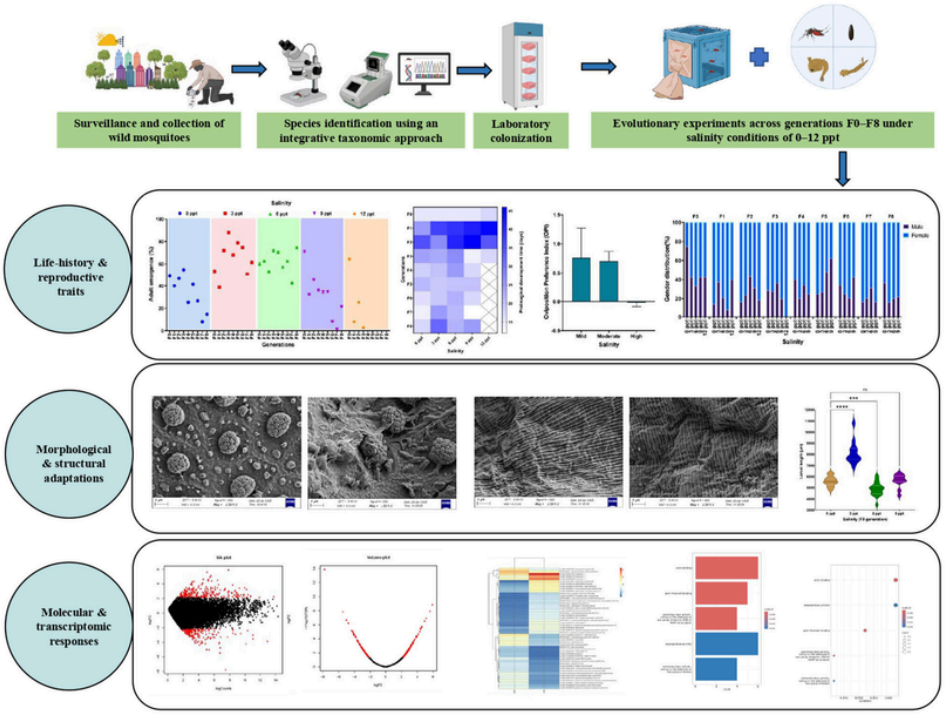
Evolving in saltwater: Adaptive responses of the dengue mosquito to saline conditions

Abstract

As sea levels rise and coastlines reshape, a new mosquito threat is quietly emerging. The dengue mosquito, *Aedes aegypti*, is adapting to survive in saltwater. Once confined to freshwater habitats, this vector of dengue, chikungunya, Zika, and yellow fever now thrives in brackish environments formed by seawater intrusion and coastal salinisation. To understand this adaptive shift, we conducted a multigenerational experiment (F0–F8), rearing *Ae. aegypt*i under increasing salinity levels from 0 to 12 ppt to mimic changing brackish conditions. Across generations, we assessed adult emergence, development time, oviposition behaviour, gender distribution, and morphological changes, supported by transcriptomic analyses to uncover adaptive mechanisms of tolerance.

The initial generation (F0) displayed successful adult emergence even at 12 ppt. However, over successive generations, emergence rates at high salinity declined sharply, indicating physiological stress and reduced fitness. Notably, the 12 ppt culture line was lost after the fourth generation due to complete female-biased emergence, preventing further breeding. In contrast, populations reared at moderate salinities (3–6 ppt) remained stable through F8 generation. They exhibited faster development, consistent survival, and a strong oviposition preference for their natal salinity, suggesting environmental imprinting and local adaptation. Microscopic and molecular analyses revealed salinity-induced remodeling of egg and cuticle structures, accompanied by shifts in gene expression from stress and detoxification pathways toward metabolic and structural adjustments.

These findings demonstrate that *Ae. aegypti* has a broad but finite capacity to adapt to saline environments. As water bodies in coastal areas become increasingly brackish due to seawater intrusion, these resilient mosquitoes may extend their range into the new habitats. Such expansion challenges freshwater-focused control strategies and reshapes the future landscape of vector-borne disease risk.



Adaptive responses of the dengue mosquito to salinity stress across generations: changes in development, morphology, and gene expression

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PDF 06

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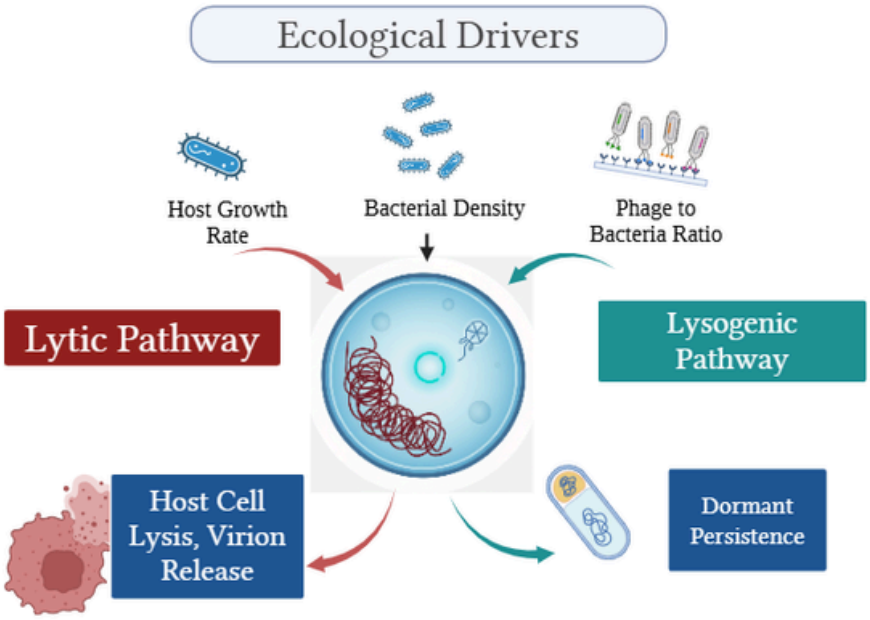
Ecological drivers of lysis-lysogeny decision in temperate bacteriophages

Abstract

The life cycle of temperate bacteriophages follows two distinct modes of reproduction. The lytic pathway involves immediate, rapid replication of the phage genome, leading directly to the destruction (lysis) of the host cell and the release of new virions. In contrast, the lysogenic pathway forms a stable association, allowing the host to remain viable. This lysogenic state serves a vital evolutionary purpose, ensuring the long-term persistence and genetic preservation of the phage until environmental conditions are optimal for activation. This strategic dual lifestyle provides phage with an optimal balance between short-term reproductive success and durable survival. Understanding the mechanisms that drive this critical decision remains a core question in the study of phages.

While genetic regulation governs the lysis–lysogeny switch, its outcome is tightly coupled to ecological cues that modulate the phage's regulatory network. Factors such as host growth dynamics, bacterial population density, and the number of infecting phages significantly influence the balance between lytic and lysogenic outcomes. By mapping phages' decision-making onto host physiology, we gain insights not only into the mechanism of phage persistence but also into their fundamental ecological roles within complex microbial communities.

References:
1. Knowles et al. Lytic to temperate switching of viral communities. Nature 7595:466-70.
2. Silveira CB, Luque A, Rohwer F. The landscape of lysogeny across microbial community density, diversity and energetics. Environmental microbiology 8:4098-111.



Ecological drivers influencing viral infection pathways

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Revisiting DNA replication-mutagenesis link: Implications for antimicrobial resistance

Abstract

The steadily rising burden of antibiotic-resistant bacterial infections poses a major global health challenge. While bacteria can acquire resistance through multiple mechanisms, mutagenesis remains a central driver of this phenomenon. Targeting mutagenesis, therefore, presents a promising approach to combat antimicrobial resistance. Since the DNA polymerases responsible for both spontaneous and stress-induced mutations are functionally coupled to the replication machinery, mutagenesis has conventionally been associated with active replication. However, in many natural and clinical settings, bacteria occupy niches where active replication is infrequent, raising the question of how mutagenesis is regulated under such conditions. By employing specific experimental designs, novel DNA-damaging agents and a model bacterium suitable for visualising DNA replication and repair via live-cell microscopy, we explored mutagenesis in previously uncharacterised contexts. We observed the conventional link between replication and mutagenesis break down in a context-specific manner. These findings underscore the importance of systematically investigating the regulation of mutagenesis and the development of antimicrobial resistance under varied contexts of stress and bacterial physiology.

It opens up possibilities for the design of improved therapeutic and diagnostic frameworks to combat antimicrobial resistance.

References:
1. Joseph AM, Badrinarayanan A., FEMS Microbiol. Rev., 2020
2. Joseph AM, Daw S, Sadhir I, Badrinarayanan A, eLife., 2021
3. Joseph AM et al., RSC. Med. Chem., 2022
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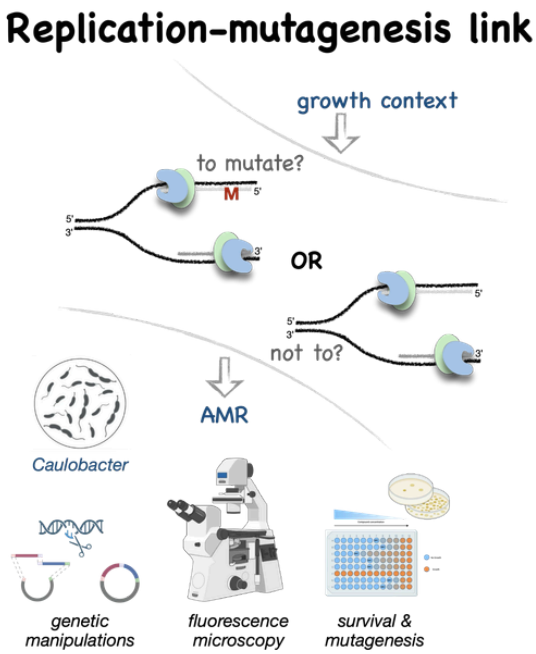

Replication-mutagenesis link

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PDF 08

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Anthropogenic structures reshape extended phenotypes to alter signal physics, collective behaviour, and fitness

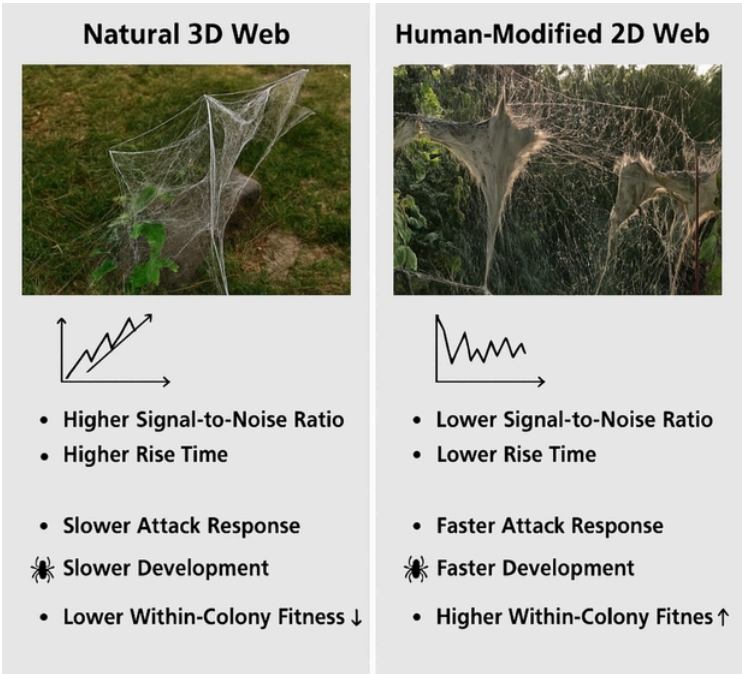
Abstract

Human activities are reshaping not only habitats but also the animal-built structures that mediate survival and reproduction. Such extended phenotypes, from nests to webs, are integral to how organisms sense and interact with their environment, yet the fitness consequences of anthropogenically induced architectural shifts remain largely unknown.

We studied the Indian social spider (*Stegodyphus sarasinorum*), which increasingly constructs two-dimensional (2D) capture webs on human-made structures instead of its ancestral three-dimensional (3D) webs. Across replicated field colonies, we show that this shift alters the physics of vibration transmission, with cascading effects on collective behaviour and reproductive success. Using a custom, field-deployable vibration analysis system, we found that 2D webs transmit prey-generated vibrations with shorter rise times, enabling faster collective attack responses, whereas 3D webs preserve higher signal-to-noise ratios, enhancing detection fidelity.

A mechanistic computational model, parameterised with field data, revealed that the 3D disadvantage emerges from the synergy between greater web tension and noise isolation. The rapid, collective response of spiders in 2D webs translated into faster development, higher within-colony reproduction, and greater prevalence in anthropogenically modified landscapes.

Our results link environmental change to physical signal transmission, collective decision-making, and fitness, providing a general framework for understanding how altered extended phenotypes mediate adaptation in a human-dominated world.



Web architecture shapes fitness trade-offs in human modified habitats

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PDF 09

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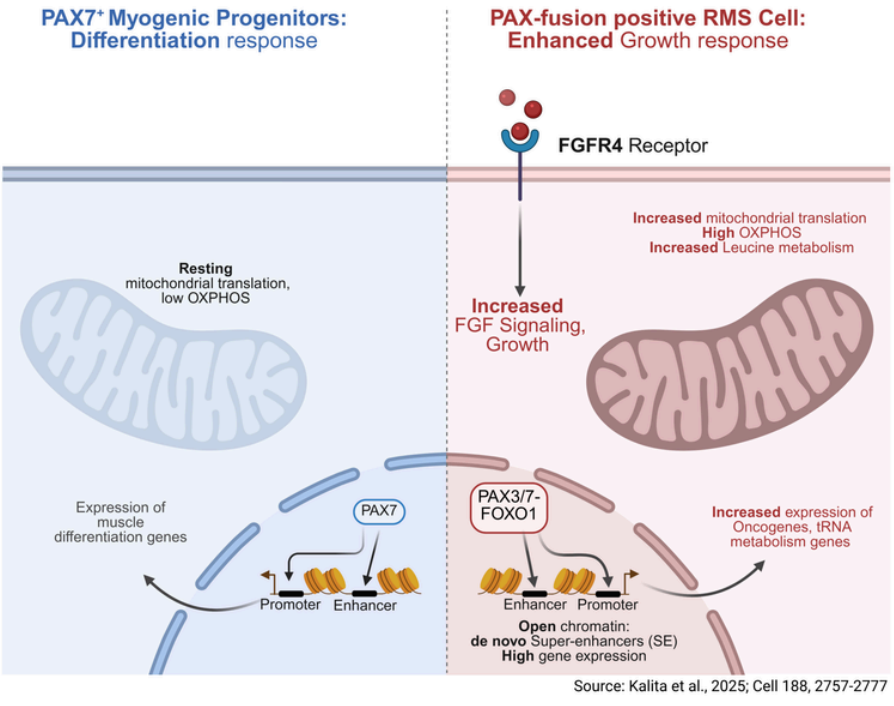
Exploring tumour-initiation events in high-risk Rhabdomyosarcoma uncovers therapeutic vulnerabilities

Abstract

Alveolar rhabdomyosarcoma (ARMS) is a high-risk cancer and among the most aggressive pediatric soft tissue sarcomas, with a poor survival outcome as the disease advances. Overall, pediatric patients with ARMS harbouring chromosomal translocation t (2;13) or t (1;13) that result in a PAX3:Fkhr or PAX7:Fkhr fusion transcription factors, exhibit a greater incidence of tumour relapse, metastasis, and poor survival outcome. This underscores the urgent need to develop effective therapies to treat this subtype of childhood cancer.

Using genomic engineering in human iPSC cells, I have developed a novel and controlled experimental model system, i.e., muscle progenitors that allowed me to understand epigenetic remodelling underlying the cancer initiation events and metastatic phenotype of high-risk pediatric sarcoma tumours. Using integrated-omics approach, i.e., RNA-seq, CUT&RUN-seq, 3D chromatin mapping Hi-C, promoter capture Hi-C (pCHi-C) and systematic comparisons with patient-derived xenografts and primary tumours approach, I uncovered how these fusion transcription factors re-organises the chromatin architecture during tumour initiation and pre-malignant events to establish super-enhancers that drive the expression of key mitochondrial target genes, altering mitochondrial metabolism to promote tumour growth and aggressiveness in high-risk rhabdomyosarcoma.

Reference:
Bhargab et al. (2025). PAX translocations remodel mitochondrial metabolism through altered leucine usage in rhabdomyosarcoma. Cell 188: 2757-2777.



Metabolism and chromatin landscape alterations during rhabdomyosarcoma progression

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PDF10

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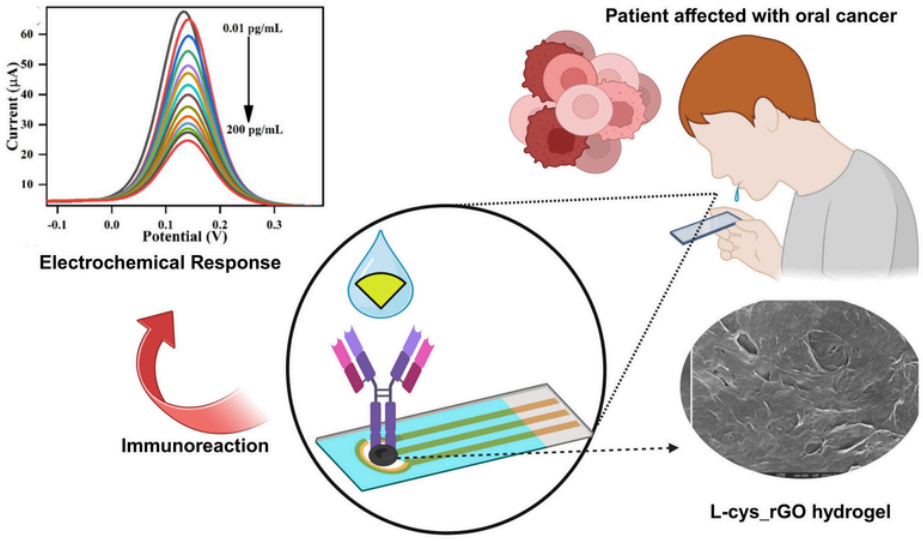
L-Cys functionalised electroactive and porous hydrogel-based biosensing nanoplatform for electrochemical detection of oral cancer biomarker

Abstract

Oral cancer is one of the most common cancers, which is often discovered at a late stage, resulting in an adverse outcome and limited treatment options for patients. Exploiting biosensors that use anti-biofouling hydrogels for early-stage oral cancer detection in non-invasive bodily fluids is becoming increasingly important in order to overcome this gap.

Here, we have developed a novel electrochemical immunosensor that can quickly, label-free, non-invasively, and affordably detect Tumour Necrosis Factor-α (TNF-α), a biomarker linked to the progression of oral cancer, in artificial saliva sample. The BSA/anti-TNF-α/L-cys_rGO hydrogel/gSPE immunosensing platform arises by modifying the gold screen-printed electrodes (gSPE) with bioinspired electroactive and porous reduced graphene oxide (rGO) hydrogel employing L-cysteine (L-cys) as a surface functionalisation and an *in situ* reducing agent. Anti-TNF-α is then covalently immobilised, and all remaining sites are blocked with bovine serum albumin (BSA). With a low detection limit of 1.20 pg/mL, a high sensitivity of 2.10 µA pg-1 mL cm-2, and a wide linear detection range of 1 to 200 pg/mL, the developed nanoplatform exhibits outstanding results when examined by differential pulse voltammetry (DPV) approach. The biosensor also demonstrated good repeatability and reproducibility, having relative standard deviations (%RSD) of 1.85% and 5.11%, respectively.

As a result, adding L-cys_rGO hydrogel to the immunosensor design improves performance and opens the door for its use in the early detection of oral cancer.



Immunosenor for oral cancer detection

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PDF11

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Investigating protein interactions directly from human cell lysates: A novel Direct-MS approach

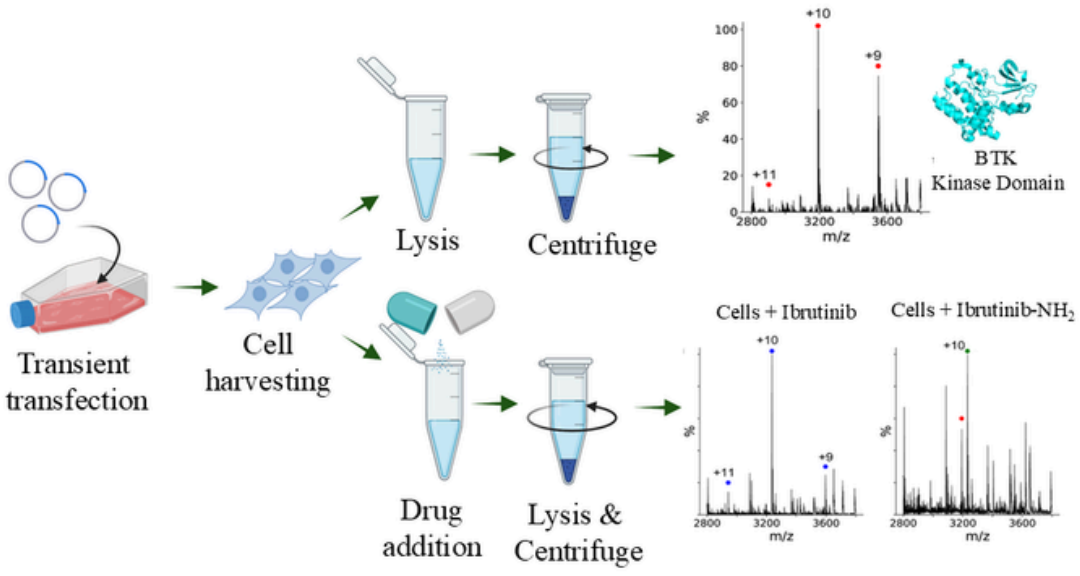
Abstract

Direct-MS, pioneered by the Sharon group, provides a fast, simple way to analyse overproduced proteins directly from crude cell lysates without purification. This approach preserves the natural cellular environment, including non-covalent interactions, saving both time and labour.

While initially used with bacterial cells, I am now expanding Direct-MS to study protein interactions in human cells. However, a challenge arises when applying this technique to human cells, as they typically produce lower levels of protein than bacterial cells. This can result in highly crowded spectra, potentially masking the target protein's signals and compromising the method's advantages. To address this bottleneck, I have developed a methodology that enables the straightforward investigation of protein interactions in human lysates.

My work focuses on improving the detection of low-abundance targets by tagging them with tandem split GFP (GFP11 and GFP1-10), which shifts their mass into less crowded regions of the spectrum. Because split GFP11 is small, fusing it to the target won't affect the expression or folding of the target. This mass shift simplifies data analysis and reveals critical insights into protein composition, structure, and post-translational modifications. Given the complexity of human cell lysates, this method offers a practical way to pinpoint and characterise specific targets amid background signals.

Ultimately, it may revolutionise the study of protein-protein interactions, drug binding, and protein modifications, offering faster, more precise approaches to discovering new therapies. In the long run, we aim to harness this technology for high-throughput screening to find drugs that modulate PPIs in desired cellular contexts. I look forward to sharing how this innovation can accelerate drug discovery and deepen our understanding of complex cellular processes.



Direct-MS is used to study protein interactions

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PDF12

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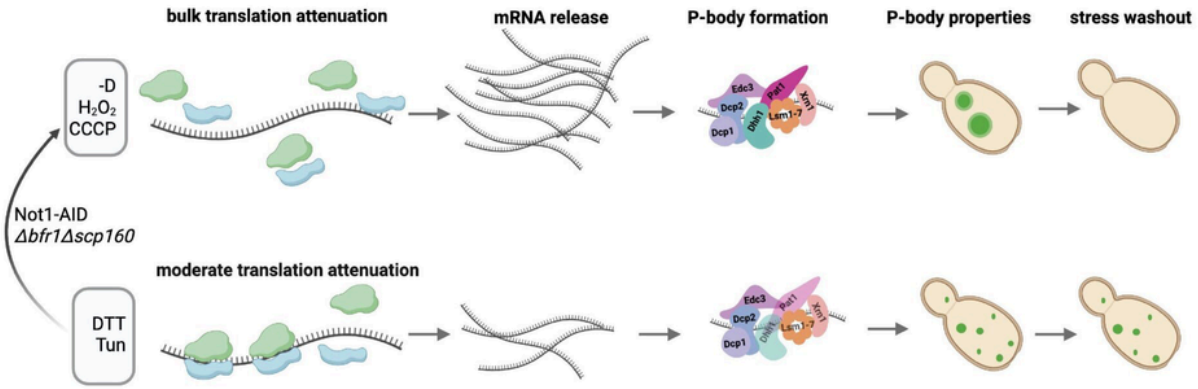
Not all processing bodies are formed equally

Abstract

One of the earliest responses to stress in eukaryotic cells is the formation of cytoplasmic granules, such as processing bodies (PBs). These membrane-less structures are dynamic, with their assembly, disassembly, and morphology influenced by the type of stress encountered. While PB formation under glucose starvation is well-studied, less is known about their behaviour under other stresses, such as endomembrane and redox stresses.

In this study, we expanded the stress repertoire in *Saccharomyces cerevisiae* and showed that PBs exhibit stress-specific differences in number, brightness, dynamics, and PB core component recruitment. ER stresses, like DTT and tunicamycin, which cause mild translation attenuation, induced dimmer PBs that sequentially recruited core components from the 5' to the 3' UTR and displayed more viscous, less fluid-like behaviour. In contrast, stresses that led to stronger translation attenuation resulted in brighter, more fluid PBs, formed by rapid en bloc recruitment of core components. To explore the relationship between translation and PB properties further, we deleted BFR1 and SCP160, two polysome-associated proteins, which shifted PBs towards a brighter phenotype and promoted earlier recruitment of 3' components, such as Pat1 and Lsm4. This reinforced the connection between translational state and PB properties. Additionally, depleting Not1 using an auxin-inducible degron to increase cytoplasmic levels of translationally inactive mRNA enhanced PB formation. Finally, an *in vitro* assay demonstrated that increased mRNA levels enlarge Dhh1-containing droplets, confirming that RNA abundance largely dictates PB characteristics.



mRNA levels define stress-specific P-body properties

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PDF13

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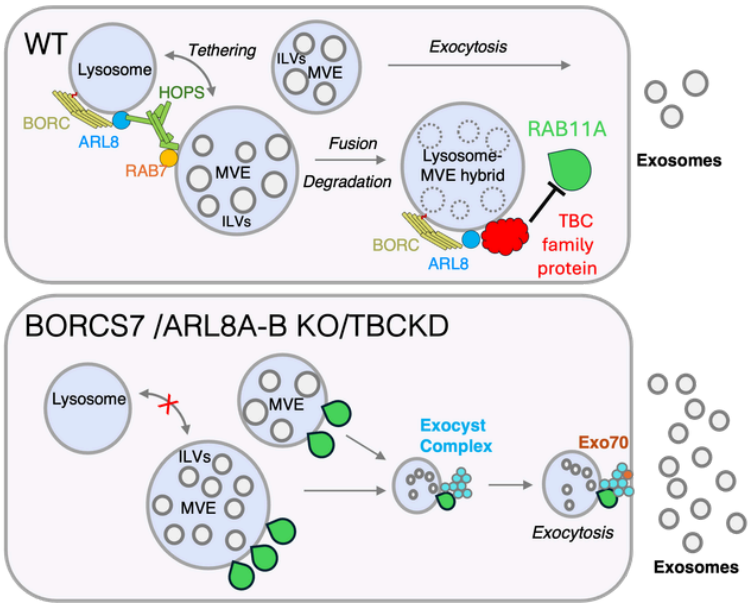
Rewiring of endo-membrane trafficking enhances exosome secretion during attenuated endosome-lysosomal function

Abstract

Cells regulate endocytosis by recycling or degrading cargo in lysosomes to maintain homeostasis. Disruptions in endolysosomal function contribute to neurodevelopmental and neurodegenerative diseases, including Alzheimer's disease, where lysosomal dysfunction increases extracellular vesicle (EV) secretion and promotes disease propagation. EVs are membrane-bound vesicles mediating intercellular communication through the transfer of bioactive molecules. A subset of EVs, exosomes, originates from multivesicular bodies that fuse with the plasma membrane to release their contents into the extracellular space. The BLOC-One-Related Complex (BORC; BORCS1-8) recruits the small GTPase ADP-Ribosylation Factor-Like 8 (ARL8), facilitating kinesin-mediated lysosomal transport and Homotypic Fusion and Protein Sorting(HOPS)-dependent fusion with late endosomes and autophagosomes. Previously, I found that loss of BORC, ARL8, or HOPS components causes perinuclear clustering of endolysosomes, impaired lysosomal clearance, and increased EV secretion, including exosomes. However, the mechanisms linking lysosomal dysfunction to enhanced EV secretion remain unclear. Here, we identify two TBC family proteins as novel ARL8 effectors using proximity biotinylation-based proteomics. Both localize to LAMP1-positive endolysosomal compartments in an ARL8- and BORC-dependent manner. These proteins act as Rab GTPase-activating proteins (Rab GAPs) that regulate Ras-related protein RAB11A, a key recycling endosome regulator. Loss of TBC-family proteins, ARL8, or BORC enhances Rab11a membrane recruitment and aberrant activation of its effectors, including the exocyst complex—a multi-protein tether essential for vesicle fusion with the plasma membrane. Knockdown of both TBCs increases Rab11a-exocyst interactions and exosome secretion, suggesting that cells compensate for lysosomal dysfunction by rerouting endosomal cargo toward exocytosis. Depletion of RAB11A or disruption of the exocyst complex suppresses this elevated exosome release, confirming that the BORC-ARL8-TBC axis regulates exosome biogenesis through Rab11a-exocyst interactions.Our findings reveal a pathway through which cells rewire trafficking to balance degradation and secretion, offering insights for biomarker discovery and therapeutic development.



A novel pathway through which cells rewire trafficking to balance degradation and secretion by acquiring RAB11A recycling membrane identity

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PDF14

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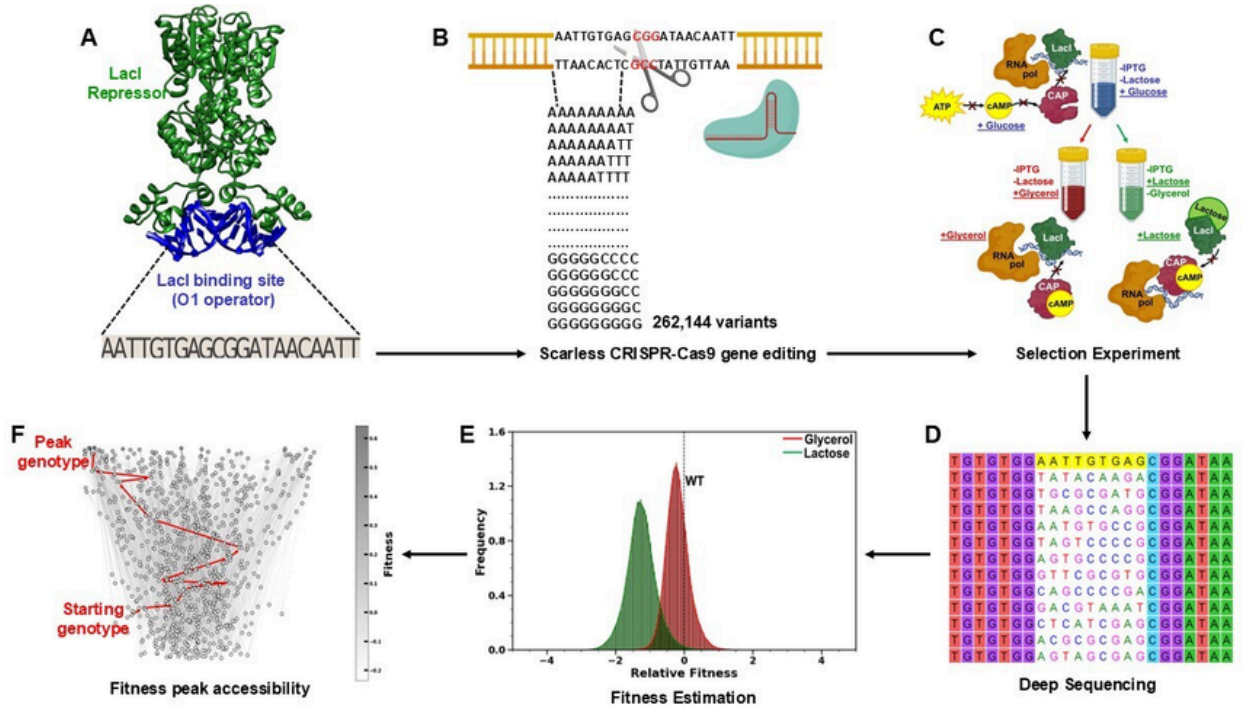
An empirical fitness landscape of gene regulation in Escherichia coli

Abstract

To metabolise lactose *Escherichia coli* cells must express the genes of the lac operon. This operon is repressed by the transcriptional regulator LacI. When lactose is absent, LacI binds to lac operator DNA and prevents operon transcription. Because few naturally occurring binding sites of this operator are known, we know little about the fitness landscape of this operator. In addition, we do not know whether adaptive evolution could easily create strong operators from weak operators or de novo from non-regulatory DNA. To find out, we used CRISPR-Cas-assisted genome editing, bulk competition, and high-throughput sequencing to map the fitness landscape of more than 140,000 lac operator variants. We did so not only in a lactose environment but also in glycerol, where lac operon expression is costly and not beneficial. Both landscapes are highly rugged and contain thousands of fitness peaks, which allow only 2 percent of evolving populations to reach one of the top 100 peaks. Both landscapes harbor a high potential for contingent evolution, but this potential is greater in glycerol, where evolving populations starting from the same genotype can reach a greater number of high fitness peaks. Our work illustrates that landscape ruggedness caused by epistasis can represent an important obstacle to adaptive evolution. It also shows that a simple environmental change can substantially affect fitness landscape topography.



A minimally invasive approach to study the adaptive landscapes of prokaryotic gene regulation

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PDF15

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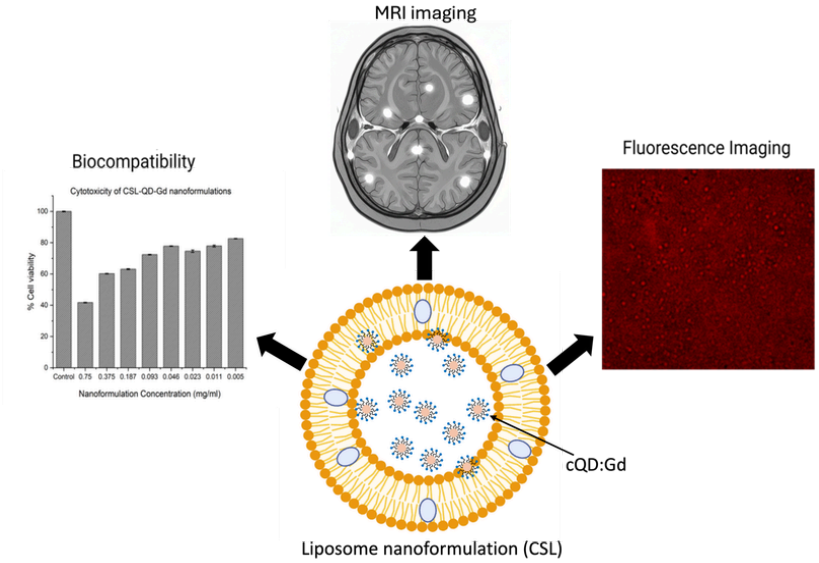
Multifunctional liposome co-encapsulating Gadolinium and carbon quantum dots for dual imaging and safer MRI contrast enhancement

Abstract

Magnetic resonance imaging (MRI) remains one of the most advanced non-invasive diagnostic tools for visualising soft tissues and organs with high spatial resolution. To enhance contrast and diagnostic precision, gadolinium (Gd)-based agents (GBCA) are routinely used; however, their free ionic forms are known to cause toxicity, tissue retention, and contraindications in patients with renal impairment and pregnant women. Liposomal encapsulation offers an effective strategy to limit direct Gd exposure.

In this study, a dual-imaging liposomal nanoformulation (CSL) co-encapsulating Gd-DOTA with carbon quantum dots (cQDs) was developed to achieve combined MRI and fluorescence imaging. The CSL:CQD-Gd liposomes were prepared by the thin-film hydration method, followed by extrusion, to obtain a uniform and stable nanoformulation. Conjugation between cQDs and Gd was established through EDC-NHS coupling before incorporation into the aqueous core of the liposomal matrix. Physicochemical characterisation using FTIR and UV-visible spectroscopy confirmed successful conjugation and encapsulation, while zeta potential measurements indicated strong colloidal stability. The formulation displayed an average hydrodynamic diameter of approximately 250 nm, suitable for systemic circulation. Fluorescence microscopy confirmed the distinct optical emission of cQDs, validating the fluorescence functionality by PL analysis of the formulation. MRI relaxivity studies showed that the liposomal formulation produced contrast intensity comparable to that of free Gd, confirming that encapsulation did not compromise imaging performance. Cytotoxicity was evaluated by MTT assay in Vero cells, demonstrating over 85% cell viability at concentrations below 0.37 mg/mL, verifying that liposomal encapsulation significantly reduced Gd-associated toxicity.

This CSL:cQD-Gd nanoformulation, therefore, presents a safe and efficient dual-imaging platform, providing fluorescence tracking and MRI contrast without sacrificing performance, offering a promising route toward next-generation biocompatible MRI contrast agents.



Schematic representation of the CSL:cQD-Gd liposomal nanoformulation demonstrating co-encapsulation of Gd-DOTA and cQD within a stable liposomal matrix (CSL). The illustration demonstrates a multi-functional liposomal formulation, which is biocompatible, preserving MRI contrast capability and showing fluorescence emission from the liposome core due to the presence of cQDs, and it confirms the dual imaging functionality of the nanoformulation, establishing it as a safer and efficient platform for combined MRI and fluorescence imaging.

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PDF16

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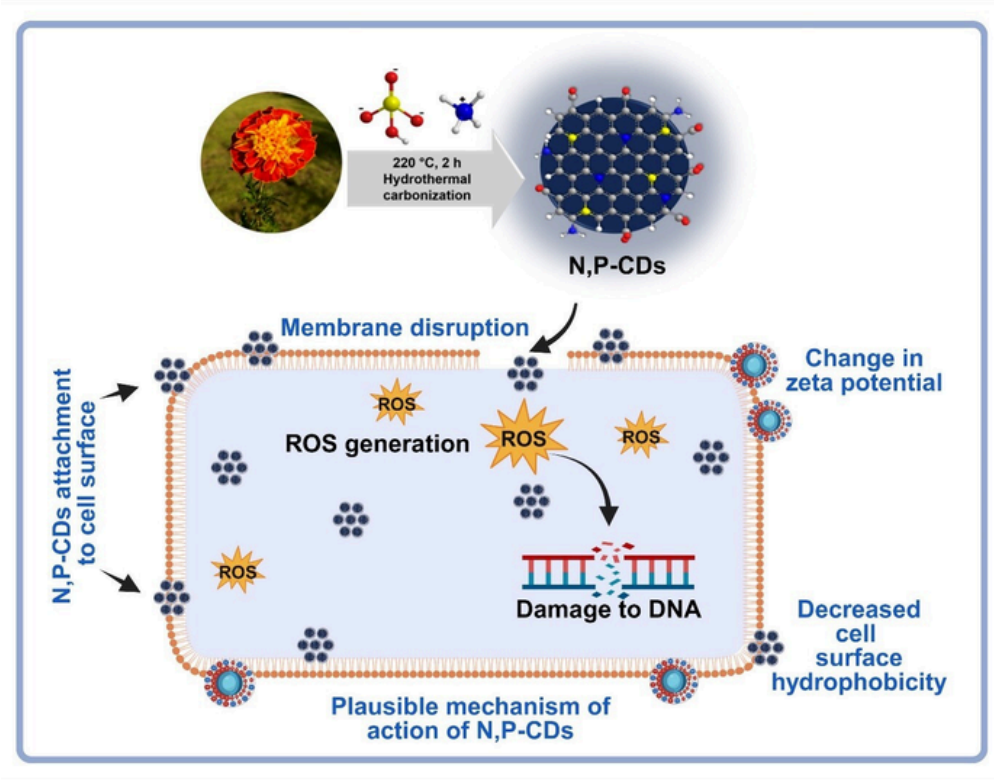
Eco-friendly N, P-doped carbon dots as safe antimicrobial and antioxidant agents with synergistic activity

Abstract

The rise of antibiotic-resistant microorganisms demands novel antimicrobial agents. Heteroatom-doped carbon dots (CDs), with strong antimicrobial and antioxidant properties, are prestigious gem for biomedical applications. In the present study, nitrogen- and phosphorus-co-doped CDs (N,P-CDs) are synthesised from an agricultural resource using a eco-friendly hydrothermal method and evaluated their antimicrobial efficacy both individually and in synergistic (1:1) combinations with silver nitrate ($AgNO_3$) and hydrogen peroxide (H_2O_2). Antimicrobial activity was assessed against *Bacillus subtilis* (Gram-positive), *E. coli* and *Pseudomonas aeruginosa* (Gram-negative), and the fungus *Candida albicans*. N,P-CDs (200 µg/mL) displayed inhibitory activity against all tested pathogens with inhibition zones ranging from 13.25 to 18.13 mm, with synergistic formulations exhibiting enhanced antibacterial activity in the presence of $AgNO_3$ and H_2O_2. Mechanistic investigations revealed that antimicrobial action was further linked with subcellular damage, including morphological destruction, alterations in zeta potential, decreased cell surface hydrophobicity, and DNA structural changes. In addition, N,P-CDs exhibited strong antioxidant capacity, as demonstrated by effective scavenging of DPPH, ABTS, and ·OH radicals, with the highest (>98 % at 1.25 mg mL-1) activity detected towards ·OH, a highly reactive species implicated in oxidative stress. Importantly, N,P-CDs demonstrated excellent biocompatibility with HT-22 and RAW cells at concentrations effective against pathogens and further mitigated lipopolysaccharide-induced cytotoxicity in HT-22 cells. This study highlights green-synthesised N,P-CDs as multifunctional nanomaterials with strong antimicrobial, antioxidant, and cytoprotective properties, underscoring their dual potential in biomedical and agri-food applications.



Anti-microbial activity of biomass-based N,P-carbon dots is evaluated against multi-drug resistant bacteria and fungi

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PDF17

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DNA nanocarrier-mediated delivery of cisplatin for enhanced efficacy and treatment of oral squamous cell carcinoma

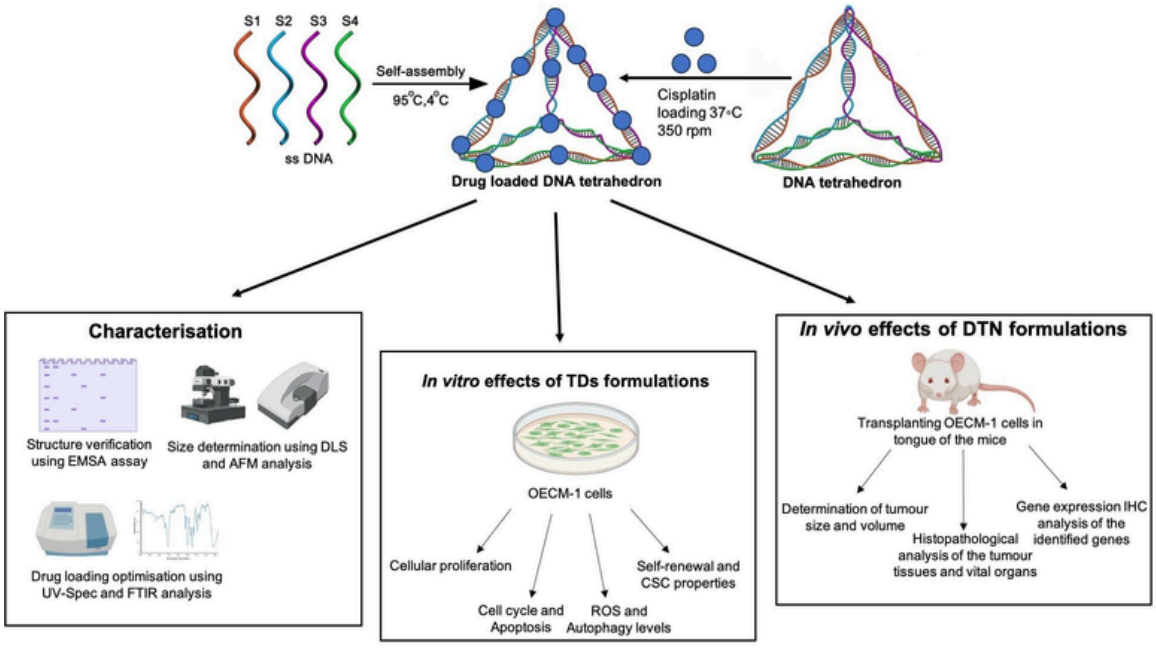
Abstract

Oral Squamous Cell Carcinoma (OSCC) is a major public health concern in Asia, largely driven by tobacco use, with a 5-year survival rate of ~50%. Poor outcomes are primarily due to minimal residual disease sustained by therapy-resistant cancer stem cells (CSCs), driving recurrence and progression. Overcoming drug resistance requires precision delivery systems that enhance efficacy, reduce toxicity, and target CSCs. DNA tetrahedrons (TDNs), owing to their biocompatibility, stability, and programmability, have emerged as promising nanocarriers.

This study reports the development of a TDN-based system encapsulating cisplatin (TDN Cis) for targeted OSCC therapy. TDN Cis was synthesised and characterised by FTIR, native PAGE, NTA, UV–Vis spectroscopy, and AFM. Drug loading efficiency, release kinetics, and stability were assessed *in vitro*. Cellular uptake was studied in OECM1 OSCC cells, followed by assays for proliferation, apoptosis, mitochondrial membrane potential (ΔΨm), cell cycle progression, ROS generation, invasion, and spheroid formation. Therapeutic efficacy was further validated in a xenograft mouse model.

TDN Cis achieved 90% drug loading, remained stable for 10 h, and provided sustained release over 24 h. Rapid cellular uptake was observed within three hours. IC50 decreased markedly to 200 nM compared to free cisplatin. G2/M arrest and Annexin V/PI staining confirmed apoptosis, with ~40% apoptotic cells. Invasion and spheroid assays showed invasive capacity reduced to ~40% vs. ~65% with free cisplatin ($p<0.05$). JC 1 staining revealed significant mitochondrial depolarisation, and ROS levels were markedly elevated. *In vivo*, TDN Cis induced superior tumour regression with minimal toxicity.

TDN Cis represents a potent, targeted nanoplatform that overcomes chemoresistance, enhances cytotoxicity, and suppresses CSC-associated phenotypes, offering a promising approach for treating recurrent and resistant OSCC.



Synthesis, characterisation, and efficiency of cisplatin-loaded DNA tetrahedrons on in vitro and in vivo OSCC models

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PDF18

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Mapping of KRAS resistance landscapes using base-editor mutagenesis

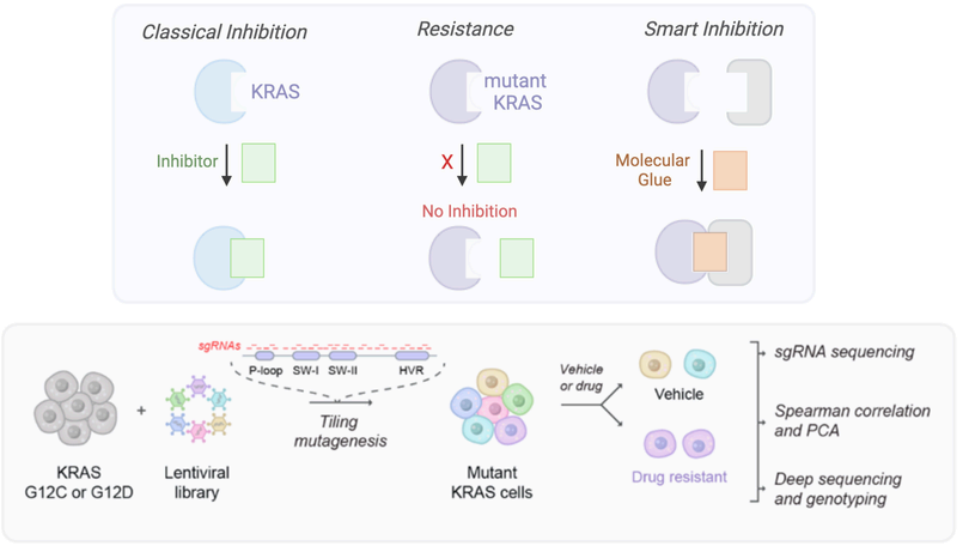
Abstract

Drug resistance remains one of the most persistent challenges in modern therapeutics. In both cancer and infectious diseases, most drugs fail due to target mutations that compromise binding or activity.

To systematically understand how mutations alter protein function and drug response, we used oncogenic KRAS as a model system. We employed CRISPR-assisted base-editor tiling mutagenesis, a high-throughput approach that enables precise, single-base editing and phenotypic screening of thousands of variants directly in mammalian cells. The strategy allowed us to map how KRAS mutations affect responses to both classical inhibitors and a newer class of induced-proximity inhibitors (molecular glues), small molecules that promote targeted protein–protein interactions to induce degradation of disease-associated proteins.

Through extensive multidrug treatments of engineered KRAS cell lines, we found that molecular glues maintained inhibitory activity against many escape mutations that rendered classical inhibitors ineffective, often preserving cell-growth inhibition at doses below 1 µM. Resistance mutations emerging from molecular glue treatment did not overlap with those from classical inhibitors, confirming distinct resistance mechanisms. Structural analyses further suggested that the extended ternary interface between KRAS and the recruited protein (CypA) provides avidity-driven stabilisation, reducing susceptibility to point mutations.

This work demonstrates how base-editor mutagenesis can serve as a systematic platform for predicting and preempting resistance. Ultimately, integrating such variant mapping with DNA-encoded library-based discovery can accelerate the rational design of next-generation therapeutics.



When inhibition gets smart: A keychain upgrade that lets the drug unlock resistant targets

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PDF19

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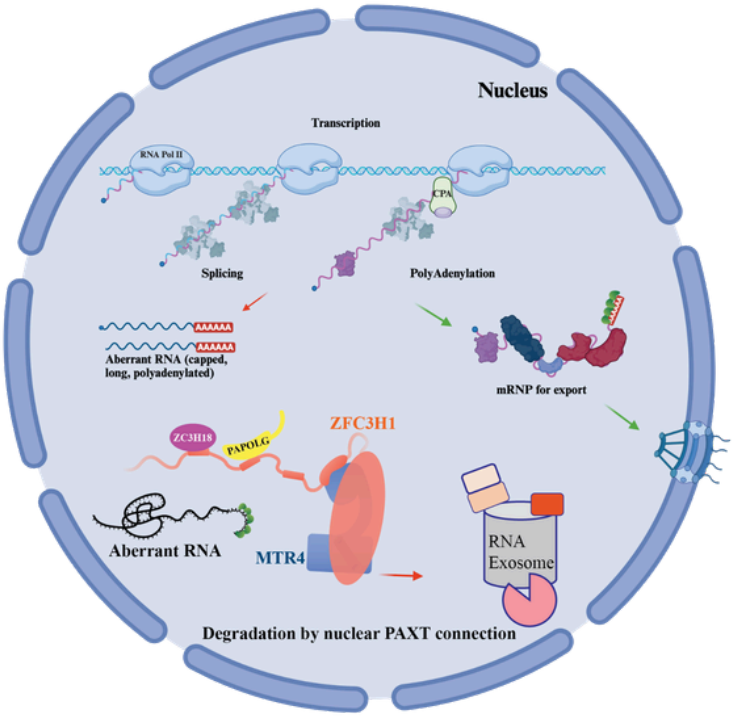
Molecular mechanism of PolyA RNA degradation by Nuclear PolyA tail eXosome Targeting (PAXT) connection

Abstract

Production of messenger RNA is intricately coordinated between different cellular machineries, from transcription of pre-mRNA to 5' capping, splicing, and 3' polyA tail to export out of the nucleus. Any defects in these processes leads to production of abnormal RNA products that must be removed. RNA exosomes are the primary molecular machinery in eukaryotes that carry out degradation of the RNA. However, the RNA exosome lacks substrate specificity and, in the nucleus, must rely on its association with adaptor complexes centered around the RNA helicase MTR4. MTR4 forms a stable heterodimer with the scaffold protein ZFC3H1 to form the PAXT connection, which targets capped, long and polyadenylated RNAs. However, how PAXT mechanistically engages with substrates and funnels the RNA to the exosome has yet to be determined. We addressed these knowledge gaps using structural and biochemical, and in silico structure predictions tools. Cryo-EM of reconstituted MTR4-ZFC3H1 revealed a conserved bi-partite mode of interaction, whereby ZFC3H1 forms extensive interactions with MTR4 that lock its helicase core in a closed, inactive state. As the helical core is predicted to bind the exosome, we recapitulated MTR4-ZFC3H1 in complex with the exosome in the presence of RNA and performed cryo-EM. Upon exosome binding, the MTR4 and ZFC3H1 undergo major structural rearrangements allowing RNA and Exosome binding to the MTR4. We next characterised the interplay between PAXT and 3' end polyA degradation machinery to understand how PAXT targets substrates. Structural predictions and biochemical studies showed PAXT binding partners, ZC3H3 and polyA polymerase Gamma, can replace and mimic subunits of the polyA degradation machinery to form an alternative complex, thus connecting PAXT to the targeted substrate. This study explains the mechanisms governing the role of PAXT in a nuclear quality control pathway.



Aberrant RNA degradation by RNA Exosome PAXT connection

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PDF 20

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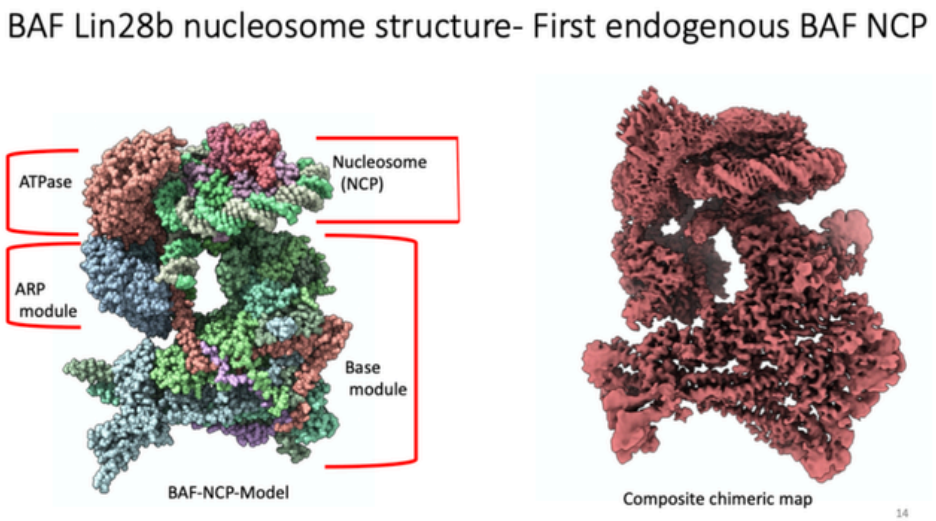
Cryo-EM structure of Human BAF complex bound to the Lin28B human enhancer nucleosome

Abstract

The remodelling of gene regulatory sequences by the human SWI/SNF complex (BAF) underpins the establishment of tissue-specific transcriptional programs and is misregulated in numerous neurological disorders as well as in over 20% of human cancers. Our current understanding of BAF structure and substrate engagement has been limited to studies involving artificial nucleosomal templates and recombinant BAF preparations lacking the full complement of core subunits and subunit domains. Here we present the cryo-EM structure of endogenous human BAF engaged with the native Lin28B enhancer nucleosome, providing evidence for ATP-independent DNA unwrapping upon the engagement of BAF with native nucleosomes. Our structural model extends beyond the ordered BAF core to include all 12 core subunits and the flexible surface domains involved in protein–protein interactions that underpin BAF biology. Importantly, we find that Oct4 binding stimulates BAF engagement and nucleosome remodelling activity at the Lin28B enhancer nucleosome. In addition, we determine the histone-binding specificity of the two major BAF reader domains, the Brg1 octamer interaction (BOI) submodule and the Baf45D double PHD domain which show a binding preference for H3K14Ac and H4K20me3 modified tails, respectively. Taken together, these findings provide structural and biophysical context to better understand the interplay between BAF and Oct4 in the activation of key metazoan enhancers.



Unlocking DNA: How BAF remodels the nucleosome

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PDF 21

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Targeting bromodomains in Plasmodium falciparum as a novel epigenetic antimalarial strategy

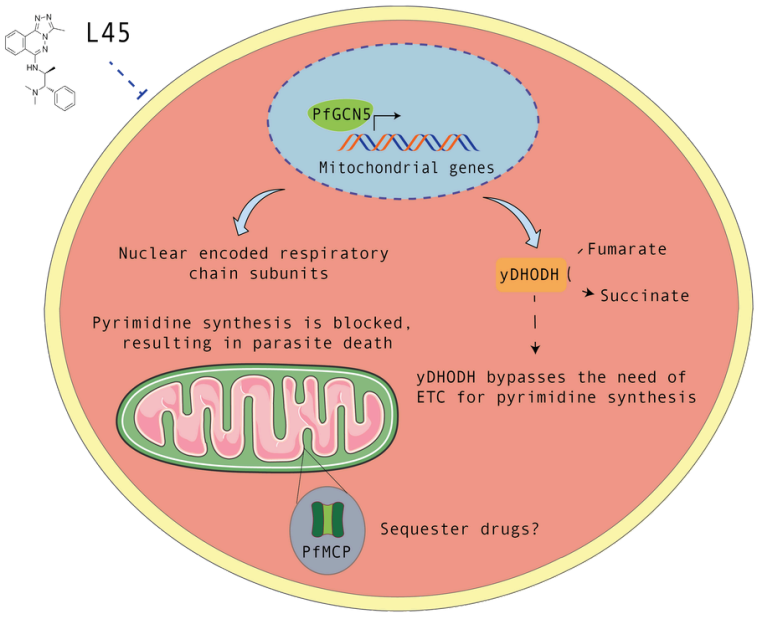
Abstract

Bromodomain-containing proteins (BDPs) regulate transcription by recognising acetylated histones [1]. They are implicated in the pathogenesis of diverse diseases and have emerged as promising therapeutic targets, particularly in cancer and inflammatory disorders [2, 3].

In this study, we evaluated BDPs in *Plasmodium falciparum* as potential druggable targets. Using conditional-knockout approaches targeting the bromodomains of PfGCN5, PfSET1, and PfBDP6, we demonstrated their essentiality for parasite survival during the blood stage. Among these, the bromodomain of PfGCN5 emerged as a particularly promising drug target due to its rapid parasite-killing effect. PfGCN5, previously implicated in invasion and virulence, has a resolved bromodomain structure in complex with the small-molecule inhibitor L45 [4]. We assessed the antimalarial activity of L45 and its therapeutic potential by targeting the PfGCN5 bromodomain. L45 showed efficacy against both blood-stage *P. falciparum* and liver-stage *P. berghei*. L45-mediated inhibition of PfGCN5 induces a compensatory resistance mechanism that offsets impaired mitochondrial function. Given PfGCN5's role in transcriptional regulation of mitochondrial pathways, including the electron transport chain [5], *in vitro* resistance selection revealed point mutations in a mitochondrial carrier protein (PfMCP-R), implicating mitochondrial adaptation in the resistance phenotype. Using CRISPR-Cas9 editing, we confirmed that a mutation in PfMCP-R conferred resistance to L45. We found that mutations in PfMCP-R render parasites hypersensitive to mitochondrial inhibitors such as atovaquone, DSM1, and myxothiazol, indicating impaired mitochondrial function. Additionally, metabolomic profiling revealed an accumulation of dUMP following L45 treatment, suggesting broader metabolic disturbances linked to bromodomain inhibition.

Together, these findings identify a novel mechanism of resistance to L45 and reveal an unexpected link between PfGCN5 bromodomain function, transcriptional regulation, and parasite metabolism.

References:
1. DOI: 10.1038/s41392-023-01647-6
2. DOI: 10.1038/nrm.2016.143
3. DOI: 10.1021/acs.jmedchem.6b01761
4. DOI: 10.1002/anie.201610816
5. DOI: 10.1093/nar/gkaf218



Transcriptional regulator PfGCN5 modulates mitochondrial function in malaria parasites

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PDF 22

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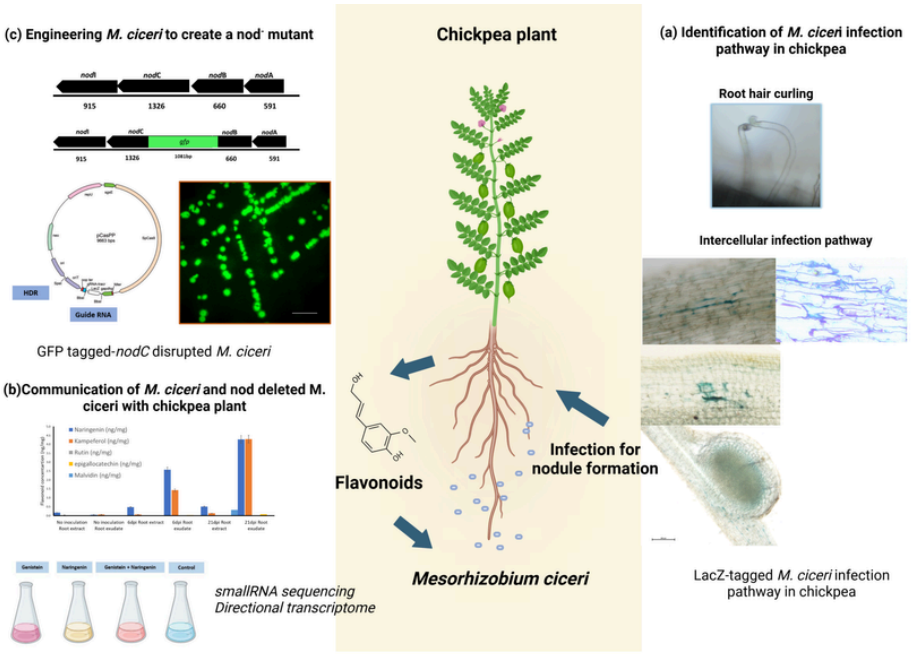
The communication network of Mesorhizobium ciceri and chickpea

Abstract

Mesorhizobium ciceri forms specialised structures on chickpea roots where they can fix nitrogen into usable compounds. This environmentally vital interaction fixes inert nitrogen, and engineering this trait into cereal crops would significantly reduce fertiliser dependency. Achieving this goal requires a foundational understanding of the early molecular events that govern the establishment of the symbiosis.

To understand the initial communication in *M. ciceri*-chickpea interaction, we first used a LacZ-tagged *M. ciceri* to trace the infection pathway in chickpea. We then used a genome-editing tool to delete the nod factor-producing enzyme nodC. The nodC-deleted *M. ciceri* was used as a control, and then the infected roots were compared with the wild type. LC-MS-based secretome analysis and comparative RNA-sequencing were performed to gain insights into the flavonoids secreted by the plant, the gene expression in the bacteria, and the small RNAs secreted by the bacteria to the plant, thereby generating a communication network.

Our findings reveal that *M. ciceri* employs an atypical infection strategy, colonising the intercellular spaces of epidermal cells rather than using canonical root hair infection threads. We identified specific flavonoids, genistein and naringenin, from the chickpea root secretome as potent and rapid inducers of the bacterial nodABC operon. Comparative transcriptomics of wild-type and mutant strains in the presence of these flavonoids suggested the genes regulated in *M. ciceri*. Further, a suite of novel small RNAs (sRNAs) was identified in *M. ciceri* whose expression is significantly modulated by the host-derived flavonoids. These results highlight a unique infection mechanism within the IRLC legume clade and position flavonoid-responsive sRNAs as critical new players in the chickpea-rhizobia dialogue. We propose that these sRNAs act as key regulators of bacterial physiology during early colonisation and are prime candidates for cross-kingdom signalling to the host plant.



The communication network of Mesorhizobium ciceri and chickpea

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PDF 23

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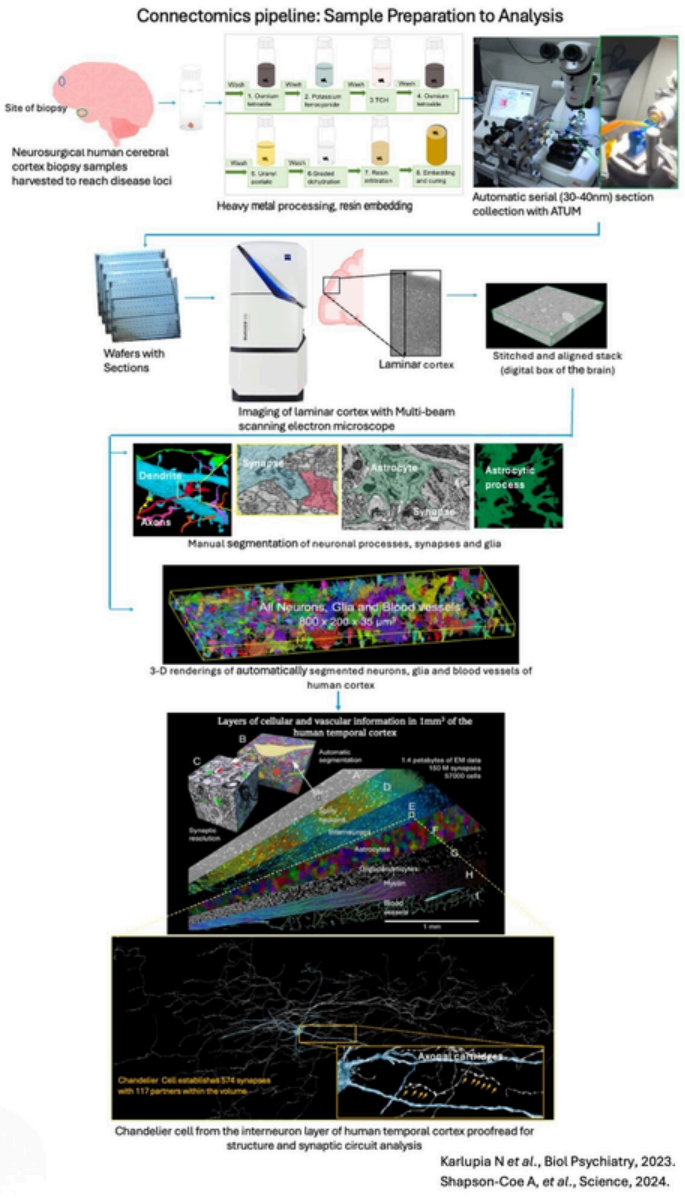
Connectomics approach to study the structure and connectivity of chandelier cell axons in the human temporal cortex

Abstract

Human cognition depends on synaptic connectivity and communication among diverse brain cell types. Connectomics, the field of large-scale electron microscopy, enables mapping of cellular circuit-level brain organisation.
I will introduce this approach for reconstructing the synaptic circuits of chandelier cells (ChCs). ChCs are inhibitory neurons known to form multiple cartridge-synapses exclusively on the axon initial segments (AIS) of pyramidal cells (PyCs) within the cortical layer of their somata. Multiple ChC axons were reconstructed in our human temporal cortex connectomics dataset to identify ChCs with somata within the volume (1). 15 ChCs were identified, and their axons were proofread and assessed for their complete synaptic connectivity.

Our results revealed the unusual structure and connectivity of ChCs. The synaptic output of ChCs was neither limited to PyC-AIS nor to the layer of their somata. 2 of 15 ChCs had two axons, which projected to non-overlapping regions but were like uni-axonal ChCs in their overall synaptic output. The average number of synapses established by these cells varied depending on the layer of their somata. The most surprising finding was that ChCs establish synapses on the AIS of other ChCs, exhibiting topographical and ChC-ChC AIS connection-based specificity in their connectivity.
At present, these results raise more questions than they answer, and I am seeking to discover whether these are associated with the patient's condition or are a feature of human ChC axonal behaviour.

Reference:
1. Shapson-Coe A, Januszewski M, Berger D, Pope A, Wu Y, Blakely T, Schalek R L, Li P, Wang S, Shepard JM, Karlupia N, Dorkenwald S, Lichtman JW et al. 2024. A connectomic study of a petascale fragment of the human cerebral cortex. Science 384: 635-648.



Connectomics pipeline: Sample preparation to chandelier cell circuit analysis

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PDF 24

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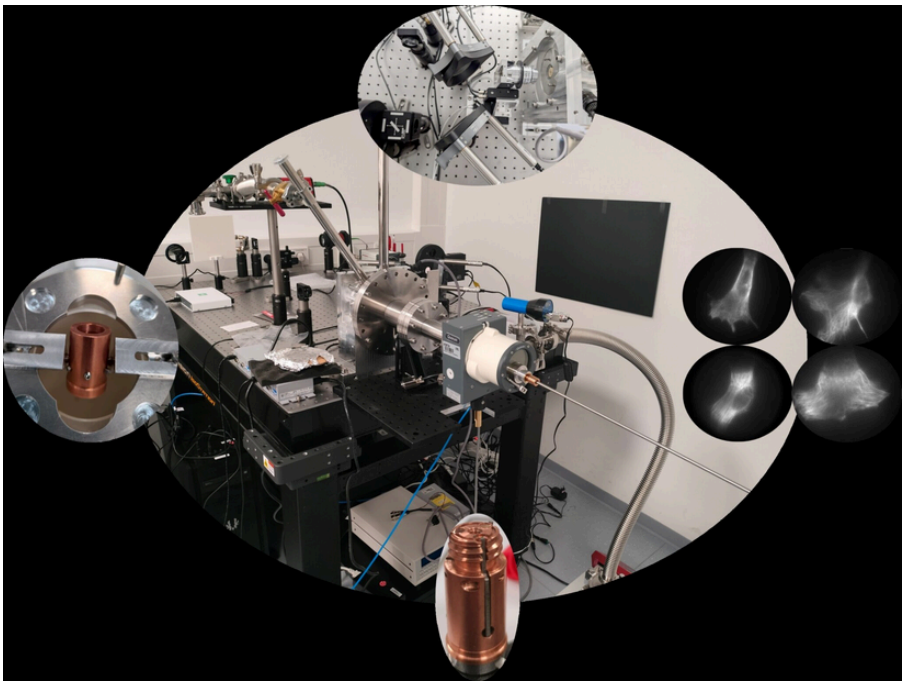
Building of a cryo-super-resolution microscope for cryo-correlative light and electron microscopy (cryo-CLEM)

Abstract

The ability to freeze biological samples in vitreous ice allows the study of macromolecules at molecular resolution with cryogenic electron microscopy (cryo-EM). Cryo-EM avoids using chemicals to fix or stain biological samples, which have been shown to perturb the native sample structure. This has further enabled understanding of structure-function relationships within the cellular context and the study of interactions between subcellular structures and macromolecules, providing deeper insights into the mechanisms governing various life processes. [1] Despite the progress made so far, cryo-EM remains limited in its ability to identify subcellular structures and rare events directly. One approach to overcome these limitations is to combine cryo-EM with light microscopy and perform correlative superresolution light and electron microscopy (CLEM). [2] For localisation-based cryo-superresolution light microscopy techniques, there are two main approaches based on the design of the cryostat used to maintain vitrified samples at close to liquid nitrogen temperature. These are open and vacuum-based cryostat designs.
In this poster, I will present a theoretical comparison between the two types of designs and discuss the planning and progress in building a microscope with a vacuum-based cryostat for cryo- single molecule localisation microscopy (cryo-SMLM) to study vitrified biological samples.

References:
1. Nogales E, Mahamid J. 2024. Bridging structural and cell biology with cryo-electron microscopy. Nature 628(8006): 47-56.
2. de Boer P, Hoogenboom JP, Giepmans BN. 2015. Correlated light and electron microscopy: ultrastructure lights up! Nat Methods 12(6): 503-13.



Cryogenic superresolution microscope

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PDF 25

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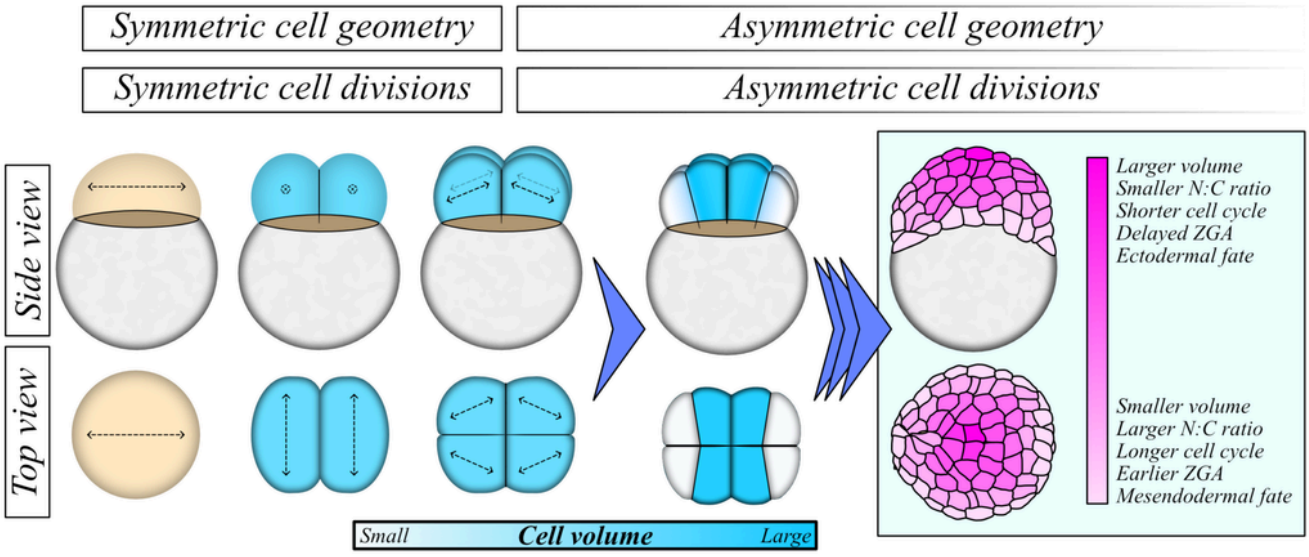
Geometry-driven asymmetric cell divisions pattern cell cycles and zygotic genome activation

Abstract

Early embryo geometry is one of the most invariant species-specific traits, yet its role in ensuring developmental reproducibility and robustness remains underexplored. Here, we demonstrate that in zebrafish, the geometry of the fertilised egg, specifically, its curvature and volume, acts as a critical initial condition, triggering a cascade of events that exert lasting influence on development. It directs asymmetric blastoderm divisions, generating radial gradients of cell volume and nucleocytoplasmic ratio. These gradients, in turn, produce mitotic phase waves, with cell cycle periods determined largely cell-autonomously by the nucleocytoplasmic ratio. Modelling and perturbation experiments, including the generation of syncytial embryos to enhance cell–cell coupling, demonstrate that reducing autonomy reshapes these waves. This highlights the instructive role of geometry-derived volume patterns in setting the intrinsic period of the cell cycle oscillator.

Remarkably, beyond organising cell cycles, early embryo geometry also patterns zygotic genome activation (ZGA) at the midblastula transition, a key step in establishing embryonic autonomy. Disrupting geometry alters ZGA patterns and causes ectopic germ-layer specification, underscoring the developmental significance of this process.

Together, these findings reveal a symmetry-breaking function of embryo geometry in coordinating the cell cycle and transcriptional patterning, establishing a blueprint for robust embryogenesis. Comparable correlations between axes of cell volume, cell cycle length, and germ layer fate exist in several other species, suggesting that geometry-driven patterning is likely a conserved principle of early development.



Zebrafish zygote geometry confers developmental robustness

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PDF 26

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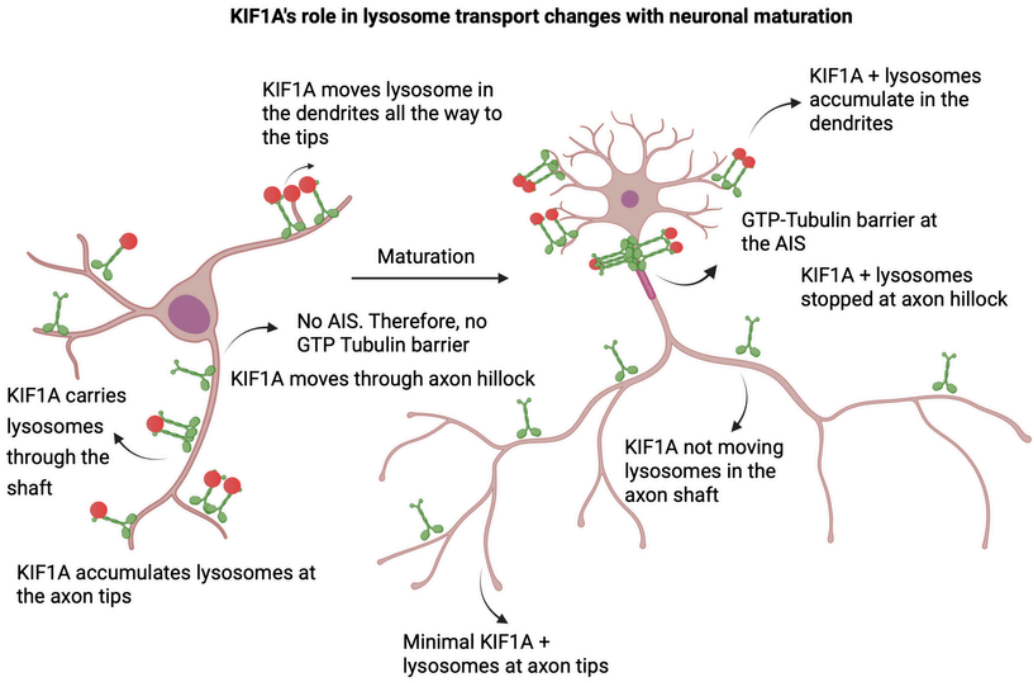
KIF1A-driven lysosome transport is regulated by microtubule modifications associated with neuronal development

Abstract

The molecular motors kinesin and dynein play an important role in the spatio-temporal organisation of cellular compartments, such as lysosomes, by moving these organelles along microtubules. Lysosomes, well known as the degradative compartments of the cell, are present in most cell types where their appropriate localisation and transport are critical for cellular function. In HeLa cells, lysosomes are moved anterogradely to the cell periphery by the kinesin family motors KIF5 and KIF1A. Interestingly, KIF1A is the primary motor protein responsible for transporting synaptic vesicle precursors in mammalian neurons. Therefore, the role of kinesin-3 motor KIF1A in lysosome transport in the neurons remains to be elucidated.

We found that KIF1A is essential for lysosome transport from the soma into the dendrites and axons of developing neurons. Surprisingly, we found that neuronal maturation, marked by Axon Initial Segment (AIS) formation, restricts KIF1A from driving lysosomes out of the soma into the axon. We discovered that the enrichment of microtubules in GTP-bound form at the AIS limits KIF1A's ability to move large lysosomes through the AIS. A KIF1A mutation, identified in hereditary spastic paraplegia (HSP), increases KIF1A's ability to bind to GTP-Tubulin microtubules at the AIS and pull the large lysosomes through the AIS, into the axon shaft and accumulate at the axon tip. Finally, we found that KIF1A is essential to move lysosomes into the dendrites and axons of developing human iPSC-derived i3Neurons. In i3Neurons, differentiated from KIF1A-KO iPSCs, the lack of lysosomes in the axons leads to increased accumulation of autophagic vesicles and development of axonal swellings.

In summary, our study reveals that neurodevelopment modulates KIF1A's role in transporting lysosomes and that disease-causing KIF1A mutations disrupt this regulation.



KIF1A-drive lysosome transport changes during neurodevelopment

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PDF 27

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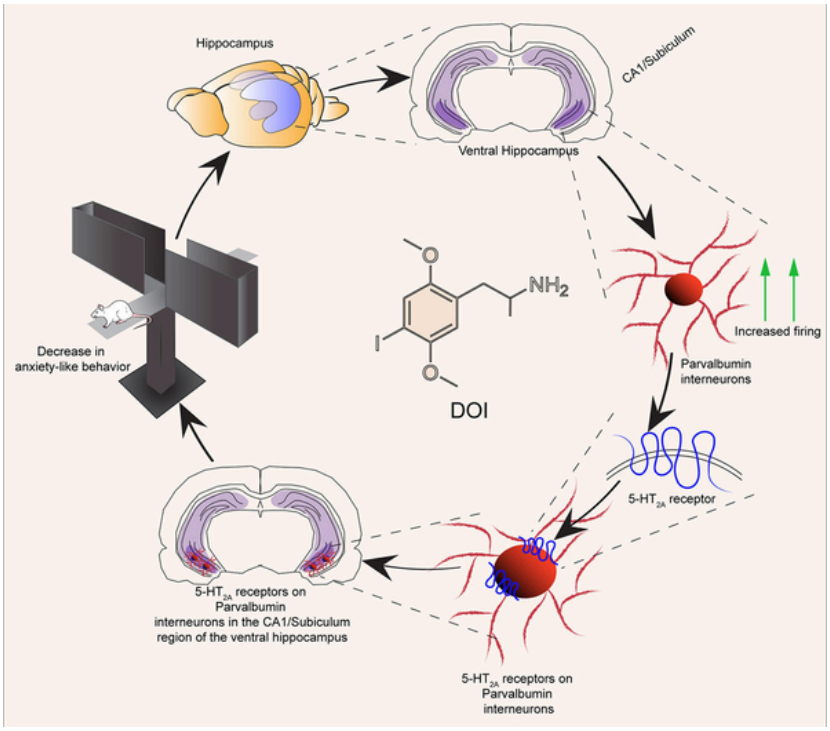
Psychedelics and behaviour: Insights into neural circuits regulating psychedelic-evoked changes in anxiety

Abstract

There has been a recent renewal of interest in the therapeutic potential of serotonergic psychedelics, but the mechanisms underlying their behavioural effects are not well understood.

This work aims to understand the neural mechanisms underlying the anxiolytic effects of the serotonergic psychedelic 2,5-dimethoxy-4-iodoamphetamine (DOI), which has a high binding affinity for the serotonin 2 (5-HT2) family of receptors, using animal models in rats and mice. Our findings reveal a critical role for a subclass of GABAergic interneurons in the ventral hippocampus (vHpc) in mediating DOI's anxiolytic action. By combining anatomical mapping, pharmacological manipulation, and genetic tools, we demonstrate that 5-HT2A receptors on the Parvalbumin (PV)-positive inhibitory neurons within the CA1/Subiculum (CA1/sub) subregions of the vHpc are essential for the anxiolytic-like effects of DOI. Using *in vivo* electrophysiology and opto-tagging approaches, we found that DOI selectively enhances the firing of fast-spiking parvalbumin (PV)-positive interneurons in the vHpc. Importantly, most of these PV interneurons express 5-HT2A receptors, suggesting a direct serotonergic modulation of inhibitory circuits. Restoring 5-HT2A receptor expression specifically in PV-positive interneurons within a loss-of-function background selectively reinstated the DOI-induced anxiolytic-like responses.

Taken together, our study localises the acute anxiolytic actions of the serotonergic psychedelic DOI to 5-HT2A receptors on the PV neurons in the vHpc CA1/sub region. It identifies PV-positive fast-spiking cells as a cellular trigger for this behavioural response. Our findings provide a mechanistic framework for the anxiolytic-like effects of serotonergic psychedelics like DOI, offering insights that could inform the development of targeted interventions for anxiety disorders.



Neural mechanism underlying the behavioural effects of the psychedelic DOI on anxiety-like behaviour

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PDF 28

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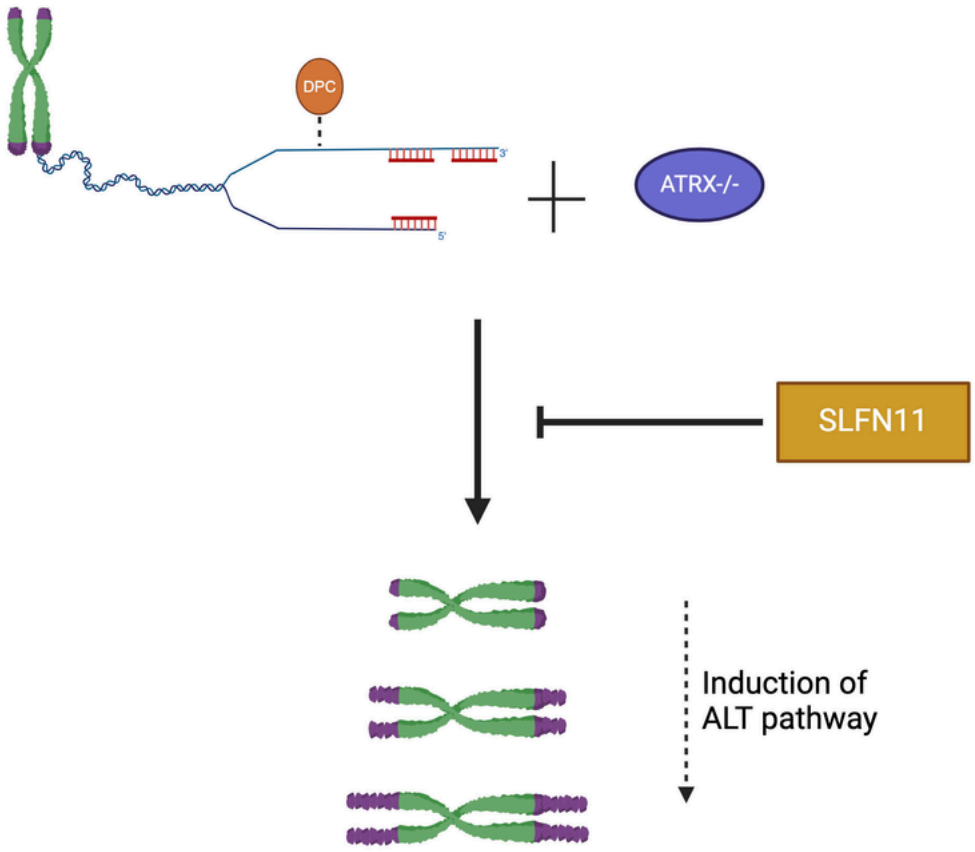
SLFN11 puts the brakes on alternative lengthening of telomeres

Abstract

Alternative lengthening of telomeres (ALT) is a unique, homologous recombination-dependent telomere elongation mechanism, primarily active in 10–15% of telomerase-negative cancer cells. While most ALT cells lack the ATRX/DAXX chromatin remodelers, ATRX/DAXX loss alone is not sufficient to activate ALT.

Our study reveals that, in addition to the ATRX/DAXX, ALT cells also lack SLFN11. Re-expression of SLFN11 in an ALT cell line led to its localisation at telomeres and decreased ALT activity, as evidenced by suppression of APBs and TERRA. Re-expression of SLFN11 also suppressed the DDR pathway at telomeres, which were activated by spontaneous DNA damage in ALT cells, leading to telomere destabilisation and ultimately killing the cells. Furthermore, SLFN11 suppresses the induction of ALT in ATRX-depleted telomerase-positive cancer cells. Notably, ATRX depletion triggered ALT in the SLFN11 knockout DU145 cell line but not in SLFN11-proficient wild-type cells.

Together, these findings identify SLFN11 as a negative regulator of the ALT pathway and indicate that its loss, alongside ATRX/DAXX inactivation, is necessary for ALT activation.



SLFN11 inhibits alternative lengthening of telomeres (ALT)

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Pharmaco-behavioural screens identify novel suppressors in zebrafish mutants of neuropsychiatric disorder genes

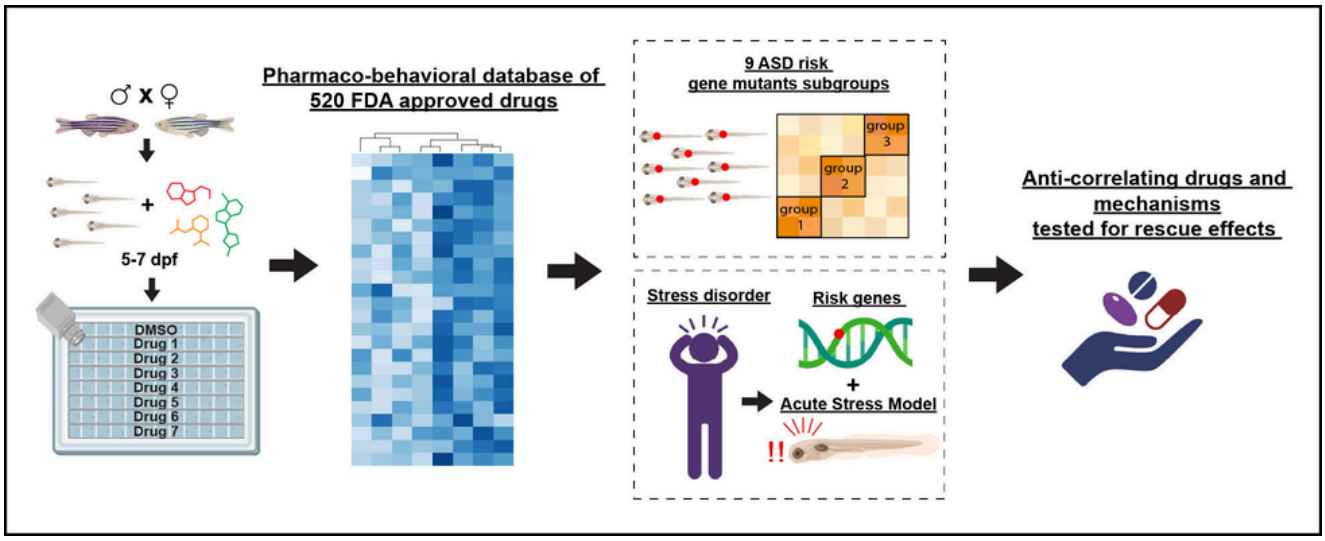
Abstract

Identifying relevant pharmacological targets in neuropsychiatric disorders remains a central challenge. Zebrafish are a scalable system increasingly used for pharmacological screens.

In this study, we use large-scale zebrafish larval behavioural assays to screen U.S. FDA-approved drugs and develop a database of the behavioural fingerprints of 520 drugs in wild-type fish. We show that drugs with more than one shared target and similar indications induce highly correlated behavioural profiles. In a previous functional screen of 10 genes strongly associated with autism, we showed that the behavioural phenotypes of these mutants clustered into three distinct subgroups. Here, we utilise our drug screening database to identify targets that significantly correlate and anticorrelate with each subgroup. Further, by screening select drugs that anti-correlate with specific mutant behavioural phenotypes, we identify top rescue drugs.

As a next step, we have created stress paradigms in larval zebrafish that model post-stress behaviours in humans. Combining the post-stress behavioural fingerprints with the behavioural phenotype in zebrafish mutants of PTSD driver genes, we will leverage our pharmaco-behavioural database to identify suppressors of specific post-stress behaviours.

Reference:
Weinschutz et al. (2023). High-throughput functional analysis of autism genes in zebrafish identifies convergence in dopaminergic and neuroimmune pathways. Cell Rep.;42(3):112243.



SLFN11 inhibits alternative lengthening of telomeres (ALT)

PDF 30

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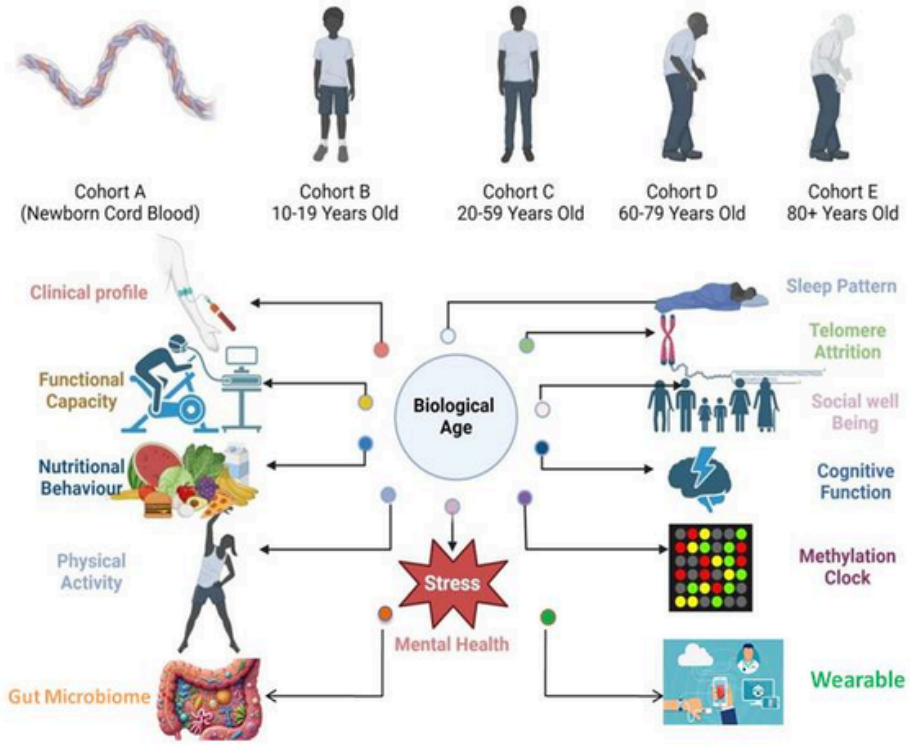
A prospective cohort study to develop multi-biomarkers panel to define biological ageing in six different cohorts from newborn to oldest adult: A study protocol

Abstract

Age-associated disease management depends significantly on chronological age and macro-level clinical data sets. However, the biological age captures bio-physiological deterioration more precisely than the chronological age. Biological ageing is the explicit reflection of functional decline. Therefore, quantifying biological age can be highly valuable for improving clinical management of age-related changes. Various epigenetic clocks have been used to quantify biological age. However, epigenetics alone cannot fully account for the complex ageing process, which involves ageing hallmarks, signalling pathways, clinical phenotypes, physiological functions, environmental exposures, and lifestyle habits. Therefore, the primary purpose of this pilot study is the feasibility testing and trajectory mapping of the ageing biomarkers across diverse age-based subgroups. This study will help to find reliable, reproducible, robust, and integrative ageing biomarkers to quantify biological age. This community-based prospective cohort study will be conducted at the National Centre of Ageing, All India Institute of Medical Sciences, New Delhi. This study will include 250 participants from six cohorts, i.e. newborns, adolescents (10–19 years), young adults (20–39 years), middle-aged individuals (40–59 years), young olds (60–79 years), and the oldest old (above 80 years). Forty individuals from each cohort will be recruited to study blood and stool biomarkers along with a comprehensive assessment of cognitive behaviour, psychological well-being, functional capacity, gut health, nutritional behaviour, and physiological measures. Multidomain data will be integrated to develop a deep learning-based multi-model algorithm for biological age estimation. This first-of-its-kind study would provide an exhaustive understanding of the ageing process throughout life, 0–100 years. Integrative biomarkers would make a precise determination of biological age.



Schematic representation of study plan

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Establishing genetic baseline: High diversity of Taenia solium in Punjab's porcine population

Abstract

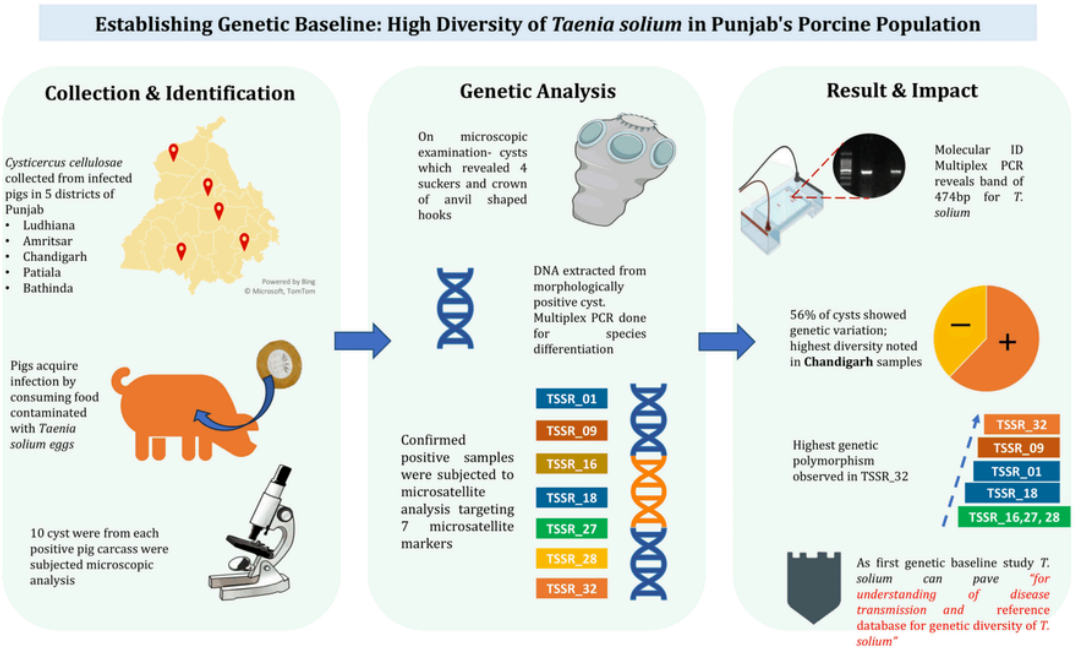
In India, neurocysticercosis (NCC), caused by *Taenia solium*, accounts for up to 30% of all epilepsy cases in endemic areas. Establishing the local genetic landscape is critical to understanding this variation and its public health impact.

The study aimed to establish the first genetic baseline and quantify the degree of genetic heterogeneity in *T. solium* cysticerci from naturally infected pigs in Punjab. Cysticerci were collected from 20 infected pigs. Morphological examination was done using microscopic analysis of the collected cysts from pig carcasses suspected positive for *T. solium* infection. Species identification was done using a multiplex PCR that differentiates *T. solium* from *T. saginata* and *T. asiatica* by amplifying a diagnostic ~474 bp fragment from the mitochondrial Val-tRNA and NADH-2 genes. Genetic analysis of 200 confirmed cysts was then performed using seven microsatellite markers (TSSR_01, TSSR_09, TSSR_16, TSSR_18, TSSR_27, TSSR_28, TSSR_32).

Fifty-six percent (112/200) of cysts showed genetic variations. The markers TSSR_32 and TSSR_09 showed the highest variability, with alterations in 60/200 (30%) and 26/200 (13%) cysts, respectively. Notably, 6% of cysts exhibited double bands, indicating a diverse population of circulating strains. Geographically, the highest rate of variation (70%) was observed in cysts from Chandigarh, indicating a potential hotspot for parasite diversity.

The high prevalence of diverse strains underscores a substantial and ongoing risk for NCC-associated epilepsy in Punjab, highlighting the need for targeted control strategies.

Reference:
Singh S P, Singh B B, Kalambhe D G, Pathak D, Aulakh R S & Dhand N K. (2018). Prevalence & distribution of Taenia solium cysticercosis in naturally infected pigs in Punjab, India. PLoS Neglected Tropical Disease, 12(11).



Schematic of this study

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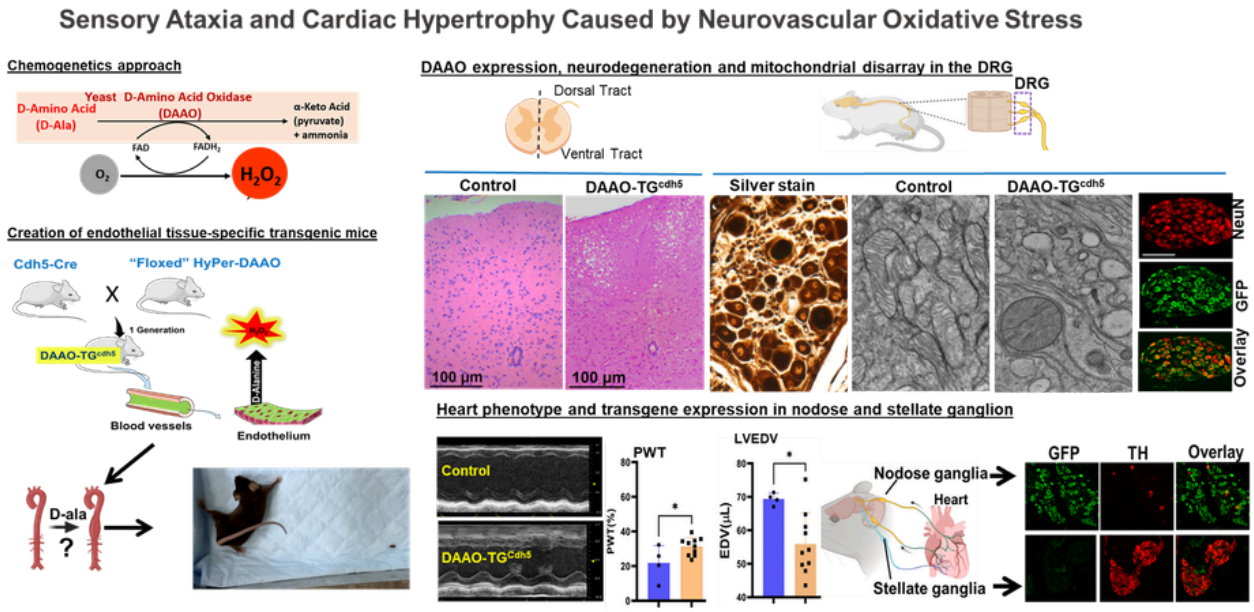
Sensory ataxia and cardiac hypertrophy caused by neurovascular oxidative stress

Abstract

Oxidative stress is associated with cardiovascular and neurodegenerative diseases. Here we report studies of neurovascular oxidative stress in chemogenetic transgenic mouse lines expressing yeast D-amino acid oxidase (DAAO) in neurons and vascular endothelium. When these transgenic mice are fed D-amino acids, DAAO generates hydrogen peroxide in target tissues. DAAO-TGCdh5 transgenic mice express DAAO under the control of the putatively endothelial-specific Cdh5 promoter. When we provide these mice with D-alanine, they rapidly develop sensory ataxia due to oxidative stress and mitochondrial dysfunction in neurons within the dorsal root and nodose ganglia innervating the heart. DAAO-TGCdh5 mice also develop cardiac hypertrophy after chronic chemogenetic oxidative stress. This combination of ataxia, mitochondrial dysfunction, and cardiac hypertrophy is similar to findings in patients with Friedreich's ataxia.

Our observations indicate that neurovascular oxidative stress is sufficient to cause sensory ataxia and cardiac hypertrophy. Studies of DAAO-TGCdh5 mice could provide mechanistic insights into Friedreich's ataxia.



Sensory ataxia and cardiac hypertrophy caused by neurovascular oxidative stress in chemogenetic transgenic mouse lines

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Transcriptional regulation of axon regrowth: NR2F6 and NR2F1 in the peripheral nervous system

Abstract

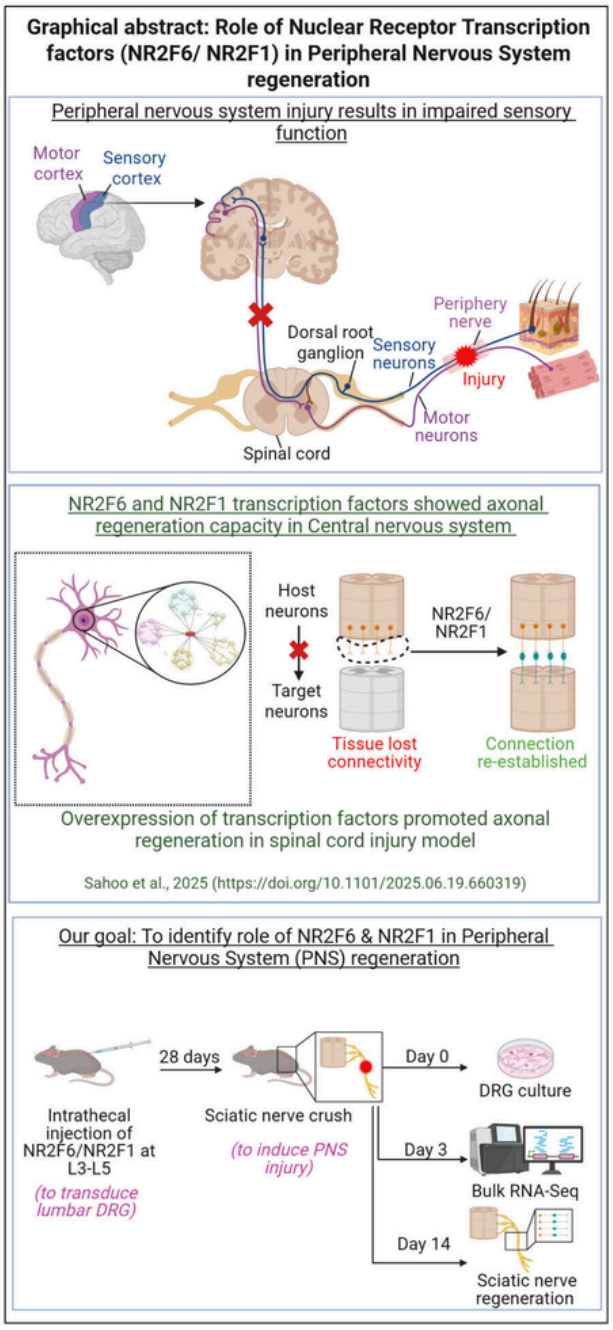
Peripheral nervous system (PNS) neurons possess limited regenerative ability after injury, driven largely by transcription factors that regulate axonal growth programs. Our laboratory recently identified two Nuclear Receptor Transcription Factors (NRTFs), NR2F6 and NR2F1, which significantly enhanced regeneration in central nervous system (CNS) injury models [1]. This raises the possibility that these factors act as general regulators of neuronal repair across systems. The present study aims to test this by examining their effects in the PNS using dorsal root ganglion (DRG) neuron assays and an in vivo sciatic nerve injury model, and to provide insights into their therapeutic potential.

Primary DRG neurons from postnatal adult mice were cultured and transduced with constructs to overexpress NR2F6 and NR2F1, and neurite outgrowth was quantified by immunocytochemistry. In vivo, adult mice received intrathecal AAV-mediated delivery of these genes following sciatic nerve crush injury, and regeneration was assessed through axon tracing and immunohistochemistry.

Our ongoing study has shown that combinatorial gene treatments result in better regeneration than single-gene treatments alone. Simultaneously, Bulk RNA sequencing will be performed on DRG tissues to identify transcriptional changes induced by NR2F6 and NR2F1 modulation.

Ultimately, our study is expected to identify key transcriptional regulators of peripheral nerve regeneration. Targeting these factors may provide novel therapeutic strategies to enhance recovery following nerve injury by modulating the intrinsic growth program of DRG neurons.

Reference:
1. Sahu et al. 2025. Nuclear Receptor Transcription factors promote axon regeneration in the Adult Corticospinal Tract. bioRxiv:2025-06. (Under Review: Nature Communications)



NRTFs promote regeneration in peripheral nervous system

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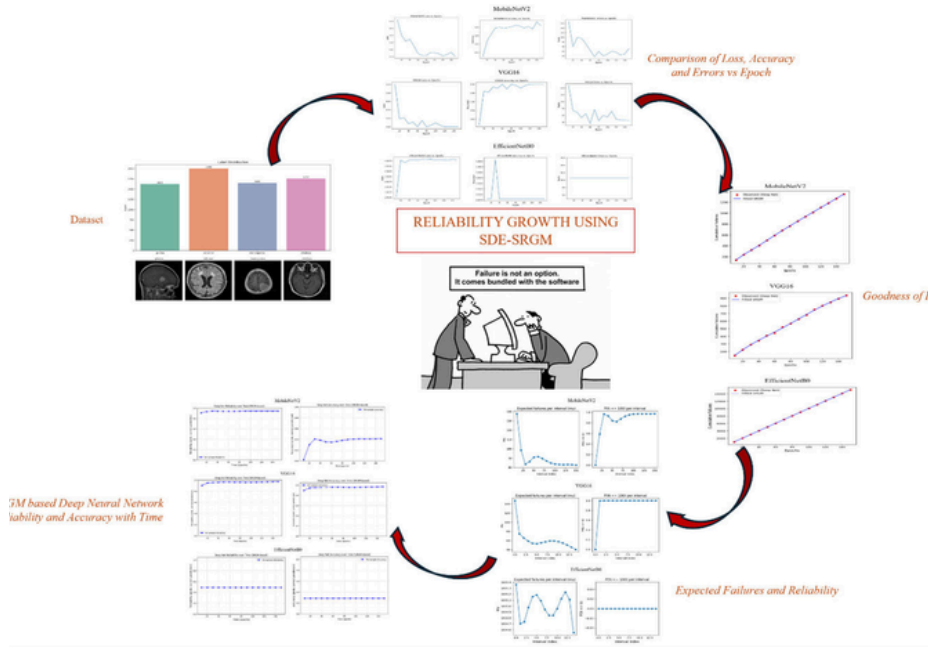
Stochastic differential equations based software reliability growth model for deep Neural networks on medical image data

Abstract

Varied Software Reliability Growth Mathematical Models (SRGMs) are extensively used in the software industry to check the reliability of the software during the testing phase, but they are still not used to check the reliability of the Deep Learning models on medical imaging data as training progresses. The objective is to check the reliability of Deep Learning Models used for classification on medical image data using SDE based SRGM. Inculcating randomness due to random weight initialisation through SDE-SRGM. In our method, misclassification predicted from networks are considered as failures and epochs as time for failure and time data purpose. Faults are segregated as simple, hard and complex based on the efforts to remove the faults. We design our SDE-SRGM on the following assumptions :
The fault detection process is designed as stochastic on continuous space.
As training progresses, faults detected are reduced.
Failure of the Deep Learning model is directly proportional to the remaining faults in the model.

SDE-SRGM :
M(t)=ap_1 [1-(((1+β))/((1+βe^(-b_1 t))))}{e^(-b_1 t+(σ^2 t)/2) }]+ ap_2 [1-(((1+β+b_2 t))/((1+βe^((-b_2 t)))))}{e^((-b_2 t+(σ^2 t)/2)) }]+ ap_3 [1(((1+β+b_3 t+(b_3^2 t)/2))/((1+βe^((-b_3 t)))))}{e^(-b_3 t+(σ^2 t)/2) }]
Notations,
M(t): Number of faults detected during time t
a: Total number of faults
p_(1,) p_2,p_3 : Proportion of simple, hard, and complex faults in total fault content.
b_1,b_2,b_3: Simple, hard, and complex fault detection rate.
β: Learning rate of the Deep Learning model.
σ: Irregular fluctuations.

We have proposed a novel method to check the reliability of deep learning network by tracking the error curve while testing as epochs increased using SDE-SRGM. Model can predict the faults at any epoch level during testing without retraining the network.



NRTFs promote regeneration in peripheral nervous system

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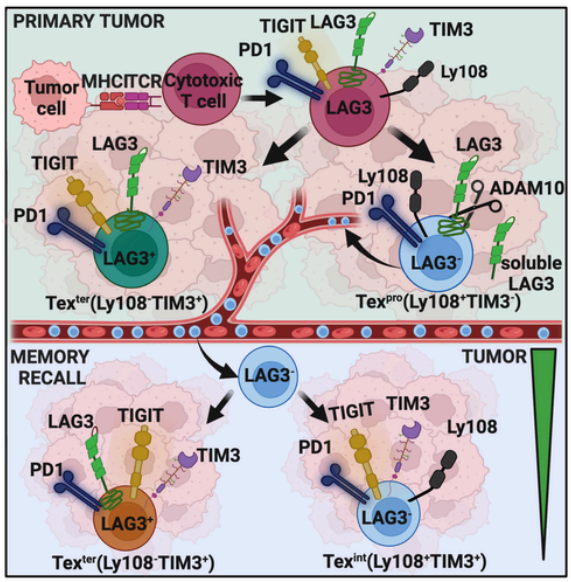
Therapeutically targeting tumour-specific stem-like progenitor exhausted CD8+ T cell subsets diverging into terminally exhausted cells and long-lived memory T cells

Abstract

Chronic T cell stimulation in tumours and chronic viral infections leads to T cell exhaustion (TEX), a state of dysfunction that remains incompletely understood from a therapeutic perspective [1,2]. As the inhibitory receptor LAG3 is expressed by all exhausted T (TEX) cells [1], we hypothesise that defining lineage heterogeneity and plasticity during cancer progression would be insightful in determining the fate and functionality and therapeutically targeting LAG3+ CD8+ T cells.
We generated a novel Lag3 lineage tracing mouse model (Lag3iCreERT2Rosa26LSL-tdTomato) to fate-map and characterise tumour-reactive LAG3+CD8+ TEX cells. We identified two distinct tdTomato+ CD8+ T cell subsets stratified by LAG3 surface expression (LAG3+tdT+ and LAG3–tdT+) that exhibit contrasting anatomical distributions, functionality and transcriptional profiles, yet share a common origin and TEX epigenetic state.3 While LAG3+tdT+CD8+ TEX cells were terminally exhausted and restricted to the tumour microenvironment, LAG3–tdT+CD8+ TEX cells were stem-like progenitors that persisted in vivo and formed the T cell memory pool.

This study highlights TEX cell functional heterogeneity and plasticity, and characterises a unique LAG3–tdT+ stem-like progenitor TEX subset that drives an anti-tumour memory response, of which LAG3+ TEX cells can be therapeutically targeted to unleash anti-tumour immunity and promote durability.

References:
1. Aggarwal V, Workman CJ, & Vignali DAA. 2023. LAG-3 as the third checkpoint inhibitor. Nature Immunology 24: 1415-22.
2. Baessler A, & Vignali DAA. 2024. T cell exhaustion. Annual Review of Immunology 42: 179-206.
3. Aggarwal V, Liu C, Cui J, Wang H, Manne S, Vignali KM, Workman CJ, & Vignali DAA. 2024. Tumor-specific stem-like progenitor CD8+ T cell subsets diverge into terminally exhausted cells and long-lived memory T cells. Immunity (Under revision, JEM 20241968).



LAG3 expression potentiates TEX terminal differentiation and tumour retention, while loss of LAG3 expression enables TEX stem memory-like phenotype

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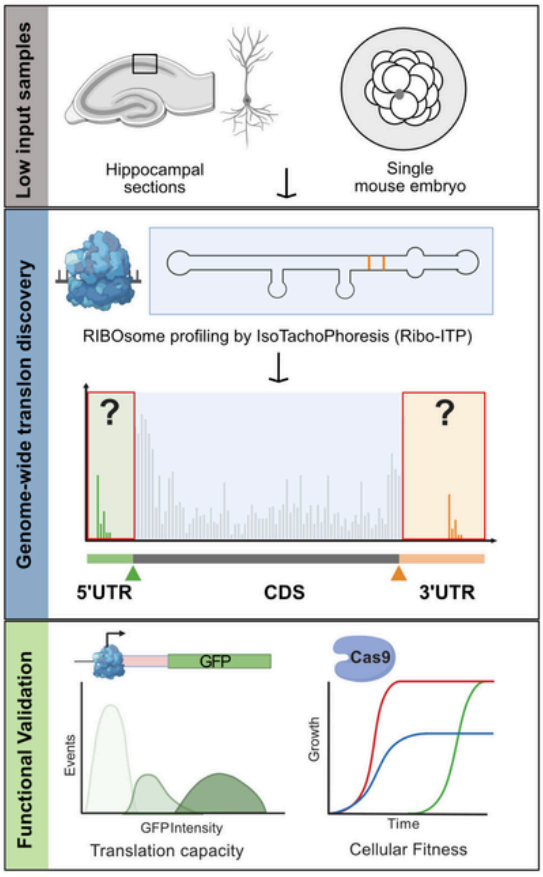
Ribo-ITP expands the translatome of limited input samples

Abstract

In the last decade, an unexpectedly large number of translated regions (translons) have been discovered using ribosome profiling and proteomics. Translons can regulate mRNA translation and encode micropeptides that contribute to multiprotein complex formation, Ca^{2+} regulation in muscle, and signalling during embryonic development. However, identification of translons has been limited to cell lines or large organs due to high input requirements for conventional ribosome profiling and mass spectrometry.

Here, we address this challenge using Ribo-ITP on difficult-to-collect samples, including microdissected hippocampal tissues and single pre-implantation embryos. Comparative analysis of more than 1,000 ribosome profiling datasets across a wide range of cell types revealed distinct sample-specific expression patterns for the detected translons. To test the translational capacity of the identified translons, we engineered a translon-dependent GFP reporter system. We detected expression of translons initiating at near-cognate start codons in mouse embryonic stem cells (mESCs). Mutating the translons in mESCs identified a small proportion that negatively impacted growth.

Taken together, we present a proof-of-concept study to identify non-canonical translation events from low-input samples, which can be applied to cell and tissue types inaccessible to conventional methods.



Novel low-input ribosome profiling technique helps identify thousands of hidden micropeptides which contribute to cellular fitness

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PDF 37

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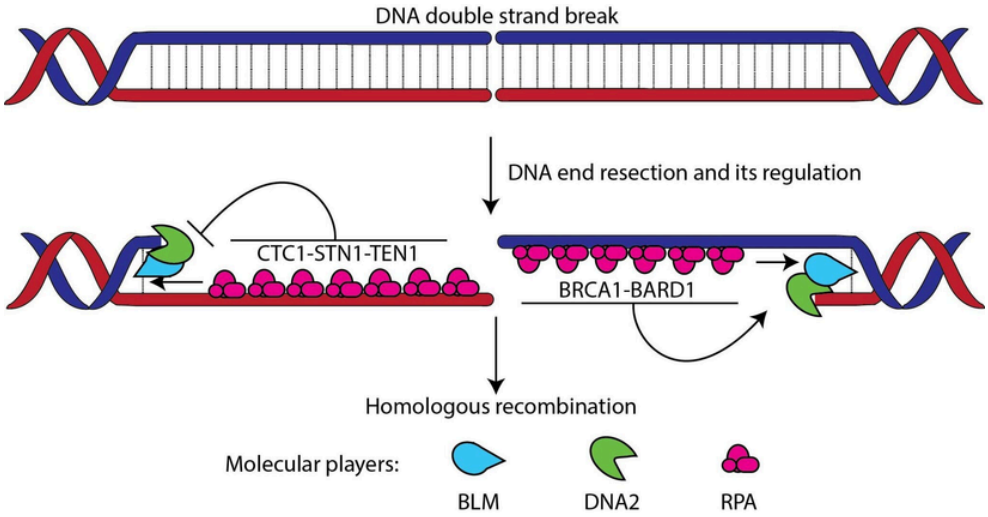
Single-molecule approaches to investigate molecular mechanisms of homologous recombination

Abstract

DNA double-strand breaks (DSBs) are one of the most cytotoxic lesions that can drive genome instability. Homologous recombination (HR) is a high-fidelity DNA repair pathway that cells use to repair DSBs accurately. However, HR must be tightly regulated to ensure physiologically appropriate outcomes.

My research focuses on understanding the molecular mechanisms that govern the balance between promoting and limiting HR. I use total internal reflection microscopy combined with single-molecule DNA curtain assays to reconstitute and directly visualise early steps of HR. Here, I will present how different molecular players, in particular crucial translocases, helicases and nucleases, act at critical steps to differentially regulate HR. As an example, I will present how we demonstrated that Srs2, a yeast HR regulatory protein, requires solely its DNA translocase activity, not its helicase activity, to regulate HR. I will also present how human proteins BLM, a helicase, and DNA2, a helicase/nuclease, combine to mediate DNA end resection, a critical early step in HR. Finally, I will show how BLM-DNA2-mediated DNA end resection is differentially regulated by BRCA1-BARD1 and CTC1-STN1-TEN1 (CST) protein complexes.



Regulation of DNA end resection

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Characterisation of innate immune responses and severity biomarkers in neonatal sepsis in India

Abstract

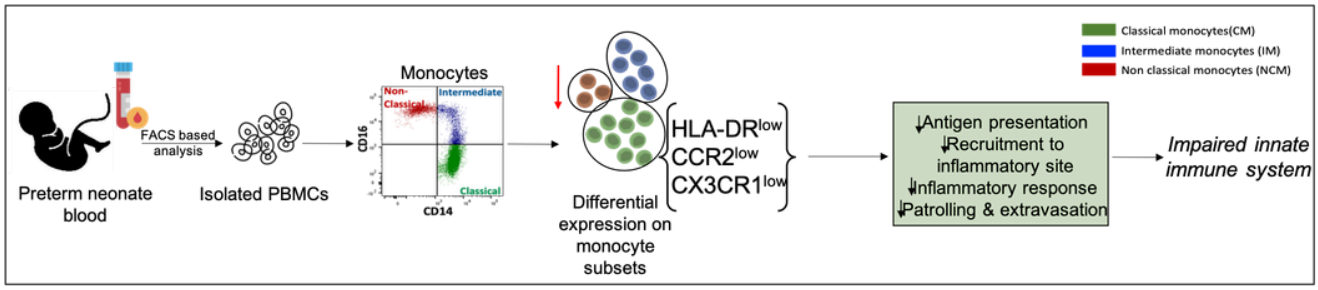
Sepsis is caused by dysregulated innate immune response to infection, leading to organ dysfunction and increased risk of mortality. India accounts for the highest incidence of neonatal sepsis worldwide with up to 65% fatality rate. To address this high disease burden, a multi-disciplinary and multi-institutional program has been established by the Government of India to collaboratively develop solutions to tackle neonatal sepsis in India.

This study forms a part of the consortium focusing on elucidating innate immune responses in preterm neonates. Aberrant production of key immune mediators compromises the neonates' ability to effectively recognise and eliminate pathogens, leading to severe clinical outcomes and high mortality. However, the factors that predispose certain neonates to adverse outcomes while enabling others to recover remain poorly understood.

Therefore, the objectives of this study are - to define the nature of dysregulation in innate immune responses; and to determine whether specific phenotypes are associated with the clinical outcomes observed in sepsis-suspected neonates.

We have collected peripheral blood from over 300 neonates with suspected sepsis and analysed monocytes and their subsets - classical, intermediate, and non-classical using multiparametric flow cytometry. Our findings reveal a significant reduction in the frequency of non-classical monocytes. Interestingly, while HLA-DR expression was found to be downregulated consistent with published literature, it did not distinguish between recovered and deceased preterm neonates. Notably, classical monocytes exhibited marked downregulation of the chemokine receptors CX3CR1 and CCR2, critical for homing, patrolling, extravasation, indicating a possible functional impairment. We are now applying machine learning to identify expression patterns that may serve as early predictors of clinical outcomes in neonatal sepsis.

From this study, we aim to identify innate immune markers associated with adverse outcomes, to enable timely and effective clinical management of neonatal sepsis.



Differential expression of monocyte subsets and their phenotype in preterm neonates

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This abstract book is designed by Moumita Mazumdar, Program Manager - Science Communication, IndiaBioscience