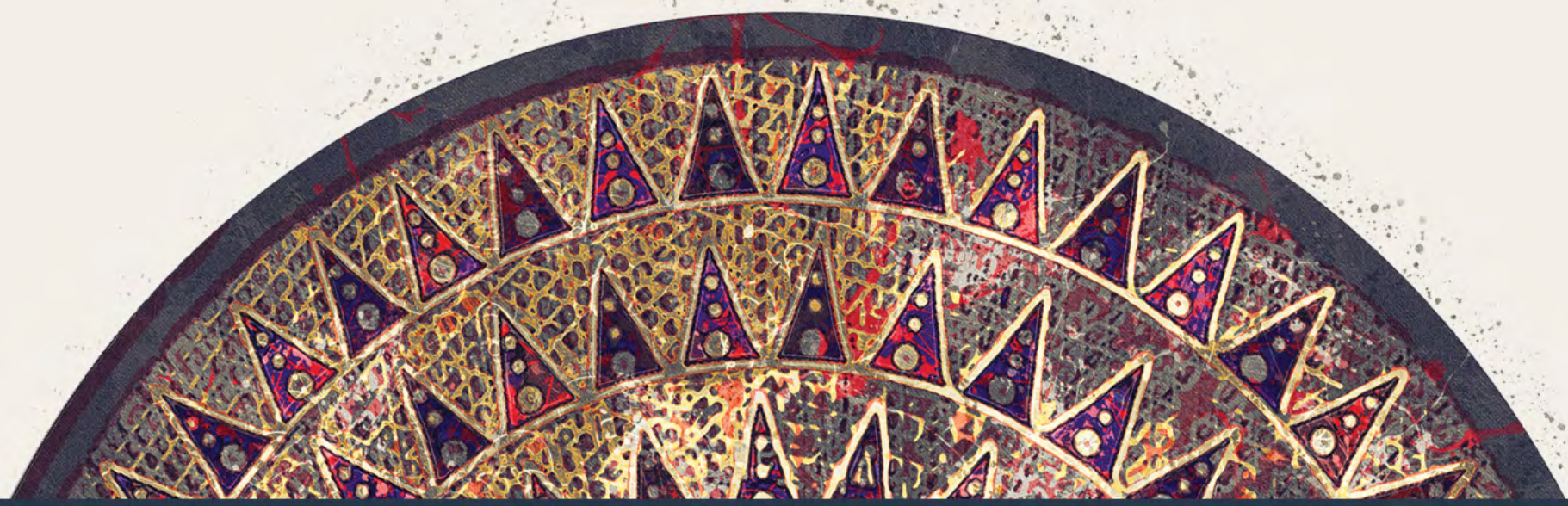


11th Young Investigators' Meeting 2019

IndiaBioscience

भारतीय प्रौद्योगिकी संस्थान गुवाहाटी
Indian Institute of Technology Guwahati

ABSTRACT BOOK

https://indiabioscience.org/meetings/yim-2019

THE YOUNG INVESTIGATORS' MEETING SERIES

Building a community of young Indian Biologists

The YIM series aims to build a community of well-networked biologists by allowing young Indian scientists from different regions and institutions to establish friendships, initiate collaborations, and learn from peer-to-peer mentoring.

The annual Young Investigators' Meeting (YIM) brings together an eclectic mix of India's best early-career life science researchers, postdoctoral fellows, reputed Indian and international scientists, administrators of institutions, funding agency representatives and science policy makers for 5 days of discussions and interactions focusing on science and careers in science.

The program features talks, posters and panel discussions where participants examine a wide variety of topics ranging from picking research problems, publishing, personnel management and mentorship. Senior scientists describe their own scientific journeys providing inspirational as well as amusing anecdotes about their experiences on of starting their scientific careers. Interactions between young Indian investigators and postdocs aspiring for jobs in India ensures that the postdocs get a first-hand account of finding jobs and setting up labs in India. Experiences shared by senior scientists enable young Indian investigators and future recruits to imbibe the scientific ethos in India and further them towards making the Indian life sciences sector internationally competitive.

Perhaps the greatest accomplishment of the YIM series is building a future community of well-networked biologists, allowing young Indian scientists from different regions and institutions to establish friendships, initiate collaborations, and learn from peer-to- peer mentoring.

Every year the YIM is organised by a different committee, comprised of young faculty members from institutions across the country. IndiaBioscience plays an administrative and advisory role in each year's YIM. YIM 2019 is the eleventh in a series that began in 2009, and has grown in popularity, size and content since then.

TABLE OF CONTENTS

YIM 2019 ORGANISERS

Dipyaman Ganguly is an immunologist who started his independent lab in CSIR-IICB six years back. Dipyaman was trained as a clinician and then moved to biomedical research. He had his first PhD (Biotechnology) from CSIR-IICB in 2006, had his second PhD (Immunology) from UT MD Anderson Cancer Center, USA, in 2010 and after his postdoctoral stint in Columbia University, USA, returned to India in 2013. His research interests are: role of dendritic cells in autoreactive inflammatory contexts, molecular regulation of innate immune response and role of mechanical cues in immune cells. Dipyaman is a recipient of National Bioscience Award (DBT, 2018), Swarnajayanti Fellowship (DST, 2017), NASI Scopus Young Scientist Award (2017) and Ramanujan Fellowship (2011).

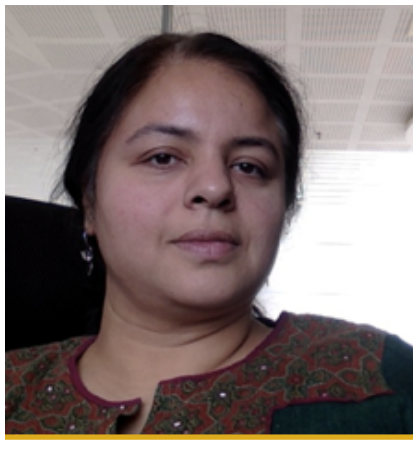
Dipyaman Ganguly
Indian Institute of Chemical Biology, Kolkata
http://iicb.res.in/dipyaman-ganguly/

Smita has a PhD from the Indian Institute of Science, Bangalore in the field of Cancer Biology. After exploring industry for couple of years, she moved into the field of scientific management. With her keen interest in management, ability to communicate, she played a key role in establishing the business and processes at C-CAMP, Bangalore. She also has experience of working as a research analyst with a digital content organization. She is deeply motivated to take the activities of IndiaBioscience to all possible corners of the country and make a strong knit network of Indian life science researchers and professionals.

Smita Jain
IndiaBioscience, Bangalore
https://indiabioscience.org/

YIM 2019 ORGANISERS

Richa Rikhy did her PhD from the Department of Biological Sciences at TIFR, Mumbai, India under the guidance of K.S. Krishnan. Subsequently she pursued her postdoctoral studies under the guidance of Jennifer Lippincott-Schwartz at the National Institutes of Health, Bethesda, USA. She is a faculty at the Indian Institute of Science Education and Research at Pune, India. Her lab uses *Drosophila* as a model system to understand questions in the area of morphogenesis and differentiation.



Richa Rikhy
Indian Institute of Science Education and Research, Pune
http://www.iiserpune.ac.in/~richa/Richa_Rikhy/Home.html

Anand is currently working as an associate professor at IIT, Guwahati. He holds a B.Tech in Industrial Biotechnology from Anna University, Chennai and a PhD in Structural and Computational Biology from IIT, Kanpur. In 2010, shortly after his PhD, he took up a faculty appointment at IIT, Guwahati and has remained there ever since. His group is fascinated by uncovering the underlying mechanism of RNA centric biological processes by employing an eclectic mix of cutting-edge computational and experimental approaches. Their current obsession is to resolve the mechanistic aspects of Ribosome assembly and CRISPR-Cas system. As a home-grown independent researcher, he takes pleasure in teaching as much as research, which continues to inspire him to unravel the mysteries of Nature and discover the eureka moments from a different perspective.

B. Anand
Indian Institute of Technology, Guwahati
http://www.iitg.ac.in/banand/

IndiaBioscience

ENGAGING COMMUNITIES.ENABLING CHANGE

From left to right: *Manoj, Manjula, Shwetha, Lakshmi, Tejeswini, Navodita, Shreya, Smita*

IndiaBioscience is an organization that fills a unique niche in the ecosystem of the life sciences in India, by being a catalyst to promote changes that affect 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 being a hub for policy discussions, science communication, and as an aggregator of information.

SUPPORTING INSTITUTIONS

Department of Biotechnology

Department of BioTechnology, Government of India
सत्यमेव जयते

This Department, set up in 1986, gave a new impetus to the development of the field of modern biology and biotechnology in India. In more than a decade of its existence, the department has promoted and accelerated the pace of development of biotechnology in the country. DBT is the largest supporter of both YIM and IndiaBioscience.

Wellcome Trust/ DBT India Alliance

IndiaAlliance
DBT wellcome

The Wellcome Trust/DBT India Alliance is an initiative funded equally by The Wellcome Trust, UK and Department of Biotechnology, India. The broad aim of the India Alliance is to build excellence in the Indian biomedical scientific community by supporting future leaders in the field.

CSIR – Indian Institute Of Chemical Biology

CSIR-Indian Institute of Chemical Biology

CSIR-IICB was established in 1935 as the first non-official centre in India for biomedical research and was included within the aegis of CSIR in 1956. CSIR-IICB today is engaged in research on diseases of national importance and biological problems of global interest.

Indian Institute of Technology (IIT), Guwahati

भारतीय प्रौद्योगिकी संस्थान गुवाहाटी
Indian Institute of Technology Guwahati

IIT Guwahati was established in 1994, with its academic programmes commencing in 1995. At present the Institute has eleven departments and five inter-disciplinary academic centres covering all the major engineering, science and humanities disciplines.

Thermo Fisher Scientific

Thermo SCIENTIFIC

Thermo Fisher Scientific is a biotechnology product development company created in 2006 by the merger of Thermo Electron and Fisher Scientific. Through its brands - Thermo Scientific, Applied Biosystems, Invitrogen, Fisher Scientific and Unity Lab Services – it provides a number of innovative technologies and research solutions.

Springer Nature

SPRINGER NATURE

Springer Nature is an academic publishing company, formed in 2015 by the merger of Nature Publishing Group, Palgrave Macmillan and Macmillan Education (held by Holtzbrinck Publishing Group) with Springer Science+Business Media. Its activities include publishing several leading scientific journals and books as well as maintaining databases and publishing platforms.

YIM 2019 SCHEDULE

Day 1 | *6th March 2019*

Young Investigators' Meeting 2019

Incharge: B Anand

12.00 - 14.00	Lunch
14.00 - 14.15	**Welcome Note** B Anand, IIT Guwahati
14.15 - 14.45	**Talk** by Ron Vale, UCSF, USA
14.45 - 15.15	**Keynote Address** Renu Swarup, Secretary, Department of Biotechnology, Government of India, New Delhi
15:15 - 15.45	**Mentor Talk 1** *Meandering of an enzymologist through genomes, methylomes and acetylomes* DN Rao, IISc, Bangalore, India
15.45 - 16.15	Tea/Coffee
16.15 - 16.45	**Mentor Talk 2** *Finding a research niche: digging deeper and digging elsewhere* Doug Koshland, Univ of California, Berkeley, CA USA
16.45 - 18.15	**Panel Discussion 1: Direction of Indian research, Collaborations and Publishing in Science** Moderator: Dipyaman Ganguly Panelists: Dominique Bergmann, Doug Koshland , Usha Vijayraghavan, B Ravindran, DN Rao, Satyajit Mayor
18.15 – 19.45	**YI Poster session** (YIs with even numbers put up the posters)
20.00 onwards	Dinner on the lawn

Day 2 | *7th March 2019*

Young Investigators' Meeting 2019

Incharge: Richa Rikhy

09.00 - 09.15	Talk by LS Shashidhara, IISER, Pune
09.15 - 09.30	Talk on 'IndiaBioscience' by Smita Jain, IndiaBioscience, Bangalore, India
09.30 - 10.00	**Mentor Talk 3** *Making a difference: stem cells, asymmetries and stomata* Dominique Bergmann, Stanford University, CA, USA
10.00 - 10.30	**Mentor Talk 4** *Experimental Ecology and Evolutionary Biology in the Laboratory* Amitabh Joshi, JNCASR, Bangalore, India
10.30 - 11.00	Tea/Coffee
11.00 - 11.30	**Mentor Talk 5** *Accidental Translation of Science* B. Ravindran, ILS, Bhubaneshwar, India
11.30 - 13.00	**Panel Discussion 2: Funding for Science in India** Moderator: B Anand Panelists: Funders: Shahid Jameel, IA; Srini V. Kaveri, CNRS; Vaishali Punjabi DBT Researchers: LS Shashidhara, IISER Pune; BK Thelma, DU; Rashna Bhandari, CDFD
13.00 - 13.45	Lunch
13.45 - 15.00	**Break Out Session 1** Setting up labs and lab management
15.00 - 16.30	**YI Poster Session** with Tea/Coffee (YIs with odd numbers put up the poster)
16.30 - 17.00	**Mentor Talk 6** *Retracing the steps of my scientific pursuit* BK Thelma, Delhi University, New Delhi, India
17.00 - 17.30	**Outreach in science, a special session: Fun of doing Science** Arvind Gupta, IUCAA, Pune
17.30 - 19.00	Transport to River Cruise
19.00 onwards	Dinner on the Cruise

DAY 3 | *8th March 2019*

Young Investigators' Meeting 2019

Incharge: Dipyaman Ganguly

09.30 - 10.00	**Mentor Talk 7** *Codes for the making of a rice flowering stem* Usha Vijayraghavan, IISc, Bangalore, India
10.00 - 11.15	**Panel Discussion 3: Science communication** Panelists: Vasudevan Mukunth, The Wire; Aashima Dogra, The Life of Science; Sarah Iqbal, India Alliance; Leslee Lazar, IIT Gandhinagar; Chitra Ravi, APU; Amitabh Joshi, JNCASR
11.15 - 11.45	Tea/Coffee
11.45 - 12.15	**Mentor Talk 8** *From immune cell development to function and back* Boris Reizis, NYU, NY, USA
12.15 - 12.45	**Mentor Talk 9** *After the malaria parasite turned out to be a plant* Saman Habib, CDRI, Lucknow, India
12.45 - 13.45	Lunch
13.45 - 15.00	**Panel Discussion 4: Mentoring** Moderator: Rashna Bhandari Panelists: Roop Mallik, Saman Habib, Boris Reizis + 3 YIs
15.00 - 17.00	**PDF Poster Session** with Tea/Coffee
17.00 - 18.00	**Session on Women in Science: Institutional Diversity, Women and Science in India and discussion coordinated** by Anitha Kurup
18.00 - 18.15	Closing remarks by Satyajit Mayor, NCBS, Bangalore, India
18.15 - 19.30	Free time/ YI + PDF Private session
19.30 onwards	Banquet Dinner on the lawn

DAY 4 | *9th March*

PDF Satellite Meeting

09.00 - 09.10	**Introduction to The PDF Satellite Meeting** Rashna Bhandari
09.10 - 09.45	**Keynote Talk** K Vijayraghavan, PSA, Government of India
09.45- 10.45	**Institutional Talks (Session 1)** Apurva Sarin, inStem; Gautam Biswas, IIT Guwahati; Santanu Chaudhury, IIT Jodhpur; LS Shashidhara, IISER Pune and Ashoka University; Santanu Bhattacharya, IACS
10.45 - 11.15	Tea/Coffee
11.15 - 12.05	**PDF Talks Session 1** (10 PDFs talk for 5 mins each)
12.05 - 12.55	**Institutional Talks (Session 2)** Roop Mallik, TIFR - Mumbai; Samit Chattopadhyay, IICB; Rajaram Nityananda, Azim Premji University; Gopal C. Kundu, NCCS; Kandala V Chary, IISER Behrampur
12.55 - 13.45	Lunch
13.45 - 14.35	**PDF Talks Session 2** (10 PDFs talk for 5 mins each)
14.35 - 15.30	**Institutional Talks (Session 3)** Debashis Mitra, CDFD; Satyajit Mayor, NCBS; Sujoy Kumar Das Gupta , Bose Institute; Vivek Tanavde, Ahemadabad University; BJ Rao, IISER Tirupathi
15.30 - 17.00	**PDF Poster Session** with Tea/Coffee
17.00 - 17.50	**PDF Talks Session 3** (10 PDFs talk for 5 mins each)
18.00 -19.00	**PDF-Institutional Heads Open Session** Moderated by Roop Mallik/Rashna Bhandari
19.30 onwards	Dinner and informal PDF-Director discussions

DAY 5 | *10th March*

PDF Satellite Meeting

09.00 - 10.00	**Institutional Talks (Session 4)** Dinabandhu Sahoo, IBSD; Sourav Pal, IISER Kolkata; Ragini Singh, Amity University; N.C Talukdar, IASST; Abhijit Chakrabarti, SINP; Sharmila Sengupta, NIBMG
10.00 – 10.50	**PDF Session 4** (10 PDFs talk for 5 mins each)
10.50 - 11.20	Tea/Coffee
11.20 - 12.00	**Institutional Talks (Session 5)** M Radhakrishna Pillai, RGCB; Satyamoorthy K, ICAS - MAHE; BK Mishra, IIT Roorkee; Tushar Vaidya, CCMB
12.00 - 12.30	Closing remarks to the PDF Satellite Meeting by Roop Mallik and open discussion
12.30 onwards	Interaction with Institutional Heads, lunch and departure

LIST OF DIRECTORS AND INSTITUTE 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 2019.

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director@rgcb.res.in

Abhijit Chakrabarti
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Roop Mallik
Tata Institute of Fundamental Research (TIFR), Mumbai
roop@tifr.res.in

Enabling Biomedical Research in India through Funding and Engagement

The Wellcome Trust/DBT India Alliance (India Alliance) is a public charity dedicated to building excellence in biomedical science by identifying the best researchers and nurturing them as future leaders in basic, clinical, and public health research. India Alliance is funded equally by the Department of Biotechnology (DBT), Government of India and Wellcome Trust, UK.

Fellowships in Biomedical Science

India Alliance envisions improving the research ecosystem of India to drive health innovations and inspire the next generation of researchers.

With a commitment to catalysing internationally competitive research, India Alliance has successfully steered three types of fellowship programs to support researchers at different stages of their career —**Early Career, Intermediate,** and **Senior**—under the tracks of **Basic Biomedical Research** and. **Clinical and Public Health Research.** Since its inception in 2009, the fellowship programme has awarded 320 fellowships at 93 different institutions in 34 Indian cities (data updated till May 2018). The focus is on funding the best people early in their careers and set them on a leadership track through a continuous system of engagement and mentoring.

Capacity Building for Research

Science Communication Workshops

Communication skills are important in research. India Alliance has developed and successfully conducted Science Communication Workshops in three formats: **Pan-India SciComm** (a two-day workshop in which participation is based on a pan-India competition), **SciComm 101** (a one-day workshop held at institutions on request), and **Science Communication and Career workshop** (a one-day workshop conducted in partnership with Nature India and Nature Careers at major scientific meetings). These workshops have trained approximately 2500 young researchers from about 100 institutions to date. India Alliance has collaborated with Nature India to organize **Visualising Science**, a two-day workshop that introduced scientists to visual tools and methods to communicate their research more effectively

Research Leadership Workshops

Scientists manage people and projects; this makes leadership skills critical to a successful career. India Alliance organizes **Research Leadership** workshops for its fellows and other young Indian researchers to help them recognize and cultivate their leadership style and develop management skills.

Developing Indian Physician Scientists (DIPS) Workshops

Developing Indian Physician Scientists (DIPS) workshops, launched in 2017, are designed to encourage young physicians to participate in research by exposure to the scientific method and inspirational role models. To date, 97 young clinicians have been trained in three workshops.

Opportunities for Interdisciplinary and International Collaborations

Finding solutions to the problems of modern society requires **interdisciplinary** and **collaborative science.** India Alliance funds major scientific events, including the **Young Investigators Meetings**, to provide young scientists the right platform to meet researchers from India and across the world, to discuss ideas and to forge interdisciplinary collaborations.

India | EMBO Symposia

India Alliance and the European Molecular Biology Organization (EMBO) co-fund up to three meetings per year in India. These meetings are designed to allow interaction of early to mid-career researchers with leading international experts. Global challenges in the context of the life sciences and driving discovery and innovations using interdisciplinary science are the focal points of these meetings. Since its launch in 2016, this initiative has supported seven scientific meetings.

Africa-India Mobility Fund

India Alliance, in partnership with the **African Academy of Sciences**, launched the **Africa-India Mobility Fund** (AIMF) in 2018. AIMF is a two-year programme designed to provide researchers from Africa and India opportunities for short visits in either direction to build and strengthen scientific collaborations. In recognition of the fact that Africa and India face similar health challenges, the AIMF initiative intends to encourage South-South collaborations, improve research capacity, and build leadership in biomedical research in India and Africa.

Strengthening Research Ecosystems in India

India Research Management Initiative

India currently lacks a well-developed research management system, which is important for institutions to navigate the high demands for funding, outreach, and governance of research. To address this lacuna, India Alliance launched the India Research Management Initiative (IRMI), a Research Management programme for India, which aims to strengthen institutional ecosystems. IRMI will also provide opportunities to Indian research managers to receive training and create a network of practitioners for serving broader career development needs.

Making Science Accessible

Open Research

Open research ensures the unbiased, instantaneous, and unhindered flow of knowledge produced by researchers, thereby promoting communication and collaborations. To keep all of India Alliance-funded research openly accessible, India Alliance joined Wellcome Open Research and Europe PMC in 2017-2018. Adoption of the open research policy is bound to improve the relationships between various stakeholders, including that between researchers and society.

Public Engagement

India Alliance aims to bridge the gap between science and society through public engagement programmes that are designed to facilitate a dialogue. Since the launch of its public engagement initiative in 2012, India Alliance actively organizes events that bring the scientific community and public together to share, debate, and deliberate on important matters of science, especially human health, which have implications for the society. Additionally, India Alliance Fellows are encouraged to undertake public engagement activities.

[redacted]rmation on India Alliance's programs and its latest initiatives visit www.indiaalliance.org.

Follow India Alliance's 10-year journey at **10years.indiaalliance.org.**

CELEBRATING 10 YEARS

IndiaAlliance
DBT wellcome

www.indiaalliance.org | Twitter @India_Alliance | Facebook @IndiaAlliance

IndiaAlliance
DBT wellcome

The Wellcome Trust/DBT India Alliance

We are a public charity dedicated to building excellence in biomedical science in India

Vision: An internationally competent research ecosystem in India
Mission: To enable biomedical research in India through funding and engagement

Objectives:
Empower researchers to be future leaders and internationally competitive
Bridge gaps in the Indian research ecosystem by designing interventions
Facilitate engagement of science with the society
Strive for excellence by encouraging diversity, inclusivity, and transparency

Funders:
Wellcome Trust, United Kingdom
Department of Biotechnology, Government of India

SPHERE OF ACTION

- BIOMEDICAL RESEARCH FUNDING
- LEADERSHIP TRAINING
- CAPACITY BUILDING
- INTERNATIONAL COLLABORATIONS
- SCIENCE COMMUNICATION
- PUBLIC ENGAGEMENT
- SCIENCE-POLICY ADVOCACY

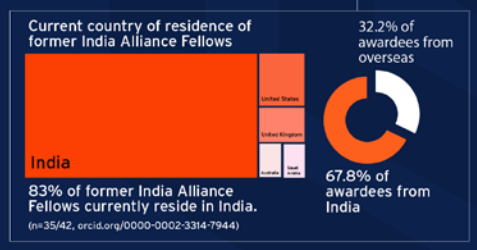
PAN-INDIA REACH

320 FELLOWSHIPS
93 INSTITUTIONS
34 CITIES

10 YEARS OF IMPACT

1 FIRST SCIENCE-FUNDING AGENCY IN INDIA WITH AN ONLINE GRANT APPLICATION PLATFORM–IASYS–TO ENSURE EFFICIENCY AND TRANSPARENCY

2 FACILITATING "BRAIN GAIN" BY ENCOURAGING SCIENTISTS TO MOVE BACK AND STAY IN INDIA



3 IMPROVING SCIENTIFIC BREADTH IN INDIA BY SUPPORTING SCIENTISTS SET UP "CENTRES OF EXCELLENCE" TO DEVELOP NEW RESEARCH AVENUES

4 RELATIVE CITATION RATIO OF INDIA ALLIANCE-FUNDED PUBLICATIONS SIGNIFICANTLY HIGHER THAN THAT OF NATIONAL COMPARATORS
(ORCID.ORG/0000-0002-3314-7944)

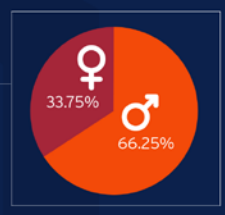
5 WORLD-CLASS ACCOLADES FOR INDIA ALLIANCE FELLOWS IN RECOGNITION OF THEIR OUTSTANDING RESEARCH WITH GLOBAL IMPACT

6 CAPACITY DEVELOPMENT BY IMPROVING SKILL-SET OF YOUNG INDIAN RESEARCHERS THROUGH TRAINING

Science Communication Workshops: ~2500 researchers
PhDs, Postdocs, Undergraduates, and Research Technicians (in labs of India Alliance Fellows): >900
Developing Indian Physician Scientists: 97 physicians
Research Leadership Workshop: ~60 researchers
Research Methodology Workshop: 36 researchers

7 FOSTERING INTERDISCIPLINARY AND COLLABORATIVE SCIENCE BY FUNDING MAJOR SCIENTIFIC EVENTS AND FACILITATING INTERNATIONAL MOBILITY

8 MEETING GENDER CHALLENGES IN SCIENCE BY ADOPTING POLICY CHANGES
One-year full-cost extension to Fellows on maternity leave during their term
Consideration of non-research career breaks, including maternity breaks, for eligibility checks



9 MAKING SCIENCE ACCESSIBLE FOR ALL THROUGH OPEN RESEARCH AND PUBLIC ENGAGEMENT

10 MOVING TOWARDS BETTER RESEARCH ASSESSMENT PRACTICES: INDIA ALLIANCE ONE OF THE TWO INDIAN SIGNATORIES OF DORA

KEYNOTE SPEAKERS

K VijayRaghavan
Principal Scientific Advisor to the
Government of India, New Delhi

Renu Swarup
Secretary, Department of Biotechnology,
Government of India, New Delhi

SPECIAL LECTURES

Anitha Kurup is a Professor and Dean, School of Social Sciences and Head of the Education Programme at National Institute of Advanced Studies (NIAS), Bangalore. She leads the National Gifted and Talented Education Program in India anchored at NIAS. Her research interests span the broad disciplines of education and gender studies. She was awarded the Fulbright Nehru Senior Research Fellowship for the year 2011-2012. Her publications "Trained Scientific Women Power: How much are we Losing and Why?" and "Trends Report: Creation and Analysis of Database of PhDs in India (1998-2007)" have been widely appreciated.

Anitha Kurup
National Institute of Advanced Studies (NIAS), Bangalore
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INSTITUTIONAL DIVERSITY, WOMEN AND SCIENCE IN INDIA

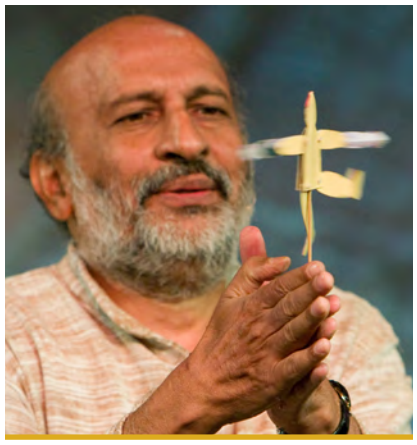
The challenges of women in science and engineering in India stand distinct from that of the West and other developed countries. Post-Independence, India has documented the glaring gender gaps spread across educational levels. India has made systematic interventions to close this gap; however, the progress made thus far further points to anomalies that have drawn the attention of researchers. India has adopted a two way strategy to promote formal education in the country. On one hand, it ensured that access to formal education was provided to all across caste, class and gender. At the same time the country also promoted science and technology in an attempt to catch up with the West. This dual policy had an interesting impact on how gender gaps played out across the years.

India presented a unique challenge, as the problem for Indian science is not increasing participation, rather it is on retaining women in science in ways which these numbers translate to visible presence of women in diverse formal work spaces. Hence there is a need to shift focus to institutional spaces and interrogate the diverse ways in which women respond to challenges in the workspace. Important dimensions of work life balance, changing caste, class & gender relations, growing number of women in these institutions, leadership styles and the history of the institutions also seem to interact in interesting ways making the field of study complex and unique. How one unfolds these complexities and attempts to provide possible explanations will be focus of the presentation.

Talk Schedule: **8th March, 17:00 – 18:00**

SPECIAL LECTURES

Arvind Gupta did a B. Tech from IIT, Kanpur (1975). He left a corporate job to pursue his passion - making science fun for children. He has written 30 books on science activities, translated 400 books into Hindi and presented 125 films on science activities on *Doordarshan*. He worked at the Children's Science Center at IUCAA, Pune for 11 years. The Centre produced 8600 short (2-minute) videos on simple experiments and science toys in 20 different languages. The videos have been viewed by over 70-million children worldwide. Every day over 12,000 books are downloaded from his popular website http://arvindguptatoys.com . He has received many honors including the Distinguished Alumnus Award of IIT/Kanpur and the Padma Shri.



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FUN OF DOING SCIENCE

Before children can understand a thing, they need experience: seeing, touching, hearing, tasting, smelling, choosing, arranging, putting things together, taking things apart. Children need to experiment with real things available in their milieu. In India most science is learnt by rote – by mugging up definitions and formulae and spitting them out in the exams. Very few children dirty their hands on making simple models. The Government of India launched the Make in India campaign two years back. They soon realized that the slogan will become a reality only when children start learning science in schools through actual experiments and projects. Now the Government of India is opening hundreds of Atal Tinkering Labs in schools.

Many science models can be made from throwaway stuff to make science fun for children. They include simple pumps, electric motors and generators. Children love toys which spin, fly, move and make sound. These toys are made with simple materials often with discarded stuff – old plastic bottles, tetrapaks, newspapers, straws, old pens etc. This lecture-demonstration will show the possibilities of using simple things to make science fun, so that the poorest children in the world can have fun.

Talk Schedule: **7th March, 17:00 - 17:30**

PANELISTS FOR PANEL DISCUSSION 1

DIRECTIONS OF INDIAN RESEARCH, COLLABORATIONS,

PUBLISHING IN SCIENCE

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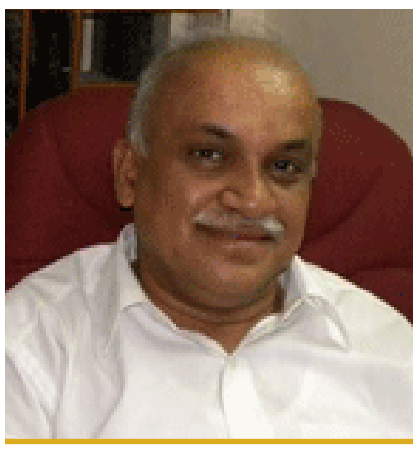

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YIM MENTOR ABSTRACTS

M01	Amitabh Joshi	Experimental ecology and evolutionary biology in the laboratory
M02	B.K. Thelma	Retracing the steps of my scientific pursuit
M03	B. Ravindran	Nitric oxide biology: accidental translation of science
M04	Boris Reizis	From immune cell development to function and back
M05	D. Narasimha Rao	Meandering of an enzymologist through genomes, methylomes and acetylomes
M06	Dominique Bergmann	Making a difference: stem cells, asymmetries and stomata
M07	Douglas Koshland	Finding a research niche: digging deeper and digging elsewhere
M08	Saman Habib	After the malaria parasite turned out to be a plant
M09	Usha Vijayraghavan	Codes for the making of a rice flowering stem

EXPERIMENTAL ECOLOGY AND EVOLUTIONARY BIOLOGY IN THE LABORATORY

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In 1996, I joined JNCASR Bengaluru, and established a lab to investigate fundamental issues in population ecology and evolutionary biology using fruitflies as a model system. I and my students and postdocs have primarily been looking at three broad, and somewhat intertwined, issues, using both experimental and theoretical methods. The first is the relationship between developmental rates and competitive ability. The second is the role of ecology in mediating the evolution of increased competitive ability through varying sets of underlying traits, and its implications for whether or not increased competitive ability is accompanied by enhanced population stability. The third is the interplay of migration rates and local dynamics in determining the dynamics and stability of spatially structured and unstructured populations, as well as the evolution of population stability. In addition, in collaboration with colleagues at JNC, and in IISER Pune and IISER Mohali, I also work on the reconceptualization of some of the foundational premises of evolutionary theory, as part of the Foundations of Genetics and Evolution Group (FOGEG). The kind of work I have been engaged in was almost non-existent in India prior to 1996. I will briefly describe some vignettes from this body of work, and use them as a scaffold around which to make some more general observations about the problems of doing science, especially evolution and ecology research, in India.

RETRACING THE STEPS OF MY SCIENTIFIC PURSUIT

Genetics and genomics in medical sciences have witnessed considerable change in utilised technology and hence in the outcome of global research efforts. Unravelling the chromosomal basis of a proportion of human genetic disorders was only the beginning; identification of disease causal genes for a small proportion of single gene disorders using recombinant DNA tools marked the next phase. The Human Genome Project at the end of the last century, with the discovery of common variants across the genome dramatically changed the scenario, paving the way for big data and hypothesis free genome-wide association studies for the challenging group of complex genetic disorders. However, due to limited insights from this approach, paradigm in complex trait genetics changed from common disease common variant to common disease rare variant search; which is greatly facilitated by next generation sequencing. While the utility of this new tool remains to be seen for complex traits, it has enabled unparalleled success in discovery genomics for single gene disorders and associated aspects edging into translational medicine. This journey, as well as the need for alternate paradigms such as Ayurgenomics for complex traits will be presented taking examples from our work over the years.

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NITRIC OXIDE BIOLOGY: ACCIDENTAL TRANSLATION OF SCIENCE

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Nitric oxide performs a wide variety of functions in cardiovascular, immune and nervous systems and Nitric oxide synthase interacting protein (NOSIP), a highly conserved protein regulates Nitric Oxide production by Nitric oxide synthases (NOS). A novel polymorphism (deletion of a four bp sequence in the non-coding region) discovered by us in human NOSIP gene determined its expression and consequently influenced NO produced by human monocytes. The polymorphism significantly influenced progression of severe disease and consequently adverse prognosis in human sepsis and Dengue infections. Recognition of NOSIP as an important regulator of inflammation by virtue of its ability to influence nitric oxide production thus opened up avenues for development of therapeutic strategies against acute inflammation. Assays for quantifying interaction between cell free NOSIP and NOS allowed us to recognize inhibitory peptide sequences blocking such interactions and consequently led to identification of 'small molecule peptide analogues' for regulation of NOSIP-NOS interactions and Nitric Oxide production in vivo. Identifying druggable molecules for regulation of inflammation was not in our agenda when we started our investigations on Nitric Oxide biology and we now appear busy doing fashionable translational science! I will take you through the story of accidental evolution of our laboratory.

FROM IMMUNE CELL DEVELOPMENT TO FUNCTION AND BACK

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The immune system comprises dozens of distinct cell types, each with a unique developmental trajectory, localization and expression profile that fit its function. While the primary evolutionary function of these cell types is to mount effective immune responses against various pathogens, their aberrant activity may contribute to autoimmune and inflammatory diseases. I studied the molecular mechanisms of autoimmunity as a graduate student and the regulation of immune cell development as a postdoctoral fellow. My lab combines these two interests and strives to apply the understanding of basic developmental pathways towards functional analysis of immune responses and autoimmunity. We have been particularly interested in dendritic cells (DCs), the key sentinel cells of the immune system that include antigen-presenting conventional dendritic cells (cDCs) and interferon-producing plasmacytoid dendritic cells (pDCs). Over the years, we have defined multiple transcription factors and signaling pathways that drive the development and functional fitness of DC subsets. The results and experimental systems developed during these studies proved useful in defining the role of DCs in autoimmune diseases and uncovering novel mechanisms of their pathogenesis. Our ultimate goal is to develop more rational, targeted approaches towards immnotherapy of autoimmune diseases such as systemic lupus erythematosus.

MEANDERING OF AN ENZYMOLOGIST THROUGH GENOMES, METHYLOMES AND ACETYLOMES

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Bacteriophages outnumber their hosts in nature and can have major effects on bacterial populations and communities. In response to viral predation, cellular defense mechanisms have evolved to prevent annihilation. They include, among others, innate immunity conferred by restriction- modification (R-M) systems, adaptive immunity mediated by CRISPR-Cas systems and an assortment of poorly studied abortive infection mechanisms that limit phage propagation.

My laboratory has been working on a class of restriction enzymes- Type III R-M systems since 1989 when I first set up an independent laboratory in the Department of Biochemistry at IISc, Bangalore. My first DST project (Rs 6 lakhs for three years) was on characterization of EcoP15 and EcoP1 restriction enzymes. My first two publications as an independent investigator were published in 1994 in Gene and JMB. My laboratory focused on these enzymes using them as model systems to understand how proteins recognize, cleave, and modify DNA. Years later we collaborated with groups in Cambridge, Edinburgh and Japan using Atomic Force Microscopy to delineate the steps starting from binding to specific recognition sequences to the actual cleavage of DNA.

Over the last 25 years or so my laboratory was interested in the structure-function relationships in several DNA methyltransfereases (MTases), particularly the ones from Helicobacter pylori that potentially code for R-M systems. Phase variation in H. pylori and in several other pathogenic bacteria is important for genetic variation, in which phase variable DNA methyltransferases seem to play a vital role. H.pylori harbors at least 10 such Phase variable DNA MTases and my research group has been working on several of these. While I was most comfortable doing biochemistry of these MTases, I decided in 2010 that I would start knocking out some of these MTases in H pylori and then look at what happens. A fantastic collaboration with Asish Mukhopadyay at NICED in Kolkata resulted in my laboratory acquiring this technology. While a lot has been said about methylation at C5 of cytosines and N6 position of adenines in bacterial genomes, work on one of the R-M systems in H pylori from my laboratory provided evidence that m4C signal acts as a global epigenetic regulator in H. pylori. We have used SMRT (single molecule real time) sequencing for the genome-wide detection of methylated cytosines or adenines which enable the capture of genome-wide profiling of DNA methylation in Helicobacter pylori strains.

A number of genome-wide characterization of lysine-acetylated proteins, or acetylomes, in bacteria have demonstrated that lysine acetylation occurs on proteins with a wide diversity of functions, including central metabolism, transcription, and chemotaxis. For the past two years my lab has embarked on deciphering the acetylome of Helicobacter pylori and therefore understanding fundamental roles of acetylation in different biological and pathological processes.

MAKING A DIFFERENCE: STEM CELLS, ASYMMETRIES AND STOMATA

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During development, multicellular organisms create a diverse set of specialized cell types and organize these cells into functional tissues. Often this process involves establishing self-renewing populations via asymmetric cell divisions. We use stomata (epidermal structures that regulate carbon dioxide and water exchange) as a model to understand integrated pattern formation in plants, and to link what happens at the cellular level to outputs at the whole organism and even whole planet levels. This has enabled my lab to interact with scientists in many different fields and to create a rewarding and holistic research program. Stomatal guard cells are created through asymmetric cell divisions whose number and orientation are dictated by the interplay of specific transcription factors, local cell-cell interactions and information from the environment. Cell-type specific bHLH transcription factors recruit entourages of more general regulators (MAPKs, chromatin remodelers, etc.). With these conserved factors as footholds, we are obtaining global information about cell specification and differentiation, showing how key transcription factors integrate information from various sources to promote discrete outcomes, and identifying the genetic networks surrounding these nodes. Current large-scale projects are to capture cell growth, division and targeted gene expression patterns in stomatal lineage cells as a whole leaf develops and to obtain cell-type-specific gene regulation trends. Much of the fate specification within the stomatal lineage involves regulatory logic and molecules conserved between plants and animals, and I will discuss themes that emerge when comparing cell fate and developmental flexibility in this lineage relative to other self-renewing populations.

FINDING A RESEARCH NICHE: DIGGING DEEPER AND DIGGING ELSEWHERE

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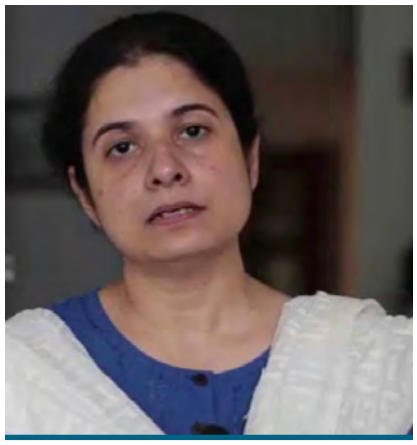
How does one make important contributions to biological research given the immense biological community and amazing pace of discovery? To answer this question, I will present two vignettes from our recent research. An answer is to dig deeper, to challenge a well-accepted conclusion with a fresh perspective. We dug deeper to understand the protein complex cohesin. Cohesin topologically entraps two different regions of DNA within or between DNA molecules to mediate higher order chromosome structure. The establishment, maintenance and release of DNA from cohesin entrapment requires regulated opening and closing of one of its four interfaces that occur between cohesin subunits. Release of DNA was thought to occur through dissociation of one interface called the exit gate. Our recent findings show that this interface is actually an evolutionarily conserved, conformational switch. This switch regulates the entry and possibly exit of DNA through one of the other interfaces. A second answer is to dig elsewhere. We have investigated stress biology by looking at the ability of rare microbes, animals and plants to survive the extreme stress of desiccation, the loss of almost all intracellular water. Using yeast as a model, we have shown that all the lethal stresses of desiccation are mitigated surprisingly by two small molecules, the sugar trehalose, and a small intrinsically disordered protein called Hsp12. These two stress effectors prevent protein aggregation and remodel membranes. I will discuss how our findings impact our understanding of stress biology and higher order chromosome structure from bacteria to man.

AFTER THE MALARIA PARASITE TURNED OUT TO BE A PLANT



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The discovery of a reduced plastid (apicoplast) in the protozoan parasite Plasmodium and related organisms in the mid-late 1990s was a revelation in many ways. Apart from providing perspectives on the evolution of this class of parasites, it offered possibilities for interference in biochemical pathways operative within the organelle as targets for discovery of anti-malarials. I joined CDRI as a fresh entrant in the field of malaria, interested in helping unravel some of the mysteries of apicoplast biology. My group addressed housekeeping processes such as organisation and replication of 35 kb circular apicoplast genome, translation mechanisms for expression of the limited (but essential) gene repertoire of the apicoplast and mitochondrion, and post-translational [Fe-S] cluster assembly by the SUF pathway. The organelles seem to make do with a translation and ribosome machinery which is both reduced and divergent from known prokaryotic/organellar counterparts and this has implications for antibiotic action. The SUF [Fe-S] assembly pathway is critical for parasite development in blood stages; we have recently provided delineation of the major steps and proteins participating in [Fe-S] assembly on important apicoplast proteins and demonstrated essentiality of SUF in mosquito stage development. Our work over the last 18 years has shown how this endosymbiont makes do with reduced components for critical functions and has offered potential sites for intervention.

CODES FOR THE MAKING OF A RICE FLOWERING STEM

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'O Tiger-lily, I wish you could talk!' 'We can talk,' said the Tiger-lily: 'when there's anybody worth talking to".
Through the Looking Glass,
Lewis Carroll

Unravelling the logic behind the development of multicellular organisms from a fertilized single cell has fascinated philosophers for millenia. In more recent times biologists use the tools of genetics and developmental biology to decipher the complex interactions between genes and environment in the making of the striking structure and patterns we see in the mature organism. Studies in convenient laboratory organisms, illustrate how a few key 'master' regulators can have large- cascading- and cumulative- effects on how growth and the emergence of form takes place. In higher plants, flowering comes about by a remarkable transformation from a basal state where cells destined to be leaves or branches are transformed to a floral fate. Our knowledge on how this transfomation is controlled comes largely from experimental studies with *Arabidopsis thaliana*, a model laboratory plant. These studies have given a remarkable insight on key regulators of flower development. Yet, given the diversity in flowering time and floral architectures that occur in nature, an outstanding question is about how these evolutionarily conserved regulators relate to the emergence of new patterns and variations in flowering stems and flowers.

We study development of the rice flowering stem (inflorescence) and rice flowers with the overall goal to understand the relationship between factors and signals that control fate of plant stem cells (meristems) and those that determine the identity of organs formed from meristems. I will summarize our studies that identified novel functions in rice flowering, stem-branching and meristem development for a gene call *RICE FLORICULA LEAFY (RFL)*, an evolutionarily conserved transcription factor whose functions in Arabidopsis thaliana are largely confined to floral meristems. We have also investigated, using functional genomic tools, the roles for other genes called MADS domain transcription factors, in floret meristem formation, organ development and meristem termination. Studies such as ours and those in leading laboratories worldwide on the genome-wide effects of rice transcription regulators would in the future allow one to build dynamic gene-regulatory networks. Comparisons with data emerging from other plant models including *Arabidopsis* will be insightful to understand how function in a specific species shapes the network properties.

POST DOCTORAL FELLOWS ABSTRACTS

P01	Ankur Sharma	Longitudinal single-cell RNA sequencing of patient-derived primary cells reveals drug-induced infidelity in stem-cell hierarchy
P02	Baskar Bakthavachalu	Ataxin-2 RNP-granule dependent translation control mechanisms in neurons
P03	Bhagawat C Subramanian	Investigating the organelle dynamics and their roles in innate immune cell response using intravital sub-cellular microscopy (ISMic) in live animals
P04	Budhaditya Mukherjee	Dissection of the fundamental roles played by apicomplexan aspartyl proteases in establishment of parasitism
P05	Debapriyo Chakraborty	Anthropogenic habitat fragmentation and gastrointestinal parasite risk not linked in a human-altered landscape in southern india
P06	Debostuti Ghoshdastidar	Reading DNA in context: Effect of flanking sequences on DNA-transcription factor binding
P07	Deepankar Singh	NFY, a trimeric transcription factor complex, is required for oogenesis and preimplantation development in mice
P08	Dhananjay Chaturvedi	Mechanisms of muscle repair: learning from drosophila
P09	Dhanashree Kelkar	Identification of enzymes regulating oxidized phospholipids using chemical genetic screen
P10	Dimple Siva	Development of recognition molecules for their use in early viral detection kit
P11	Gaurang Mahajan	Metaplasticity mediated by internal calcium stores in hippocampal dendritic spines

Post Doctoral Fellows Abstracts

P12	**Gauri Achari**	Zinc chitosan nanoparticles have anti-quorum sensing activity in the plant pathogen *Ralstonia solanacearum*
P13	**Harish Katti**	Monkeys can't read but their brains can: Compositionality and CAPTCHA decoding in IT neurons.
P14	**Harshad Ingle**	Viral complementation of immunodeficiency confers protection against enteric pathogens via IFN-Î»
P15	**Herschel Dhekne**	A pathway for Parkinson's Disease LRRK2 kinase to block primary cilia signaling in the brain
P16	**Jagajjit Sahu**	Unravelling Indian publication patterns; A 5-year scientometric profiling of PubMed reports
P17	**Joubert Kharlyngdoh**	Improvement of kidney function and preservation of glomerular filter rate in Diabetic rats ZSF1 following treatment with low doses of GDT-01
P18	**Joyshree Chanam**	Effect of environmental factors on floral odour and consequently plant-pollinator interactions in alpine meadow plants in the Eastern Himalayas
P19	**K. Iyer**	Mechanosensitive binding of p120-Catenin regulates E-Cadherin turnover and Viscoelastic behavior of Tissues
P20	**Kaustuv Nayak**	Human T cell responses to chikungunya infection
P21	**Mangesh Suryavanshi**	Exposure of Lactobacillus sp. retains functional eubacteria species along with trans-domain healthy gut diversity

Post Doctoral Fellows Abstracts

P22	**Neelja Singhal**	Comparative genomics of Î²-lactamase genes â€“ a goldmine for rapid antimicrobial resistance (AMR) detection and newer Î²-lactamase inhibitors ?
P23	**Parthasarathy Kalyanasundaram**	Odor arrival side discrimination in *Manduca sexta*
P24	**Pooja Kumari**	Spatio-temporal regulation of RNA during development and disease
P25	**Poonam Thakur**	Alpha-synuclein pre-formed fibrils disrupt the spontaneous firing patterns of substantia nigra neurons
P26	**Rajat Anand**	Omics technologies for drug discovery and disease diagnosis/prognosis
P27	**Rajiv Kangabam**	Water quality Index and role of microbial community in the Loktak lake, the largest fresh water lake in Indo-Burma biodiversity hotspot
P28	**Rishita Changede**	Geometry of the cell niche regulates architecture of cell matrix adhesions that dictates cellular response
P29	**Rittik Deb**	Host dietary specialization and neutral assembly shape gut bacterial communities of wild dragonflies
P30	**Saravanan Palani**	Phospho-regulation of tropomyosin is crucial for actin filament turnover
P31	**Sarita Saraswati**	High mitogenic stimulus induced by VEGF or Notch inhibition arrests physiological and tumor angiogenesis
P32	**Sarmistha Mahanty**	Epidermal differentiation- a survival adaptive response is mediated by lysosome biogenesis

Post Doctoral Fellows Abstracts

P33	Senthil Thangaraj	Unmasking the amyloid face of ALS and FTLD associated TDP-43 fibrils
P34	Shantanu Shukla	Microbiome mediated dietary specialization in a necrophagous insect
P35	Sharath Chandra A	Pro-tumorigenic phenotype of p53 in Cancer Associated Fibroblasts
P36	Sonia Sen	Integration of spatial and temporal cues in Drosophila embryonic neuroblasts
P37	Sundar Naganathan	Emergence of bilaterally symmetric somites during zebrafish embryogenesis
P38	Venkatakrishnan Ramaswamy	An algorithmic barrier to neural circuit understanding
P39	Vikash Singh	PAK to the Future

LONGITUDINAL SINGLE-CELL RNA SEQUENCING OF PATIENT-DERIVED PRIMARY CELLS REVEALS DRUG-INDUCED INFIDELITY IN STEM-CELL HIERARCHY

Single Cell Genomics, Systems Biology, Tumor Evolution, Stem Cells, Personalised Medicine

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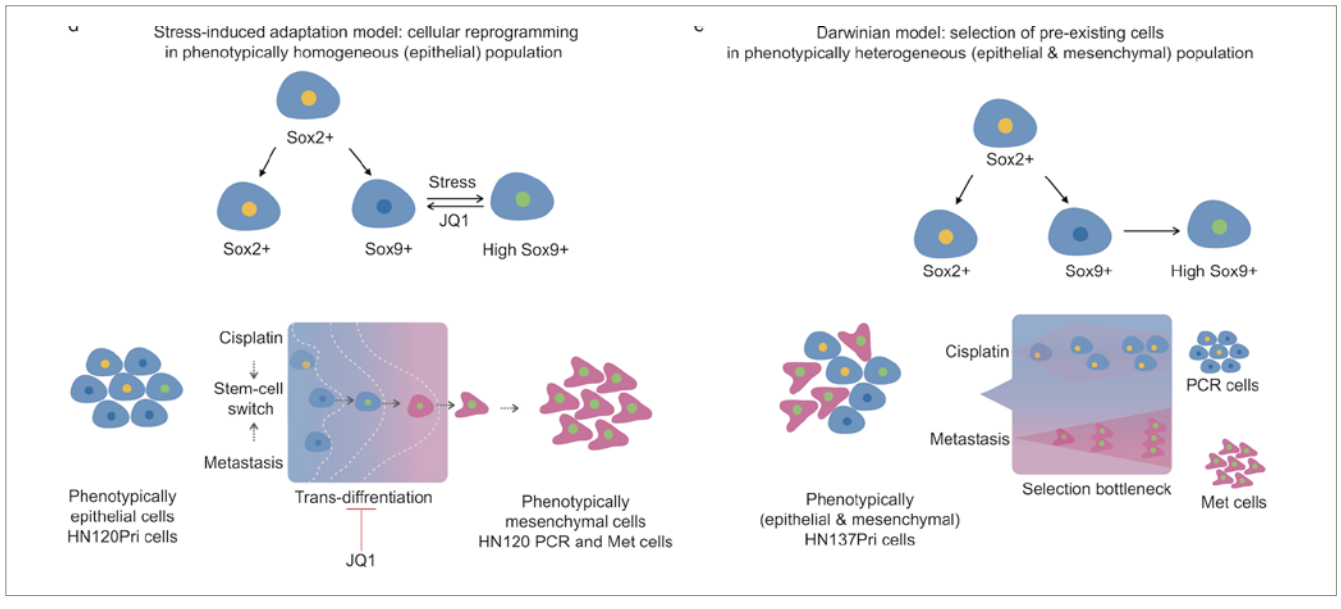
Chemo-resistance is one of the major causes of cancer-related deaths. Here we used single-cell transcriptomics to investigate divergent modes of chemo-resistance in tumor cells. We observed that higher degree of phenotypic intra-tumor heterogeneity (ITH) favors selection of pre-existing drug-resistant cells, whereas phenotypically homogeneous cells engage covert epigenetic mechanisms to trans-differentiate under drug-selection. This adaptation was driven by selection-induced gain of H3K27ac marks on bivalently poised resistance-associated chromatin, and therefore not expressed in the treatment-naïve setting. Mechanistic interrogation of this phenomenon revealed that drug-induced adaptation was acquired upon the loss of stem factor SOX2, and a concomitant gain of SOX9. Strikingly we observed an enrichment of SOX9 at drug-induced H3K27ac sites, suggesting that tumor evolution could be driven by stem cell-switch-mediated epigenetic plasticity. Importantly, JQ1 mediated inhibition of BRD4 could reverse drug-induced adaptation. These results provide mechanistic insights into the modes of therapy-induced cellular plasticity and underscore the use of epigenetic inhibitors in targeting tumor evolution.



ATAXIN-2 RNP-GRANULE DEPENDENT TRANSLATION CONTROL MECHANISMS IN NEURONS

RNA Biology, Gene Regulation, Translation Control, Neuroscience, Drosophila Genetics

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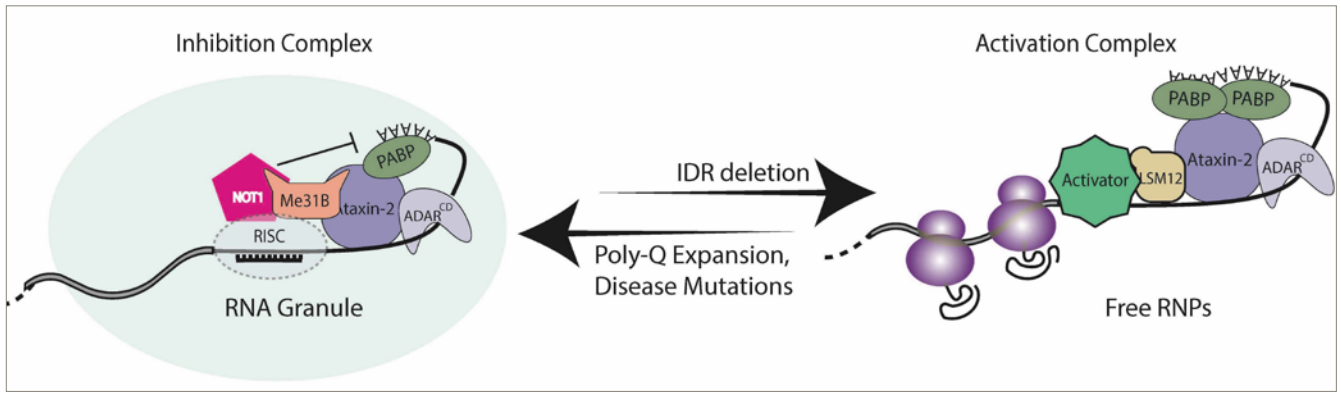
Ataxin-2 is an RNA binding protein with important neuronal functions. Our recent work in Drosophila has demonstrated that distinct domains of Ataxin-2 play essential roles in the RNA-granule formation and translational control in the fly brain. We demonstrated Ataxin-2 associated with CaMKII mRNA and repress translation. While studies from other groups have shown Ataxin-2 as a translation activator for period mRNA in circadian neurons. So, Ataxin-2 is thought to control translation, determined by the associated protein complex. There are previous studies that show the global mRNA partners of Ataxin-2 in cultured mammalian cells, but the neuronal Ataxin-2 mRNA target list is unknown. To understand how Ataxin-2 control neuronal mRNA, we used TRIBE (Targets of RNA-Binding Proteins Identified by Editing) approach to express ADAR tagged Ataxin-2 adult specifically in fly brain and identified the mRNA targets. We find Ataxin-2 strongly bind several important neuronal mRNAs previously shown to have roles in synapse formation and memory, preferentially on their 3'UTRs. Our further analysis show Ataxin-2 share several of these mRNA targets with other neuronal RNP granule proteins. We speculate Ataxin-2 along with other RNP granule protein bind neuronal mRNAs and regulate translation. We are currently in the process of understanding the Ataxin-2 translation control mechanism.



INVESTIGATING THE ORGANELLE DYNAMICS AND THEIR ROLES IN INNATE IMMUNE CELL RESPONSE USING INTRAVITAL SUB-CELLULAR MICROSCOPY (ISMIC) IN LIVE ANIMALS

Innate Immune Cells, Intravital Microscopy, Receptor Signaling, Vesicle Trafficking, Organelle Homeostasis

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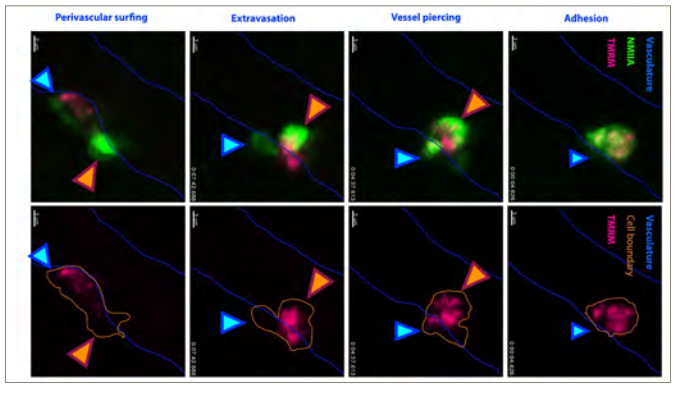
The innate immune cells such as neutrophils rapidly respond and dictate the outcome of host response to inflammatory insults. Such cells exhibit specialized responses during inflammation, which include extravasation, fast migration, phagocytosis, burst of reactive radicals, release of enzymes, formation of the extra-cellular traps etc. Such dynamic responses require extreme plasticity at the level of cellular morphology and organelle dynamics. In fact, altered organelle morphology is associated with impaired functioning of innate cells in many clinical scenarios. For example, in patients with Pelger-Huet anomaly or myelokathexis, neutrophils display marked abnormalities in their nuclear shape and lobation. Also, in patients with Chédiak-Higashi syndrome or May-Hegglin anomaly, neutrophils exhibit giant cytoplasmic granules or inclusions respectively. Not surprisingly, such patients suffer from a deficient immunity as well as other physical abnormalities. Our research focuses on the characterization, regulation and functional roles of organelle dynamics in driving innate immune cell response during homeostasis and inflammation. We have now established an in vivo model to image neutrophil responses to sterile infection, at sub-cellular resolution, in the mouse hind foot-pad using two-photon microscopy in live animals. For the first time, we document the in vivo sub-cellular dynamics of key regulatory molecules -1) b2 integrin and its regulation by leukotriene B4 signaling during neutrophil extravasation, whose absence results in leukocyte adhesion deficiency syndrome (LADS); and 2) non-muscle myosin IIA (NMIIA) and its role in promoting neutrophil extravasation, given the role of NMIIA in the May-Hegglin anomaly. We will also present our results on the dynamics and roles of mitochondria and other organelles during neutrophil extravasation. Our approaches will unravel molecular mechanisms that shape innate immune cell responses to complex diseases.



Visualizing innate cell organelle dynamics in live animals using ISMic

DISSECTION OF THE FUNDAMENTAL ROLES PLAYED BY APICOMPLEXAN ASPARTYL PROTEASES IN ESTABLISHMENT OF PARASITISM

Leishmania/Plasmodium, CRISPR-Cas9, Post-Translational Modifications, Transmission, Drug-Resistance

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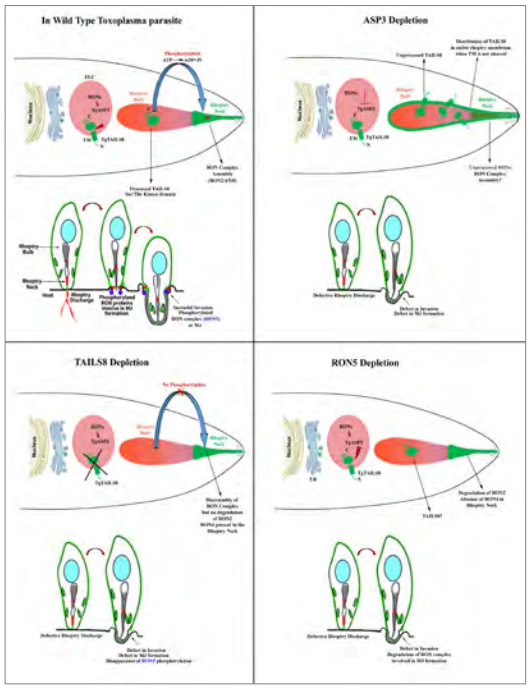
Apicomplexans are life-threatening human & animal pathogens. Survival & dissemination of these obligate intracellular parasites depends on their capacity to actively invade & egress from host. These obligate intracellular parasites possess an arsenal of secretory proteins, sequentially discharged from specialized secretory organelles (micronemes & rhoptries) during egress/invasion. Host attachment leads to discharge of rhoptry proteins, RONs & ROPs, that critically participate in entry process by forming moving junction (MJ) & subversion of host cellular functions respectively. We previously showed that aspartyl protease3 (TgASP3) acts as maturase of numerous RONs/ROPs & ASP3 depletion blocks rhoptry discharge/invasion1. Orthologues of TgASP3, PMIX & PMX, fulfil equally fundamental functions in all invasive stages of malaria parasites2. Terminal amine isotopic labeling of substrates (TAILS) analysis identified TgTAILS8, a type II transmembrane RON protein with putative Ser/Thr kinase domain among novel TgASP3 substrates. Conditional depletion of TAILS8 causes a severe decrease in rhoptry discharge leading to defective MJ formation & invasion. Depletion of TAILS8 resulted in disappearance of phosphorylated RON5 & absence of RON4 two critical components of MJ, conserved across Apicomplexa. Recombinant TAILS8 exhibits kinase activity in vitro, presumably relevant for the assembly of the RON complex in MJ. Apicomplexan possesses a large family of ROP kinases, several of which are dispensable for growth in vitro but critically affecting virulence. In contrast, TAILS8 is crucial RON kinase implicated in organelle discharge & invasion.



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ANTHROPOGENIC HABITAT FRAGMENTATION AND GASTROINTESTINAL PARASITE RISK NOT LINKED IN A HUMAN-ALTERED LANDSCAPE IN SOUTHERN INDIA

Disease Ecology And Epidemiology, Emerging Infectious Diseases, One Health, Public Health, Urbanisation

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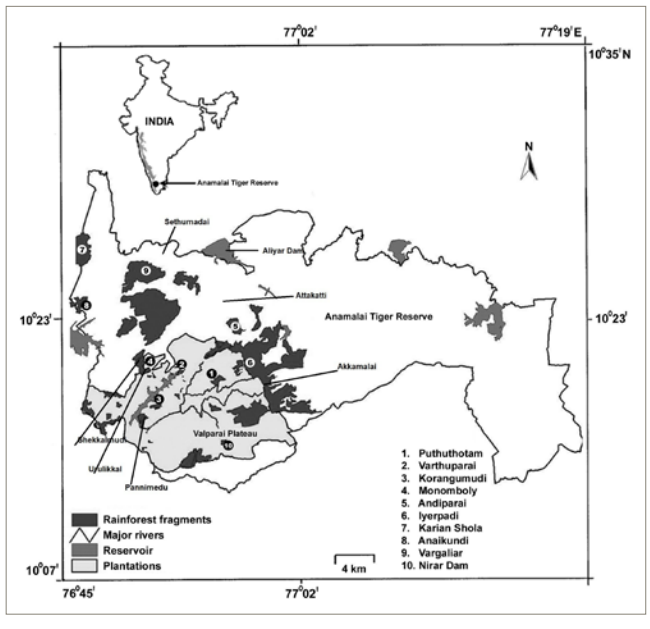
Anthropogenic habitat fragmentation through biodiversity loss and altered species interactions is postulated to increase zoonotic disease risk, which can then potentially be reduced by increasing overall ecosystem health through conservation measures [1]. According to this hypothesis, disease risk is predicted to rise in highly fragmented habitats in contrast to less fragmented ones [2]. To test these predictions, we noninvasively collected faecal samples from 17 mammal host species across 20 different habitat fragments in Anamalai Tiger Reserve, India. The size of forest fragments ranged from two to 20,000 ha. Employing a community ecological framework, we compared gastrointestinal parasite risk (richness and prevalence) across the range of habitat sizes. After adjusting for sample size differences and differences in parasite abundance, we could not detect any significant difference in parasite risk between smaller and larger fragments. Unexpectedly, parasite community compositions across fragments also did not considerably differed across fragments indicating a largely homogenised parasite community distribution across the altered landscape. We however found difference in parasite community distribution based on host species ecology and trait. Overall, these results indicate that the link between habitat fragmentation and gastrointestinal parasite risk is far from a general one but depends on context and host ecology and highlight the usefulness of a community ecological framework in synthesizing landscape level patterns in host-parasite interactions.



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READING DNA IN CONTEXT: EFFECT OF FLANKING SEQUENCES ON DNA-TRANSCRIPTION FACTOR BINDING

Computational Biophysics, Nucleic Acid Structure, Transcriptional Regulation of Gene Expression, Transient Dynamics in Nucleic Acids, Big Data Analyses

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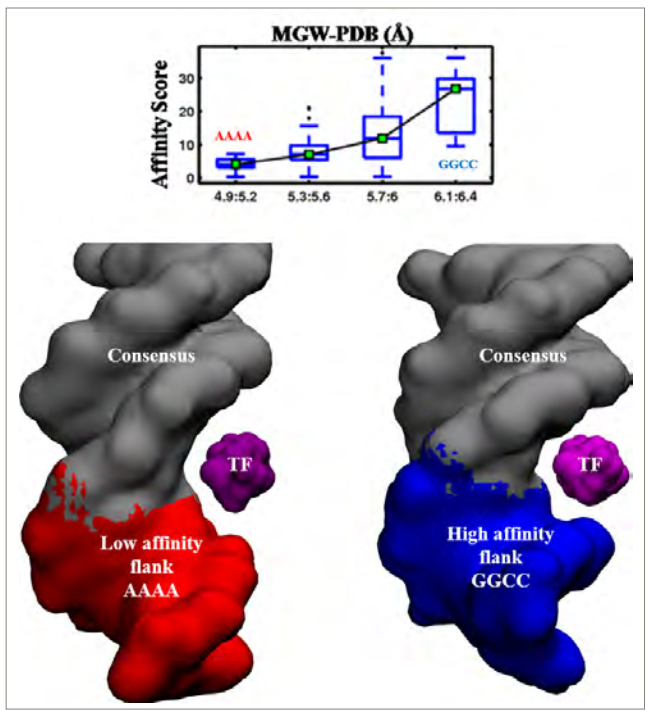
The complexity of an organism is not determined by the number of genes but in how the expression of these genes are regulated. Transcription factors (TFs) are DNA-binding proteins that regulate gene expression patterns by binding to specific DNA sequences (consensus motif) in the genome. Based on the target sequence alone it is not possible for the TF to locate a functional consensus site among the billions of base pairs in the genome. Along with the consensus binding motif, the flanking sequence context is believed to play a role in DNA-TF recognition. In this study, we employ high-throughput in vitro and in silico analyses to understand the influence of sequences flanking the cognate sites in binding of the three most prevalent eukaryotic TF families (Zinc finger, homeodomain, and bZIP). In vitro binding preferences of each TF toward the entire DNA sequence space were correlated with a wide range of DNA structural parameters. Results demonstrate that the flexibility of flanking regions modulates binding affinity of certain TF families. Furthermore, DNA duplex stability and minor groove width play important role in DNA-TF recognition but differ in how exactly they influence the binding in each specific case. Our analyses clearly indicate that the structural features of preferred flanking sequences are not universal, as similar DNA-binding folds can employ distinct DNA recognition modes. Further, molecular dynamics simulation studies reveal that preferred flanking sequences make the shape of the consensus motif more conducive for TF recognition and binding.



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NFY, A TRIMERIC TRANSCRIPTION FACTOR COMPLEX, IS REQUIRED FOR OOGENESIS AND PREIMPLANTATION DEVELOPMENT IN MICE

Epigenetics, Zygotic Transcription, Embryogenesis, Oogenesis, Ciliates

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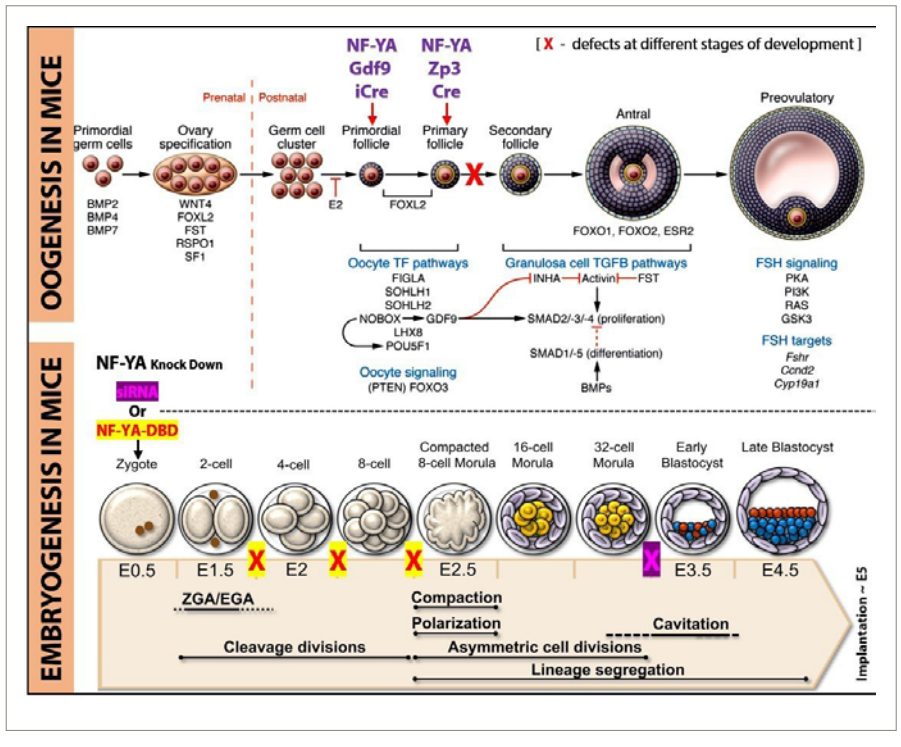
Life in mammals starts with the fusion of two dimorphic haploid germ cells, an oocyte and a spermatozoon, which later give rise to a totipotent zygote. This totipotency is achieved by extensive epigenetic reprogramming consisting of two molecular events, i.e. (a) clearance of maternal mRNA and proteins and (b) onset of transcription from newly form zygote, called as Zygotic Genome Activation (ZGA). My work focuses on identifying factors controlling the activation of genes specifically expressed at the onset of the ZGA. Using an integrated approach of computational biology, we identified NFY, a trimeric transcription factor complex composed of NF-YA, YB and YC, bears a potential of key ZGA regulator. Here we show that siRNA mediated depletion of NF-YA, or in combination with NF-YA DNA binding domain mutated RNA led to developmental arrest at various stages of preimplantation development. During ZGA, MuERVL transposons, which can act as an alternative promoter for number of 2-cell stage transcripts, is required for successful ZGA and it is tightly regulated. Interestingly, we also find de-repression of MuERVL transposable elements and 2-cell transcripts in NF-YA depleted morula embryos. We propose that NF-YA is required for successful ZGA, and it may also be directly/indirectly required for regulation of transposable elements and hence, indispensable for mouse preimplantation development. Furthermore, using a conditional knock out model, we show that depletion of NF-YA during early folliculogenesis could lead to premature activation of primordial follicle pool causing premature ovarian failure (POF), a common cause of infertility and premature aging in women. Therefore, we suggest that NFY is an integral part of germ cell specific program during oogenesis and an important regulator of early embryo development.



MECHANISMS OF MUSCLE REPAIR: LEARNING FROM DROSOPHILA

Regenerative-Biology, Drosophila, Muscle repair, Stem Cells, Development

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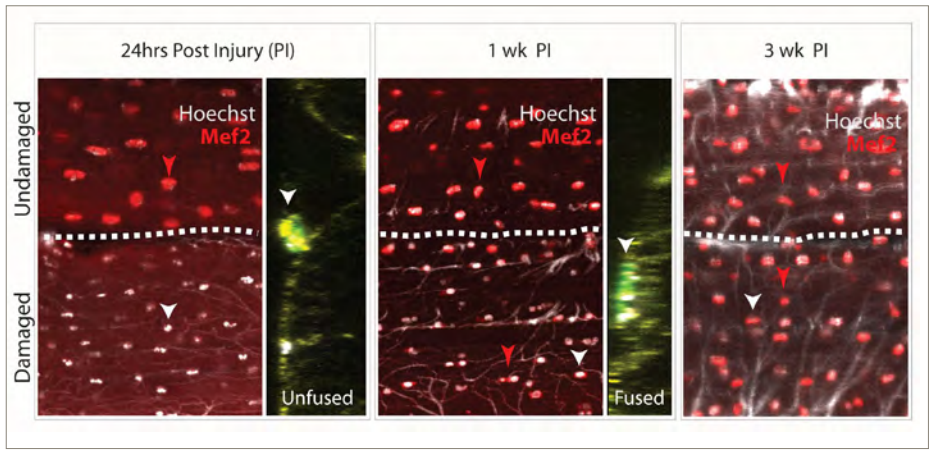
In recent work, we were the first group to demonstrate the presence of adult muscle stem-cell like cells in adult Drosophila flight muscle syncitia1. In mammals, the homologous cell population which is necessary for muscle repair was identified five decades ago. This cell type is referred to as Satellite cells. In Drosophila, we have demonstrated the lineage of these mononucleate cells, and shown that they proliferate upon injury and that they fuse with injured fibers. We focus on Dorsal Longitudinal Muscles (DLMs). They share architectural hallmarks with mammalian skeletal muscles. Our findings allow for the genetic tools available in Drosophila to be exploited to study molecular mechanisms of muscle repair in vivo for the very first time.

Current investigations focus on the effects of muscle injury on muscle morphology through every step of recovery. We have standardized MicroCT scanning and volumetrics for DLMs that affords accurate volume measurements in situ on scales not seen in this field yet. We find considerable asymmetry in wildtype DLM volumes about the midline. This may indicate a preference in wing usage in indivdual animals. Consistent changes in actin fibers arrangements at specific intervals in adult life, suggest age depedent organellar volumes.

In addition to contradicting conjectures in the field about uninjured DLMs, we find drastic changes in muscle fiber volumes and cellular processes in response to injury, that recover with repair. Further, the role of Drosophila satellite cells in these processes is being investigated.



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IDENTIFICATION OF ENZYMES REGULATING OXIDIZED PHOSPHOLIPIDS USING CHEMICAL GENETIC SCREEN

Cellular Signaling, Lipid Signaling, Phospho-Proteomics, Lysophosphatidylserine

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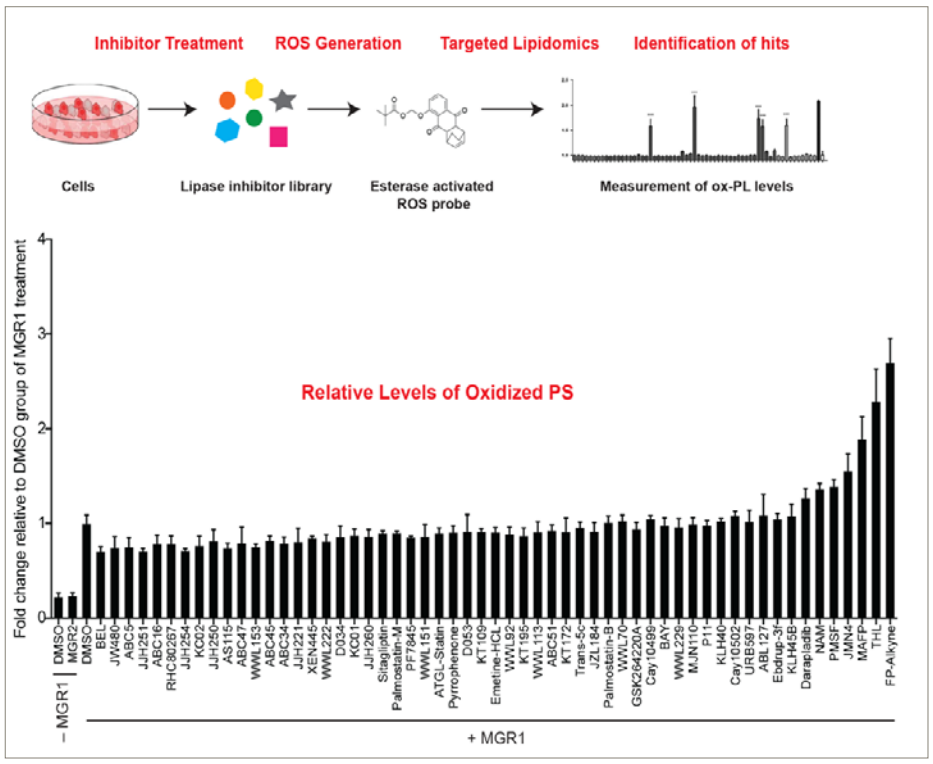
Reactive oxygen species (ROS) are transient, highly reactive chemical intermediates or by-products generated during oxygen metabolism. (1) Unchecked ROS species cause damage and destruction of cellular components like lipids, proteins and even DNA, which eventually lead to cell death via apoptosis or necrosis. (2) When ROS are generated due to oxidative stress close to the membranes, lipids containing the polyunsaturated fatty acid (PUFA) chains are oxidized. The resulting lipid oxidation products can disrupt the local structure and integrity of the membrane, and in turn impair cellular functions by modulating the activity of a wide array of cellular proteins like enzymes, receptors and channels. (3) At present, little is known about the chemical composition and metabolism of the oxidized phosphatidylserines. Towards this, we employed chemical genetic screen to identify lipases that metabolize oxidized PS in mammalian cells. We have characterized oxidized PS in mammalian cells and studies its metabolic regulation. A serine hydrolase enzyme ABHD12 was identified as oxidized PS metabolizing enzyme in cells. We validated these findings in different cellular assays including macrophages derived from Abhd12 knockout mice, and thus annotated an enzyme capable of regulating oxidized PS in vivo.



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DEVELOPMENT OF RECOGNITION MOLECULES FOR THEIR USE IN EARLY VIRAL DETECTION KIT

Viral Diseases, Diagnostic Kits, Development of Recognition Molecules, Smart Production, Delivery to Public

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Introduction

Chikungunya is a well-known disease. It's popular due to the pain it causes after the virus infects us. Scientific people around the world are seriously trying their best to stop the spread of the virus. Preventive medicines like vaccine and therapeutics like antivirals are at different stages of development but none of them is still handy to the patient. He cannot go and pick it right now from the medical store. In case of chikungunuya, there is no early detection kit that can detect the disease in acute phase. There are methods like RT-PCR to do this task but it's not rapid. Hence we planned to make an early detection kit with the use of oligonucleotide based recognition molecule that can catch the virus in the blood within 1-4 days of infection.

Method

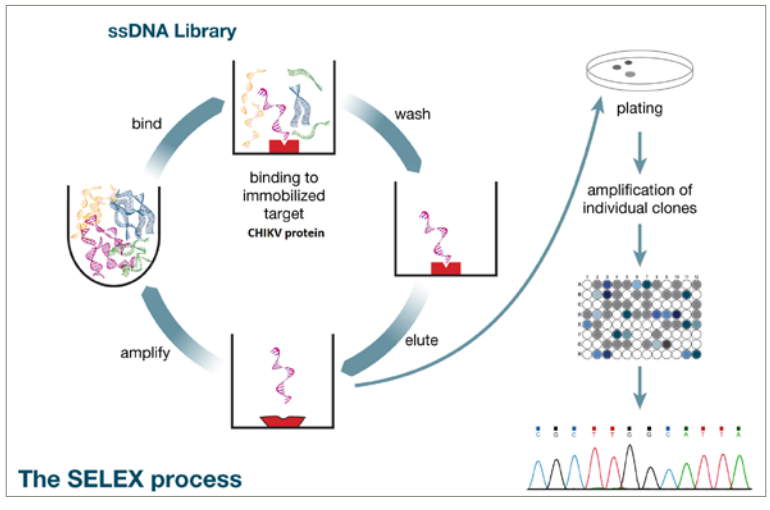
We used a most popular and published method called SELEX (Systematic evolution of ligands by exponential enrichment) to bring out the best binder to the target CHIKV.

Results

Using SELEX we brought out the best binder, tested its affinity to the target by binding assays. It's working, doing its job perfectly. It's binding specifically to target. We used this recognition molecule to develop a rapid kit that can catch the virus in the blood. The kit was also tested for its efficiency. It's working.

Conclusion

We planned to make the kit for detecting CHIKV. We made it by using a published method with some customization. The kit is tested and verified. Now the kit is waiting to serve the public and to tell them whether the virus within them is CHIKV or not.



METAPLASTICITY MEDIATED BY INTERNAL CALCIUM STORES IN HIPPOCAMPAL DENDRITIC SPINES

Mathematical Models Of Biochemical Signaling, Networks In Neuroscience, Analysis Of Large Data Sets In Biology, Synaptic Function And Plasticity, Computation In Neuronal Dendrites

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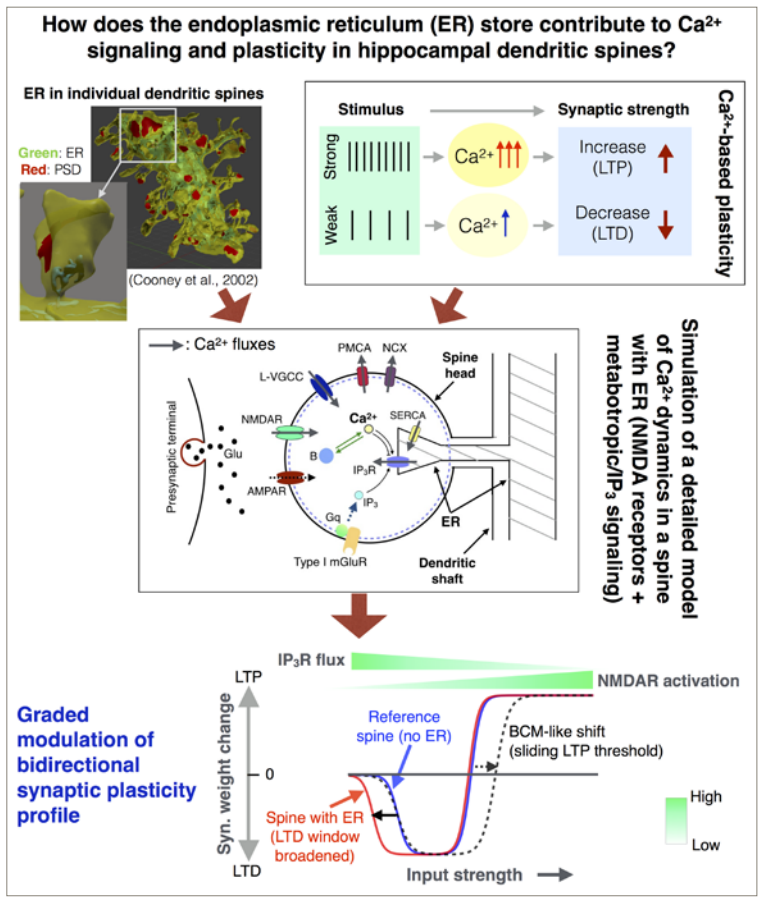
Postsynaptic calcium (Ca) entry regulated by ionotropic (NMDA) receptors is considered central to the induction of activity-dependent changes in the strength of excitatory synapses in the hippocampus. This generic description of a synapse does not, however, take into account the potential contribution made by internal Ca stores associated with the endoplasmic reticulum (ER). Imaging studies indicate a heterogeneous distribution of ER in dendritic spines on hippocampal pyramidal neurons, which may introduce localized functional differences between synapses in Ca signaling, as suggested by experimental work in the context of metabotropic receptor (mGluR)-dependent synaptic depression. Aberrant ER Ca regulation may also have pathological consequences. We use biologically detailed computational modeling of an ER-bearing dendritic spine head to address the involvement of IP3-sensitive stores in spine Ca dynamics during activity patterns mimicking the induction of long-term potentiation (LTP) and depression (LTD). Our simulations characterize in detail the dependence of store contribution on the temporal profile of synaptic input and the activation of NMDA receptors. Our analysis suggests a graded modulation of the NMDA receptor-dependent plasticity profile, with ER selectively enhancing LTD induction, whereas its modulatory effect is diminished at LTP-inducing stronger stimulation. Given the observed association of ER with stronger synapses, we propose that spine ER may locally tune Ca-based plasticity on an as-needed basis, providing a "braking" mechanism to temper runaway strengthening of potentiated synapses. Broadly, our study contributes a biophysically more complete picture of postsynaptic Ca regulation in relation to synaptic plasticity and stability, and offers a novel perspective on functional role of intracellular stores on the level of individual synapses.



ZINC CHITOSAN NANOPARTICLES HAVE ANTI-QUORUM SENSING ACTIVITY IN THE PLANT PATHOGEN *Ralstonia solanacearum*

Biofertilizers, Nanoparticles, Quorum Sensing, Capsicum annuum, Ralstonia solanacearum

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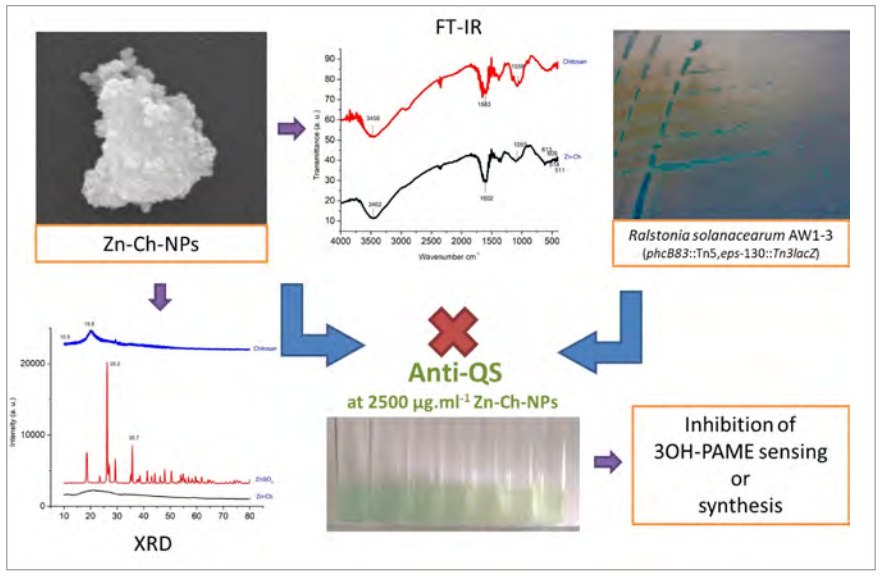
Ralstonia solanacearum causes bacterial wilt (BW) in solanaceous vegetables worldwide leading to 80-100% crop loss. In Goa, the locally cultivated Capsicum annuum L. (Chilli) varieties are susceptible to BW. Quorum sensing (QS) system with 3-hydroxy palmitic acid methyl ester (3OH-PAME) autoinducer regulates virulence factor expression in this pathogen. This study is focused on determining the anti-QS activity of Zinc-Chitosan nanoparticles (Zn-Ch-NPs) in *R. solanacearum*. Zn-Ch-NPs were synthesized using ZnSO4 as the precursor and characterized by Scanning Electron Microscopy (SEM), X-Ray Diffraction, Fourier-Transform Infrared Spectroscopy, and Zeta potential analysis. Anti-QS assay was performed using the indicator strain *R. solanacearum* AW1-3 (phcB83::Tn5,eps-130::Tn3lacZ) which does not produce 3OH-PAME; but responds to its exogenous presence with an expression of β-galactosidase which acts on X-gal causing appearance of blue color. The results indicate that the Zn-Ch-NPs are nanospheres (31.41 nm in diameter); have a zeta potential of +30.57 and a Zn content of 112.2 µg.mg-1. At a concentration of 2500 µg.ml-1, the Zn-Ch-NPs inhibited QS in *R. solanacearum* strain AW1-3 without affecting its growth as indicated by absence of blue coloration and a population of 10.91 Log CFU.mL-1. Bulk ZnSO4 inhibited growth of *R. solanacearum* at a concentration 250 µg. ml-1 (Zn2+) and no effect on QS was observed in presence of bulk ZnO and chitosan. Further studies confirming the exact anti-QS mechanism, possibly by inhibition of signal sensing or synthesis are under progress. The study highlights importance of Zn-Ch-NPs for anti-virulence strategies for BW management.



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MONKEYS CAN'T READ BUT THEIR BRAINS CAN: COMPOSITIONALITY AND CAPTCHA DECODING IN IT NEURONS

Electrophysiology, Object Perception, Scene Perception, Computer Vision, Neural Mechanisms

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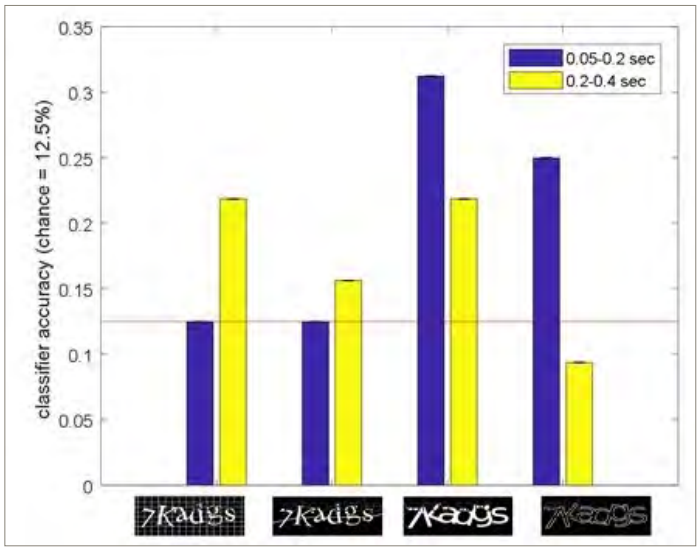
Primates excel at object recognition. An everyday example of this is the distorted letter tests that we see on websites (CAPTCHAs). CAPTCHA strings are used to deny access to malicious computer programs. What makes us so good at reading distorted letters? One possibility is that we may have specialized, invariant letter detectors that combine systematically to represent words. Alternatively, we may have specialized detectors not only for single letters but also for combinations of letters, thereby leading to efficient decoding. Either type of representation may exist de facto in the primate visual system or may emerge because of learning to read.

We investigated this by characterizing the representation of single Latin letters and combinations of letters in the inferior temporal (IT) cortex of monkeys. We selected uppercase and lowercase English letters as well as numbers and combined them systematically into strings that were up to 6 characters long. To these, we applied local, global as well as CAPTCHA-like shape distortions and obtained a dataset of 432 unique stimuli. We recorded multi-channel extracellular activity from 141 visually responsive neurons from two monkeys.

First, can the population activity of monkey IT neurons be used to solve CAPTCHAs? This was indeed the case: linear classifiers trained to identify characters at each retinal location were sufficient for above-chance decoding of CAPTCHAs. Second, can the neural response to letter strings be predicted as a composition of single-letter responses? We find linear models can predict neural responses to longer strings using single characters.



Taken together, our results show for the first time that generic object recognition mechanisms in the primate brain are sufficient to process and decode CAPTCHAs, and that this ability arises from simple compositional rules that govern how word responses relate to letters.

VIRAL COMPLEMENTATION OF IMMUNODEFICIENCY CONFERS PROTECTION AGAINST ENTERIC PATHOGENS VIA IFN-Î»

Gut microbiota, Virology, Innate immunity, Infectious diseases, MicroRNA

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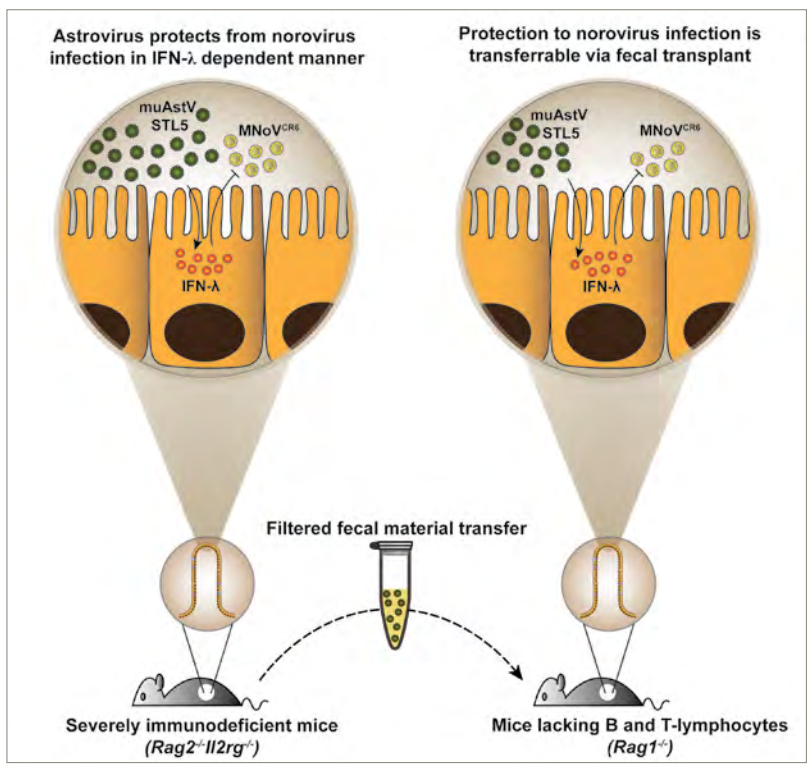
Commensal microbes profoundly impact host immunity to enteric viral infections. We have shown that the bacterial microbiota and host antiviral cytokine interferon-lambda (IFN-λ) determine the persistence of murine norovirus in the gut. However, the effects of the virome in modulating enteric infections remain unexplored. Here we report that murine astrovirus can complement primary immunodeficiency to protect against murine norovirus and rotavirus infections. Protection against infection was horizontally transferable between immunocompromised mouse strains by cohousing and fecal transplantation. Furthermore, protection against enteric pathogens corresponded with the presence of a specific strain of murine astrovirus in the gut, and this complementation of immunodeficiency required IFN-λ signaling in gut epithelial cells. Our study demonstrates that elements of the virome can protect against enteric pathogens in an immunodeficient host.



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A PATHWAY FOR PARKINSON'S DISEASE LRRK2 KINASE TO BLOCK PRIMARY CILIA SIGNALING IN THE BRAIN

Microscopy, Signaling, Neurodegeneration, Membrane Trafficking, Biomarkers

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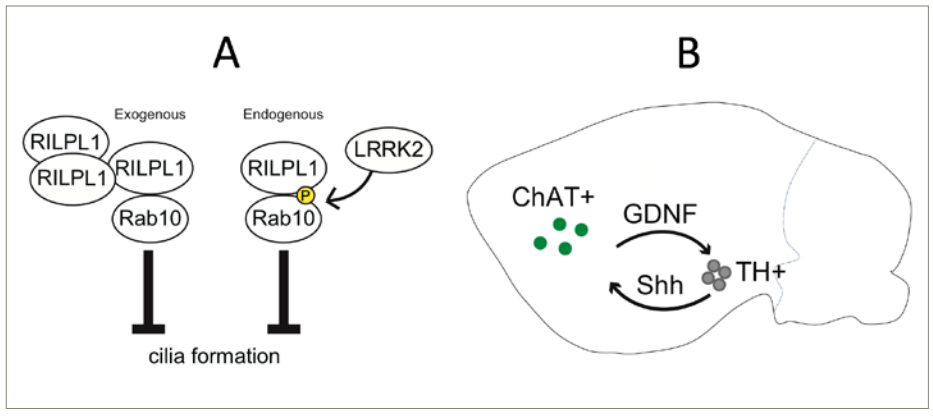
Parkinson's disease-associated LRRK2 kinase phosphorylates multiple Rab GTPases, including Rab8A and Rab10. We show here that LRRK2 kinase interferes with primary cilia formation in cultured cells, human LRRK2 G2019S iPS cells and in the cortex of LRRK2 R1441C mice. Rab8A phosphorylation blocks its ability to promote ciliogenesis, whereas Rab10 phosphorylation strengthens its intrinsic ability to block ciliogenesis by enhancing binding to RILPL1. Importantly, the ability of pathogenic LRRK2 to interfere with ciliogenesis requires both Rab10 and RILPL1 proteins. Pathogenic LRRK2 influences the ability of cells to respond to cilia-dependent, Hedgehog signaling as monitored by Gli1 transcriptional activation. Moreover, cholinergic neurons in the striatum of LRRK2 R1441C mice show decreased ciliation, which will decrease their ability to sense Sonic hedgehog in a GDNF neuro-protective circuit that supports dopaminergic neurons. These data reveal a molecular pathway for regulating cilia function that likely contributes to Parkinson's disease-specific pathology.



UNRAVELLING INDIAN PUBLICATION PATTERNS; A 5-YEAR SCIENTOMETRIC PROFILING OF PUBMED REPORTS

Bioinformatics, Complex Network Analysis, Scientometrics, Mathematical Modeling, Social Network Analysis

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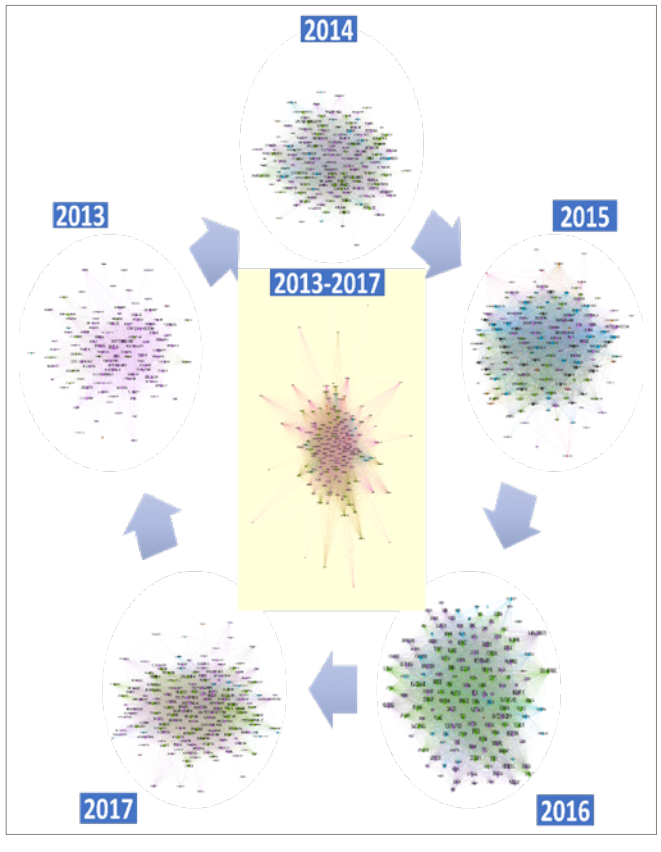
Scientometrics, more popularly known as bibliometrics is a meta-analysis approach to evaluate and understand the research productivity and patterns. The practices in this approach have been improved a lot in the past decades due to the advancement of data analytics. R is the most widely used open source tool which has the ability to perform scientometrics on huge literature data in an automated fashion. The aim of this study was to infer into the year wise changes in publication patterns of India in all disciplines of biology. PubMed was the initial source of raw data which were screened through Web of Science (WoS) server and only the Thomson Reuters indexed reports were chosen. The bibliometric data were downloaded from 2013 to 2017 and processed separately for individual years. With the help of bibliometrix package and R scripts, we performed the scientometric analysis on each year's data. The highest 31068 number of articles were obtained in 2016 and 2013 had 18644, the lowest. The annual growth percentage ranged from -58.51 to 383.46 which were observed in the year 2016 and 2015 respectively. The Average citations per documents were found to be in a descending order with the growth in years; lowest in 2017 which is 2.28 and highest with 12.75 in 2013. Among the foreign countries collaborating in these publications, the USA was found to produce the most number of reports. The country collaboration networks were constructed and the change in network topologies across all the 5 years was observed. The collaboration indexes were 2.18, 2.59, 2.5, 2.66 and 2.68 for the year 2013 to 2017 respectively. The network diameter was 2 for all the 5 years whereas the average degree ranged from 33.12 to 79.93. This piece of study provides insight into the publication patterns, especially collaborations of Indian institutes with foreign organizations.



IMPROVEMENT OF KIDNEY FUNCTION AND PRESERVATION OF GLOMERULAR FILTER RATE IN DIABETIC RATS ZSF1 FOLLOWING TREATMENT WITH LOW DOSES OF GDT-01

Molecular Biology, Molecular Endocrinology, Molecular Toxicology, Diabetic Nephropathy, Glomerular Diseases

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https://www.rushu.rush.edu/research/departmental-research/internal-medicine-research/nephrology-research/glomerular-disease-therapeutics-laboratory

Background

Study on rat diabetic nephropathy with the previously published sialic acid precursor N-acetyl D-mannose amine (Clement et al. Nature Medicine Jan 2011), worsened hyperglycemia in ZDF rats indicating a harmful long term effect. A 7-month period study was conducted using the compound GDT-01, to investigate the improvement of in vivo glomerular sialylation with the goal of studying its effect on chronic kidney diseases and hyperglycemia in rat diabetic nephropathy.

Methods

Five months old male ZSF1 rats (n= 6 rats per group) were treated for a period of over 7 months with tap water or GDT-01. Weekly measurement of proteinuria, BUN, creatinine, blood glucose, and other blood parameters, while renal histological changes were assessed upon termination of the study.

Results

A declining dose regimen of the compound GDT-01 was used, and the actual doses delivered (mg / Kg; mean + SE) over 3 separate periods were as follows: Period A, Days 0 to 95, 136.8 + 11.8; Period B, Days 96 to 122, 21.5 + 5; and Period C, Days 123 to 222, 3.7 + 0.2. Proteinuria was consistently lower in the GDT-01 treated group with in Period C (10/14 readings, $P<0.05$), occasionally in Period B (1/4 readings) and rarely in Period A (2/14 readings). However, blood glucose levels were similar between the GDT-01 and tap water groups, while BUN and creatinine were consistently lower in the GDT-01 treated group in Period C (BUN, 7/14, $P < 0.05$ to 0.01; creatinine, 5/14, $P<0.05$), and only occasionally in Periods A and B. A detailed renal histology and sialylation analysis are ongoing.

Conclusions

Low oral doses of the sialylation inducing compound GDT-01 improve GFR and proteinuria over a prolonged period without increasing hyperglycemia in ZSF1 diabetic rats.

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EFFECT OF ENVIRONMENTAL FACTORS ON FLORAL ODOUR AND CONSEQUENTLY PLANT-POLLINATOR INTERACTIONS IN ALPINE MEADOW PLANTS IN THE EASTERN HIMALAYAS

Plant-Insect Interactions, Chemical Ecology, Pollination, Herbivory, Climate Change

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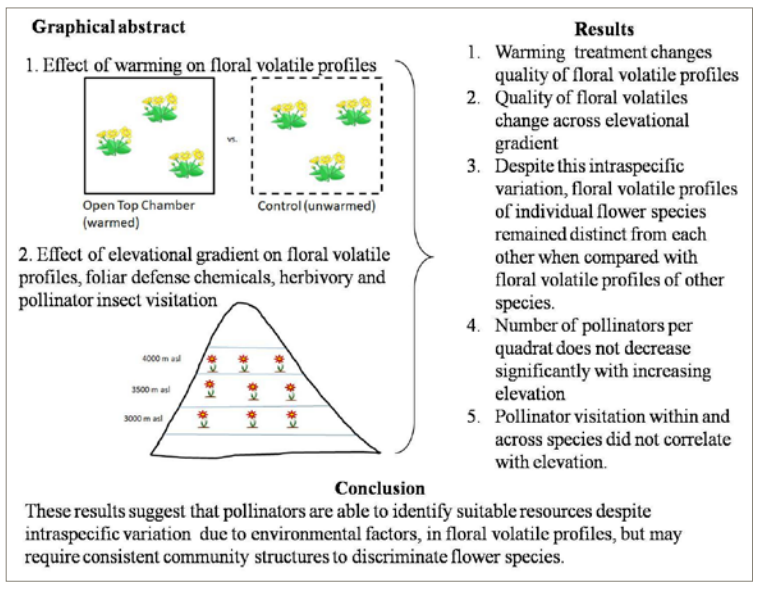
Many plant-pollinator interactions are mediated by floral odours which signal not only the identities of desired plant species, but also location and in some cases, even information regarding rewards such as nectar and pollen. The floral odour of a plant species therefore needs to be constant for the pollinators to recognize them. However, numerous environmental factors could potentially affect the emitted odour profile.

We investigated the effects of two factors, viz., elevational gradient (four species) and temperature (one species, using in situ warming chambers) on floral Volatile Organic Compounds (VOCs) of alpine meadow flowers of the Eastern Himalayas. This is because tropical alpine ecosystems are far less studied than temperate ones, and are also more vulnerable to global warming. We also investigated whether elevation affects pollinator visitation on these flowers.

We found intraspecific variation in floral VOCs across elevations. But floral VOCs of each species were more similar to each other, independent of elevations, than to those of other species, indicating constancy of species specific floral odour identities that overrides effect of habitat environments. Further, we observed no trend in pollinator visitation on same flower species across elevations, indicating that despite the intraspecific variation in floral VOCs, pollinators might still recognize a flower species as long as the plant community structure is stable. We also found that warming led to intraspecies variation in floral VOCs at the same elevation. This study contributes to our understanding of effect of climate warming on future plant-pollinator interactions in tropical alpine ecosystems.



MECHANOSENSITIVE BINDING OF P120-CATENIN REGULATES E-CADHERIN TURNOVER AND VISCOELASTIC BEHAVIOR OF TISSUES

Mechanotransduction , Tissue Mechanics, Physical Propagation Of Forces, Viscoelasticity, Developmental Biology

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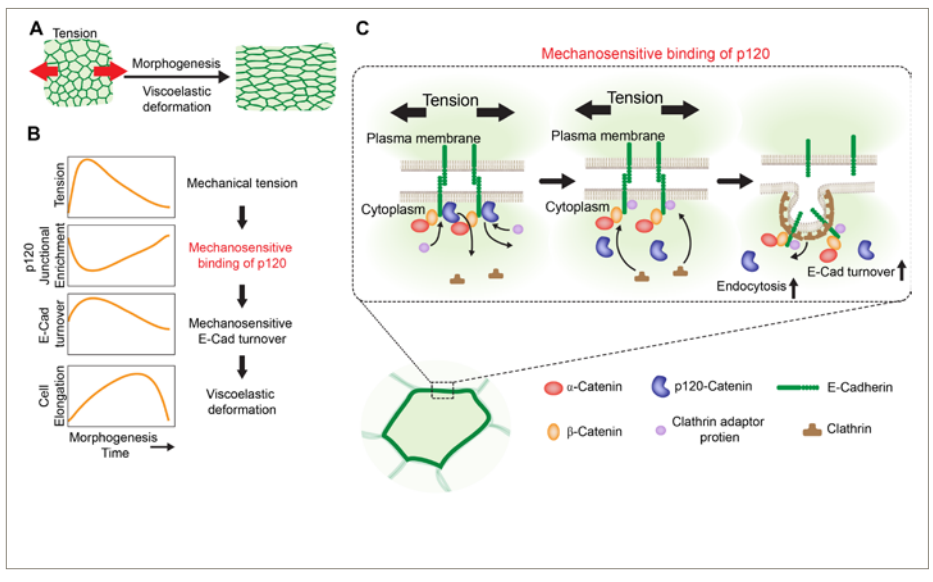
Studying how epithelia respond to mechanical stresses is key to understanding tissue shape changes during morphogenesis. Here, we study the viscoelastic properties of the Drosophila wing epithelium during pupal morphogenesis, by quantifying mechanical stress and cell shape as a function of time [1]. We find a delay of 6 hours between maximal tissue stress and maximal cell elongation indicating a viscoelastic deformation of the tissue. We show that this viscoelastic behavior emerges from the mechanosensitivity of endocytic E-Cadherin turnover. The increase in E-Cadherin turnover in response to stress is mediated by mechanosensitive relocalization of the E-Cadherin binding protein p120 Catenin from cell junctions to cytoplasm. Mechanosensitivity of E-Cadherin turnover is lost in p120 mutant wings, where E-Cadherin turnover is constitutively high. In this mutant, the relationship between mechanical stress and cell elongation is altered. Cells deform more rapidly in response to stress, indicating a lower viscosity. Unlike wild type, p120 mutant cells show much lower viscosity, reverting to their original shape when stress is relaxed. Taken together, our findings reveal that p120-dependent mechanosensitive E-Cadherin turnover regulates viscoelastic behavior of epithelial tissues, allowing mechanical stresses to generate stable cell shape changes during development.



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HUMAN T CELL RESPONSES TO CHIKUNGUNYA INFECTION

Dengue, Chikungunya, Epitope Mapping, Pathogenesis

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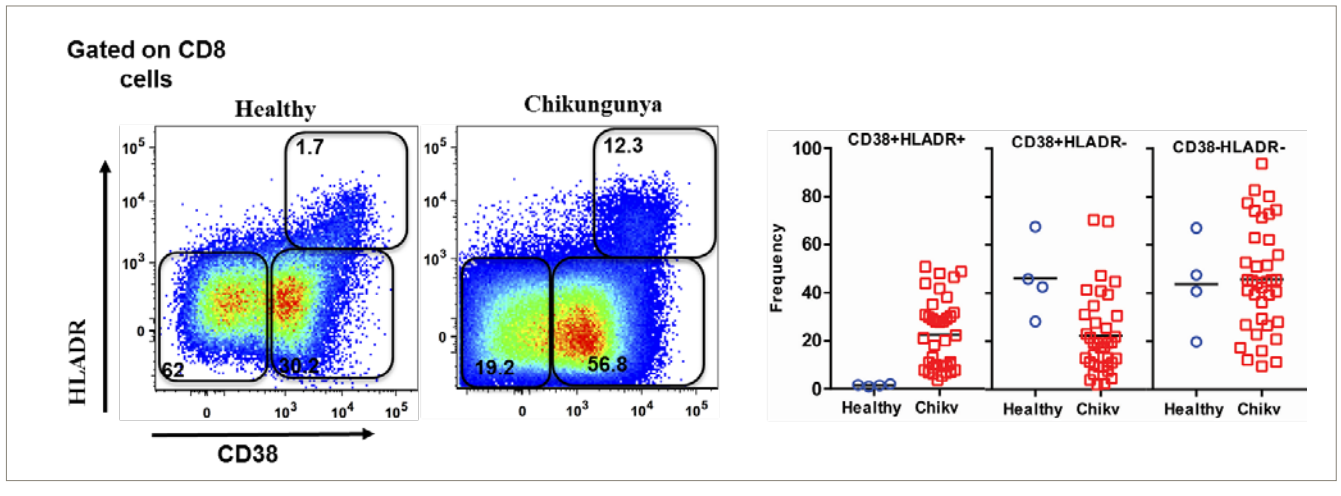
Chikungunya virus is expanding globally and continue to cause major public health threat to Indian populations. Vaccine efforts are underway, and it is hoped that these will eventually progress to human evaluation. However, currently we have little understanding of the phenotypes and functions of the human T cells in chikungunya patients, a knowledge that is essential for improving vaccine design/ testing and evaluation efforts. Here, we provide a detailed analysis of the CD8 T cell responses in chikungunya patients from India. We found that CD38+HLADR-38+ CD8 T cell subset expanded dramatically in chikungunya febrile patients with frequencies averaging about 20% of the total CD8 T cells, and reaching as high as 50% of the CD8 T cells in some patients. The frequencies of these activated CD8 T cells were substantially low and barely above background levels in afebrile patients reporting to the clinic with persistent arthralgia/ arthritis that was lasting for more than 30 days. These massively expanding CD8 T cells observed in the acute febrile patients were highly proliferating (ki67+), robustly expressing markers indicative strong Th1 differentiation (T-bet+), cytotoxic functions (Perforin+) and inflammatory/ synovial tissue homing characteristics (CX3CR1 and CXCR4). Interestingly, antigen-stimulation mediated IFN-g producing functions of these cells was highly compromised, reminiscent of the "cytokine stunned" phenotype seen in other situations such as human dengue or hepatitis C infections. Taken together, these results suggest that these highly differentiated effector CD8 T cell that were massively expanding during acute chikungunya febrile infection might be involved in protection by homing to infected tissues and eliminating infected targets rather than causing inflammation.



EXPOSURE OF LACTOBACILLUS SP. RETAINS FUNCTIONAL EUBACTERIA SPECIES ALONG WITH TRANS-DOMAIN HEALTHY GUT DIVERSITY.

Bacterial Genomics, Human Microbiome, Functional Foods, Metabolic Disorder, Clinical Trials

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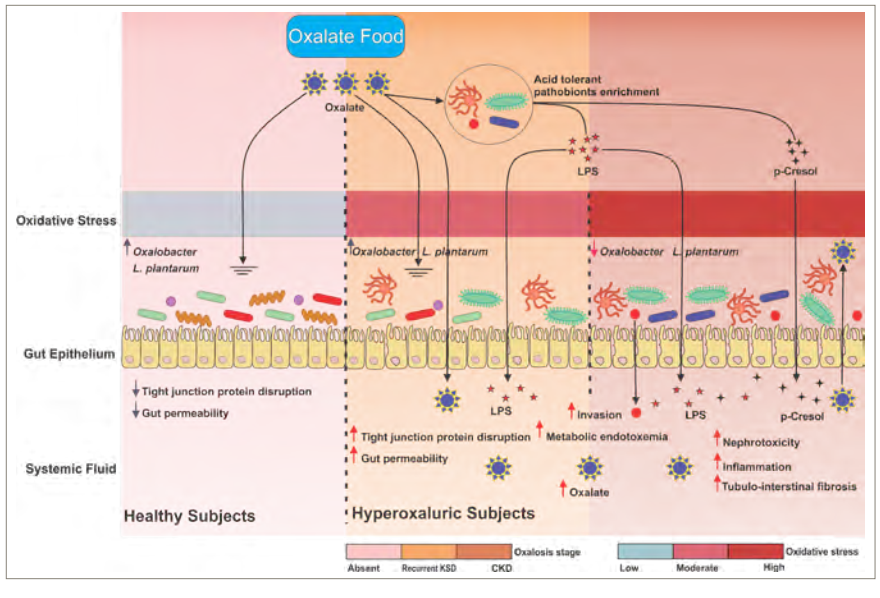
Lactic acid bacteria (LAB) are the abundant and coherent species in the human gut, and most of times demonstrated for disease correlation in microbiome studies. Certain LAB is known as Oxalate metabolising Bacterial Species (OMBS) and their colonization are inversely linked to the prevalence of hyperoxaluria condition. Such OMBS are now being focused for research and some are formulated as the therapeutics for the metabolism related complications. In the present study, we evaluated the colonization pattern of *L. plantarum* in the tested Indian cohort through the bioinformatics and their exposure to shape in other eubacterial species diversity under in-vivo condition. In silico surveillance of *L. plantarum* for their OTUs abundances has been covered from selected Indian population study (Under review at Scientific Reports, 2018). Whereas biome file was used for the statistical exploration. We utilised fully characterized oxalotrophic *L. plantarum* E2C2 and E2C5 strains from Indian gut (PMID: 28163824) for the hypothesis testing. In pre-clinical trial experimentation used animal models for the hyperoxaluria condition (C57BL/6 mice, n=25, treatment group=10) whereas induction of hyperoxaluria through sodium oxalate ingestion. All the parameters including like urine oxalate, gut microbiome and kidney histology were recorded and compared with control group (n=10). We analysed the sequences to their OTUs and observed that the dysbiosis in gut microbiota is not just limited to eubacteria species, but also to other domains like archaea and eukaryotes. We found that 20.08% of healthy eubacterial population retained wherein *Oxalobacter formigenes* and Lactobacillus sp. colonization in disease condition observed. Oxalate metabolizing bacterial species (OMBS) and butyrate producing eubacteria species were found to be decreased in Lactobacillus non-colonizers and in recurrent episodes. Our study underscores the need of genomics for further intervention studies.



COMPARATIVE GENOMICS OF Î²-LACTAMASE GENES Â€“ A GOLDMINE FOR RAPID ANTIMICROBIAL RESISTANCE (AMR) DETECTION AND NEWER Î²-LACTAMASE INHIBITORS ?

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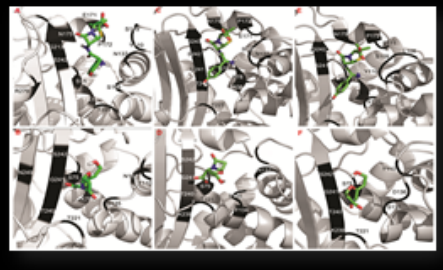
Antimicrobial resistance, Microbial pathogenesis, Comparative genomics, Diagnostics, Proteomics

Î²-lactam antibiotics have been a prime choice for treating a number of infectious diseases. However, their widespread & indiscriminate use has resulted in microbial resistance towards this important class of antibiotics. Bacteria hydrolyze these antibiotics using their intrinsic/acquired antibiotic modifying enzymes, the Î²-lactamases. Studies from our laboratory using comparative genomics of Î²-lactamase genes and their promoters in a large number of Y. enterocolitica and E.coli strains revealed that, though the promoters were conserved, point mutations were present in different Î²-lactamase genes. Similar observations were also made while compiling a database of Î²-lactamase genes. Identification of consensus sequences among the Î²-lactamase genes of different bacteria could be useful for developing rapid and simple methods for detection of pathogens harbouring these genes. Our studies also revealed that mutations at sites other than active site of the enzyme may create diverse local changes in the 3D structure of the enzyme which might affect its binding affinity with Î²-lactam antibiotics as well as Î²-lactamase inhibitors. These findings might be useful for designing better Î²-lactamase inhibitors with improved efficiencies in future. Currently, we are working on developing a rapid and simple Loop Mediated Isothermal Amplification (LAMP) test using Î²-lactamase genes for detection of food-borne pathogens. Primer sequences and reaction conditions have been identified for detection of Y. enterocolitica. The work would be extended to detection of other food-borne pathogens. We are also working to identify novel sequences in Î²-lactamase genes which can be used as ideal targets for designing newer Î²-lactamase inhibitors. These studies would surely help us make assessments of the true potential of Î²-lactamase genes to serve as markers for rapid detection of AMR and salvaging several Î²-lactam antibiotics by designing novel Î²-lactamase inhibitors.

Molecular docking of blaA variants present in different biotypes



Molecular docking analysis of the blaA variants of *Y.enterocolitica*. Molecular interactions of docked blaAx (A & B), blaAy (C & D), blaAz (E & F) with amoxicillin and clavulanic acid respectively. The two antibiotics are represented by stick and the interacting aminoacids are shown in dark color

ODOR ARRIVAL SIDE DISCRIMINATION IN *Manduca sexta*

Animal behavior, Sensory Ecology, Pheromone Communication, Multisensory Integration, Neural Basis of Behavior

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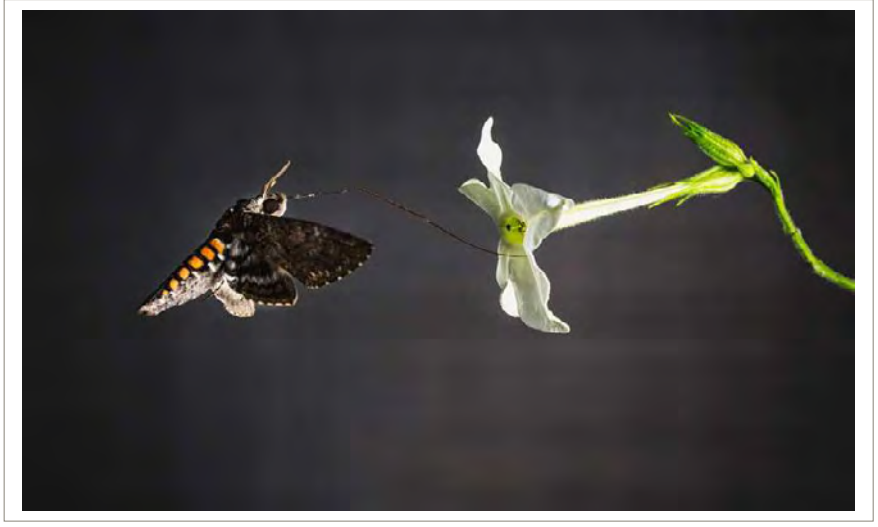
The hawkmoth *Manduca sexta* uses odors to find mates, food and egg-laying sites. Odor molecules are distributed by moving air into a non-uniform, patchy cloud known as a plume. While tracking an odor plume, *M. sexta* drive their antennae through the odor plume along a zigzagging flight track in a relatively narrow range of flight speeds. Their flapping wings also draws air and odor through their antennae. These behaviors could work together to sample the odor environment both in space and time and this information may be used to alter steering maneuvers to maintain contact with the plume. One element of this spatial odor information could come from bilateral comparisons between the two antennae. The prerequisite for this strategy to work is that the moth must be able to discriminate which antenna is being stimulated. To address this question, we designed an odor arrival side discrimination task based on the proboscis extension reflex conditioning. Proboscis extension was monitored by recording electric potentials generated by the cibarial pump muscle that is involved in drawing nectar up the moths' proboscis. Initially, the moths were presented 10-12 trials of conditioning stimuli i.e., odor stimulation to one of the antennae was associated with sucrose reward. Following this, moths were tested for cibarial muscle activity when odor was presented from either the associated or unassociated side. The moths were expected to generate cibarial muscle potentials when presented with odor on the associated side and remain relatively quiet during odor presentation to the other side. Moths discriminated the odor arrival side with an accuracy of >70%. These results show that moths may be able to determine the odor plume location in 3D space, and use this information to control the turning maneuvers used during plume tracking.



SPATIO-TEMPORAL REGULATION OF RNA DURING DEVELOPMENT AND DISEASE

RNA Granules, Post Transcriptional Gene Regulation, Reprogramming/ Pluripotency, RNA Binding Proteins, Neurodegeneration

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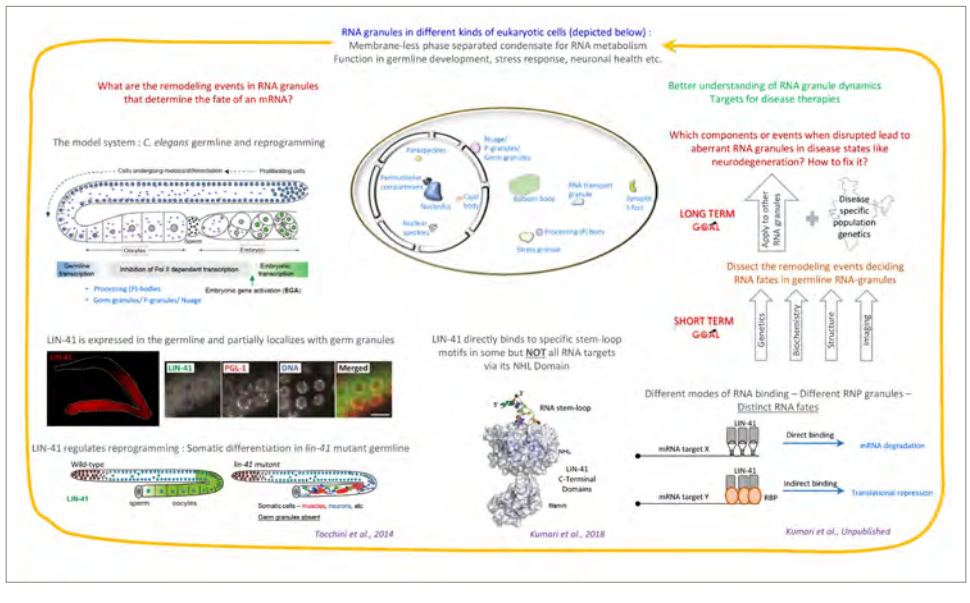
RNAs that are specifically regulated in time and space are captured in ribonucleoprotein (RNP) complexes. Certain components of RNPs promote assembly into phase separated granules that function as membrane-less organelles for RNA metabolism. Some specific examples are P-bodies, stress granules and germ granules. RNA granules function in post-transcriptional gene regulation in many cellular contexts ranging from developmental programs in oocytes and embryos to synaptic plasticity in memory formation. They are also implicated in viral infection and neuro-degeneration. Different RNA granules share mechanisms underlying their assembly and disassembly. However, very little is known about the dynamic remodeling events that underlie entry and exit of mRNAs thereby regulating their fate. I aim to study germline RNPs that regulate pluripotency as a model to dissect these remodeling events.LIN-41, an RNA binding protein, regulates the onset of pluripotency in the *C. elegans* germline. In addition to direct RNA binding via its NHL domain, the specificity of LIN-41 towards majority germline mRNA targets is mediated by another RBP functioning in a complex with LIN-41. Moreover, while direct RNA targets of LIN-41 are degraded, the indirect targets are translationally repressed. My results suggest that LIN-41 works in different RNP granules to confer distinct fates to RNA targets. Therefore, I will use LIN-41, its partner proteins and RNA targets as candidates to study remodeling events in RNP granules. Components of the siRNA pathway that localize to germ granules and have been implicated in pluripotency, will provide additional candidates for analysis. I plan to use a combination of genetics, structural biology and imaging approaches to dissect the dynamic events in RNA granules. The findings of this project will potentially lead to better understanding of the different types of RNA granules and their organization in the context of normal cell fate specification and in disease.



ALPHA-SYNUCLEIN PRE-FORMED FIBRILS DISRUPT THE SPONTANEOUS FIRING PATTERNS OF SUBSTANTIA NIGRA NEURONS

Parkinson's Disease, Alpha-Synuclein, Animal Models, Neurodegeneration, Dopamine

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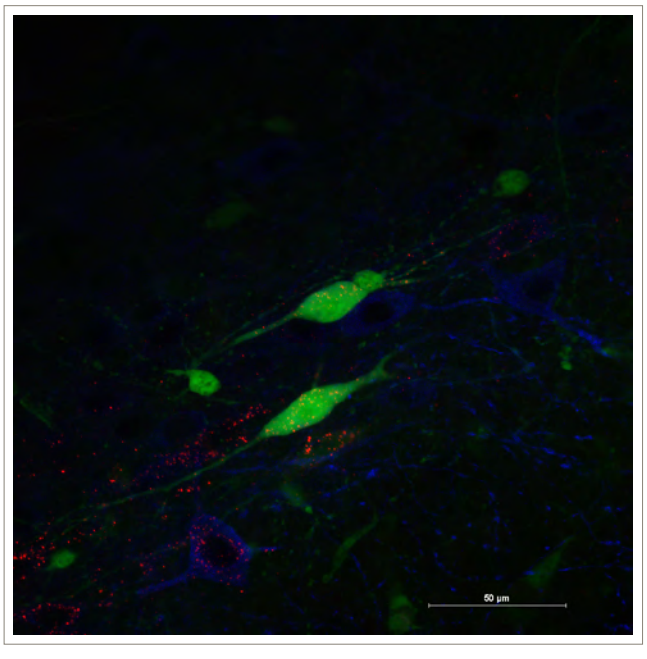
Background
Alpha-synuclein aggregation is a well-known pathophysiological hallmark of Parkinson's disease (PD). Present study aimed to delineate how the alpha-synuclein pre-formed fibrils (pff) affect the firing patterns of dopaminergic (DA) substantia nigra (SN) neurons, which is the main neuronal population affected during PD. SN DA neurons are a heterogenous group of neurons with lateral SN neurons being more vulnerable to neurodegeneration compared to medial neurons.

Methods
Male C57Bl/6N mice (3-4 month) were used for the study. Medial SN neurons project mainly to dorso-medial striatum while lateral SN neurons project to dorso-lateral striatum. To identify these two subpopulations red fluorescence beads were injected in either dorso-medial striatum or dorso-lateral striatum that retrogradely travel to SN. After 3-4 days, acute mid-brain slices were prepared and autonomous tonic firing of DA SN neurons was studied by whole-cell patch-clamp recordings for 300 seconds. Some neurons were also exposed to 0.5 nM pff during recording.



Results
Medial SN neurons were characterized by an average firing frequency of around 2 Hz which was mostly unaffected by the fibrils. On the other hand, lateral SN neurons fired at an average frequency of 3 Hz. However, lateral SN neurons exposed to 0.5 nM pff displayed a significant decline in firing frequency (1 Hz). Drop in firing frequency was also accompanied by occasional bursting and hyperpolarization of the membrane. Further, pff caused a significant decrease in the regularity of firing in lateral SN neurons.

Conclusion
Preliminary data suggests a disruption of normal firing patterns by pff in lateral SN neurons but not in medial SN neurons. This higher vulnerability of lateral SN neurons to alpha-synuclein is in line with their higher propensity to degenerate during PD. Mechanisms of firing pattern disruption are also being further explored.

OMICS TECHNOLOGIES FOR DRUG DISCOVERY AND DISEASE DIAGNOSIS/PROGNOSIS

Mathematical Modelling In Biology, High Throughput Data Analysis, Systems Biology, Diagnostics, Drug Discovery

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Omics technologies like genomics, proteomics, metabolomics etc. are nowadays routinely used in drug discovery applications[1,2] as well as in disease diagnosis[3] and prognosis[4]. The presentation will elaborate on the work done to find drugs against tuberculosis using proteomics data from Mtb infected macrophage cultures. A novel methodology of integrating proteomics data with biological networks will be elaborated. On disease diagnosis/prognosis side, the usage of metabolomics data from healthy human subjects to predict future risk of diabetes will also be discussed. Here also, state of art machine learning algorithms used to connect metabolomics data with disease state will be discussed.

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WATER QUALITY INDEX AND ROLE OF MICROBIAL COMMUNITY IN THE LOKTAK LAKE, THE LARGEST FRESH WATER LAKE IN INDO-BURMA BIODIVERSITY HOTSPOT

Wetland, Water Quality, Land Use Land Cover Changes, Microbial Biotechnology, Sustainable Development

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Water quality is an important factor touching on all aspects of ecosystems and human well-being and a significant tool in determining the state of human poverty, wealth and education levels. The understanding and relationships between the microbial communities and environmental factors is critical for understanding the stability and functioning of wetlands. The aim of the present study was to assess the water quality and microbial diversity in Loktak Lake. Water and sediment samples from five locations were collected and analysis for 20 physicochemical parameters. Metagenomic DNA was extracted using 1 g of the sample using the commercial kit following manufacturer's protocol to unravel the composition of the microbial community at different land use. The result of the water quality index range from 64 to 77 indicating that the lake water is polluted and not fit for drinking purpose even though the local people are using it for drinking purposes. The bacteriological analysis shows the presence of Coliform and E coli in all the samples which are beyond the permissible limit of BIS, which indicates the contamination of fecal materials in the lake water. The people perception study also identified the increasing in pollution. Among the bacteria Proteobacteria was found to be the most dominant bacterial phylum with 34.62% follow by Acidobacteria (25.8%), Actinobacteria (9.34%) and Plantomycetes (6.55%). The study identified the need to mitigate the anthropogenic activities in and around the lake and proper disposal of waste. This approach will help in improving the water quality of the lake and prevent health issues due to drinking of the polluted water and sustainable conservation of the Loktak Lake.

Description	Lotkak L1 (%)	Lotkak L2 (%)	Lotkak L3 (%)	Lotkak L4 (%)	Lotkak L5 (%)
Phylum	Proteobacteria (34.62)	Proteobacteria (35.11)	Proteobacteria (32.15)	Proteobacteria (34.65)	Proteobacteria (38.53)
Class	Alphaproteobacteria (20.17)	Alphaproteobacteria (21.22)	Alphaproteobacteria (17.22)	Alphaproteobacteria (15.61)	Alphaproteobacteria (24.24)
Order	Rhizobiales (9.99)	Rhizobiales (10.6)	Rhizobiales (11.97)	Rhizobiales (6.98)	Rhizobiales (10.89)
Family	Hyphomicrobiaceae (6.58)	Hyphomicrobiaceae (7.39)	Hyphomicrobiaceae (6.54)	Unclassified family from iii1-15 order (5.51)	Rhodospirillaceae (5.23)
Genus	*Rhodoplanes (5.42)*	*Rhodoplanes (5.94)*	*Candidatus Solibacter (5.44)*	*Unclassified genus from iii1-15 order (5.51)*	*Unclassified genus from Rhodospirillaceae family (4.71)*
Species	*Unclassified species from Rhodoplanes genus (5.42)*	*Unclassified species from Rhodoplanes genus (5.94)*	*Unclassified species from Candidatus Solibacter genus (5.44)*	*Unclassified species from iii1-15 order (5.51)*	*Unclassified species from Rhodospirillaceae family (4.71)*

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GEOMETRY OF THE CELL NICHE REGULATES ARCHITECTURE OF CELL MATRIX ADHESIONS THAT DICTATES CELLULAR RESPONSE

Cell Matrix Adhesions, Functional Architecture, Super Resolution Microscopy, Cell Material Interactions, Stem Cells

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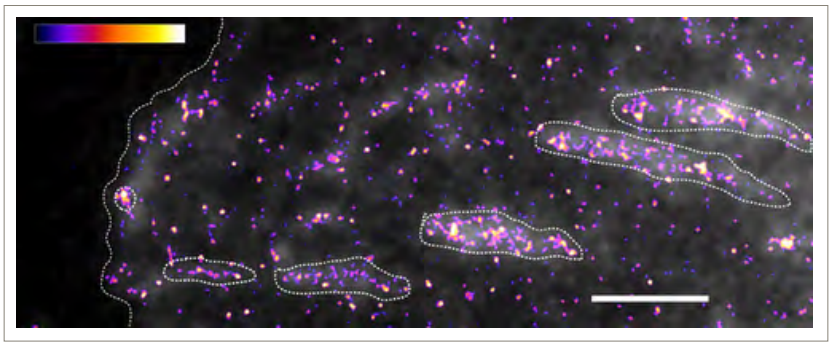
https://mbi.nus.edu.sg/mechanisms/rishita-changede/

Attachment to the extracellular matrix (ECM) via integrins plays a major role in stem cell maintenance and fate. Mutations in integrin or alterations in ECM of the stem cell niche leads multiple disease in several organs. Biophysical features of the ECM such as geometry and force are sensed by integrin mediated cell-matrix adhesions that modulate cell behavior via an enigmatic process of mechanotransduction [1]. In this study, we mimicked small nanofiber geometry of stem cell niche ECM. We observed that adhesions cannot form on a single fiber but forms on two or more closely spaced fibers. These adhesions are formed by modular integrin clusters containing liagnd bound and unbound integrins that bridged the fiber mesh. This bridging organization of integrins was confirmed using multiple integrin point mutants. Using Talin mutants, we show that Talin is a major crosslinker that can bring integrins together. We discovered that these clusters are the primary signaling centers within the adhesion. We observed that the cluster regions are long lived and provide a plaque for activation of several proteins such as focal adhesion kinase (FAK). Non-phosphorylatable mutant of FAK was not retained at these cluster indicating that they are sites for mechanosignaling. In support of this hypothesis, we also observed that integrin clusters recruit ligand independent EGFR specifically on rigid substrates and not on soft substrates. This EGFR signaling was necessary for cell mechanoresponse [2]. Taken together, these results demonstrated that a mesh of ECM fibers is required that seeds modular integrin cluster. Using mutants we showed that these clusters are the sites of differential signaling events depending upon rigidity suggesting that they could be novel targets for therapy.



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HOST DIETARY SPECIALIZATION AND NEUTRAL ASSEMBLY SHAPE GUT BACTERIAL COMMUNITIES OF WILD DRAGONFLIES

Microbial Ecology, Community Structure and Assembly, Metagenomics and Statistical Modelling, Host-Gut Microbiome Interaction, Host Behaviour and Evolution, Bioacoustics

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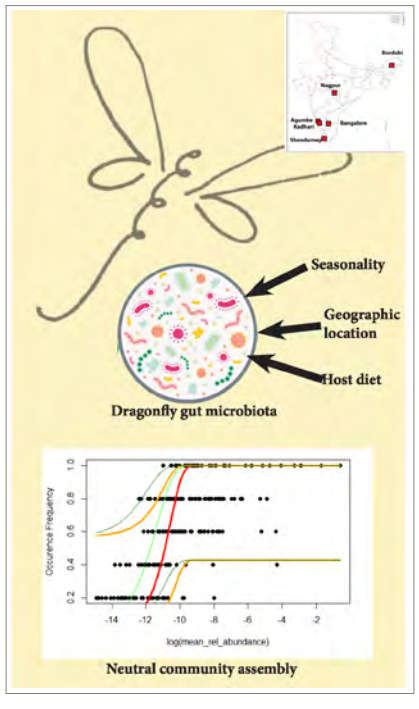
Host-associated gut microbial communities can have large impacts on host ecology and evolution and are typically shaped by host taxonomy and diet [1] . Different host species often harbour distinct microbial communities, potentially because (1) host dietary specialisation determines microbial colonisation, (2) host-specific selection acts on diet-acquired microbiota, and (3) a combination of both processes. While the first possibility involves passive community structuring, the other two may arise from a functional association and should produce stable microbial communities. However, these alternatives have rarely been tested in wild host populations. We used 16S rRNA amplicon sequencing to characterise the gut bacterial communities of six dragonfly species collected across multiple seasons and locations. We found that variation in bacterial community composition was predominantly explained by sampling season and location, and secondarily by host species. To distinguish the role of host dietary specialisation and host-imposed selection, we used insect-specific primers to identify prey in the gut contents of three focal dragonfly species. We found that these dragonflies –considered to be generalist predators– consumed distinct prey, with seasonal diet variation. Together, the patterns of host dietary specialisation and spatial and temporal variation suggest a strong role of passive processes in shaping the gut bacterial community. Indeed, the abundance and distribution of ~76% of the bacterial community members were consistent with neutral community assembly. Our results contradict the pervasive expectation that host-imposed selection shapes gut microbial communities, and highlight the importance of joint analyses of variation in host diet and gut microbial communities of natural host populations.



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PHOSPHO-REGULATION OF TROPOMYOSIN IS CRUCIAL FOR ACTIN FILAMENT TURNOVER

Cell Division, Genetic Code Expansion, Synthetic Biology, Actomyosin, Cardiomyopathies

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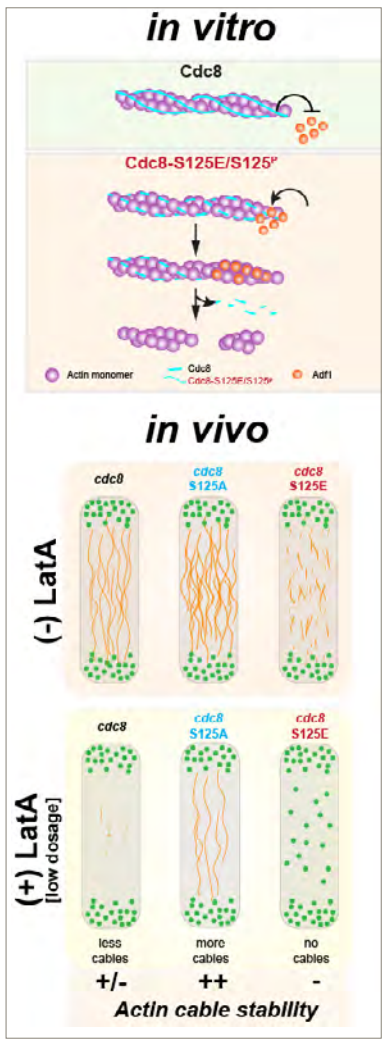
Actin filaments, which constitute one of the major cytoskeletal networks in eukaryotes, are known to exhibit a high rate of turnover. The mechanisms that regulate the half-life of actin filaments in various cytoskeletal structures are not well understood, although actin monomer pools, actin nucleators, and actin severing proteins, are all involved. Here, we describe a phosphorylation-controlled mechanism for actin cable turnover in *Schizosaccharomyces pombe*.



We show that the *S. pombe* tropomyosin (Cdc8) is phosphorylated on serine-125. Phosphorylation of Cdc8 does not affect its structure and stability, but leads to a weaker interaction with actin filaments, as judged by total internal reflection fluorescence (TIRF) microscopy and co-sedimentation assays. Furthermore, phosphorylation-mediated release of Cdc8 from actin filaments facilitates occupancy of filaments by the actin severing protein Adf1 and subsequent filament disassembly. In in vivo experiments, phospho-mimetic mutants of Cdc8 showed decreased actin cable stability and, conversely, a non-phosphorylatable Cdc8 mutant show increased actin cable stability. These data indicate that phosphorylation of tropomyosin causes its release from the actin filament, permitting association of Adf1 and actin cable disassembly. In muscle cells, the interaction between tropomyosin and F-actin is regulated by calcium mediated allosteric movement of tropomyosin, which in turn allows myosin II to access F-actin. Non-muscle tropomyosins, including *S. pombe* tropomyosins, are not under calcium regulation. Our work shows that phosphorylation of tropomyosin may provide a mechanism for a regulated interaction between F-actin and actin binding proteins such as Adf1.

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HIGH MITOGENIC STIMULUS INDUCED BY VEGF OR NOTCH INHIBITION ARRESTS PHYSIOLOGICAL AND TUMOR ANGIOGENESIS

Inducible Fluorescent Genetic Mosaics, Angiogenesis, Cell Signaling, Notch/Dll4, DNA Damage

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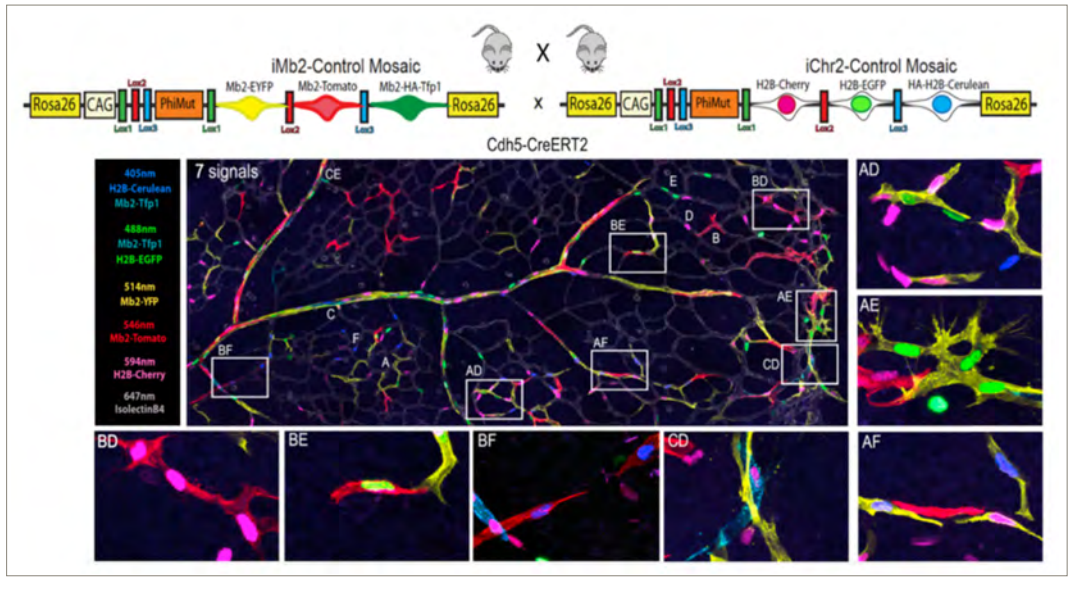
VEGF elicits endothelial sprouting and proliferation, whereas physiological Dll4/Notch signaling acts as a rheostat that inhibits this effect. In a genetic pulse-chase mosaic experiments we found that endothelial cells (ECs) with higher VEGF or lower Notch signaling are outcompeted during physiological and tumor angiogenesis. We show that a high VEGF input in sprouting ECs is associated with high ERK activity and Cdkn1a expression, which induces cell cycle exit. This process is normally attenuated by Notch activity in angiogenic stalk-ECs, enabling a longer-lived cell cycle. Accordingly, inhibition of Notch activity in angiogenic ECs induces premature cell cycle exit, whereas in quiescent vessels it induces cell cycle entry. Mechanistically, we show that these VEGF and Notch context-dependent effects involve tight regulation of ERK and Cdkn1a, which control the balance between proliferation and cell cycle arrest. We have used new inducible fluorescent genetic mosaic (ifgMosaic) mouse lines and different pharmacological treatments to understand with high cellular and temporal resolution the role of Notch, VEGF, ERK and p21 in the proliferative behavior of angiogenic tip and stalk cells, and quiescent ECs. This approach allowed us to find that in contrast to physiological angiogenesis, tumor angiogenesis is highly inefficient. Modulating this endothelial cell cycle gatekeeper mechanism in tumors might be a useful way to reinforce the effects of angiogenesis inhibitors in cancer. Whereas anti-angiogenesis therapy targets mainly angiogenic vessels, pro-mitogenic drugs can target also the more mature and functional tumor vessels, inducing biological changes that abnormalize them, which ultimately reduce their ability to nurture tumor growth.



EPIDERMAL DIFFERENTIATION- A SURVIVAL ADAPTIVE RESPONSE IS MEDIATED BY LYSOSOME BIOGENESIS

Cell Biology, Protein Trafficking, Organelle Biogenesis, ER Stress and Autophagy, Tissue Homeostasis

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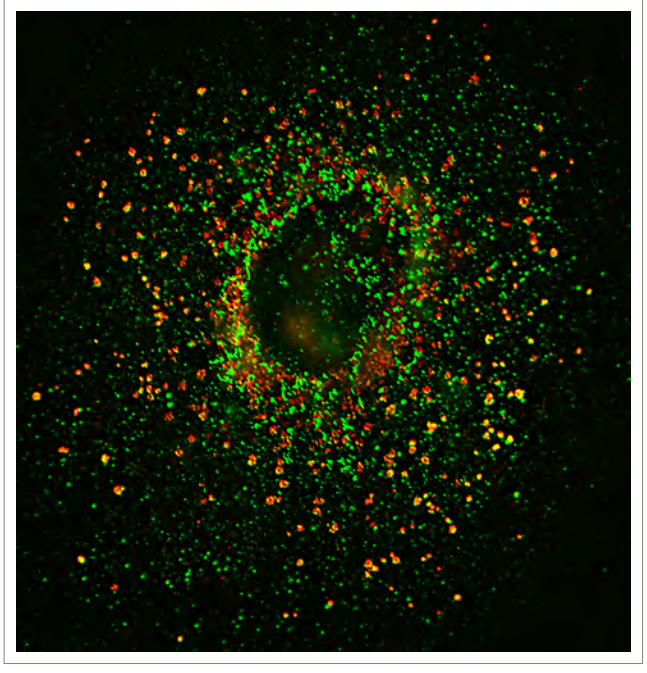
Biological systems work to maintain homeostasis. Epidermis of human skin provides protection and maintains homeostasis of the body. Cells in the upper epidermis are constantly exposed to ionic/radiation/pathogenic stresses including nutrient scarcity. We have questioned how keratinocytes, the predominant cell type of epidermis survives with these adversities and maintain its function. Previous studies have shown that keratinocytes differentiate in presence of high extracellular calcium in vitro, by an unidentified cellular mechanism/s [1]. By employing primary human keratinocytes, we found a correlation between intracellular calcium - differentiation and organelle biogenesis/distribution.



Keratinocytes undergo characteristic differentiation of stratum spinosum/ granulosum layers of skin upon incubation with 2 mM CaCl2 for more than 48 h. Continued incubation of calcium for 6-9 days results in terminal differentiation which resembles cornified envelope of skin, creating an in vitro epidermal model system. Interestingly, differentiated keratinocytes generate large number of globular lysosomes distributed throughout the cytosol and tethered with Golgi-tethering factors such as GM130/Golgins, named as Golgi-associated lysosomes (GALs).

Intracellular free Ca2+ measurement revealed an initial flux which then decreases and stabilized with the differentiation process. Further, this initial calcium flux generates ER stress, possibly increases the lysosome biogenesis through ATF6 transcription factor independently of mTOR signaling. In line, chemical modulation of intracellular calcium or ER stress, potentially block the epidermal differentiation and concomitant lysosome biogenesis. Taken together, our results suggest that increased organelle biogenesis possibly attain intracellular ion/nutrient homeostasis, which in turn maintains skin integrity.

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UNMASKING THE AMYLOID FACE OF ALS AND FTLD ASSOCIATED TDP-43 FIBRILS

Protein Biotechnology. Amyloid Aggregation, Neurodegenerative Disorders, Phase Separation, Therapeutics

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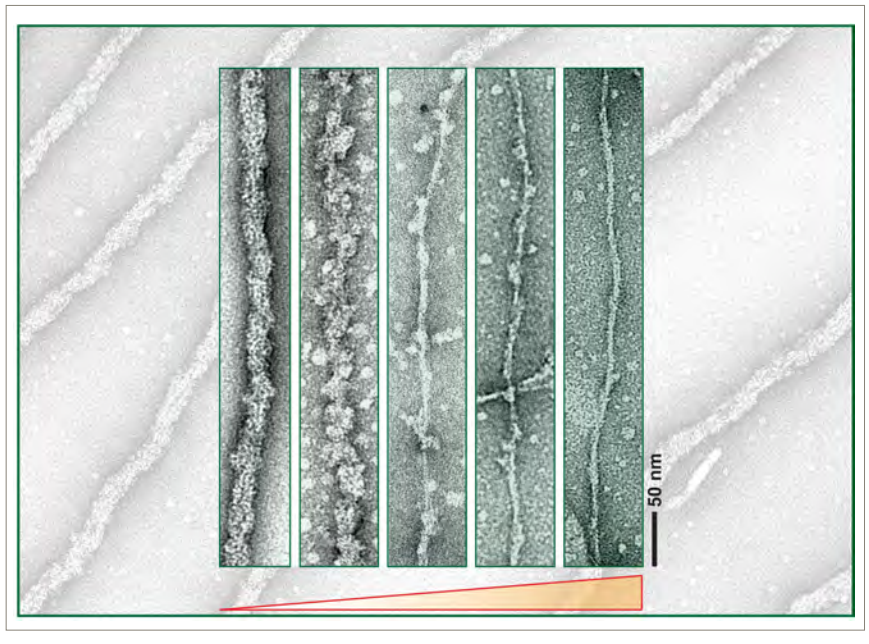
Amyotrophic Lateral Sclerosis (ALS) and frontotemporal lobar degeneration (FTLD), classified under motor neuron diseases, are fatal neurological disorders pathologically characterized by the presence of abnormal neuronal inclusions containing TDP-43 protein aggregates. Unlike other pathological amyloids such as Tau or alpha-synuclein, amyloid properties of TDP-43 in vitro and in vivo are poorly understood. The high aggregation tendency of TDP-43 has hampered designing studies to identify the molecular players keeping the protein in active form physiologically and regions forming the amyloid aggregation core in pathological inclusions. Therefore, it is utmost important to investigate the molecular determinants of the functional TDP-43 and the structural insights into how TDP-43 ends up as aggregates in the dying neurons to understand the disease mechanism and to develop novel strategies for diagnostic and therapeutic purposes. Here, we show full-length TDP-43, under established conditions, formed fibrils comprising beta-sheet as their secondary structure but binds very weakly to an amyloid binding dye called Thioflavin T (ThT). Upon limited proteolysis of fibrils, we identified the thinner but protease resistant fibrillar core that binds readily to ThT and thereby exposes the amyloid character which is buried within. Following MS-MS analysis, we identified the possible peptide region forming the amyloid core of TDP-43 fibrils. Synthesised peptides from the fibrillar core readily form the fibrils that recapitulated the bonafide amyloid properties. Our study explains the ambiguity in the amyloid nature of TDP-43 aggregates witnessed in ALS/FTLD cases. The development of a robust system for producing native TDP-43 combined with the identification of the amyloid core also offers unique opportunities to screen for small molecule and antibodies that modulate TDP-43 aggregation and toxicity, thus paving way for the novel therapies for the treatment of ALS and FTLD.



MICROBIOME MEDIATED DIETARY SPECIALIZATION IN A NECROPHAGOUS INSECT

Ecology, Evolutionary Biology, Entomology, Microbiome, Symbiosis

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Ephemeral resources such as carrion have high nutritive value and are severely contested, which select for rapid exploitation by organisms. Proliferation of microbial decomposers can render the resource unpalatable and toxic, reducing the fitness of competing animals. However, some insects specialize in feeding on decomposing carrion, but the effects of insect behavior on its microbial communities and its biochemical properties remain poorly understood.

Here, we demonstrate that the burying beetle *Nicrophorus vespilloides*, which uses small carcasses for breeding, successfully manages carrion microbiota by preventing microbial succession that is typically associated with putrefaction. Instead, beetles inoculated a symbiotic microbial community that prevented the buildup of toxic metabolites in carrion on which the larvae fed. The beetles suppressed the growth of certain microbial decomposers, but promoted the growth of a fungal symbiont that produced extracellular digestive enzymes on the carcass [1]. Apart from a source of nutrition, the carrion serves to transmit a core symbiotic community to the larvae [2], which is required for optimal larval development. Such adaptive modification of the environment sheds light on the role of the microbiome in enabling dietary specialization in insects, and demonstrates how insects modify their habitats to enhance fitness.

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PRO-TUMORIGENIC PHENOTYPE OF P53 IN CANCER ASSOCIATED FIBROBLASTS

Cancer Biology, Tumor Microenvironment, P53, Molecular Biology

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Introduction

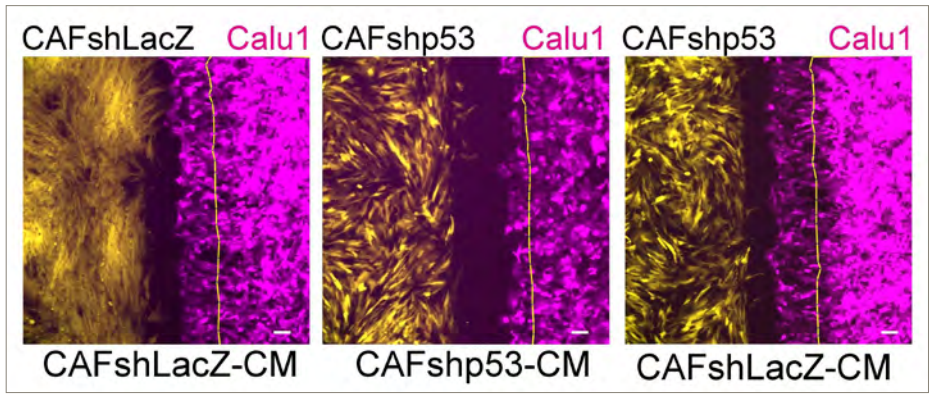
p53 is a seminal tumor suppressor protein, acting as a major barrier against cancer. Cancerous mutations in the TP53 gene, resulting in production of mutant p53 proteins, can lead not only to loss of its tumor suppressive functions, but often also to gain of tumor-promoting activities. Of note, alterations in the regulatory networks that impinge on p53 may cause genetically wild type (wt) p53 to acquire features resembling bona fide mutant p53. However, the extent and functional impact of p53 conversion from its canonical tumor suppressive state into such "pseudomutant" states in actual human tumors remains to be determined.

Hypothesis

So far, p53 research has focused primarily on its cell-autonomous functions. Yet, p53 can also exert non-cell-autonomous effects on tumor development. Within the tumor microenvironment, cancer cells are surrounded by non-cancerous adjacent cells. Specifically, Cancer-Associated Fibroblasts (CAFs) contribute in multiple ways to tumor progression. To identify the possible roles of p53 in CAFs, we investigated the contribution of p53 to the gene expression landscape and the biological properties of CAFs, relative to normal fibroblasts (NFs).

Results and Discussion

Global transcriptome analysis displayed substantial differences in the transcriptional impact of p53 between CAFs and NFs. Functional studies revealed that CAF p53, while remaining genetically wt, contributes to the activated fibroblast phenotype of CAFs and promotes non-cell-autonomously tumor cell migration and invasion. Furthermore, the cancer-promoting role of CAF p53 was confirmed by co-injection of fibroblasts and tumor cells into SCID mice. Remarkably, co-cultivation with cancer cells rendered the transcriptional impact of NF p53 more similar to that of CAF p53. Overall, our study highlights for the first time a novel function of non-mutated p53 in the tumor microenvironment.



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INTEGRATION OF SPATIAL AND TEMPORAL CUES IN DROSOPHILA EMBRYONIC NEUROBLASTS

Development, Neurobiology, Evo-Devo, Circuits, Drosophila

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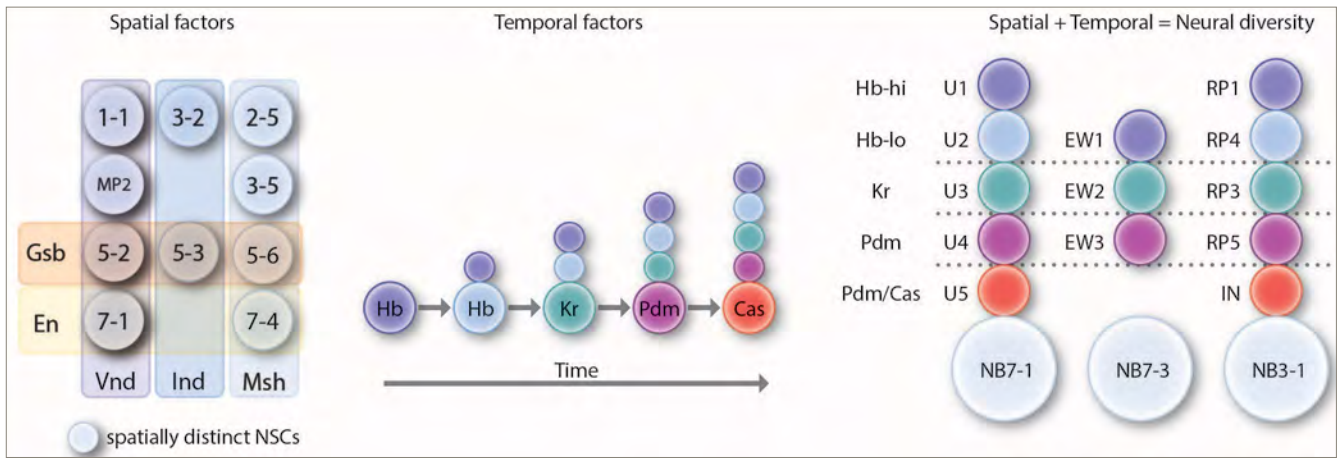
During mammalian and Drosophila neurogenesis, spatial cues generate regionally distinct progenitor populations, and subsequent temporal cues generate further diversity within each progenitor lineage [1] . Yet in no organism is it known how spatial and temporal cues are integrated. They could act independently - their combinatorial activity specifying unique neural identities - or sequentially such that one axis influences the activity of the other (e.g. spatial transcription factors, STFs, influence temporal transcription factors, TTF, binding). We test these models by assaying the targets of the TTF Hunchback/Ikaros (Hb) in two adjacent Drosophila progenitors using Targeted DamID (TaDa [2]) and find that Hb targets are different in different progenitors. Profiling chromatin accessibility using TaDa (CaTaDa [3]) we find that the two progenitors have distinct chromatin landscapes, and while NB5-6 specific Hb loci are enriched for open chromatin in that lineage, the same loci in NB7-4 have closed chromatin and vice versa. We propose that neuroblast-specific chromatin organization, likely established by STFs, biases subsequent TTF binding to produce different neurons in each lineage. Consistent with this model, the STF Gsb/Pax3, essential for NB5-6 specification, shows enrichment at open chromatin and Hb enriched loci in NB5-6 but not NB7-4.



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2. Southall, T. D. et al. Cell-type-specific profiling of gene expression and chromatin binding without cell isolation: assaying RNA Pol II occupancy in neural stem cells. Dev. Cell 26, 101–112 (2013).
3. Aughey, G. N., Estacio Gomez, A., Thomson, J., Yin, H. & Southall, T. D. CATaDa reveals global remodelling of chromatin accessibility during stem cell differentiation in vivo. eLife 7, (2018).

EMERGENCE OF BILATERALLY SYMMETRIC SOMITES DURING ZEBRAFISH EMBRYOGENESIS

Symmetry Breaking, Body Axes Establishment, Embryo Shape And Form, Quantitative Developmental Biology, Tissue Mechanics

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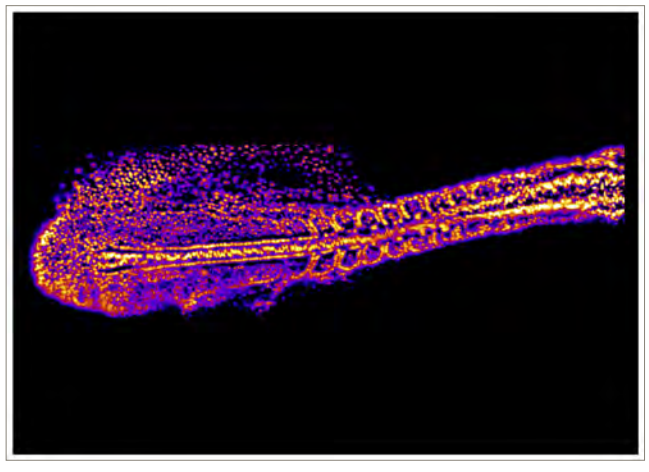
How does a left-right (LR) symmetric body plan emerge during embryonic development in vertebrates? LR symmetry in embryos is first observed during a process called somitogenesis, where the body axis is periodically segmented into epithelial blocks known as somites. Somites, which give rise to the musculoskeletal system, form bilaterally on either side of a tissue called the notochord. The size, shape and anteroposterior position of bilateral somites need to be symmetric across the notochord to ensure a LR symmetric musculoskeletal system. However, it is unknown how this precise coordination between the two sides is achieved during embryonic development.



To investigate emergence of LR symmetries in developing zebrafish embryos, we imaged somite boundary formation using a custom-built light-sheet microscope (SPIM). Imaging the spherical embryo for 8 hrs from 6 angles (30Â° apart) and fusing the different views allowed us to visualize in 3D the formation of the first 15 LR somites. To quantify somite size and shape, we map projected the 3D images of the spherical embryo on to a 2D surface. By following somite properties over time, we could show that many bilateral somite pairsform in an asymmetric fashion. Interestingly, these somite asymmetries are transient and are resolved over time suggesting that the embryo performs error correction. Uncovering mechanisms that correct these errors would be key towards understanding emergence of body form symmetry, which is fundamental to vertebrate development.

References:

1. Doe, C. Q. Temporal Patterning in the Drosophila CNS. Annu. Rev. Cell Dev. Biol. 33, 219–240 (2017).
2. Southall, T. D. et al. Cell-type-specific profiling of gene expression and chromatin binding without cell isolation: assaying RNA Pol II occupancy in neural stem cells. Dev. Cell 26, 101–112 (2013).
3. Aughey, G. N., Estacio Gomez, A., Thomson, J., Yin, H. & Southall, T. D. CATaDa reveals global remodelling of chromatin accessibility during stem cell differentiation in vivo. eLife 7, (2018).

AN ALGORITHMIC BARRIER TO NEURAL CIRCUIT UNDERSTANDING

Theoretical Neuroscience, Neuroscience, Machine Learning, Computer Science, Mathematics

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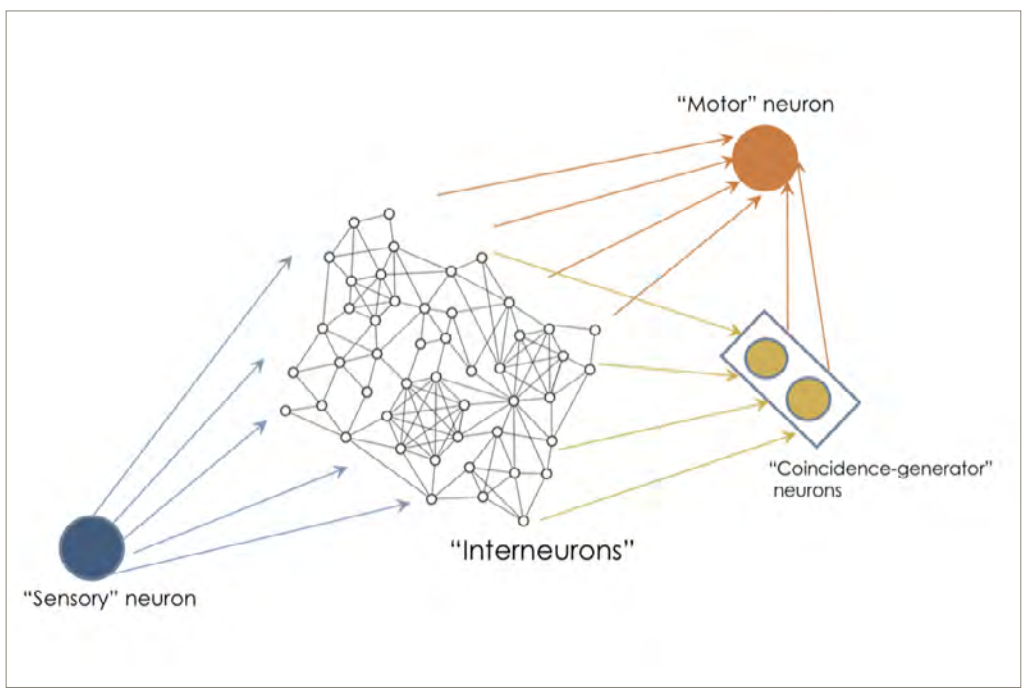
Neuroscience is witnessing extraordinary progress in experimental techniques, especially at the neural circuit level. These advances are aimed at ultimately enabling us to understand mechanistic circuit computation leading to behavior. Here, using techniques from Theoretical Computer Science, we examine how many experiments are needed to obtain such an empirical understanding. It is proved, mathematically, that establishing the most extensive notions of understanding need exponentially-many experiments in the number of neurons, in general, unless a widely-posited hypothesis about computation is false (i.e. unless P=NP). Worse still, the feasible experimental regime is one where the number of experiments scales sub-linearly in the number of neurons, suggesting a fundamental impediment to such an understanding. Determining which notions of understanding are algorithmically tractable in which contexts, thus, becomes an important new endeavor in Neuroscience.



PAK TO THE FUTURE

Actin Cytoskeleton, Small Gtpases, Membrane Dynamics, Bacterial Infections, Lysosomal Processes

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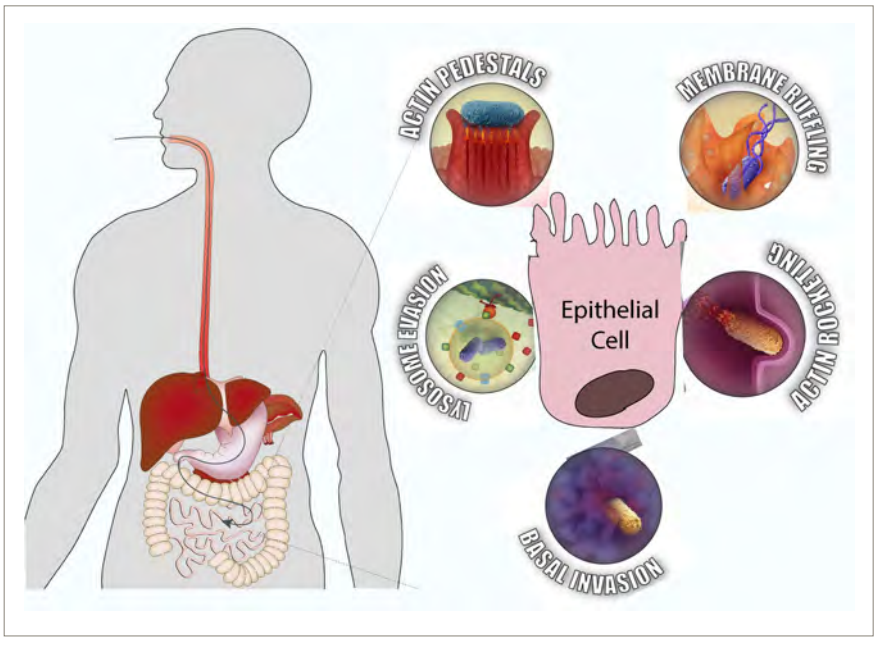
Reorganization of actin cytoskeleton is essential for many cellular processes including the control of cell architecture, motility, and membrane transport. Eukaryotic cells employ the Rho GTPases Rac1, Cdc42 and RhoA that act as master cytoskeleton regulators coordinating the formation of actin rich structures. These proteins are often hijacked by infectious pathogens in order to promote their own colonisation of the host.

As well as activating the nucleation promoting factors N-WASP (Cdc42) and the Wave Regulatory complex (Rac1), Rac1 and Cdc42 are capable of binding and activating the family of serine/threonine kinases known as p21 activated Kinases (PAK). Once activated PAK is able to phosphorylate numerous proteins including those that regulate the actin cytoskeleton. These include Filamin A, Myosin Light Chain Kinase (MLCK) and Lim Kinase a protein which in turn regulates the actin depolymerisation proteins Cofilin and ADF to modify actin dynamics.

Current work outlines the importance of PAK in the pathogenicity of both Salmonella Typhimurium, and pathogenic E. coli. The latter of which employs a dedicated effector protein to scavenge activated PAK in the cell for its own use. The identification of the Kinase domain as redundant in promoting infection stimulates future research that will attempt to identify precisely what PAK is really doing to manipulate actin assembly.



YOUNG INVESTIGATORS ABSTRACTS

Y01	Ambadas Rode	tRNAs modulate oncogene expression by shifting the hairpin-G-quadruplex conformational equilibrium in RNA
Y02	Amitava Sengupta	Epigenetic plasticity in human acute myeloid leukemia pathobiology
Y03	Amlan Das	A novel triazole NMK-T-057 induces autophagic cell death in breast cancer cells and in vivo by inhibiting γ-secretase-mediated activation of Notch-signaling
Y04	Anand Krishnan	Toward an integrative understanding of acoustic signals and signalling strategies in diverse animals
Y05	Anup Padmanabhan	Repurposing Cell Adhesion Receptors for Actomyosin Cortex Regulation : The curious case of non-junctional E-cadherin
Y06	Bilal Ahmad Mir	Engineering plants for advanced biofuels
Y07	Channakeshavaiah Chikkaputtaiah	Development of sustainable tolerance to brown leaf spot (*Bipolaris oryzae*) and leaf blast (*Magnaporthe oryzae*) fungal diseases of rice through multiplex-multigene CRISPR-Cas9/Cpf1 genome editing system
Y08	Charanya Ramachandran	Culture of human corneal endothelium for transplantation
Y09	Dhiraj Bhatia	Dna based emerging technologies for biological and bioengineering applications
Y10	Divya Uma	The architecture, dynamics, and silk investment in social spider webs
Y11	Geetanjali Sundaram	Mitotic timing in fission yeast: novel regulatory mechanisms

Young Investigators Abstracts

Y12	Gireesha Mohannath	Epigenetics of ribosomal RNA (rRNA) gene regulation and their genomic stability
Y13	Gyan Modi	Naturally inspired novel neuroprotective molecules for the treatment of alzheimer's disease
Y14	Imroze Khan	Cannibalism rescues the negative fitness impacts of biased sex-ratios in red flour beetles
Y15	Jawaid Ahsan	Sense or Non-sense? Decision making in *Drosophila melanogaster*
Y16	Karishma Kaushik	Increased production of the extracellular polysaccharide Psl can give a growth advantage to Pseudomonas aeruginosa in low-iron conditions
Y17	Kaushik Biswas	Histone H3 acetylation and loss of Sp1-HDAC1 complex contribute to derepression of GM2-synthase gene in renal cell carcinoma (RCC)
Y18	Kaviraja Udupa	Investigating the efficacy of dual stimulation of tDCS and rTMS neuro-modulation therapy of Major depression
Y19	Komalapriya Chandrasekaran	In-silico analysis of morphogenesis network of fungal pathogen candida albicans
Y20	Lipi Thukral	The same yet different: unique structural dynamics distinguish six atg8 human orthologs
Y21	Madhura Kulkarni	Hippo signaling in breast cancer progression
Y22	Maitreyee Sharma	The human Y chromosome: biological relatedness and the route of migration of the three tribes of North East India

Young Investigators Abstracts

Y23	Manish Kumar	Using high-throughput biology to tackle antibiotic resistance
Y24	Mohammad Imtiyaj Khan	Investigations on the expression of betalains in planta vis-a-vis antioxidant metabolites and enzymes
Y25	Mohankumar K Murugesan	Therapeutic genome editing for beta-hemoglobinopathies
Y26	Neha Garg	Disruption of myc/max protein-protein interactions in medulloblastoma stem cells: hot spot detection, structure-based virtual screening, biophysical and in vitro testing.
Y27	Prasada Rao	Impeding dna break repair enables oocyte quality control
Y28	Pratigya Subba	Dissecting the molecular mechanisms of cross-talk between root development process and salt stress signalling
Y29	Priyanka Singh	Molecular mechanisms of polo-like kinase, PLK4, recruitment and functioning during centriole duplication
Y30	Priyanka Upadhyai	Modeling skeletal ciliopathies *in vitro* and in *Drosophila* to dissect the role of IFT52 and EXOC6B in primary cilia mediated regulation of osteoblast and chondrocyte differentiation
Y31	Rakesh Joshi	Mechanistic insights into enzymatic catalysis by trehalase
Y32	Rama Krishna Kancha	Structural analysis reveals differential conformational diversity in druggable enzymes

Young Investigators Abstracts

Y33	**Sachin Kumar**	Regulation of neutrophil infiltration; implication in meta-inflammation and metabolic syndromes
Y34	**Shilpak Chatterjee**	CD38-NAD+axis regulates immunotherapeutic anti-tumor t cell response
Y35	**Smarajit Polley**	Yin and yang of IKK signaling: targeting beyond the active site to tame NF-Kappab
Y36	**Sravanti Uppaluri**	Operant conditioning and retention following amputation in *Schmidtea mediterranea*
Y37	**Timir Tripathi**	Unusual linked thiol-based redox metabolism of parasitic helminths: novel pathway for drug discovery
Y38	**Trinath Jamma**	Role of host-microbiota-derived metabolites in modulating intestinal inflammation
Y39	**Vinita Sharma**	Forensic class characterization from seized rhino horn: a tool for combating transboundary illegal wildlife trade
Y40	**Vinothkumar Kutti Ragunath**	Membrane protein structure and dynamics

TRNAS MODULATE ONCOGENE EXPRESSION BY SHIFTING THE HAIRPIN-G-QUADRUPLEX CONFORMATIONAL EQUILIBRIUM IN RNA

Synthetic Biology, Non-coding RNAs, Conformational transition , G-quadruplex, Riboswitches

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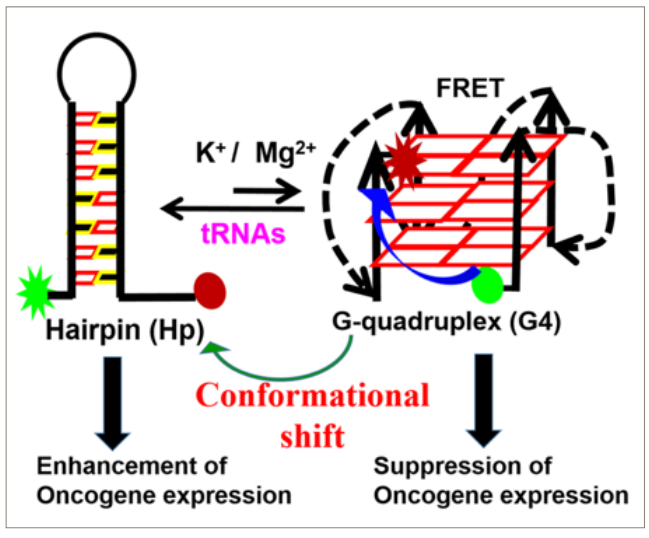
The functions of several RNAs are regulated by conformational transitions occurred in response to set of effector molecules. RNA G-quadruplexes (G4s) are non-canonical structures found in the coding and non-coding regions and are involved in translation regulation. In 5′ untranslatd region (UTR) of several oncogenes mRNA, formation of G4 conformer competes with the formation of hairpin conformer. The hairpin-G4 conformational equilibria would affect oncogene expression. Thus, factors that influence the conformational equilibria are potentially regulators of the oncogene expression and cancer development. Herein, we investigated effect of high concentration of transfer RNA (tRNA), which mimics the overexpression of tRNA in certain cancers, on the hairpin-G4 conformational equilibria in the 5′ UTR sequences derived from oncogenes. Our kinetic and equilibrium analyses of the hairpin to G4 conformational transitions using FRET indicated that tRNA at physiological concentration (20 μM) shifted the equilibria up to 60 % toward the hairpin conformer. Moreover, in vitro translation experiments showed enhancement of reporter gene expression due to shift of the conformational equilibria. These findings suggest that tRNA could be a possible therapeutic target for cancer development.immunodeficient host.



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EPIGENETIC PLASTICITY IN HUMAN ACUTE MYELOID LEUKEMIA PATHOBIOLOGY

Hematopoiesis, Hematopoietic Stem Cell Aging, Leukemia Pathobiology, Bone Marrow Microenvironment, Immunogenetics And Cancer Immunotherapy

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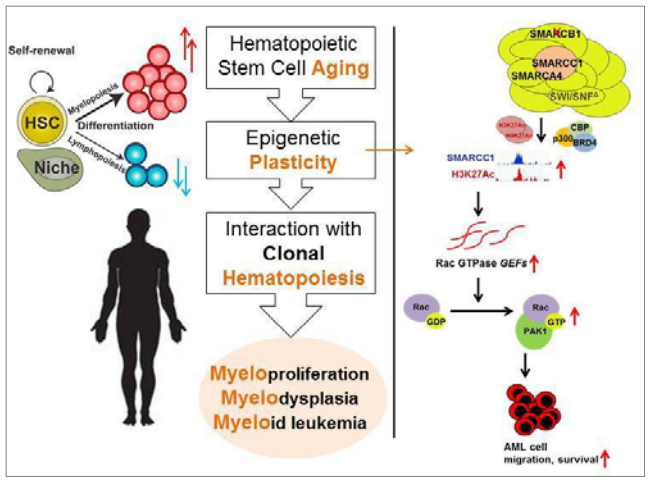
Cancer, once considered as a genetic disorder, is now increasingly being perceived as an admixture of genetic and epigenetic perturbations. Acute myeloid leukemia (AML) is the second most common leukemia worldwide with a median age of ~65 years at diagnosis. SWI/SNF and NuRD are multi-subunit ATP-dependent chromatin remodelers that regulate epigenetic architecture and cellular identity. Although SWI/SNF genes are frequently altered in human tumors, evidences showing their involvement in tumor cell-autonomous transcriptional plasticity, one of the key hallmarks in tumorigenesis, are limiting. Rac GTPases regulate myeloid leukemia cell engraftment. Although leukemia cells generally display an elevated Rac GTPase, mechanism of Rac activation in AML is incompletely understood. We have identified that, loss of specific subunits in SWI/SNF and NuRD in human primary AML cells associates with nucleation of neo-oncogenic chromatin remodelers, which augments Rac GTPase-GEFs expression, Rac activation, migration, and survival of AML cells. Mechanistically, this involves sub-stoichiometric interaction and locus-specific occupancy of chromatin remodelers along with defined histone modifying enzymes. Rac GEFs inhibition selectively attenuated survival and migration of AML cells. These findings inform novel transcriptional dependency, and connect epigenetic regulation with trafficking of hematopoietic progenitors in human AML. In unison, our results highlight that overly permissive chromatin causes stochastic activation of oncogenic gene expression program in tumorigenesis.possible therapeutic target for cancer development. immunodeficient host.



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A NOVEL TRIAZOLE NMK-T-057 INDUCES AUTOPHAGIC CELL DEATH IN BREAST CANCER CELLS AND IN VIVO BY INHIBITING γ-SECRETASE-MEDIATED ACTIVATION OF NOTCH-SIGNALING.

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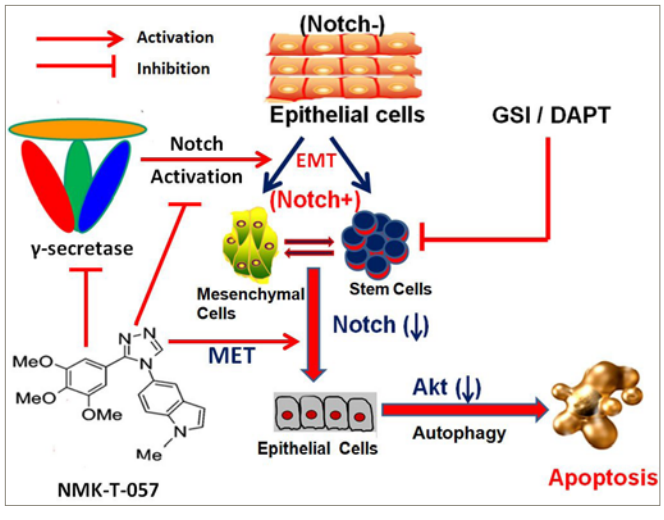
Hematopoiesis, Hematopoietic Stem Cell Aging, Leukemia Pathobiology, Bone Marrow Microenvironment, Immunogenetics And Cancer Immunotherapy

Notch signaling is reported to be deregulated in several malignancies, including breast, and the enzyme γ-secretase plays important role in the activation and nuclear translocation of Notch intracellular domain (NICD). Hence, pharmacological inhibition of γ-secretase might lead to the subsequent inhibition of Notch signaling in cancer cells. In search of novel γ-secretase inhibitors (GSIs), we screened a series of triazole-based compounds, for their potential to bind γ-secretase and we observed that 3-(3´4´,5´-Trimethoxyphenyl)-5-(N-methyl-3´-indolyl)-1,2,4-triazole compound (also known as NMK-T-057) can bind to γ-secretase complex. Very interestingly, NMK-T-057 was found to inhibit proliferation, colony forming ability, motility in various triple negative breast cancer cells (TNBCs) such as MDA-MB-231, MDA-MB-468, 4T1 and also MCF-7 (ER/PR positive cell line), with negligible cytotoxicity against non-cancerous cells such as MCF-10A, and PBMC. NMK-T-057 also showed limited toxicity in Swiss albino mice, as determined by measuring the hematological and clinical parameters. The in silico study revealing the affinity of NMK-T-057 towards γ-secretase, was further validated by fluorescence based γ-secretase activity assay, which confirmed inhibition of γ-secretase activity in NMK-T-057 treated TNBC cells. Very interestingly, it was observed that NMK-T-057 induced significant autophagic responses in TNBCs and administration of the autophagy inhibitor 3-MA, attenuated NMK-T-057 induced cell death. Hence, it may be concluded that NMK-T-057 could be potential drug candidate against breast cancer, specifically TNBCs, which can trigger autophagy-mediated cell death in breast cancer cells by inhibiting the γ-secretase–mediated activation of Notch-signaling.



TOWARD AN INTEGRATIVE UNDERSTANDING OF ACOUSTIC SIGNALS AND SIGNALLING STRATEGIES IN DIVERSE ANIMALS

Birds, Ecology, Bioacoustics, Ornithology, Communication

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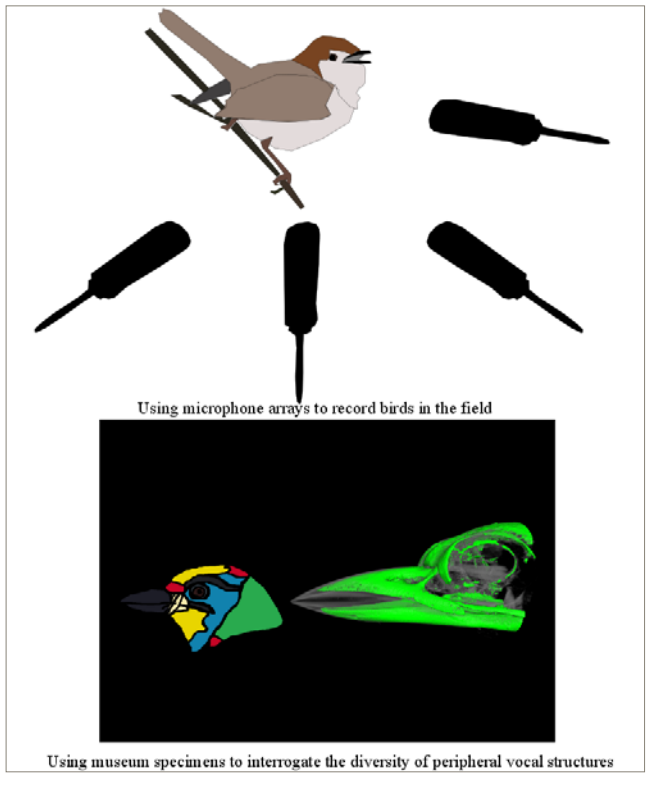
Animal vocalizations function to communicate information in diverse contexts. In order to convey this information, sound must first be produced, then modified by vocal structures such as beaks or mouths, propagated across complex environments, and must be distinct to its intended recipient over a chorus of other vocalizing animals[1]. To accomplish this, animals may produce sounds that occupy distinct regions of acoustic space (analogous to ecological space)[2], or may adopt behavioral strategies to avoid overlap with neighbors, such as singing at different times or at different heights. Together, these represent an organism's acoustic niche, a concept that integrates the physics of the environment and sound-producing structures with the ecology and natural history of signaling animals[3]. We aim to study how diverse signaling strategies influence larger-scale ecological patterns in acoustic communities. Our research adopts a top-down perspective, first examining community-level acoustic niche patterns to understand how coexisting birds and bats occupy the sound spectrum across seasons and across habitats at different altitudes. Next, we focus on groups of closely related coexisting birds to understand the behavioral strategies employed by simultaneously vocalizing conspecifics and heterospecifics to communicate information. Finally, we study the Asian barbets, a group of birds that vocalize without opening their beaks, to understand how the evolutionary diversification of beak form and material has influenced sound production in these highly vocal birds.



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REPURPOSING CELL ADHESION RECEPTORS FOR ACTOMYOSIN CORTEX REGULATION : THE CURIOUS CASE OF NON-JUNCTIONAL E-CADHERIN

Actomyosin cortex, Mechanobiology, Cytokinesis, C. elegans Development, Pathogenesis

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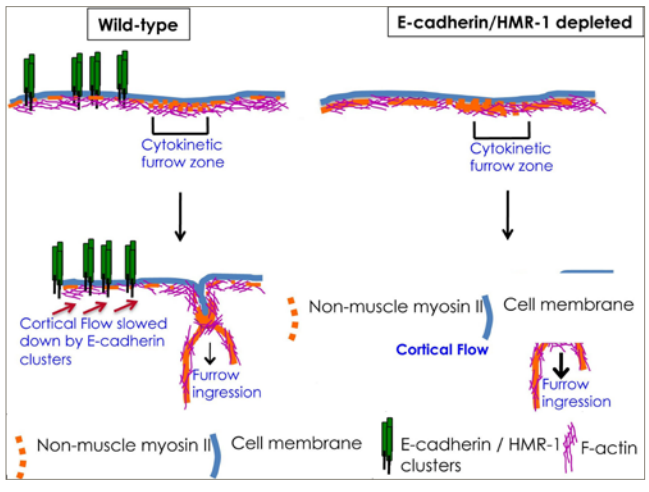
Cell-cell adhesion is a hallmark of multi-cellularity. E-cadherin, by far the most well studied cell-cell adhesion receptor, is essential for epithelial tissue integrity, morphogenesis and development in all-multicellular organisms. Its functional disruption is associated with cancer metastasis. Our current understanding of the E-cadherin function is limited to its role as an adhesion receptor within cell-cell junctions despite the fact that clusters of E-cadherin have been observed at cell surfaces outside of junctions. What cellular roles, if any, may these non-junctional clusters have? During the course of my analysis of HMR-1, the E-cadherin ortholog in early C. elegans development, I discovered that these non-junctional clusters slowed down cytokinetic furrow ingression. Intriguingly this inhibitory effect was independent of E-cadherin's cell adhesion function and instead mediated by its cytoplasmic domain via two mechanisms : (1) E-cadherin/HMR-1 diminishes type-II myosin levels at the cortex by inhibiting Rho activity, and (2) by physically associating with the cortical F-actin, E-cadherin/HMR-1 resists cortical shape changes such as during cytokinesis.



These findings reveal a non-junctional, adhesion-independent role of E-cadherin in regulating the actomyosin cortex and cell proliferation, in addition to its established role in adherens junctions.

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ENGINEERING PLANTS FOR ADVANCED BIOFUELS

Metabolic Engineering, Genetic Profiling, Biofuels, Metabolomics, Plant Tissue Culture

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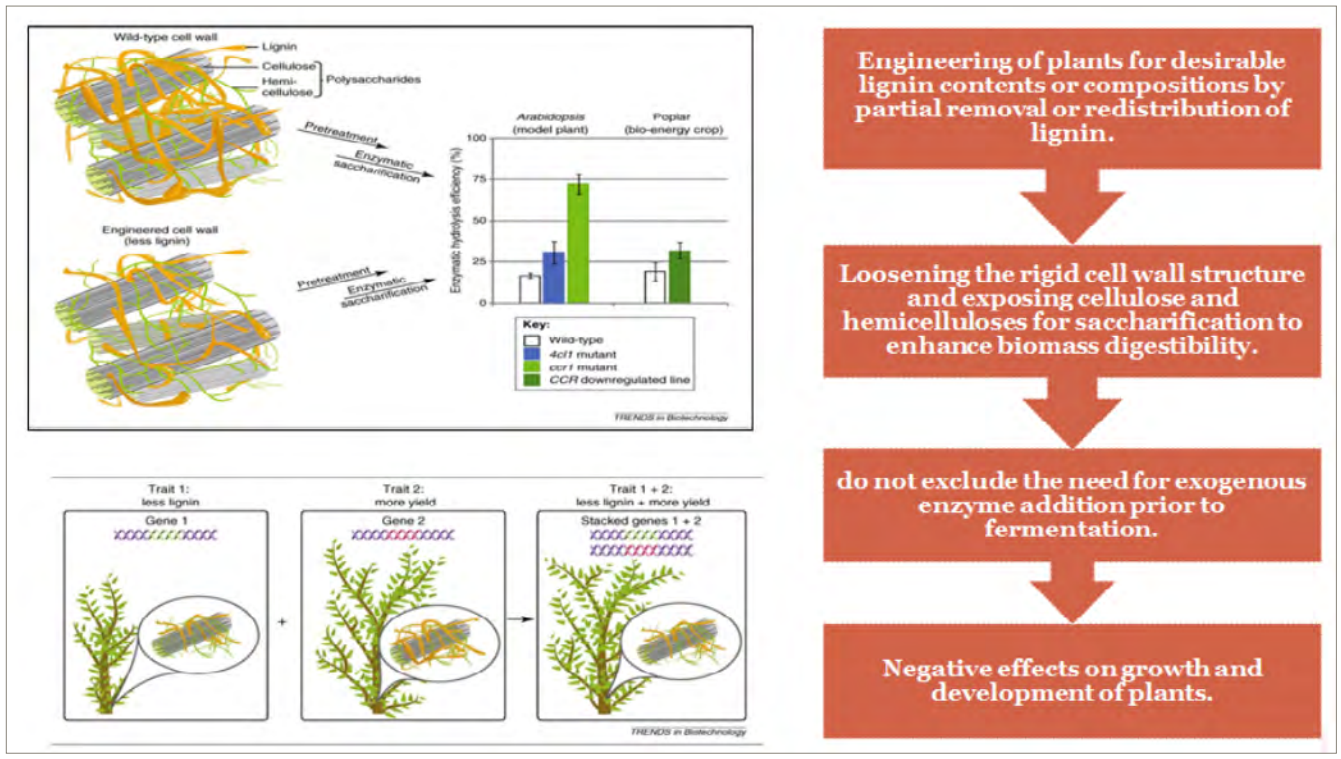
Dwindling fossil reserves and global warming has catalyzed a worldwide trend to utilize plant biomass for the production of biofuels and other biomaterials. Plant biomass is the most abundant renewable resource on the earth. However there are currently various limitations to use this biomass for biofuels. Towards this end our research team focuses on the engineering of plants for biofuel production. We are trying to tailor a biofuel feedstock that would be easily converted to bioethanol and for this our current approach is to express some thermophilic enzymes directly in plants.



Linging Engineering and Gene Stacking

DEVELOPMENT OF SUSTAINABLE TOLERANCE TO BROWN LEAF SPOT (*BIPOLARIS ORYZAE*) AND LEAF BLAST (*MAGNAPORTHE ORYZAE*) FUNGAL DISEASES OF RICE THROUGH MULTIPLEX-MULTIGENE CRISPR-CAS9/CPF1 GENOME EDITING SYSTEM

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Multiple Abiotic Stress, CRISPR-Cas9, CRISPR-Cpf1, Genome Editing, Crop Stress Tolerance, Crop Improvement

Brown leaf spot (BLS) & leaf blast (LB) caused by Bipolaris oryzae and *Magnaporthe oryzae* respectively are two of the major fungal diseases of rice affecting yield and grain quality in most of the rice growing areas of India especially North East and Eastern India. BLS was once recorded as the major contributing factor to the Bengal famine in 1943, is still a devastating disease. The yield loss of rice in India mainly due to these two diseases accounts to about 35% annually. Presently, no sustainable BLS & LB tolerant rice cultivars available and their management in the field partially rely on the use of fungicides which is environmentally harmful. Although molecular breeding efforts are directed towards mitigating the BLS & LB fungi, so far unsuccessful due to complexity of the diseases which are regulated by multiple genes. A sustainable research solution is essentially required to mitigate huge yield losses of rice due to these two major fungal diseases. Breakthrough CRISPR genome editing technology has several advantages over conventional biotechnological approaches by allowing highly precise mutations in elite cultivars. Through systematic transcriptome RNA Seq analysis and qRT-PCR validation, we have identified three major negative regulatory genes of rice which are down-regulated for BLS & LB infection. Using the state of the art CRISPR-Cas9/Cpf1 genome editing approach, we aim to carry out target specific multiplex-multigene editing of negative regulatory disease susceptible genes of rice for BLS & LB fungi through Gibson assembly & multisite gateway recombination approach. We characterize the CRISPR-edited rice lines through molecular genetic and biochemical approaches to develop sustainable tolerance to BLS & LB in rice crop model. The outcome would be highly significant for translational research to target multiple genes simultaneously for the improvement of qualitative and quantitative traits of crop plants.

CULTURE OF HUMAN CORNEAL ENDOTHELIUM FOR TRANSPLANTATION

Corneal Endothelium, Stem Cells, Translational Research, Tissue Engineering, Ocular Development

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Purpose

Human corneal endothelial cells are known to undergo endothelial to mesenchymal transition (EnMT) when cultured in vitro. This study aimed to determine when the cells undergo EnMT in culture by systematically assessing their growth characteristics and functional properties at every passage (P) or sub- culture. This information is critical for us to assess the safety of the cells before taking them for human transplantation.

Methods

Corneas from donors 5 days to 45 years old were cultured from P1-P8. Growth characteristics such as population doubling time, cell proliferation and total cell density (at confluence) were quantified at every passage. Similarly, changes in the putative EnMT and endothelium gene and protein expression was quantified using real time PCR and immunostaining at different passages. Finally, barrier function of the cultured cells was measured as permeability to FITC dextran (10kDa). Statistical analysis was performed using GraphPad prism ($p \leq 0.5$ was considered significant).

Results

Between passages 1-3 the following were noted a) young donors (≤ 10 years) showed a population doubling time of 41.4±3.9 hours which was comparable to adult donors (11-30years) averaging 46.2±13.6 hours and significantly lesser than older donors of 40-50 years (90.2±8.6 hrs) b) 60±23% of the cells were proliferating in cultures from young donors when compared to 42±11% in older donors c) the average cell density obtained from young donors was significantly more (2000±353 cells/mm2) than older donors (754±287 cells/mm2). Interestingly, in all the cultures, irrespective of age of donor, there was a sudden increase in the doubling time at P4 (to 121.1±35.7hrs) when compared to P3 (31.08±8.6) and reduced to 53.69±13.3 hrs at P5. Concomitant with this, we noticed that the morphology of cells altered from polygonal and homogeneous to more fibroblastic confirmed by the increased expression of α-SMA in P6 cells compared to P2 cells. Further, late passage (P6 or 7) cells were more permeable to FITC-dextran than early passage cells (P1-3).

Conclusion

Our data is in agreement with earlier studies that the yield of cells from young donors is better than from older donors. However, we have shown here that at passage 4 the cells start undergoing a transition from endothelium to more fibroblastic phenotype. This suggests that at present only early passage (1-3) cells are safe to take to the clinic for transplantation.

DNA BASED EMERGING TECHNOLOGIES FOR BIOLOGICAL AND BIOENGINEERING APPLICATIONS

Structural DNA Nanotechnology, Cell Biology And Membrane Traffic, Stem Cells Programming, Advanced Microscopy, Biomedical Applications

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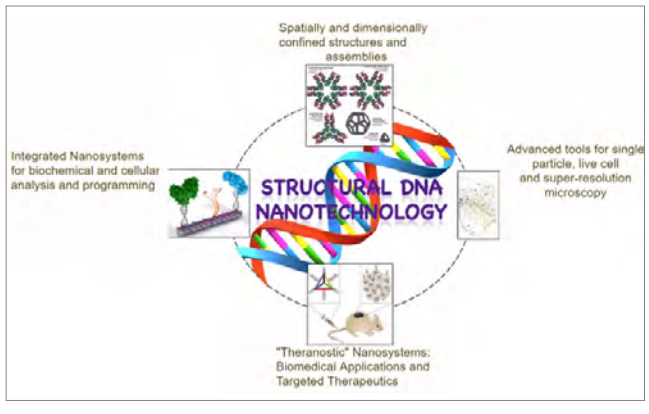
Structural DNA nanotechnology explores various nanoscale structural and functional properties of DNA to manipulate matter at nanoscale for diverse applications. Three-dimensional architectures based on DNA polyhedra have raised particular interest in biomedical applications. DNA polyhedra possess an internal void bounded by a well- defined three-dimensionally structured surface. The internal void can house cargo, and the designer DNA scaffold can facilitate molecular display to program biological targeting. While the delivery of designer DNA particles bearing surface ligands has been achieved, the successful demonstration of their full potential of targeted delivery when housing an internal payload remains an outstanding challenge.
I will present the first successful delivery of quantum dots (QDs) as the internal payload of DNA icosahedra. A long- standing challenge for QDs has been the inability to achieve their monofunctionalization in bulk. We resolve this by encapsulating QDs within molecularly identical icosahedral DNA particles in bulk where the DNA shell is mono-functionalized with different endocytic ligands. We demonstrate the monofunctionalization and successful specific, endocytic uptake of QDs, using multiple endocytic ligands like folic acid, Galectin-3 (Gal3) and Shiga toxin B-subunit (STxB). Single particle tracking of Gal3/STxB-bearing, QD-loaded icosahedra reveal new observations of compartment dynamics along the endocytic pathways. QD-loaded DNA polyhedra bearing ligands of unique stoichiometry represent a new class of high-precision molecular imaging tools for quantitative approaches to complex biological phenomena arising from receptor clustering. Our results highlight the emerging potential of DNA devices in cell biology and biomedical applications that could enable probing and programming various biological systems as well as developing next generation tools for targeted delivery of molecular payloads within living systems.



THE ARCHITECTURE, DYNAMICS, AND SILK INVESTMENT IN SOCIAL SPIDER WEBS

Predator-Prey Interactions, Mimicry, Collective Behavior, Behavioral Ecology, Plant-Insect Interactions

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Animal architecture is diverse in form and structure, and extraordinarily intricate, often facilitated by collective behavior of hundreds of individuals. Webs built by social spiders are one such example of animal architecture. We discuss the architecture, dynamics and silk investment in a tropical social spider *Stegodyphus sarasinorum* whose webs are built within a few days but maintained for several months. One of the benefits of sociality in spiders is to conserve silk. We test this hypothesis by allowing spiders in different group sizes (1, 5, 10 and 25 spiders per group) to build webs in a pre-defined space over 10 days and tracking web evolution through image analysis. Specifically, we ask a) if spiders in larger group size produce more silk, and b) if per capita silk investment reduces with increase in group size. Our results indicate that spiders in larger group invest more silk compared to spiders in smaller groups. However, spiders do not save silk by living in larger colonies. Additionally, we discuss the spatio-temporal evolution of web of *S. sarasinorum.* From an architectural point of view, unlike manmade structures, these webs achieve moderate stability and functionality even when they are not fully complete.

MITOTIC TIMING IN FISSION YEAST: NOVEL REGULATORY MECHANISMS

Mitosis, Cell cycle, Atf1, Spc1, S.pombe

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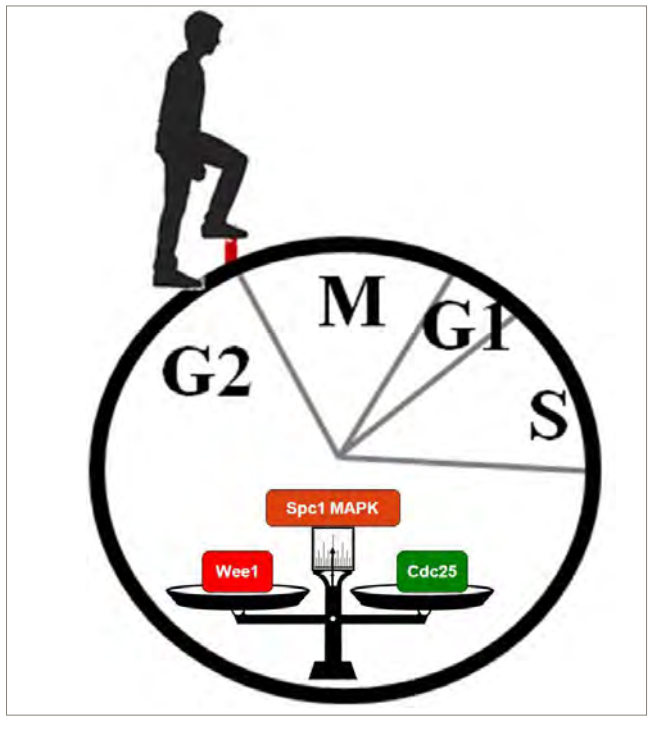
In eukaryotes Mitogen Activated Protein Kinases (MAPKs) play vital roles in multiple physiological processes including cell division. The MAPK Spc1 (human p38 homolog) in the fission yeast *Schizosaccharomyces pombe* regulates both cell division and stress response .Since p38 is a well-established target for cancer therapy, understanding its role in regulation of the cell cycle is very important. Here, we present interesting findings about role of the MAPK Spc1 (p38 homolog) in regulating mitotic timing in the excellent model system *S. pombe*. Our study reveals that high Spc1 activity can sense aberrant activity of classical regulators of mitotic timing which include the positive regulator Cdc25 and the negative regulator Wee1. Not only can it sense these perturbations, but can initiate suitable responses to restore their optimal activity and thereby rescue the cells from premature deleterious entry into mitosis. We show that this restoring mechanism operates via Rad24 (14-3-3 homolog) and this is the first report linking Spc1 activity with Rad24.



A hallmark of this mechanism is the fact that it is exclusive to cells which have a pre-existing defect in regulation of mitotic timing. The Spc1-Rad24 dependent pathway therefore represents a novel secondary mechanism for regulating G2-M progression which serves as a backup for the primary Wee1-Cdc25 dependent pathway. Detailed investigation of this new mechanism is important to understand how Spc1 co-ordinates the response to environmental changes with cell cycle progression in *S. pombe*. Here we present results that help to understand the intricacies of this novel mechanism.

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EPIGENETICS OF RIBOSOMAL RNA (RRNA) GENE REGULATION AND THEIR GENOMIC STABILITY

Epigenetics, rRNA Gene Regulation, Chromatin Biology, Genome Instability, DNA Methylation and Histone Modifications

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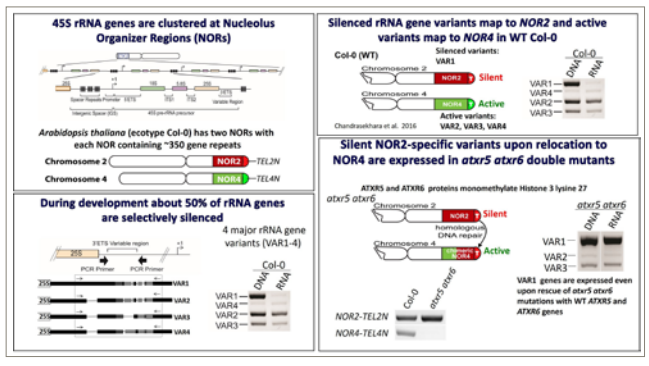
Eukaryotes carry hundreds to thousands of ribosomal RNA (rRNA) genes that are arrayed as tandem repeats at loci called nucleolus organizer regions (NORs). These rRNA genes are transcribed in the nucleolus by RNA polymerase I (Pol I), yielding primary transcripts that are processed into 18S, 5.8S, and 25-28S RNAs of ribosomes, the protein synthesizing machines of cells. The number of transcribed rRNA genes is known to vary with developmental and physiological demands, but the mechanisms dictating the on or off states of specific subsets of rRNA genes remain unclear. We recently found that rRNA gene subtypes, that are silenced during development genetically map to NOR2, whereas active rRNA genes map to NOR4 (1). In an atxr5 atxr6 mutant line deficient for histone H3 lysine 27 (H3K27) monomethylation, a translocation of rRNA genes on a multimegabase scale from one chromosomal location (NOR2 on chromosome 2) to the other (NOR4 on chromosome 4) has occurred and this translocation causes the translocated genes escape silencing in their new location (NOR4), independent of the atxr mutations (2). Collectively, these data support our new hypothesis that chromosomal context determines activity status of rRNA genes at the level of NORs. Moving forward, we intend to this hypothesis for human rRNA genes because of the significance of their regulation in cancer biology. The other objective of our group is to understand the molecular basis for megabase-scale rDNA instability that occurred in atxr5 atx6 double mutants of Arabidopsis.



Epigenetics of Ribosomal RNA (rRNA) gene regulation and their genomic stability

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NATURALLY INSPIRED NOVEL NEUROPROTECTIVE MOLECULES FOR THE TREATMENT OF ALZHEIMER'S DISEASE

Alzheimer's disease, Natural Products, Imaging agents

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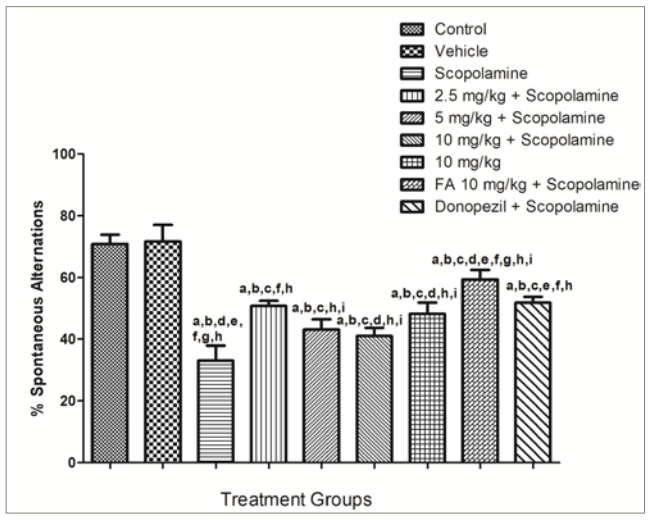
Alzheimer disease (AD) is a multifactorial progressive neurodegenerative disorder characterized by gradual loss in memory coupled with other changes. Several lines of evidences including the publications from our research group indicated critical pathological role played by genetics, amyloid beta (AÎ²), metals and oxidative stress in the neurodegeneration. Our laboratory has been developing novel multifunctional neuroprotective molecules of varying selectivity towards cholinergic system. Several natural products including trans-4-hydroxy-3-methoxycinnamic acid (FA) and their hybrid analogs are under investigation as neuroprotective agents for AD. In in-vitro experiments, FA has shown promising neuroprotection property, however, its ability to cross blood brain barrier is limited due to it lower lipophilicity (Log P ~1.25). The low logP value of FA is also responsible for poor aqueous solubility. To develop naturally inspired in-vivo active neuroprotective molecules and to overcome the limitations associated with the natural compounds, we embarked on the development of FA analogs. In *in-vitro* enzyme inhibitory studies, lead molecule F24 was found to be the most active with IC50 value of 5.74Î¼M against acetyl cholinesterase. In scopolamine induced AD mice model, F24 is able to effectively reverse the memory loss at very low dose compared to FA. The detailed In-vitro and in-vivo studies will be presented.



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CANNIBALISM RESCUES THE NEGATIVE FITNESS IMPACTS OF BIASED SEX-RATIOS IN RED FLOUR BEETLES

Aging, Environmental Stress, Experimental Evolution, Immune Responses, Infection

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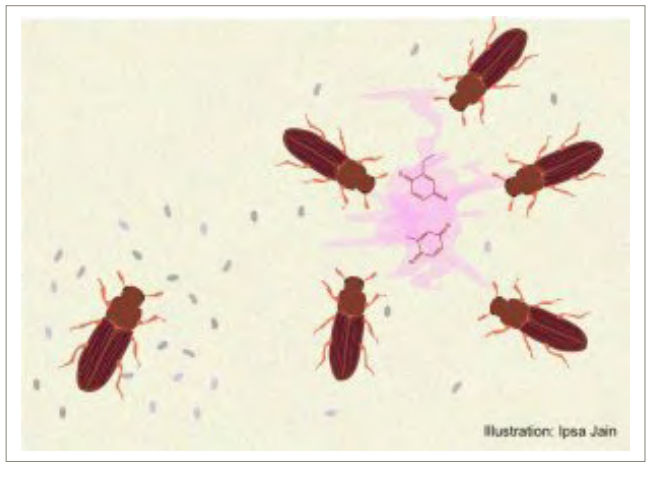
Theory and experiments predict that cannibalism increases fitness under stressful conditions such as limited access to resource in many animal species. However, empirical evidence where cannibalism directly manipulates their fitness by providing additional nutritional sources under resource competition is limiting. Our recent work highlights an example where skewed sex ratios altered resource competition in beetle Tribolium castaneum[1]. Females that lived in female-biased (FB) groups suppressed each other's fecundity by using toxin quinones secreted from their stink glands, suggesting a form of nonsexual interference competition in populations with high female density. Does cannibalism rescue the negative fitness impacts of female density? In a proof-of-principle study, we tested this possibility by adding the pupae as potential food sources to adult beetles under different sex-ratio groups. We found that the access to additional nutritional sources via cannibalism quickly relaxed the competition in female-biased groups, with a rapid increase in their fitness. In contrast, cannibalism had no impacts on female fitness in male-biased groups. Next, we tested whether experimental reduction of cannibalism can reverse the fitness benefits of FB females. We could reduce the cannibalism rate by adding pupae, infected with high dose of bacterial pathogen Bacillus thuringiensis to female-biased groups. FB adults avoided cannibalizing infected pupae. As a result, their fitness could not be rescued. Overall, our data suggest a causal link between cannibalism, nutrition and fitness effects under distinct contexts of population structure such as biased sex ratios. Our data also opens up an exciting possibility where cannibalism could impact disease spread and infection prevalence in natural populations.



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SENSE OR NON-SENSE? DECISION MAKING IN *Drosophila melanogaster*

Drosophila, Behavior, Olfaction, Learning, Memory

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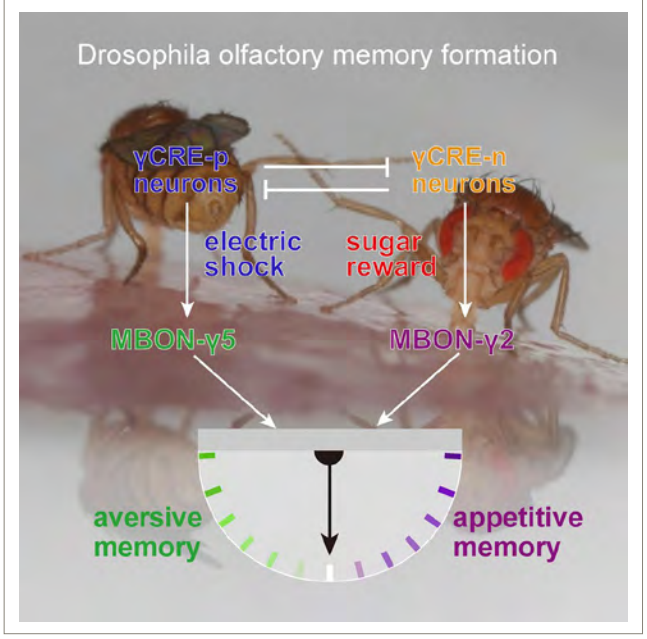
Brains have to decide whether and how to respond to detected stimuli based on complex sensory input. The vinegar fly *Drosophila melanogaster* evaluates food sources based on olfactory cues. Here, we performed a behavioral screen using the vinegar fly and established the innate valence of 110 odorants. Our analysis of neuronal activation patterns evoked by attractive and aversive odorants suggests that even though the identity of odorants is coded by the set of activated receptors, the main representation of odorant valence is formed at the output level of the antennal lobe. The topographic clustering within the antennal lobe of valence-specific output neurons resembles a corresponding domain in the olfactory bulb of mice. The basal anatomical structure of the olfactory circuit between insects and vertebrates is known to be similar; our study suggests that the representation of odorant valence is as well.



INCREASED PRODUCTION OF THE EXTRACELLULAR POLYSACCHARIDE PSL CAN GIVE A GROWTH ADVANTAGE TO PSEUDOMONAS AERUGINOSA IN LOW-IRON CONDITIONS

Infectious Diseases, Antibiotic Resistance, Pathogen Detection Assays, Host-Microbial Dynamics , Biomimetic Models

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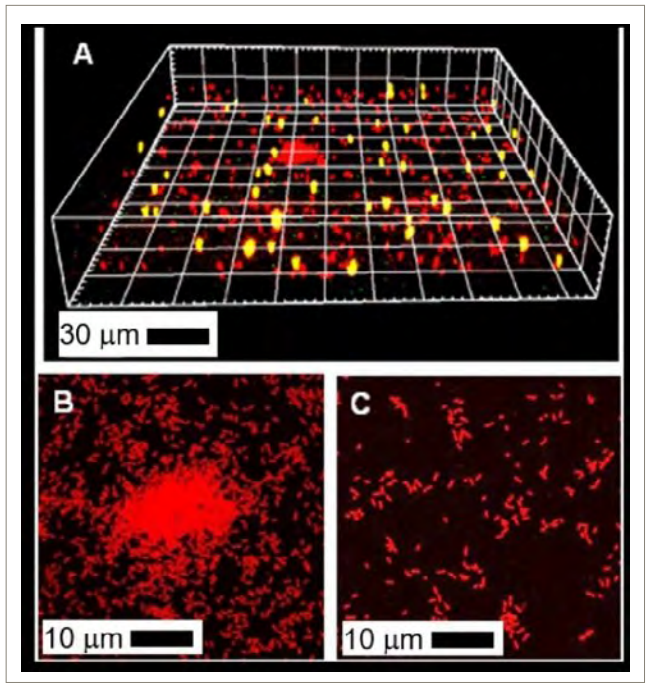
In infections, biofilm formation is associated with a number of fitness advantages, such as resistance to antibiotics and to clearance by the immune system. Biofilm formation has also been linked to fitness advantages in environments other than *in vivo* infections; primarily, biofilms are thought to help constituent organisms evade predation and to promote intercellular signaling. The opportunistic human pathogen *Pseudomonas aeruginosa* forms biofilm infections in lungs, wounds, and on implants and medical devices. However, the tendency toward biofilm formation originated in this bacterium's native environment, primarily plants and soil. Such environments are polymicrobial and often resource-limited. Other researchers have recently shown that the *P. aeruginosa* extracellular polysaccharide Psl can bind iron. For the lab strain PA01, Psl is also the dominant adhesive and cohesive "glue" holding together multicellular aggregates and biofilms. Here, we perform quantitative time-lapse confocal microscopy and image analysis of early biofilm growth by PA01. We find that aggregates of *P. aeruginosa* have a growth advantage over single cells of *P. aeruginosa* in the presence of Staphylococcus aureus in low-iron environments. Our results suggest the growth advantage of aggregates is linked to their high Psl content and to the production of an active factor by *S. aureus*. We posit that the ability of Psl to promote iron acquisition may have been linked to the evolutionary development of the strong biofilm-forming tendencies of *P. aeruginosa.*



HISTONE H3 ACETYLATION AND LOSS OF Sp1-HDAC1 COMPLEX CONTRIBUTE TO DEREPRESSION OF GM2-SYNTHASE GENE IN RENAL CELL CARCINOMA (RCC)

Glycosphingolipids, Cancer Biology, Cellular Signaling, Gene Editing, Cancer Epigenetics

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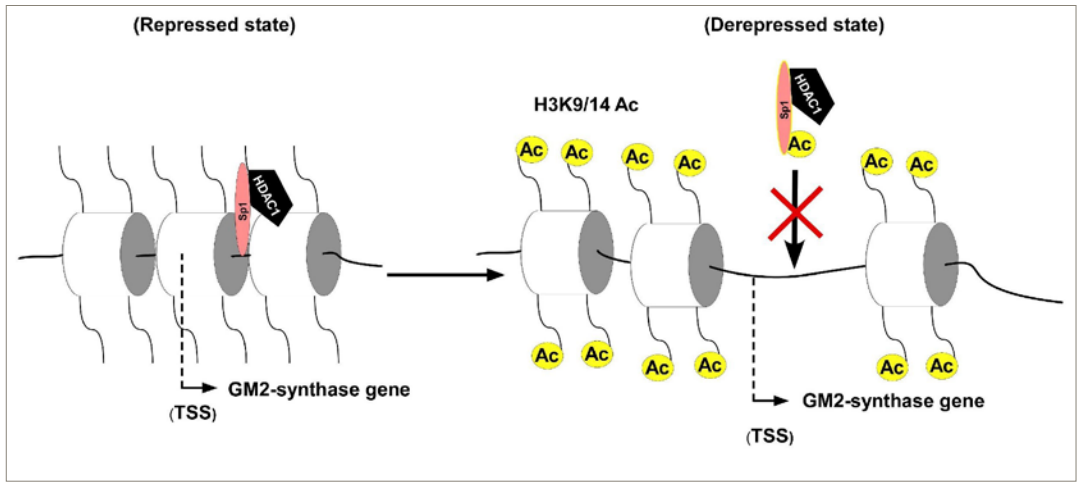
Over-expression of GM2-synthase and its corresponding gangliosides are linked with tumor progression, migration and suppression of tumor specific host immune response. However, the mechanism underlying the de-repression of GM2-synthase in renal cell carcinoma (RCC) is poorly understood. Here, we demonstrate that higher GM2-synthase mRNA expression in various cancer cells as well as human RCC tumors correlate with higher histone acetylation levels (either H3K9 or H3K14 or both) at region +38/+187 with respect to the transcription-start site (TSS) of GM2-synthase gene, compared to either normal kidney epithelial cell line (NKE) or normal adjacent tissues. Again, increase of GM2-synthase mRNA expression in cells treated with HDAC inhibitor is accompanied with increased histone acetylation levels at this region. However, DNA methylation around the TSS is absent in both RCC cell lines and NKE. Increased association of Sp1 and HDAC1 with region +38/+187 was observed in repressed GM2-synthase gene in NKE and tumor-adjacent tissues relative to RCC cell lines and tumor tissues respectively, indicating a plausible site-specific repressive role of HDAC1 and Sp1. Site directed mutagenesis of the Sp1 binding site within +38/+187 region relieved the repressed luciferase activity of this region by limiting HDAC1 recruitment. Moreover, knock down of either Sp1 or HDAC1 increased GM2-synthase transcription. Additionally, activation of GM2-synthase in SK-RC-45 cells by butyrate was accompanied by a loss of Sp1 and HDAC1 from +37/+187 region. Taken together, we identified an epigenetic mechanism involved with de-repression of GM2-synthase gene in RCC.



INVESTIGATING THE EFFICACY OF DUAL STIMULATION OF tDCS AND rTMS NEURO-MODULATION THERAPY OF MAJOR DEPRESSION

Clinical Neurophysiology, Movement And Mood Disorders, Non-Invasive Brain Stimulation, Cortical Excitability And Plasticity, Cardiac Autonomic Functions

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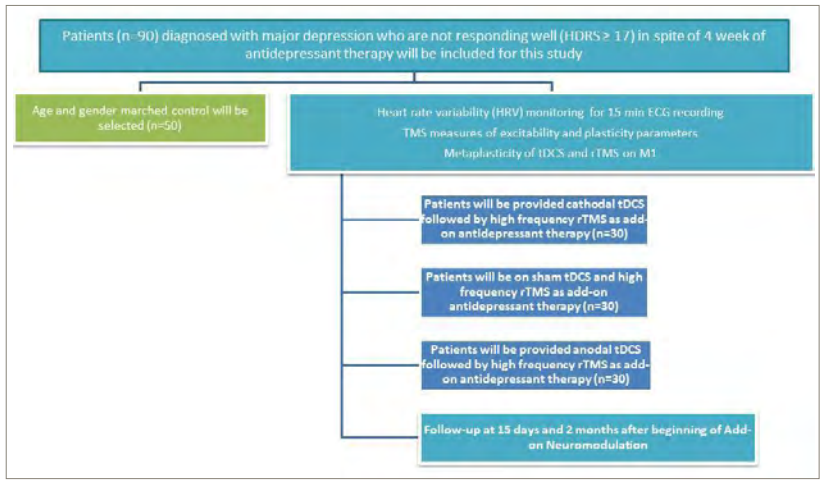
Major depression is the commonest psychiatric disorder affecting 3-5% of Indian population. Although the drugs acting on several neurotransmitter systems clinically improve the condition in 30-50% patients, recurrence or relapses are common in third of patients requiring add-on/adjuvant therapies. In this regard, there is expanding interest in repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS) as add-on antidepressant therapies. Although these two modes of non-invasive brain stimulation (NIBS) have been successfully considered as second or third lines of antidepressant managements, we are still lacking the clear understanding of combining these two modes of NIBS. Further, the neurobiological factors and patient characteristics contributing to efficacy of these add-on antidepressant therapies in refractory depression is not established. Hence we planned this study to investigate the efficacy of dual stimulation of tDCS and high frequency rTMS in patients with Major depression and investigate the factors affecting medical refractoriness in depression by using a comprehensive battery of clinical and investigative modalities including clinical scales of Depression (primary objective) and Cortical excitability measures using single paired pulse and plasticity measures and cardiac autonomic function tests (secondary objectives). We plan to recruit 90 patients diagnosed with major depression who are not responding well in spite of at least one course of antidepressant therapy and 50 age- and sex-matched healthy controls. Their baseline measures of clinical scales, autonomic function tests and cortical excitability measures will be determined. Patients will be divided into three groups randomly into dual stimulation of cathodal/anodal/sham tDCS-rTMS therapy. Patients will be studied with same battery of tests at 15 days and 2 months after start of dual stimulation therapy.



IN-SILICO ANALYSIS OF MORPHOGENESIS NETWORK OF FUNGAL PATHOGEN CANDIDA ALBICANS

Systems and Synthetic Biology, Mathematical Biology, Computational Biology, Big Data Analytics, Dynamical Systems Theory

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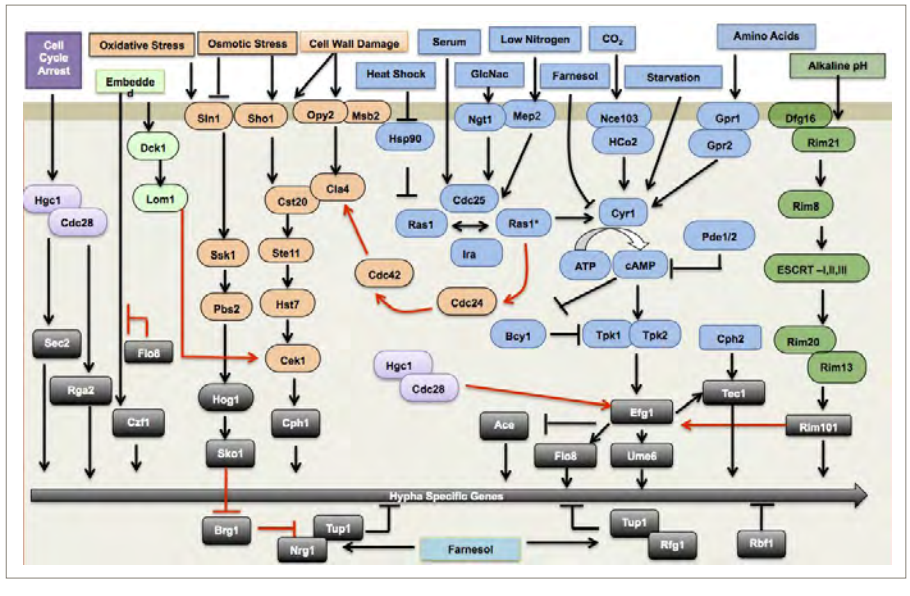
Predicting how pathogenic microbes behave in response to host stimuli is a difficult but an essential biomedical challenge, as it is key to drug discovery and therapeutic processes. I aim address this Grand Challenge in the context of understanding the key pathogenesis factor of human fungal pathogen Candida albicans (C.albicans) – Morphogenesis. C. albicans switch between yeast, hyphae and pseudo-hyphae forms in response to a range of host stimuli. Morphogenetic plasticity is an essential virulence factor of C. albicans as it confers the pathogen the ability to adhere, colonise and invade host-tissues; and disseminate infection. Morphogenesis is crucial for biofilm formation, altering host-pathogen outcomes and anti-fungal drug resistance. Morphogenetic mutants are also less virulent. Morphogenetic transition in C. albicans is primarily governed by following six signalling cascades: (1) cAMP-Protein Kinase A pathway (2) mitogen activated protein kinase pathways (HOG1 and CEK1) (3) pH pathway (4) embedded pathway (5) cell cycle arrest pathway and (6) negative regulation pathway. Environmental signals stimulating these signalling cascades, their mechanisms of regulation as well as the existing cross talks between the six morphogenetic cascades are reasonably well established. Despite our elaborate knowledge about cellular pathways/components involved in morphogenesis and its importance in Candida pathobiology, there are huge gaps in our understanding of this phenomenon. This is because, traditional reductionistic investigations conducted so far, have only given us a linear view of this complex phenomenon. An inter-disciplinary systems biology approach is absolutely crucial to dissect such a system. A grand mathematical model of Candida's morphogenesis network is constructed and analysed to provide the much-needed quantitative and holistic understanding cellular-processes governing morphogenesis.



THE SAME YET DIFFERENT: UNIQUE STRUCTURAL DYNAMICS DISTINGUISH SIX ATG8 HUMAN ORTHOLOGS

Computational Biology, Biophysics, Structural Biology, Structural Bioinformatics, High-performance Computing

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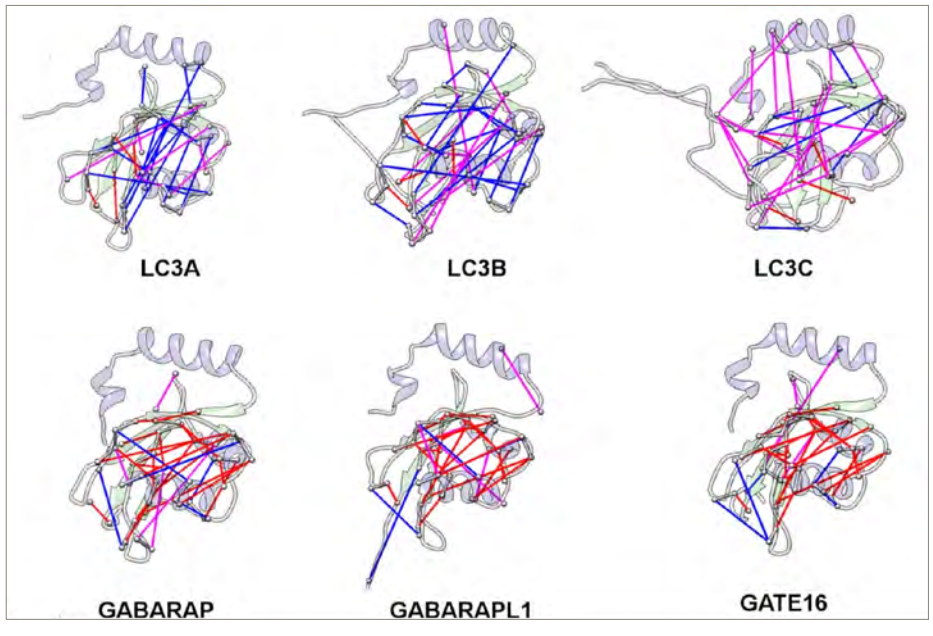
During autophagy, six distinct members of LC3 homologs are involved in autophagosome initiation and expansion. Although LC3B labelling is perhaps the most well-established and representative marker for autophagy, the ambiguity regarding its functionally diverse evolutionary partners (LC3A, LC3C, GABARAP, GABARAPL1, and GATE16) is striking. Significant questions have persisted about how these structurally identical proteins exhibit different binding partners and what are factors leading to preferential substrate recognition. We set out to compare and contrast the six Atg8 human orthologs using a detailed computational framework that relied on experimentally resolved crystal structures. We found that structural divergences arise from non-covalent interactions between microclusters (functional modules), whereas direct backbone movements rarely occurred. Comparisons of binding modes of these proteins further indicated that the differences in substrate specificities are achieved by subtle rearrangements in pocket volume, hydration pattern, and contributions of specific residues in defining unique binding. Our results thus illustrate the significance of these weak albeit specific non-covalent interactions within under-studied LC3 family members, and provides the molecular-basis of understanding their properties that plays a fundamental role in expanding autophagic machinery.



HIPPO SIGNALING IN BREAST CANCER PROGRESSION

Hippo Signalling, Breast Cancer, Metastasis, Translational Research

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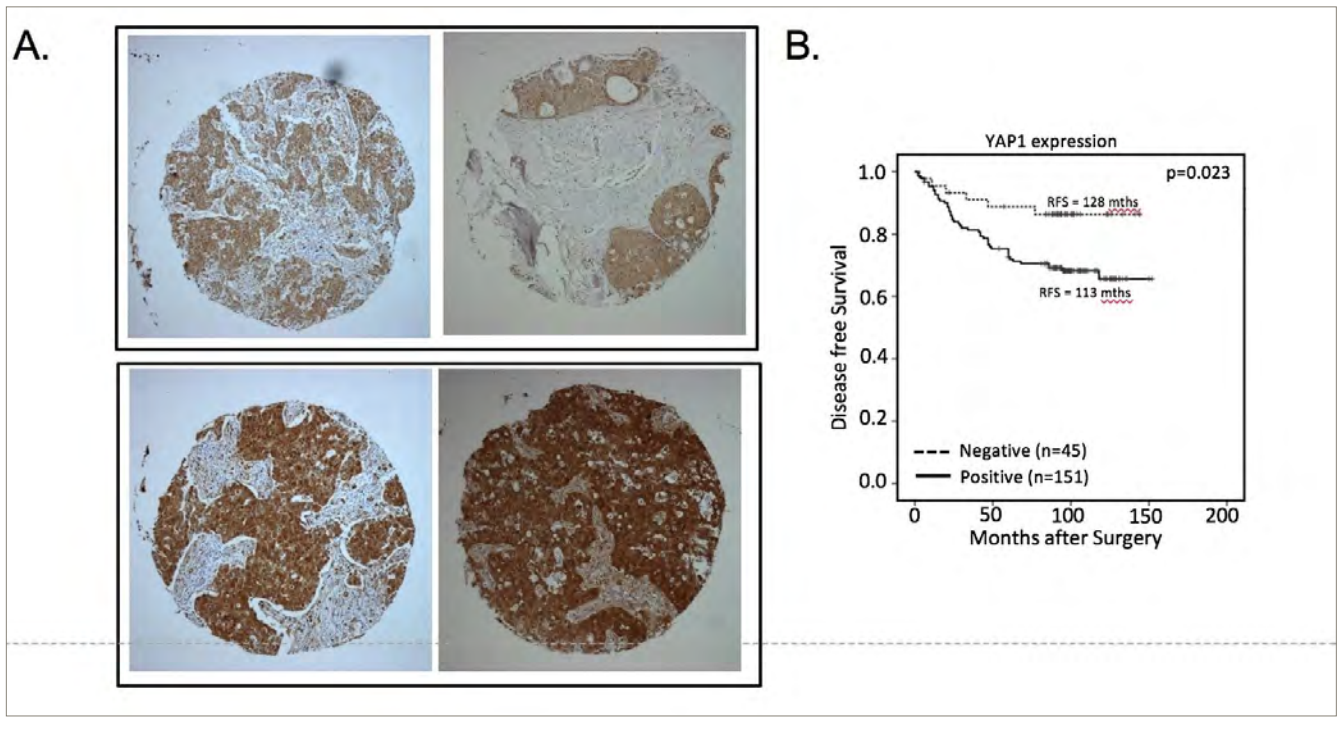
Breast cancer is one of the commonest cancers and causes of cancer death amongst women. Breast cancer incidence has increased by 20% in the last five years (GLOBOCAN 2012). Breast cancers are assessed based on their expression status of the hormone receptors. About 70-85% of the cancers are hormone receptor positive for Estrogen (ER), Progesterone (PR) and/or HER2. 10-15% of all breast cancers that lack expression of these three hormone receptors are triple negative breast cancer. A subset of these breast cancers present with aggressive parameters that carry a higher risk of recurrence and mortality. Such subtypes, pose a therapeutic challenge to the clinicians till today.

In our preliminary analysis we have discovered a novel molecular marker, Yes-associated protein; YAP, to be associated with earlier recurrence status and we have confirmed its association with reduced survival outcomes in breast cancer patients using a 248-breast cancer patient tissue microarray (TMA) availed from NUH, Singapore. Phenotypic manifestations of YAP expression in the cell-based model system and further characterization of subtype-specific expression data in breast cancer led us to hypothesize YAP's involvement in initiating epithelial-mesenchymal transition (EMT) and stemness. Thereby promoting metastatic progression in breast epithelial cell lines. Currently, we are validating the association between YAP-gene signature and metastatic and recurrence status of breast cancers using Indian Breast cancer tissue samples.



THE HUMAN Y CHROMOSOME: BIOLOGICAL RELATEDNESS AND THE ROUTE OF MIGRATION OF THE THREE TRIBES OF NORTH EAST INDIA

Molecular Biology, Demographic Anthropology, Human Ecology and Adaptation, Human Biological Diversity, Public Health

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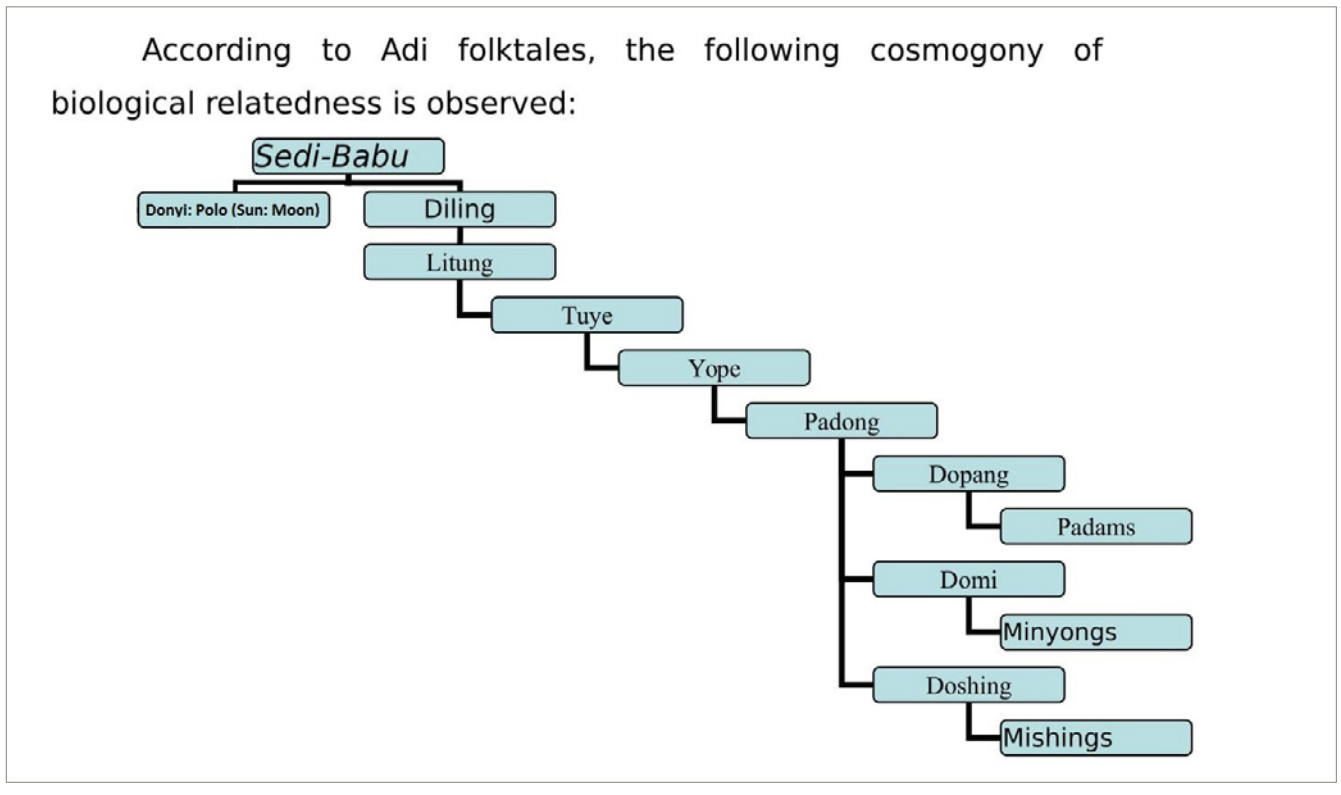
The various socio-cultural and ecological studies done on the Adi tribes of Arunachal Pradesh (the Easternmost hilly state of North-East India) and the Mishing tribe of Assam (neighbouring plains state to Arunachal Pradesh) refer that the Minyong and the Padam tribes who are within the broader framework of the 'Adi', show immense socio-cultural and linguistic affinities which have led to the belief that these are biologically cognate tribes and had derived from the same ancestral stock. Such kind of biological relatedness in also popular in the Adi traditional folklore as well as in the pristine Mishing folk tales. This investigator would like to keep these pristine beliefs of cosmogony in the background in order to lead to the genomic study of these three tribes to know their biological affinities as well as their route of migration. Very recently, apparently from the beginning of 2000-2001, human origin and migration stories have been revealed by the studies of Y chromosome haplogroups. Very few such kind of studies are conducted among the North East Indian tribes to know about their genetic structure.



USING HIGH-THROUGHPUT BIOLOGY TO TACKLE ANTIBIOTIC RESISTANCE

Computational Biologist, High Throughput Data Analysis, Microbial Pathogenesis, Antimicrobial Resistance

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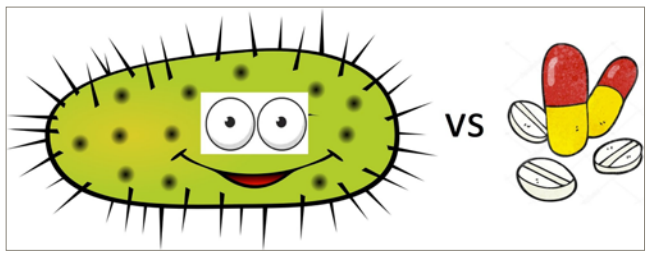
Whole-genome sequencing and large-scale gene expression profiling technologies have revolutionized biological research. The large amount of data produced by them has shifted the trend of focusing one gene or protein at a time, to investigate the behavior and relationships of functioning molecules simultaneously. To facilitate this it is crucial to develop suitable and realistic models and efficient algorithms that can extract useful information from data. We have constructed a library of antibiotic resistance genes (ARGs) to find the diversity of ARGs in metagenomic samples. This library is being used in a collaborative study with a virologist to understand the role of bacteriophages in dissemination of ARGs among bacteria.



INVESTIGATIONS ON THE EXPRESSION OF BETALAINS IN PLANTA VIS-A-VIS ANTIOXIDANT METABOLITES AND ENZYMES

Betalains, Biosynthesis, Bio-mimetic Synthesis, Bioavailability, Bioactivity

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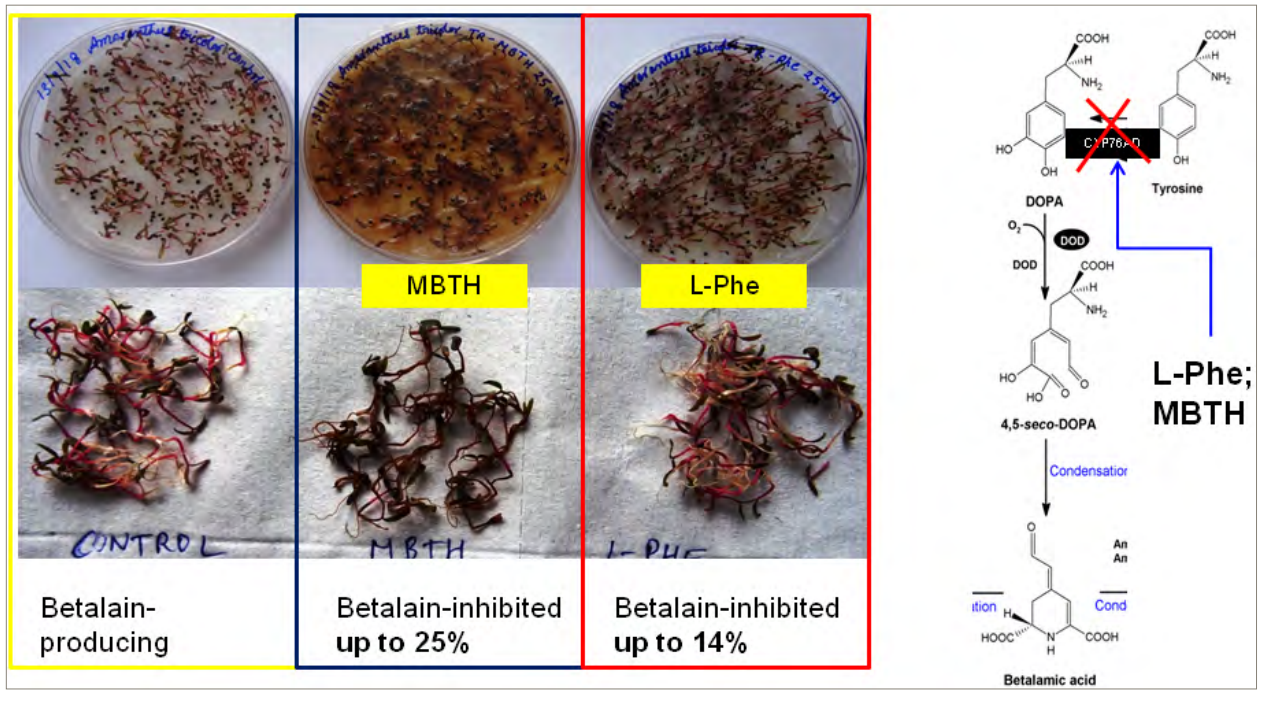
Edible leafy vegetable Amaranth (Amaranthus spp.) has received considerable attention because of its antioxidant property, high nutritional value and rich presence of natural pigment such as betalains, and others with potential for food colourant applications. Betalains are hydrophilic and high tinctorial pigments having pharmacological properties. Compared to the available literature on clinical efficacy of betalains, the role of betalains in plants is still a subject of investigation. In this study, Amaranthus spp. seedlings were used as model for investigating the effect of betalain accumulation on other antioxidant metabolites and enzymes in the presence of its biosynthetic inhibitors such as L-phenylalanine (L-Phe; 14% inhibition) and MBTH (25% inhibition). Though betalains have been known to be antioxidative, its effect on the expression of other antioxidants in the plant is yet to explain. The results of this study reveal that when there is decrease in betalain content, other antioxidants increase significantly. Our findings to decipher the role of betalains in planta will be presented.



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THERAPEUTIC GENOME EDITING FOR BETA-HEMOGLOBINOPATHIES

Genome Editing for Gene and Cell Therapy, Cancer Stem Cells, Tumor Modeling using the, CRISPR/CAS9 system, Cancer Immunotherapy

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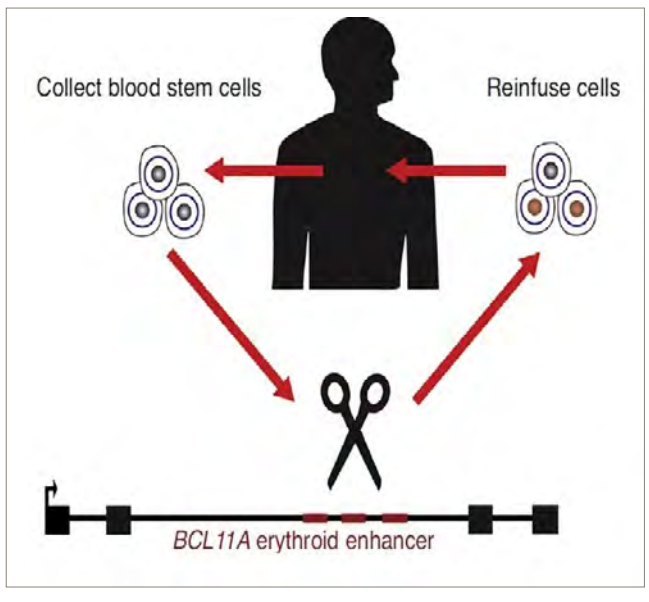
The switch from fetal to adult hemoglobin is a very important developmental event that occurs in erythroid cells during the months following birth. Reversing the fetal to adult hemoglobin switch is substantial therapeutic interest for sickle cell disease, since persistence high levels of fetal hemoglobin (HbF) ameliorates clinical symptoms of sickle cell disease and β -Thalassemia. Genome wide association studies identified transcriptional repressor BCL11A as a major regulator of the hemoglobin expression. Inactivation of BCL11A in mice carrying a human β-globin cluster transgene leads to profound delay in globin switching and impaired HbF silencing. Knockout of Bcl11a alone is sufficient to rescue phenotype of mouse model of sickle cell disease. BCL11A is dispensable in non-erythroid functions such as for normal lymphoid and neural development. Genomic analysis identified the enhancer that is specific in erythroid but not B-lymphoid cells for BCL11A expression. Functional mapping of the Bcl11A enhancer identified the minimal critical sequence that is necessary for its function. The effects of deletions of these specific regions of enhancer have the same effect as altering the whole enhancer. In this study, we utilized genome engineering platform based on CRISPR/Cas9 system to edit BCL11A erythroid specific enhancer in human hematopoietic stem cells for the treatment of β-Hemoglobinopathies.



DISRUPTION OF MYC/MAX PROTEIN-PROTEIN INTERACTIONS IN MEDULLOBLASTOMA STEM CELLS: HOT SPOT DETECTION, STRUCTURE-BASED VIRTUAL SCREENING, BIOPHYSICAL AND IN VITRO TESTING.

Brain Tumours, Cancer Stem Cells, Novel Molecules/Compounds Discovery To Target Cancer Stem Cells, Micrornas And Epigenetic, Nanoparticles For Drug Delivery

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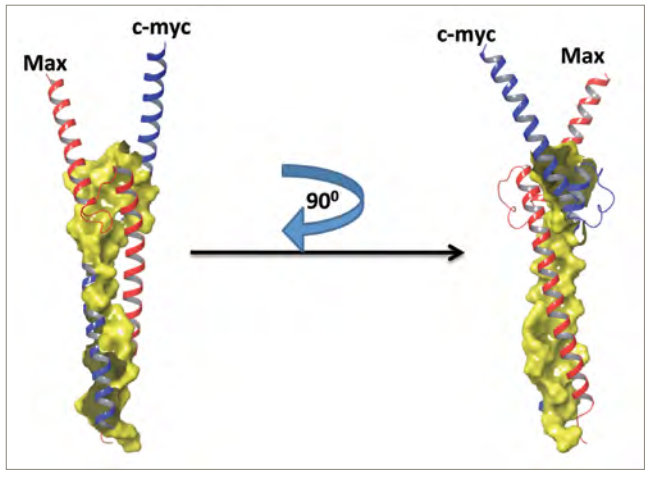
The oncogenic protein myc is a master regulator of pediatric brain cancer stem cells and therefore is among the most attractive of cancer targets. C-myc is identified to play a central role in the pathophysiology of the highly metastatic group C Medulloblastoma cancer stem cells. C-myc is involved in protein-protein interactions (PPI) with Max, and disruption of these interactions could be a possible means to target the function of myc in cancer stem cells. In this work, we have detected the hot spot residues in myc/max dimer using hot-spot computational predictors like HotSpot Wizard, PredHS , KFC and Robetta. Extensive molecular dynamics simulations were applied to generate an ensemble of conformations and were used to quantitatively estimate the binding free energy of the identified hot spot residues using MM-PBSA alanine scanning mutagenesis and per-residue energy decomposition. As predicted by computational predictors and estimated binding free energy, the myc/max residues were designated as "hot spots". Virtual screen of potential inhibitor that could disrupt myc/max dimer was done and inhibitor was further investigated for biophysical and kinetic studies and later tested for its inhibitory potential on Medulloblastoma group C cancer stem cells. The peptidomemitic compounds of ChemDiv and 2P2I shows appreciable binding affinity toward the hotspot myc target residues. The compounds with utmost binding affinities were further analyzed for their binding stability with Molecular Dynamic simulation. The most stable compounds showed promising anticancer potential toward the Medulloblastoma cancer stem cells with low IC50 value. The present study provides a confidence to disrupt the protein-protein interaction with peptidomemitic compounds by specifically targeting the hot-spot target residues. (Grant Acknowledgements: Ramanujan SERB grant, India (SB/S2/RJN-072/2015)).



IMPEDING DNA BREAK REPAIR ENABLES OOCYTE QUALITY CONTROL

Reproductive Biology and Disorders, DNA Damage Response, Oocyte Atresia, Post Translational Modifications In Meiosis

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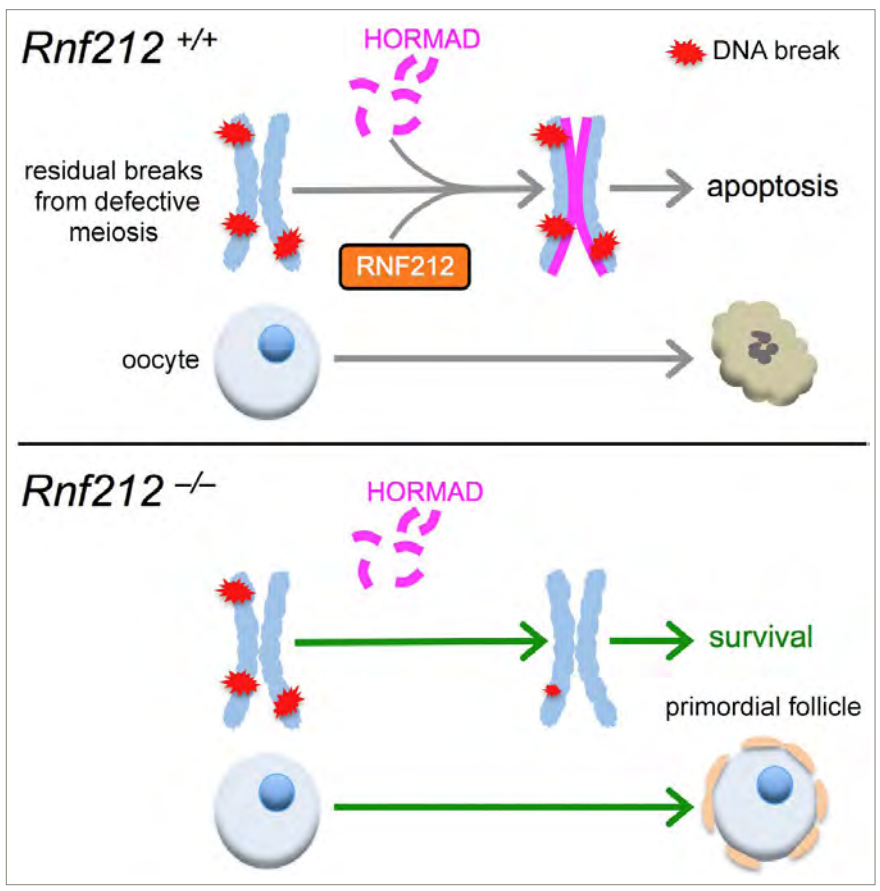
Oocyte quality and number are important determinants of reproductive success (1). These attributes are influenced by the selective elimination of oocytes that experience problems during the early stages of triggering perinatal loss of more than two thirds of all fetal oocytes in a conserved process called fetal oocyte attrition (2). Defects in the chromosomal events of meiotic prophase also trigger oocyte loss, typically later than fetal oocyte attrition, during the early postnatal period before oocytes arrest and become quiescent (3). This second wave of oocyte death is mediated by interrelated pathways that signal defects is DSB repair and homolog synapsis (3-5). Together, these perinatal and postnatal processes balance the quality and size of the ovarian follicle reserve to maximize reproductive success. In this study, we implicate a new factor, RNF212, in postnatal oocyte apoptosis and show that it functions in a counterintuitive process that helps oocytes to gauge whether meiotic prophase was defective.



Ramanujan SERB grant, India (SB/S2/RJN-072/2015)).

DISSECTING THE MOLECULAR MECHANISMS OF CROSS-TALK BETWEEN ROOT DEVELOPMENT PROCESS AND SALT STRESS SIGNALLING

Arabidopsis, Salinity, Kinase, Mass spectrometry, Phosphorylation

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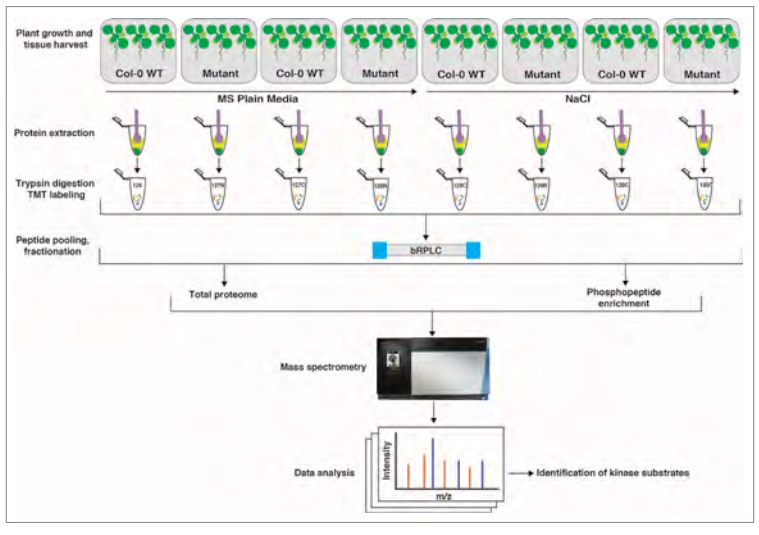
Salinity stress is one of the most devastating factors that adversely affect crop yields worldwide. Responses to salt stress are known to be initiated through cascades of phosphorylation reactions on multiple plant proteins.The roles of few kinases have been implicated in the signal transduction process of salt stress [1]. Similarly, a network of kinases is known to regulate root development [2]. This indicates an intricate mechanism governing root development and stress tolerance. Here, we screened A. thaliana mutants that were defective in root morphology and displayed differential sensitivity to NaCl stress. Search for these mutants was carried out using literature survey through PubMed and the eplant tool that also contains mutant information. Interestingly, our search led to the identification of one kinase mutant (At_Kin1) that belong to the Salt Overly Sensitive (SOS) Pathway. The mutant displayed both, abnormal root morphology and hypersensitivity to salt stress. The mutant was ordered from TAIR and screened for homozygosity. We carried out an in depth phosphoproteome analysis of At_Kin1 under control (MS plain) and salt stress conditions using a combination of TiO2 enrichment and high-throughput mass spectrometry. The analysis revealed the identity of several potential At_Kin1 targets. The data provides a detailed understanding of molecular mechanisms of cross-talk between root development process and salt stress signalling at the phosphoproteome level. The identified molecules can be utilized as targets to develop innovative strategies to manage salt stress for agricultural benefits.



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MOLECULAR MECHANISMS OF POLO-LIKE KINASE, PLK4, RECRUITMENT AND FUNCTIONING DURING CENTRIOLE DUPLICATION

Centrosome, Cell Division, Microtubules, Cilia, Mitosis

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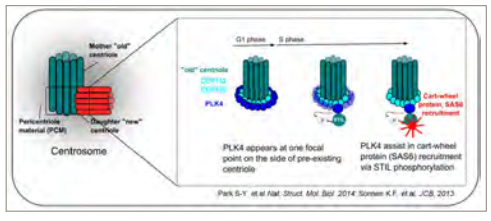
Centrosomes are microtubule-organizing centers of the cell. It is consisting of a pair of barrel-shaped structures, called centrioles, exhibiting nine-fold symmetric (cartwheel-like) arrangements of microtubules and they are surrounded by a cloud of proteins known as pericentriolar material (PCM). They are involved in several cellular functions, ranging from spindle formation, cell migration to cell polarization. The centriole duplication is coordinated with the cell cycle. Interesting parallels can be drawn between centrosome duplication and DNA replication, e.g. i) a new centriole is made adjacent to a pre-existing "old" centriole; ii) centriole duplicates during S-phase of the cell cycle; and iii) cell-cycle kinases like cyclin-dependent kinases (CDKs) and Polo-like kinases (PLKs) are also involved in centriole duplication, thus ensuring that each resulting daughter cell inherits only one centrosome (i.e. one pair of centriole). PLK4, belonging to the family of Polo-like kinases (PLKs) is a master regulator for centriole duplication, as its overexpression has been shown to result in flower-like arrangement i.e. unregulated centriole duplication in the S-phase arrested cells (Kleylein-Sohn, J et al., 2007). Both, increase (Ko M A. et al., 2005) or decrease (Kuriyama, R et al. 2009; Pellegrino, R. et al. 2010) in the expression of PLK4 has been reported in variety of cancers (Shinmura, K. et al. 2014) but its functional relevance remains to be tested. We are interested in investigating: *1) How PLK4 gets recruited at the right place and at the right time to duplicate centrioles? and, 2) Exploration of the molecular implications of its kinase activity during centriole duplication.*



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MODELING SKELETAL CILIOPATHIES *in vitro* AND IN *Drosophila* TO DISSECT THE ROLE OF IFT52 AND EXOC6B IN PRIMARY CILIA MEDIATED REGULATION OF OSTEOBLAST AND CHONDROCYTE DIFFERENTIATION

Primary Cilia And Ciliopathies, Mechanisms Of Eukaryotic Transcriptional Regulation, Human Population Genetics, Ancestry Information And Disease Biology, Drosophila And Mammalian Cell Culture Models Of Human Diseases

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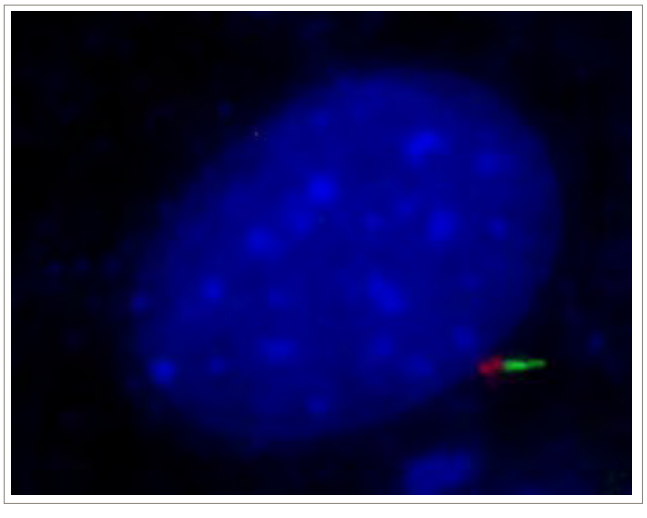
Using whole exome sequencing our center had reported pathogenic variants in IFT52, a component of the intraflagellar transport (IFT) complex B of primary cilia and EXOC6B, a constituent of the exocyst complex that facilitates vesicular transport to be associated with two novel skeletal ciliopathies. To delineate the role of IFT52 and EXOC6B primary cilia dependent modulation of osteogenesis and chondrogenesis, mammalian cell culture and *Drosophila* models are being optimized. Osteoblastic (OS) and chondrogenic (CH) differentiation were induced *in vitro* and ascertained by assaying for known differentiation markers. In each context primary cilia and basal bodies were assessed. OS differentiation was evident at 7 days post induction via Alizarin red (AR) staining to reveal extracellular matrix (ECM) mineralization. It was correlated with elevated expression of osteoblast differentiation marker - Alkaline phosphatase, upregulated by ~ 23 and 80 fold at 7 and 14 days post induction. CH differentiation was first detected at 14 days post induction by sulfated proteoglycan deposition and ECM mineralization using Alcian blue and AR staining, respectively. Known ciliary genes, ARL13B, a membrane associated small GTPase critical for ciliary axoneme structure and IFT88, a core component of the IFT-B complex and an IFT52 interacting partner co-localized with the primary cilia that were significantly enriched at 7 and 14 days following OS and CH differentiation, respectively. Importantly during OS differentiation, IFT52 transcript levels were nearly doubled at 7 and 14 days, while EXOC6B expression increased ~ 3 fold at day 7 but diminished to basal levels subsequently. These studies will be followed by lentiviral-based RNA interference to silence IFT52 and EXOC6B and assay their roles in primary cilia dependent control of osteogenesis and chondrogenesis. We are also examining the role and regulation of IFT52 in cilia assembly and function in *Drosophila.*



MECHANISTIC INSIGHTS INTO ENZYMATIC CATALYSIS BY TREHALASE

Carbohydrate Metabolism, Insect, Gustatory Receptor, Lepidoptera, Trehalose Metabolism

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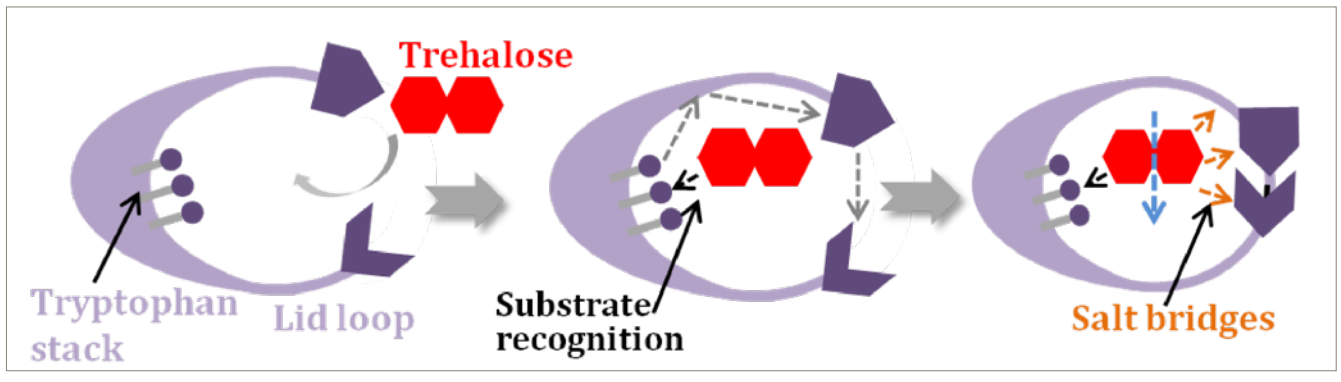
Energy metabolism in the diamondback moth, *Plutella xylostell*a is facilitated by trehalase from the predominant gut bacterium *Enterobacter cloacae*, by assisting in trehalose hydrolysis. We report the biochemical and structural characterization of recombinant trehalase from *Enterobacter cloacae* (Px_EclTre). Crystal structures of Px_EclTre were determined in the ligand free form and bound to the inhibitor Validoxylamine A. The crystal structure of the ligand free form, unavailable till date for any other bacterial trehalases, enabled us to delineate the conformational changes accompanying ligand binding in trehalases. Multiple salt bridges stabilised closure of a hood over the substrate- binding site. A cluster of five tryptophans lined the -1 substrate binding subsite and interacted with crucial active site residues, and contributed to both trehalase activity and stability. Importance of these residues in enzyme activity was further validated by mutagenesis studies. Structural analysis of the conformational changes led to the assignment of functional roles for many of the putative sequence motifs in trehalases. This information can be further explored for the design of effective inhibitors against trehalases.



STRUCTURAL ANALYSIS REVEALS DIFFERENTIAL CONFORMATIONAL DIVERSITY IN DRUGGABLE ENZYMES

Cancer Biology, Chaperone Biochemistry, Medicinal Chemistry, Human/Mouse Genetics , Computational Biology

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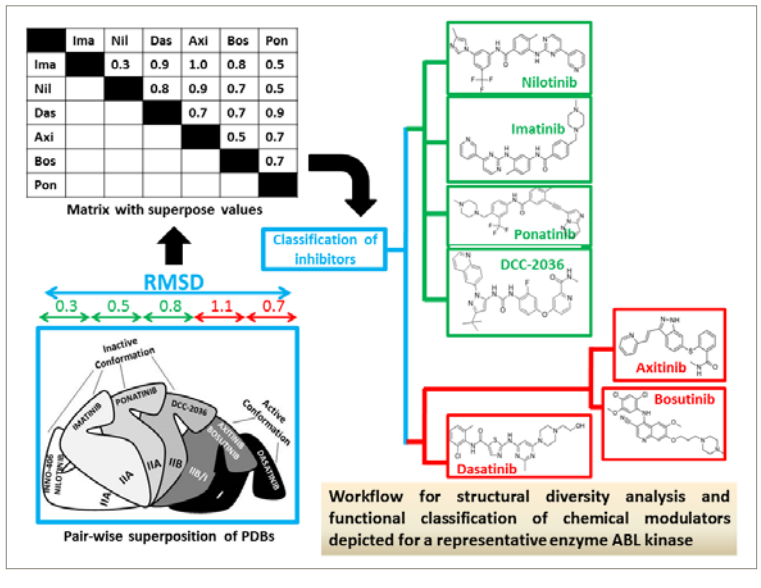
The importance of protein conformation in an enzyme's function is well understood. The presence of an ensemble of conformations for a given enzyme enables the modulation of its function by diverse chemical entities, each of which is selective/specific to a particular conformation. Therefore, co-crystal structures of enzyme-inhibitor complexes reveal the variety in an available conformational ensemble for a particular enzyme. The enzyme's conformational diversity thus reflects chemotype diversity among the available chemical modulators for that particular enzyme.

We have analyzed the conformational diversity of each of the 149 druggable enzymes and compared between enzymes of multiple classes. Our results indicate that a greater structural diversity is present in kinases than that of non-kinase enzymes. We further show that a greater percentage of tyrosine kinases exhibited a high conformational diversity implying the availability of a broader spectrum of chemotypes for this class of enzymes. Apart from establishing conformational continuum for druggable enzymes, we also classified chemical modulators based on the conformation they stabilized.

Given the role of enzymes in pathogenesis of multiple diseases, our results indicate a need to explore for novel chemical entities that might result in achieving broader chemotype diversity in non-kinase enzymes. The data provided in this study may serve as a resource for chemical biology studies aimed at delineating the relationship between an enzyme's structure (conformation) and its function. For example, we have previously used a chemical biology approach and showed that the conformation of multiple oncoproteins impact their interaction with HSP90 chaperone using kinase inhibitors that are specific for either inactive or active conformations (Kancha et al., 2013). Furthermore, the information regarding unexplored conformational landscape for non-kinome may elicit interest among drug development programs.



Workflow for structural diversity analysis and functional classification of chemical modulators depicted for a representative enzyme ABL kinase

REGULATION OF NEUTROPHIL INFILTRATION; IMPLICATION IN META-INFLAMMATION AND METABOLIC SYNDROMES

Metabolic syndromes, Inflammation, Neutrophil Heterogeneity and Type 2 Diabetes, Hematopoiesis, Ageing

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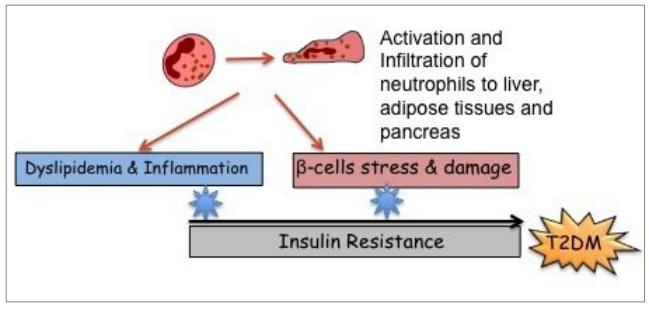
Leukocytes, particularly neutrophils, reach to site of infection to combat intruders. However, aberrant neutrophil activation and accumulation into tissues trigger tissue damage due to release of a plethora of toxic oxidants and proteases. Furthermore low grade chronic or meta-inflammation is associated with metabolic syndromes including type 2 diabetes. Although, neutrophil recruitment cascade multi-steps are well defined with rolling, adhesion, polarization, activation and diapedesis, but molecular mechanisms of these steps are not fully defined. In our recent studies, we investigated role of small GTPase, which regulates cytoskeleton, in neutrophil migration. Using structure/ function, live cell microscopy and biochemistry approaches, I reported that Rho GTPase, Cdc42 via its effector Wiskott-Aldrich Syndrome protein (WASp), regulates the crosstalk of integrin CD11b and microtubules- a key process in neutrophil migration towards site to infection/ inflammation (Blood J, 2012, 2014). We also identified that Rap1b works as negative regulator of neutrophil infiltration during acute lung injury (JEM, 2014). The small GTPase Rap1b is generally viewed as a positive regulator by controlling bidirectional integrin signaling. Unexpectedly, We found that Rap1b-deficient mice exhibited enhanced neutrophil recruitment to inflamed lungs and enhanced susceptibility to endotoxin shock. My lab is current focusing on differential activation and infiltration of neutrophils in diverse organs including in initiation and development of type 2 diabetes. We also got some interesting data about redox regulation of immune cells and their association with type 2 diabetes.



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CD38-NAD+AXIS REGULATES IMMUNOTHERAPEUTIC ANTI-TUMOR T CELL RESPONSE

Tumor Immunology, T Cells, Metabolism

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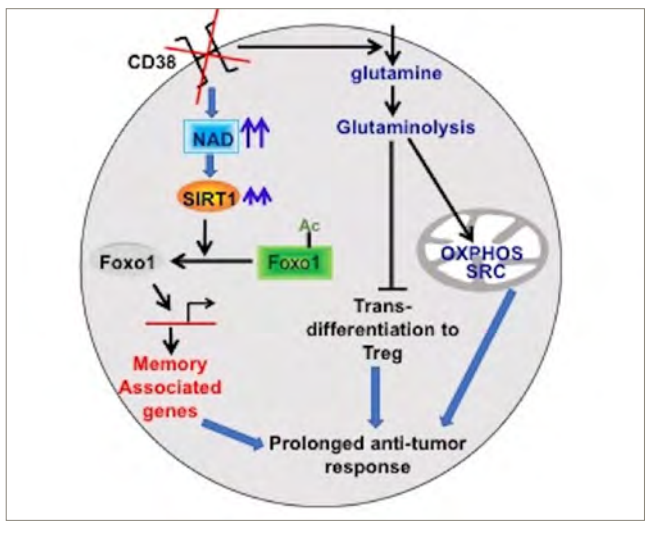
Adoptive T cells therapy (ACT) has shown great prominence in immunotherapy of cancer. However, ACT is only able to cure a small proportion of the patients because of the rapid functional exhausting and short-term persistence in tumor bearing host.Therefore, identifying factors governing the maintenance of functional phenotype and survival of anti-tumor T cells is of utmost interest. Recently, we found that Th1/17 cells which can be differentiated by combining the culture conditions of Th1 and Th17 cells, exhibited durable and superior anti-tumor activity in vivo as compared to Th1 and Th17 alone. Since metabolic fitness affects the persistence and functionality of the adoptively transferred T cells, we comprehensively characterized the metabolomic profile of Th1, Th17 and Th1/17 cells and identified that increased level of intracellular NAD+ is a key in regulating the anti-tumor potential of the Th1/17 cells. NAD+ mediated this effect mainly through providing substrate for the SIRT1 deacetylation activity since pharmacological blockade or genetic ablation of SIRT1 in Th1/17 cells completely abolished its anti-tumor activity. We further provided data in support of this observation by using CD38 deficient T cells which has very high level of NAD+ due to absence of NAD+ lyase CD38. We found that CD38 deficient T cells without any in vitro differentiation could mount potent anti-tumor response in mice bearing melanoma. These data together suggest that strategies to maintain high level of intracellular NAD+ in anti-tumor T cells by blocking CD38 will have a potential therapeutic application in improving ACT.



YIN AND YANG OF IKK SIGNALING: TARGETING BEYOND THE ACTIVE SITE TO TAME NF-KAPPAB

Protein Kinases And Phospho-Proteome, Signaling Modularity, Chemical Biology, Cryo-EM, Mass Spectrometry

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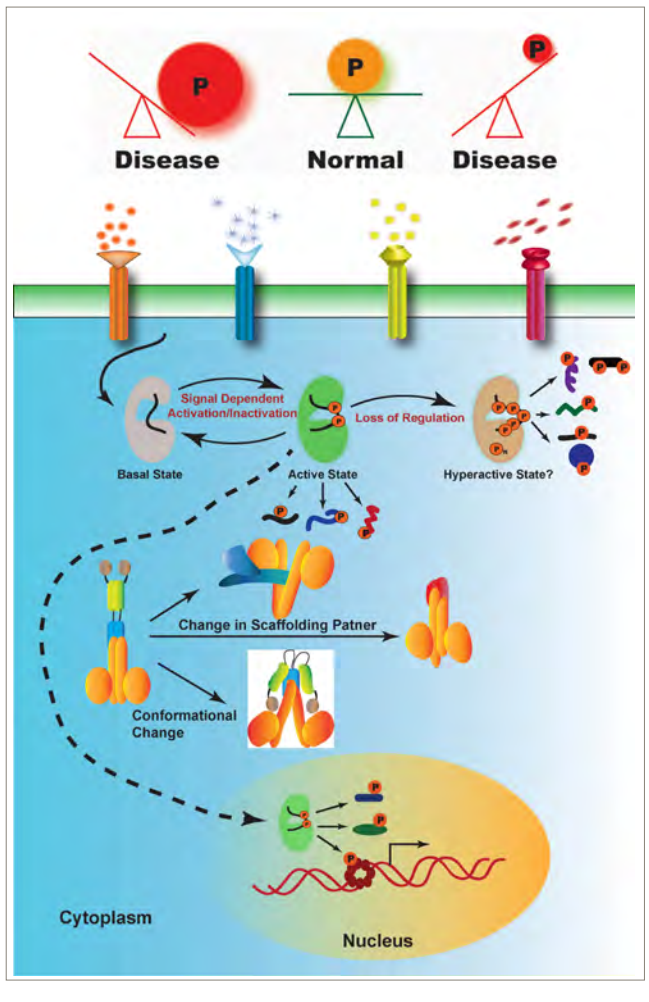
Activation of IKK-complex is the gateway to NF-kappaB activation. Canonical IKK-complex is composed of two catalytic subunits, IKK1/IKKalpha and IKK2/IKKbeta that bear Ser/Thr kinase domains and a non-catalytic regulatory scaffolding subunit, NF-kappaB Essential Modulator (NEMO)IKK activity is tightly controlled in cells and loss of this regulation has been associated with many human diseases, and its potential as a drug target was readily realized. Subsequently, many highly specific and potent active-site directed IKK-inhibitors were developed with little clinical application owing to intolerable side effects upon prolonged treatment. It is important to inhibit aberrant IKK-activity leaving the basal and protective IKK-activity intact, an aspect that these inhibitors failed to achieve. In another approach, peptides targeting the protein:protein interaction interface between IKK and NEMO were designed and used in many disease models with promising outcomes. As the importance of higher order assemblies in signal transduction is becoming increasingly clearer, it will be beneficial to realize the structural and biochemical differences between the IKK-complexes (minimal and higher order), before and after induction by a stimulus. We are also exploring the possibility that these kinases may have a broader substrate pool than anticipated. We are using chemical genetics and Mass Spectrometry in this pursuit. We have identified at least two proteins that can be phosphorylated by IKKs in vitro. Currently efforts are undertaken to find out if they could be phosphorylated by IKKs in a signal dependent manner, and what impact would these phosphorylations have on the cellular well-being. Targeting novel protein:protein interaction interfaces in the activated complex as well as their interaction with novel substrates may lead to better clinical outcomes.



OPERANT CONDITIONING AND RETENTION FOLLOWING AMPUTATION IN *Schmidtea mediterranea*

Development, Regeneration, Memory, Biophysics & Systems Biology, Size And Growth Control

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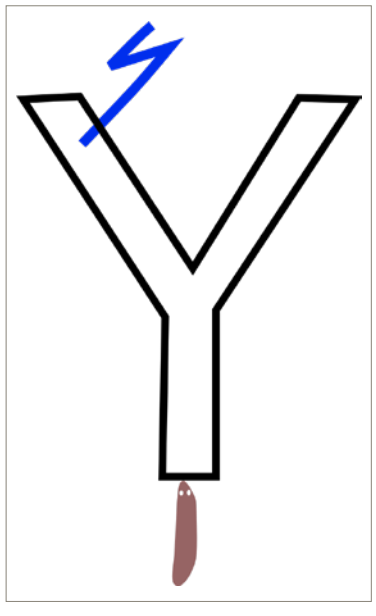
Spatial memory plays a crucial role in survival and hunting alike for a large portion of the motile animal kingdom. Planaria, flatworms with a primitive nerve system, are a well-known model organism in the area of regeneration studies. Over 60 years ago, planaria also gained fame over their ability to be trained using associated learning paradigms. Any memory that was imposed on the animals was also observed to remain in progeny that regenerated from large body amputations.



However, the scientific community quickly lost interest in these memory studies when some of the work proved to be irreproducible. More recently, some bold new work, with the use of a custom built automated training paradigm, free of investigator bias, has shown that planaria are indeed capable of associate learning. We examine whether simple spatial memory formed after training in a Y-shaped maze is maintained in all amputated parts following regeneration. Irrespective of the initial size of the amputation, we find that that the resultant progeny maintain their training - i.e. the directional biased imposed on them. Thus our results indicate that neither memory formation, nor inheritance of memories is mediated by a particular location of the body of the planaria. In many other organisms, memory is located primarily in a central control system such as brain. However, in planaria despite the presence of a thick nerve system in the cephalic ganglion, there does not appear to be a central memory and information processing system.

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UNUSUAL LINKED THIOL-BASED REDOX METABOLISM OF PARASITIC HELMINTHS: NOVEL PATHWAY FOR DRUG DISCOVERY

Protein Science, Molecular Biophysics, Drug Discovery, Biochemistry, Parasite

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Fascioliasis, a food-borne disease, caused by Fasciola hepatica or Fasciola gigantica infection to domestic ruminants causes major economic losses (> US$3 billion p.a.) in livestock production. In humans, fascioliasis is also an important zoonotic disease that has infected 2.4–17 million people and has put approximately 180 million people at risk globally. WHO recommends triclabendazole for the treatment of fascioliasis. However, recent studies have suggested that the endoparasites have gained resistance to triclabendazole in several regions of the world. Parasitic flukes are exposed to free radicals during their life cycle. Despite being relentlessly exposed to ROS released by activated immune cells, these parasites can survive for many years in the host. Cellular thiol-based redox metabolism plays a crucial role in parasite survival within their hosts. One of the redox-sensitive enzymes, thioredoxin glutathione reductase (TGR), has been accepted as a drug target against blood fluke infections, and related clinical trials are in progress. We have performed molecular and structural studies on the proteins involved in thiol-based antioxidant system of the liver fluke- F. gigantica. The thiol-disulfide redox metabolism in platyhelminth parasites depends entirely on a single selenocysteine (Sec) containing flavoenzyme, thioredoxin glutathione reductase (TGRsec). We investigated the catalytic and structural properties of different variants of F. gigantica TGR to understand the role of Sec, the co-factor FAD, and various domains in maintaining the structure-function relationship of TGRsec. Biochemical studies revealed that Sec is responsible for higher thioredoxin reductase (TrxR) and glutathione reductase (GR) activity of FgTGRsec. The N-terminal Grx domain was found to positively regulate the TrxR activity of FgTGRsec and also stabilized the overall FgTGRsec structure. Alteration in the FAD microenvironment was directly proportional to the loss of TrxR and GR activities. Based on these results, we concluded that the Grx domain stabilizes the full-length FgTGRsec protein for efficient catalysis. Thus, we suggest that in platyhelminth parasitic flukes, during evolution, the Grx domain merged with the TrxR domain to confer higher catalytic activity and provide additional structural stability to the full-length TGR.

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ROLE OF HOST-MICROBIOTA-DERIVED METABOLITES IN MODULATING INTESTINAL INFLAMMATION

Macrophage Biology, Innate Cell Signaling, Microbial Pathogenesis, Immune Homeostasis, Metabolism

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Derailed inflammation associated malignancies are alarmingly taking prominent position in the rankings of origin of cancer and related deaths. Of which, Colitis associated cancer (CAC) is emerging to be a third form of malignancy and alarmingly, the second most causative factor of cancer-related deaths worldwide. Adding to the existing underlying reasons such as inherited mutations familial adenomatous polyposis and hereditary nonpolyposis colorectal cancer syndrome, there is also increasing evidence suggesting that high-calorie diet significantly contributes to the incidence of CAC with clinically detectable signs of inflammatory bowel disease (IBD). Supporting, the patients with IBD are found to more likely develop CAC than patients without IBD. Deeper insight into the basis of IBD and CAC deciphered that a fascinating relationship exists among immune cells; gut microbiota and host-gut microbiota derived secondary metabolites. Under physiological conditions, intestinal microbiota has been identified to profoundly influence host intestinal immune cell composition by producing secondary metabolites and considered to be one of the regulatory factors in maintaining gut immune homeostasis. In this context, we are interested in understanding the ability of liver secreted bile acids and their corresponding microbiota-derived secondary metabolites regulating not only the immune cell function but also maintain intestinal barrier integrity.

FORENSIC CLASS CHARACTERIZATION FROM SEIZED RHINO HORN: A TOOL FOR COMBATING TRANSBOUNDARY ILLEGAL WILDLIFE TRADE

Wildlife forensics, Animal systematics, Conservation biology, Behavior, Evolution

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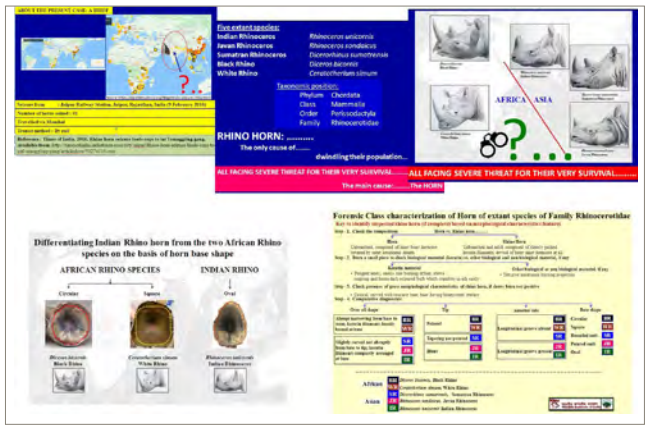
Illegal wildlife trafficking of five rhinoceros species for their parts and products is continuously declining their populations throughout its distributional ranges in the world. The Rhino horn forms a major part in illegal trade of this species. For the proper implementation of National and International wildlife laws the species identification is a prime requisite in wildlife forensics and for which development of standard operating protocols (SOP) is must. In the present study we describe the identification of a seized horn in illegal wildlife trade using morphometry and DNA-based analysis.



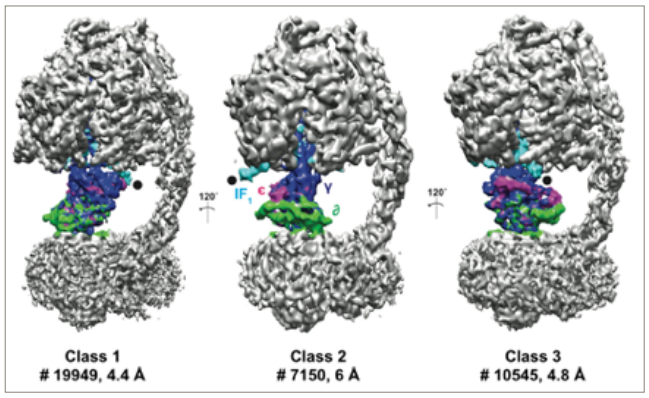
Forensic Class Characterization from Seized Rhino Horn: A tool for comnating transboundary illegal wildlife trade

A set of morphological parameters of rhino horn namely outer profile, shape of tip and basal shape characterized all five extant species of rhino. The abrupt narrowing outer profile from base to tip, loosely bound keratin filament at the base, pointed tip and circular base of the seized horn proved that it was of African Black Rhino. DNA analysis was carried out using cyt b (274 bp) and 12S rRNA (326 bp) genes. A BLAST search and comparison with the reference database at Wildlife Institute of India indicated that the seized horn was of African Black Rhino. This study documents the first ever record of presence of African Black Rhino horn in illegal wildlife trade in India and the SOP devised here using a combined approach of morphological characteristics and DNA-based analysis can be used for forensic class characterization of horn of extant species of *Rhinocerotida*e family throughout the world.

MEMBRANE PROTEIN STRUCTURE AND DYNAMICS

Membrane Proteins, Electron Cryomicroscopy, X-Ray Crystallography

Our research focuses on how things move across the membrane and how membrane proteins function. The hydrophobic nature of membrane proteins presents several unique challenges in structure determination including their low abundance or difficulty in over-expression of functional material and stability in detergents. Some of these issues can be mitigated with use of single particle cryo electron microscopy (cryoEM) as there is no need to grow crystals and very little protein is required. Using cryoEM and when necessary X-ray crystallography, we aim to determine the structure and elucidate the function of a range of membrane proteins including enzymes and channels. In the near future, we would like to expand into imaging some of these proteins in their native cellular environment by electron tomography.
A second line of our research interest focuses on improving the images that can be obtained with electron microscopes thus realizing the full potential of cryoEM.



In Single particle cryoEM, the molecules in solution are rapidly frozen and different functional states of macromolecules that might exist can be trapped and computationally classified to obtain many different structures and thus the dynamics of macromolecules can be studied by cryoEM. Shown below is an example of the mitochondrial F1FO-ATP synthase from Pichia angusta in three different states. ATP synthase's are dynamic enzymes that use the proton gradient to synthesise ATP and can exist in multiple different states. Using a protein inhibitor found in mitochondria, the enzyme has been trapped in different states and computationally these states can be separated from a mixture of population. The central stalk that consists of three proteins is colored in different colors (blue, magenta and green) and the inhibitor protein in cyan. The three states are related roughly by 120° rotation. Previously, we had obtained maps for state 1 to 7 Å with 42,771 but with the new detector, a 4.4 Å map can be obtained with ~20,000 particles for the same state. This example is used to emphasize how higher resolution maps with lesser particles can be obtained with better detectors.

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Ashok Rao, *NCBS*

Ramanathan, *NCBS*

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