CELL BIOLOGY NEWSLETTER

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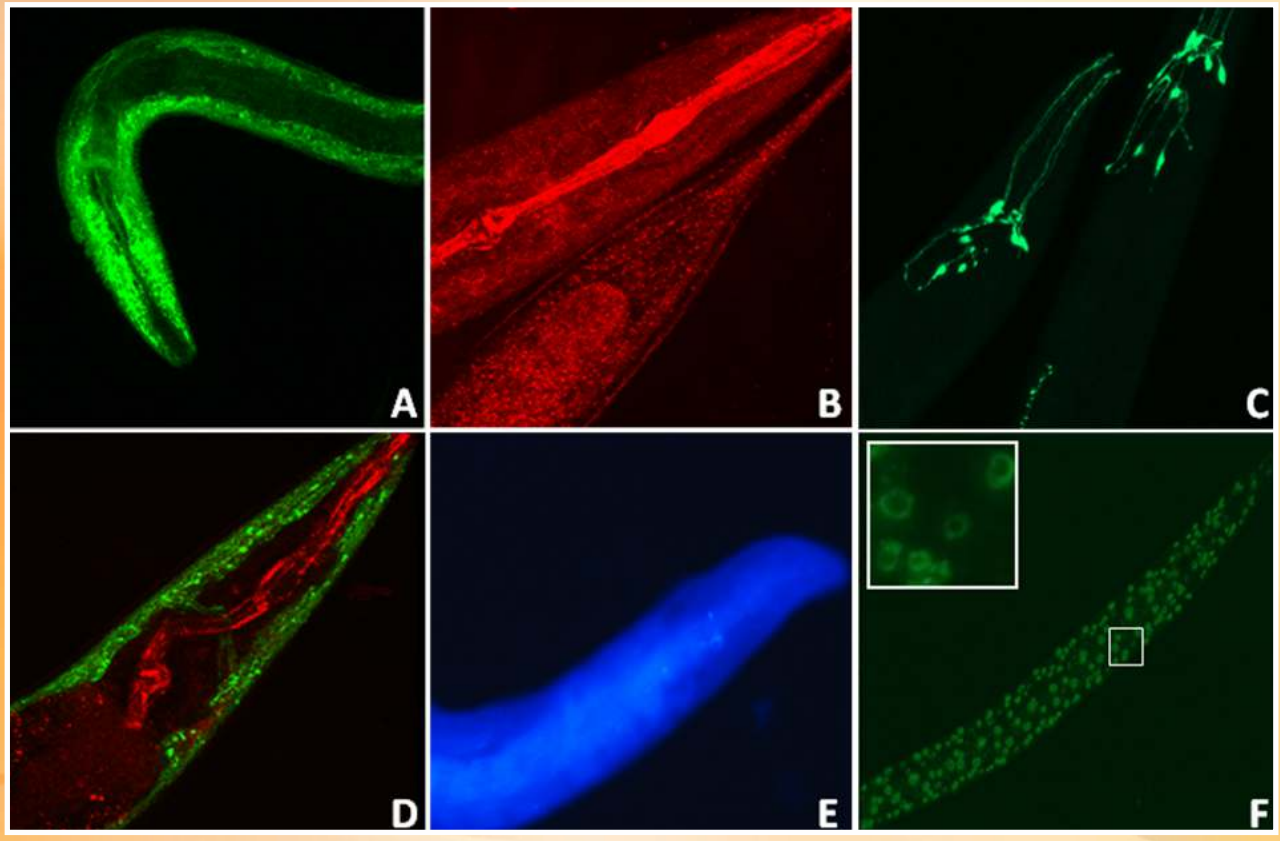

Dedicated to Late Prof S RV Rao

INDIAN SOCIETY OF CELL BIOLOGY

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Cover Page: Transgenic *C. elegans* expressing various reporter genes/fluorophores: *C. elegans* expressing "human" alpha synuclein tagged with YFP (A), *C. elegans* stained with lipid specific dye Nile Red (B), Transgenic *C. elegans* strain expressing GFP in subset of neurons (C), *C. elegans* expressing YFP stained with Mirotracker Red (D), *C. elegans* stained with Thioflavin S (E), Transgenic *C. elegans* expressing FOXO Transcription factor Daf-16 tagged with GFP, Specific Cytoplasmic presence of the protein is highlighted in the box (F). For detail see Article by Dr Aamir Nazir (page 37-38).

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(Extended till March 2023)

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Executive Secretary:	Dr. Madhu Tapadia (BHU, Varanasi)

Presidential Remarks

Dr. Jyotsna Dhawan, Hyderabad

Dear fellow cell enthusiasts:

What a strange few years we have lived through. A pandemic has effects great and small. Among the more trivial of these was the extension of the term of the ISCB executive committee which was to end in 2021: the newly elected office-bearers will now take over later this month. At the global level, the scale of human suffering caused by 30 kb of viral RNA, some intermediate hosts (perhaps bats) that unwittingly promoted zoonotic transmission, and a series of undetermined once-in-lifetime events caused by the cumulative effects of human intervention on the planet, is as yet unmeasured and may take a generation to absorb and assess. Closer to home, the Covid-19 pandemic waxes and wanes and leaves us wondering where our scientific endeavours will take us. A historical view of pandemics reveals their impact not only on scientific knowledge and sociological structures, but in a philosophical understanding of what it means to be human.

Viruses are tiny, almost insignificant in their coding capacity and yet, they can incapacitate organisms whose genomic and physical size dwarfs the invader. Students of cell biology will be familiar with the notion that in their ability to subvert cellular processes, viruses are windows into the working of the cell. Starting with the foundational concepts of molecular biology uncovered by studying phage invasions of bacteria, the revelations on DNA replication and transcription given to us by DNA tumor viruses, new biology by RNA tumor viruses that provided much of the framework of our current understanding of the cell cycle, growth factors and signaling, and the cytoplasmic trickery of many viruses that taught us about translational control and trafficking, using viruses to inform us about our cells has been a recurring theme.

And so it is with SARS-Cov2. Cell surface molecules such as Ace2 and the unmusically named Tmprss2 after a long lag of sporadic publications, have achieved notoriety in that the total publications on these two molecules number ~10,000 in 2.5 years! All of us have been amazed by the genome sequencing output of thousands of viral genomes per month, structural biology tracking every mutation in the spike RBD at atomic resolution with warp speed as they appear, revealing the dance of those residues which bind the receptor and permit infection. The understanding of distinctions between epithelial cells lining the respiratory tract has intensified, and the involvement of organelles from the motile cilia and to the Golgi ribbon in viral internalization and life cycle is sure to give us a better understanding of the normal functioning of these subcellular assemblies. Inflammation and the immune response too have been intensely studied revealing new information on pyroptosis and the cytokine storm. Integrative studies and modelling on levels from molecular to host level have been needed to create frameworks that inform public health decisions.

Two things emerged from my reflection on the past year. The first is something that has been emphasized by many scientists and physicians: let us not neglect the diseases that are endemic and continue to affect far more people than oscillating pandemics like Covid19. The second is the opportunity that has given us when disasters like pandemics disrupt our normal functioning. It has been the experience of many that such disasters make one takes stock of one's assumptions more readily than during "times of peace" when the power of the status quo numbs us.

If we reflect on what Covid19 taught us about what it means to be human, surely we must raise our eyes from the deep absorption we have had in understanding pleasing mechanisms, symmetric molecules, stunning organelles, beautiful cells, integration of previously unconnected concepts: all the intellectual satisfaction that comes from the privilege of being scientists, and acknowledge that much needs to be done about our human scale interactions with

other humans. Whether it is ensuring that entry into higher education is more equitable, that we eschew prejudicial stereotypes and encourage diversity, that young people are supported and properly acknowledged for their contributions, that institutional culture is open, we can all do more to ensure that our scientific culture is unbiased, and enables critical thought and inquiry.

Science gives us the understanding and the tools to work towards mitigating human suffering, philosophy emphasizes that human frailty encompasses a far wider domain. While celebrating the manner in which people got together to understand the virus and stem its impact, the lessons for us in dealing with the human problems that face us, with or without a virus in pursuit, are worth considering.

I look forward to meeting you all at 45th AICBC in Banaras.

Jyotsna Dhawan
Hyderabad
Jan 2023

Secretary Remarks

Dr. Bhupendra N Singh, CDRI, Lucknow

Dear Colleagues and Students,

We are delighted to bring out the first issue of the society Newsletter in 2023. After two consecutive years (2020-21) of viral pandemic we could organize a mini All India Cell Biology Symposium in University of Kashmir, Srinagar in 2022. And a full three days All India Cell Biology Conference is being organized at Banaras Hindu University, Varanasi in January 20-22, 2023. The present newsletter has compilation of both All India Cell Biology Conferences, in 2019 at IISER, Mohali and in 2022 at University of Kashmir, Srinagar. We have included the conference reports of both meetings and the details of society awards lectures delivered by eminent scientists. Many awards were given to the young students for their oral and platform presentations whose details are being provided. This year we have lost one of the most revered cell biologists of the country, Prof. S R V Rao, who was like father and guru for all of us. His contributions to the society are immense and, therefore, we have dedicated this newsletter to him and included his obituary written by one of his beloved student Prof. B K Thelma, Department of Genetics, South campus, Delhi University. She has also shared some of her memorable moments with him captured in photographs. In addition to that we received encouraging response from our students and the senior members of the society as they submitted good number of write ups. The issue covers articles on topics like Cancer biology, Autophagy, *Drosophila* Malpighian tubules, human cells exposure to metal oxide nanoparticles, C. elegans, tiny worms for studying human diseases and E3 ligases and host-pathogen interaction.

Outreach Program: A hands-on workshop on “Use of Quantitative PCR in Biomedical Research and Diagnostics” was organized by Dr. Anirudh Singh, AIIMS, Bhopal. We have put efforts towards increasing society membership and have inducted students as well as life members to the society. We are eager to meet you all at the forthcoming annual meeting at Banaras Hindu University, Varanasi, January 20-22, 2023.

Wish you all A VERY HAPPY NEW YEAR 2023.

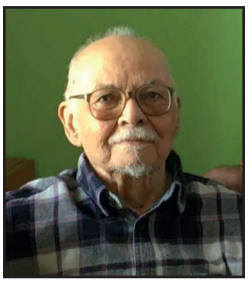
Dr. Bhupendra N Singh
Secretary, ISCB

Prof S R V Rao OBITUARY

Prof SRV Rao and Mrs Rao in their house in NOIDA (1993)

Prof SRV Rao at AICBC meeting in Ahmedabad (1982)

An Ode to a Life Guru Prof. S R V Rao



(1927-2022)

I sat down on a quiet Sunday morning in silos and began to pen my enriching, enviable and invaluable memories, being with, learning from and trying to emulate the qualities of my academic mentor and more importantly a Guru for life, Professor S R V Rao. The lessons are practical- the 'Practice what you Preach' principle of our dear teacher, fondly referred to as SRV, made everlasting impression on me and undoubtedly also on all my dear lab mates of yore. Through this memoir, I hope to convey the essence of Professor Rao's simple yet most beautiful life enabled by his persona; his yester years' observational/paradigm driven science; his notable contributions thereof; and his unpenned teachings relevant for scientists and laypersons alike. Outside of the scientific world he was an ardent lover of classical music, a cricket fan, an extrovert who inspired and inculcated lifelong lessons in everyone around him.

SRV's scientific achievements: Prof. Rao, a renowned and respected cytogeneticist was born to Mrs Rukmini Devi and Dr. Salem Ramachandra Rao on December 27, 1927 in Nagamangala, a small town in Mandya district, Karnataka and was an unusual eight months pregnancy survivor. He completed his early education there and moved on to obtain his Master's degree at Central college, Bangalore under the tutelage of teachers like Professor B R Seshachar, a doyen of cell biology. At his insistence, Prof. Rao went to BITS Pilani for his Ph. D and worked on Cytology of Homoptera with Prof. M. A. Moghe. After a brief stint at Cancer Research Institute, Chennai as a Post-doctoral fellow with Dr Shanta Rao, SRV joined the Department of Zoology, University of Delhi as a lecturer which was his academic abode until he superannuated in 1992. He then served as an INSA senior scientist in the Department of Genetics, Delhi University South Campus until 1995 (and I admit I felt very blessed).

The science of SRV's time was different, funding extents were different but intensity and passion reigned supreme. SRV quietly established himself and to date probably remains one of the very few who excelled in both mammalian and insect cytogenetics. His doctoral training helped him in the former, as well as selection and participation in an early international workshop on human lymphocyte culture and air-dried chromosome preparations at AIIMS, New Delhi with leaders such as Prof. Harold Klinger, Albert Einstein school of Medicine, New York, USA; followed by a brief training in chromosome banding techniques at W. Alton Jones cell science centre, Lake Placid, New York, USA in 1974 set the ball rolling for SRV and cytogenetics was the main stay of his scientific pursuit through his career. He prepared beautiful air-dried metaphase chromosomes from bone marrow for a variety of small mammals (and trained students in this art) which remain classical papers of the 1970s and 80s. Demonstration of Robertsonian rearrangements in the speciation of *Funanbulus* (squirrel) species (Rao et al., 1972); extensive X and Y chromosome polymorphism in *Nesokia indica,* the Indian mole rat (Rao et al., 1983) are just a few of SRV's landmark papers.

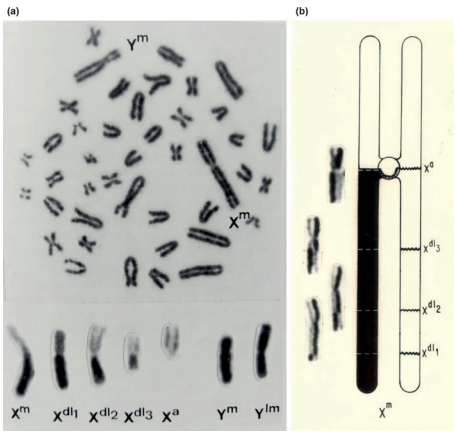


Figure 1. a) Metaphase chromosomes of *Nesokia indica* with polymorphic X and Y chromosomes due to deletions (marked dl 1, 2, 3 and a-acrocentric) shown in the lower panel; X^m is the full length metacentric X chromosome; **b)** Diagrammatic representation of the X chromosome with deletion sites marked; X-chromosome cut outs (left) showing sister chromatid exchanges (captured with 5-BrdU labelling) and folate sensitive fragile sites (right) corresponding to the deletion sites.

With late Prof. Vinod C Shah, a long time friend, colleague and collaborator, SRV established tritium labelling of mammalian metaphase chromosomes and autoradiography for the first time in the country. Using this powerful technique, he documented the rare spontaneous reactivation/reversal of the mammalian inactive X chromosome observed in a very small proportion of bone marrow cells of *Nesokia* (Rao and Jhanwar, 1975). His continued work on *Nesokia*, showed that polymorphic X and Y chromosomes originated due to deletions at specific chromosomal regions in the constitutive heterochromatin arm of the X and distal regions of the Y chromosome respectively; and went on to lending evidence to the inherent fragility of these regions by Bromo-deoxy uridine labelling of the metaphase chromosomes and detection of sister chromatid exchanges at the exact same chromosomal sites (Thelma and Rao, 1982); and subsequently showing that folate sensitive fragile sites (now associated with fragile X syndrome in humans) correspond to the same sites on the X-chromosome (Tewari et al., 1987) (Fig 1); and importantly, demonstrated the influence of heterochromatin on spermatogenesis using fertile and infertile males with X and Y chromosome polymorphism (Juyal et al., 1989).

In the area of insect cytogenetics that SRV pursued, the most exciting and revolutionary finding was

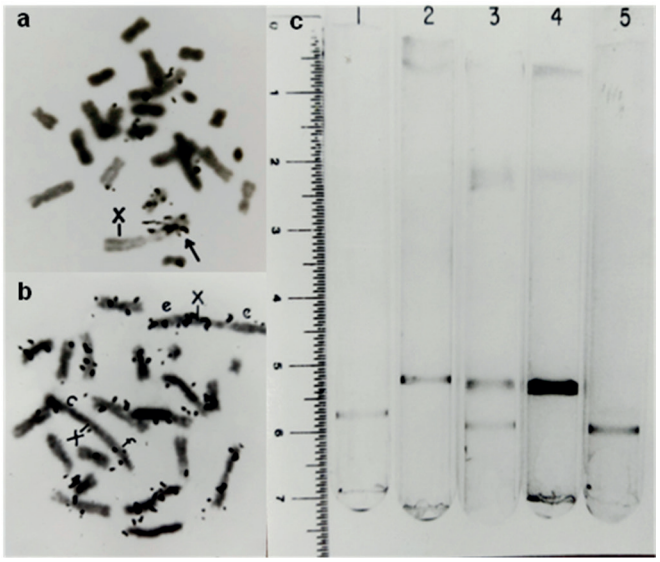


Figure 2. X-chromosome inactivation in somatic cells of female *Gryllotalpa fossor.* Tritiated thymidine labelled metaphase chromosomes showing late replication of the inactive X chromosome in female - with unlabeled facultative arm of the X-chromosome (bottom X) in female (b); and unlabeled constitutive heterochromatic arms in both male (a) and female (b). Note labelling in the euchromatic arm of the single X-chromosome in male (a). (c) Biochemical evidence for random X-chromosome inactivation in female somatic cells - Cylindrical PAGE profile of Glucose 6-phosphate dehydrogenase in hemizygous male [either fast (lane 1) or slow (lane 2) forms]; heterozygous female (with both slow and fast forms, lane 3); homozygous female (slow, lane 4); and homozygous female (fast, lane 5).

the first ever evidence of X chromosome inactivation in a non-mammalian system, *Gryllotalpa fossor,* the Indian mole cricket, belonging to Family Orthpptera (Arora and Rao, 1979). To address the scientific skepticism of this unusual yet authentic observation of X-chromosome inactivation- particularly in an insect system with XX/XO sex determination, SRV designed and performed beautiful biochemical experiments using azacytidine, a demethylating agent to reverse the inactivation and capture the dose difference in G6PD, an X-linked gene and proved developmental reactivation of the inactive X chromosome thereby providing clinching evidence for his novel observation that X-chromosome inactivation occurs beyond the realm of mammals (Rao and Padmaja, 1992) (Fig 2).

These remain some of SRV's prized original contributions in cytogenetics. It was only natural that these fascinating findings attracted a lot of international scientists including Prof. Susumu Ohno, Jacob Wahrman, Stanley Gartler etc to SRV's lab which also gave an exposure and wonderful opportunity for the students to have excellent discussions. These might mean little in the face of contemporary genomics or molecular biology research now but undoubtedly represent the quality science and remain classic contributions achieved with small grants through big passion and unwavering commitment in the chosen path of scientific pursuit. These scientific contributions only added to SRV's humility. Besides several awards that he received during his career, the Hari Om trust, J C Bose, visiting senior scientist at Medical Research Council, UK to name a few, he was duly recognised for his contributions by the fellowship of the Indian National Science Academy (1983) and National Academy of Sciences India (1990). He was a founding member of the Indian Society of Cell Biology (ISCB) and had also served as the President of the Society.

SRV's persona: Through his life and career with family, students and colleagues he remained a simple, lovable, happy and cheerful person with no hatred or anger, greed or malice, and though practical and pragmatic, was governed by his heart. His warm demeanour and the way he viewed this world with happiness and positivity, instilled a lot of confidence and independence – he allowed us to grow. His optimism with the right mix of chalk and real life scientific challenges, his charm to transform us and teach us to settle for nothing but the best, to reach the highest potential at every step will always be cherished. He repeatedly told us how strict his mentor was and made him rewrite manuscripts several times (throwing several versions in the dustbin). Self-sufficiency was another important lesson we learnt- he insisted that we make our own distilled water for experiments, develop films, print the

photographs, and even type our research manuscripts (pre computer era). If we missed turning off autoclaves, oven or lights, we were levied a fine that went to the kitty for birthday celebrations of lab members.

SRV's outreach: His home was a modern Gurukula and his lab at DU was an open house- anytime was tea time for students, friends and colleagues with long drawn discussions on the latest (!!) paper – two-three months after its publication – marked first on the current contents, request for reprints sent out and reprints received through air or surface mail by the responsive authors. With only four or five labs pan India working on cytogenetics in those days and all mostly friends with each other - a faculty or student visiting Delhi was an assured recipient of SRV's hospitality. With no dhabas, canteens or good eating places around, food for the guests would always come from SRV's home painstakingly prepared and packed by dear aunty Sharada Rao, herself an endearing, charming and adorable human being. Several of the guests staying over at their home on these visits were a rather common feature. Though everyone was very dear to him, this note would be incomplete without the mention of some of the regular visitors like late Prof. Tikaram Sharma, BHU, Ardhendu Mukherjee, Calcutta University, Vinod Shah, Gujarat University and Abbas Musavi of AMU; Prof Sharat Chandra, then of IISc, Bangalore among his contemporaries; & Professors Subash Lakhotia, Lalji Singh, Rajiva Raman and Mercy Jacob (all BHU) and HA Ranganath (Mysore Univ) of gen next. SRV just had the warmth and great charm to make everyone feel at home no matter the circumstances, including his childrens' ongoing exams. Among his over 20 doctoral students too, specially the middle lot and later ones, they were a few of us – Suresh Jhanwar, Ramesh Juyal, Sher Ali, Padmaja, Rita and others who were very lucky to have been associated very closely and also had an opportunity to serve/help him in different capacities. Through his life and science, he actually taught us that 'What you do is not important but how you do is'. If doctoral students complained or grumbled at work he always said 'Don't tell the world about the labour pains, show them the baby'. He was gifted with good eyes and deft hands and wanted everyone of his students to develop that. To reiterate the importance of observation at work and since the work was largely microscope centered, he always said 'Fortune favours the prepared mind' and his own novel findings bear testimony.

Lighter moments: This narrative would be incomplete without some anecdotes that we recall being his students, which always makes us smile. When SRV joined Delhi University, he was given a room and a new almirah and when the carpenter asked him "Where should I place it Saab?" SRV replied "GHODE ke pass rakho beta". In Hindi, GHODA means horse and so the carpenter was very perplexed and said "Saab, GHODA kahaahai?" and asked him to show the place quickly as it was heavy and a colleague had to come to his rescue. GHODE means wall in Kannada!!!! SRV and his colleague late professor MRN Prasad had a project on cytology of the Indian elephant for the first time very early on. SRV being the younger one, was asked to climb the ladder to get the glass slide scrape of buccal cells while the mahout helped open the mouth of the elephant. SRV told us that it was the first time, and, in his anxiety, the first slide went into the elephant's mouth and he mustered the courage and ultimately got the buccal smear. He also told us that the whole night he prayed for the elephant which fortunately survived the slide in the stomach. The manuscript describing the findings was published in Nature (Venkatasubba Rao and M. R. N. Prasad. 1963. The nuclear sex in the Indian elephant, Elephas maximus L. Naturwissenschaften, 50(7):313). About his sarcasm (of course to make us perform better), culturing *Nesokia* lymphocytes to make chromosome preparations was tough then and finally when Rita found one metaphase plate in her preparation and all of us were super excited, he told her to take a picture and hang it as a pendent.

SRV's later life: Both SRV and aunty Rao were the most practical people that I have ever come across in my life. They moved to Bangalore after completion of his academic commitments to be closer to the extended family. The day they realised that they cannot manage their household, and having lived their lives as well as they could, they with peace and contentment, moved to a home for the elderly in Bangalore. With no major health issues they both lived happy and content. They enjoyed their stay there with no complaints on food, co-habitants, or anything else and in fact always encouraged me to book a place in time if I chose to live that way. Reading, music and cricket- his life companions stood him in good stead. He continued to read avidly and carefully put aside newspaper cuttings which he felt was useful to us - his own kids and lab kids (till his end). Aunty Rao passed away very peacefully one morning in July 2013. SRV took it in his stride and just said everyone has to go one day and I only hope I will also have as good and easy an end as aunty. God obviously heard his prayers and just gave him the leeway for his children to reach from USA to wish him the final goodbye on October 25, 2022. For the goodness they had, the love and respect they earned from just about everyone they came across in their lives, they are endowed with two daughters and a son, well-educated and settled in USA. They took turns to visit and be with parents three to four times in a year, take them out for a few days and give them a good time and for SRV to have his food and drink of choice! Parents need to be blessed to have children

who care and children need to be blessed to have parents whom they can emulate as role models. What more could parents have asked – HE in his unseen ways ensured that the children were around when both of them moved on their eternal journey. May our dear SRV's soul rest in eternal peace knowing that his life lessons are imprinted in his students' hearts with love and everlasting gratitude; and be a guiding light; and may there be many more SRVs.

Finally, as for myself, I think I got the best and enviable of everything - was a part of the family and stayed in their University accommodation (when the hostel seat was not provided to post docs), had the good fortune of longest academic interactions- thanks to my getting a job at DU, inherited a lot of books and quaint lab paraphernalia, and during their stay in Bangalore, got to meet them so often, and in fact at least three to four times in a year. When I visited him on October 07, 2022 and spent over three hours talking to him about a lot of different things, he was as cheerful as always but just said not once but twice- that as one gets old one should not live too long and should just go. Slight hearing impairment which had set in and inability to move around freely (especially post- pandemic) may have prompted

Prof S R V Rao (with author) Oct 07, 2022

him to say this. When I took his blessings and left the room he called out to say 'All the best Putta' (child- the way he called me) and called out a second time and said 'God bless you'. That image and voice are etched in my mind...Did I know that would be the last time!

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XLIII All India Cell Biology Conference, December 19-21, 2019: A brief report

INDIAN INSTITUTE OF SCIENCE EDUCATION AND RESEARCH (IISER) MOHALI

The XLIII All India Cell Biology Conference (annual meeting of the Indian Society of Cell Biology, ISCB) was held at IISER Mohali from 19-21 December 2019. Apart from the series of stellar talks by eminent scientists across India, embedded within the conference were two panel discussions that highlighted several issues related to science communication and ethics in research.

This meeting brought together around 250 registered participants that included research scientists, post-doctoral fellows, PhD students and a large number of undergraduate students from different parts of India engaged in investigating the basic principles of cell biology and the mechanisms underpinning them.

The conference started with the presidential address by Dr. Jyotsna Dhawan, President, ISCB. She talked about how cilia in quiescent muscle stem cells limit signaling and specify the type of cell-cycle arrest. Prof. Sumantra Chatterjee, an eminent neurobiologist from NCBS, Bangalore, delivered the Inaugural Lecture. In his spectacular presentation, Prof. Chatterjee described the importance of astrocytes in determining the electrophysiological state of the neuron or neuronal function in the context of autism.

The following three days were a celebration of science with a bouquet of fabulous scientific sessions. There were 20 invited talks delivered by leading workers of their respective fields. For the invited talks, emphasis was given to showcase the research work of the relatively younger investigators from both universities and research institutes. Every effort was made to have representations from different corners of our country. Importantly, the organizers successfully achieved to maintain a gender balance in the list of invited speakers with nine out of twenty speakers being women. Apart from these invited talks, we had short presentations by young colleagues, who are in their initial years of their independent careers. Five such short talks were selected from a list of twenty by a national committee. All the speakers delivered fantastic presentations on different disciplines that represented a full spectrum of cell biology research from organismal level to cell lines and from plant cell biology to stem cell biology.

Day 1: The first scientific session was chaired by Prof. S C Lakhotia. This session had four invited talks and one short talk. Dr. Deepa Subramanyam from NCCS, Pune discussed her work on the involvement of endocytosis in molecular control embryonic stem cell pluripotency. Work from her lab have underpinned how clathrin mediated endocytosis (CME) regulates levels of e-cadherin and TGFβR1 in mESCs. Dr. Ram Kishore Yadav from IISER Mohali described the details of *wuschel* mediated auxin biosynthesis in *Arabidopsis* shoot apical meristem. The talk revolved around stem cell differentiation that occurs in the periphery of the shoot apical meristem and how stem cells protect themselves from differentiation pathways. The next talk, delivered by Dr. Ravi Manjithaya from JNCASR Bangalore, dwelled upon the different mechanisms of modulation of the autophagic flux by small molecule autophagy modulators and identification of novel regulators of autophagosomes. Dr. Siddhartha S Jana from IACS, Kolkata illustrated his findings perinuclear non – muscle myosin II in tumorigenesis. Dr. Jana concluded the talk by providing insights into the perinuclear NMM II role in tumor progression. Dr. Puja Singh from CSIR-CCMB Hyderabad delivered the short talk of this session. Her work unearthed the effect of altered microtubule polyglutamylation on centriole assembly/function and provided evidence for hyper-glutamylation of centriolar microtubule associated centriole duplication.

The second scientific session of Day 1 was chaired by Dr. Surendra Ghaskadbi. This session had four invited talks and two short talks. Dr. Sandhya P Koushika from TIFR Mumbai talked about the regeneration response in the axons of C. elegans. Her work demonstrated that UNC-16/JIP-3 plays DLK1-mediated inhibitory role in neuronal regeneration, and the slower regeneration rate is preferable for functional restoration of neurons. Dr. Biman B Mandal from IIT Guwahati delivered his talk on the advancement of bioengineered human tissues and organs for tissue/organ graft. He emphasized on the uses and benefits of naturally occurring scaffolds for generating human tissues/organs for future implications in biomedical human transplantation. The next talk, delivered by Dr. Indumathi Mariappan from LVPEI, Hyderabad dealt with the role of Retinal Degeneration 3 (RD3) gene in early onset of retinal dystrophy. Dr. Pradhyumna K Singh from CSIR-NBRI, Lucknow talked about a non-GM approach to protect edible crops from whitefly and also on the development of transgenic cotton in his laboratory that would serve as an attractant/ toxic trap for white flies. The two short talks of the session were delivered by Dr. Lakshmi R Perumalsamy

from SRIHER, Chennai and Dr. Shubra Majumder from Presidency University, Kolkata. While Dr. Perumalsamy spoke about the novel role of RASSF7 in notch signaling in breast cancer, Dr. Majumder talked about how Voltage Dependent Anion Channel proteins (VDACs) regulate centriole and cilia assembly in mammalian cells.

The Executive Committee Meeting of the Indian Society of Cell Biology was held in the evening.

Day 2: The second day of the conference started with the third scientific session chaired by Dr. Jyotsna Dhawan from CSIR-CCMB, Hyderabad. In this session there were three invited talks. Dr. Puran Singh Sijwali from CSIR-CCMB Hyderabad talked about WIPI2, the Atg18 autophagy marker homolog of *Plasmodium* and demonstrated that the autophagy of malaria parasite adopts a non-canonical pathway. Dr. Raj Ladher from NCBS, Bangalore talked about organization of hair bundles in a step-wise fashion in the inner ear hair cells and the signaling patterns that instruct this organization of pattern formation. Dr. Chandrima Das from SINP, Kolkata delivered the last talk of this session. She described the role of UBR7 as an EMT suppressor and regulator of the Wnt signaling and how it can be used as a potential therapeutic target of triple negative breast cancer.

The next session in the morning was a panel discussion on Science Communication. Dr. Jyotsna Dhawan from CSIR-CCMB Hyderabad served as the moderator. The eminent panelists were Dr. Archita Bhatta from Department of Science and Technology, Government of India, Mr. T V Jayan from The Hindu Business Line, and Mr. Ratnesh Thakur from NIPGR, New Delhi. The panelist emphasized the effective communication of science, especially in the regional language, to sensitize people of all backgrounds towards science. It was also felt that though removing scientific jargons can make the process easier but it also has the possibility of scientific miscommunication.

This was followed by one of the most exciting events of the conference - the platform presentations by the research students. A total of forty-eight students applied for platform presentations. From them twelve students were selected by a national selection committee based on the evaluation of their submitted abstracts. Six students presented their work in this session. All the students presented their work with lots of passion and excitement and did extremely well in defending their works. Each and all enjoyed these six talks. A panel of five judges evaluated their talks.

The fourth scientific session was chaired by Prof. Anand Bachhawat from IISER Mohali. There were two invited talks and one short talk. Dr. Jonaki Sen from IIT Kanpur talked about the role of microRNA–19b controls patterning by restricting the expression of Wnt 7b to the cortical hem which gives rise to the hippocampus in the avian brain. Her work also demonstrated that miR-19b inhibits the function of NeuroD1 and hence inhibits their differentiation while promoting progenitor cell proliferation. Dr. Anuradha Ratnaparkhi from ARI, Pune talked about the identification of Heartless (Htl) as a negative regulator of Fog signaling in Drosophila. She also explained how Smog regulates this interaction among these two distinct signaling via post-transcriptional mechanisms. Dr. Sandip Kar from IIT Bombay delivered the short talk of this session. He elucidated dynamic nature of cell-cycle variability in a serum dependent manner. In combination with mathematical modeling and quantitative live cell imaging, he showed that low serum concentration leads to higher G1 population whereas higher serum dosage increases the variance in S/G2/M duration.

The last lecture of the day was the 10th Professor Jyotirmoy Das Memorial Lecture. Prof. Subramaniam Ganesh, Department of Bioengineering, IIT Kanpur delivered the prestigious lecture. Prof. Ganesh described the timeline of his research on the rare neurodegenerative disease known as Lafora disease (LD) and highlighted his recent work that unraveled the role of the genes *malin* and *laforin*, identified by his group, in LD. He explained how alteration of these cellular pathways leads to the manifestation of specific LD associated phenotypes. The award, consisting of a citation and a plaque, was handed over to him by Dr. Bhupendra N Singh, General Secretary, ISCB.

In the evening, the general body meeting was held. All the members attending the conference were involved in the discussion to provide feedback and suggestions for the benefit of the society as well for any upcoming events that can be organized by the society. Prof. S. C. Lakhotia mentioned that the conference is acting as an emerging forum to acquire learning experience and that it is not only restricted to learning but extends to scientific networking. He expressed his happiness on seeing younger people in science joining the conference. Dr. Jyotsna Dhawan added to Prof. Lakhotia's comment and said that the culture of the present conference presents a form of evolved tradition. She also added that content developing for outreach is to be looked at, and emphasized the need for more contribution to the ISCB newsletter. Inclusion of issues of mental health of graduate students as one of the panel discussions in the upcoming meetings was also suggested. Dr. S. Ghaskabdi encouraged the younger participants to apply for lifetime membership. Prof. S.C. Lakhotia stressed on the part that fee of participation should not be increased in the coming future. Several participants expressed that the organizing committee has done an

excellent job by using double-blind methods for platform presentation selections. Closing remarks were given with the announcement that next AICBC will be held at ACRTEC Mumbai.

Day 3: The third and the final day of the conference started with the fifth scientific session chaired by Prof. Pradeep Burma from DU South Campus, New Delhi. This session had three invited talks and one short talk. Dr. Kalika Prasad from IISER Thiruvananthapuram talked about how the gradient of transcription factor Plethora2 defines the competence zone till which the root can withstand an abuse. He also elucidated how Plt2 maintains its endogenous levels by suppressing of activating its expression when either in high or low dosage respectively. In his talk, Dr. Rahul Roy from IISc, Bangalore dealt with the different facets of flavi-virus life cycle and demonstrated the involvement of replication compartmentalization as a main event during flavi-viral infection. Dr. Rasna Bhandari from CDFD, Hyderabad presented her work on the inositol pyrophosphate mediated pyro-phosphorylation of MYC protein and its role in cell survival. Pyro-phosphorylation of MYC maintains its cellular levels by poly-ubiquitination and degradation. Dr. Amaresh C Panda from ILS, Bhubaneswar delivered the short talk of this session. He explained how circular RNA–rolling circle amplification can identify circRNA splice variants.

This session was followed by platform presentation by six more research students. Similar to that observed for day 2, all the speakers delivered fantastic presentations with lots of passion and excitement. Importantly all of them finished their presentations within time and defended their work very well. The same panel of five judges who evaluated the previous round on day 2 evaluated their presentations.

The panel discussion of the day was on Scientific Integrity and Ethics of Publication. Dr. Rashna Bhandari from CDFD, Hyderabad moderated the second panel discussion. The eminent panelists were Dr. Jyotsna Dhawan from CSIR-CCMB, Hyderabad, Dr. Raj Ladher from NCBS, Bangalore, and Dr. Ravi Manjithaya from JNCASR, Bangalore. They discussed the process and nuances of educating mentees on processing data correctly, the decision on authorship, and how to keep away from predatory journals and different aspects of plagiarism. The panel discussion experienced active participation of the audience and was highly appreciated by the students.

Dr. Bhupendra N Singh from CSIR-CDRI, Lucknow, chaired the sixth and the final scientific session of the conference. In this session there were two invited talks. Dr. Sanjeev Shukla from IISER Bhopal talked about the crosstalk of signaling pathways with cellular processes in mediating breast cancer. His work demonstrated how hypoxia mediated TGF-β signaling leads to alternative splicing of genes, which are involved in global EMT in cancer cells. Dr. Surajit Sarkar from DU South Campus, New Delhi illustrated genetic means to alleviate poly(Q) mediated neurotoxicity in *Drososphila*. He concluded that the combinatorial down regulation of CREB binding protein (CBP) and MYC as effective measure to counter poly(Q) disorders.

A significant portion of the conference time, consisting of three hours on the first as well as on the second day, were kept aside for the poster presentation by research students. More than 180 posters were presented by the students, postdoctoral fellows and even by scientists. All the posters were displayed for all the three days of the conference for maximum visibility and interactions. The poster sessions experienced a large footfall and were vibrant with scientific discussions, arguments and suggestions. A panel of twelve judges visited all the posters and evaluated them.

On the final day, prizes were given to the students for their platform and posters presentations. While Ms. Rutambhara Purohit (IIT Madras) and Mr. Aniketh Bishnu (ACRTEC, Navi Mumbai) received the awards for platform presentation, the awards for poster presentation were given to Dr. Akhouri Kishore Raghawan (CCMB, Hyderabad), Ms. Prerna Aggarwal (DU, South Campus), Mr. P. Vineet Daniel (IIT Mandi), Mr. Gautam Chandra Sarkar (NII Delhi), Ms. Shivangi Gupta and Ms. Prerna Budakoti, both from IISER Mohali.

In the closing ceremony, Prof. Subhash C. Lakhotia mentioned that this conference was successful in providing a platform that inspired and enriched the younger generation and provided them with ample opportunity for scientific networking. Prof. J K Roy and Dr. S. Ghaskadbi appreciated the organization and committee members, convener and the volunteers for organizing a successful conference loaded with lots of information and interaction. Participants also extended a vote of thanks and appreciated the quality of work and lectures delivered. The organizers, in their vote of thanks, expressed their sincere thanks and gratitude to all who were involved in organizing the Conference, and all the funding agencies for their generous support.

On 22nd and 23rd December 2019, two post-conference events were organized. The first one was a Career Crafting workshop co-organized with India Biosciences that gave a hands-on experience in writing CV/resume, and discussions on strategies for career development to 70 participants (Ph.D. students, postdocs, and early-career investigators). The second event was for high school students. Along with DBT Welcome Trust India Alliance, the Explorer series of

public scientific lectures was organized to inspire the young minds and ignite the excitement for science in them. This thrilling event was attended by 500 high school students and students from ten local colleges.

The most awaited announcement of results for the best poster and platform presentations were announced. The following students received the awards:

Best paper presentation in poster session:

1. Prof. S. R. V. Rao Award – Dr. Akhouri Kishore Raghawan (CCMB, Hyderabad)
2. Prof. B. R. Sheshachar Memorial Award – Mr. P. Vineet Daniel (IIT Mandi)
3. Dr. Manasi Ram Memorial Award – Ms. Prerna Aggarwal (DU, South Campus)
4. ISCB award – Mr. Gautam Chandra Sarkar (NII Delhi)
5. ISCB award – Ms. Shivangi Gupta (IISER Mohali)
6. ISCB award – Ms. Prerna Budakoti (IISER Mohali)

Best paper presentation in oral session:

1. Professor V. C. Sah Award – Mr. Aniketh Bishnu (ACRTEC, Navi Mumbai)
2. Professor A. S. Mukherjee Memorial Award – Ms. Rutambhara Purohit (IIT Madras)

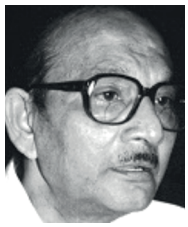
Each student received a certificate and a set of books from the following titles:

1. Sapiens: A Brief History of Humankind by Yuval Noah Harari
2. Who We Are and How We Got Here by David Reich
3. The Gene: An Intimate History by Siddhartha Mukherjee
4. Gene Machine: The Race to Decipher the Secrets of the Ribosome by Venki Ramakrishnan

Award lectures at the XLIII All India Cell Biology Conference, 20 December 2019

The Tenth Prof. J Das Memorial Lecture Award

Prof. Jyotirmoy Das



Prof. Jyotirmoy Das lecture is in the memory of late Prof. Jyotirmoy Das who made pioneering contributions in *Vibrio cholerae* research. He worked on elucidation of genetics of cholera phages, physical and genetic maps, repair processes and stress biology of *Vibrio cholera*. He did his B.Sc. and M.Sc. in Physics from Calcutta University and Ph.D. in Biophysics from the Baylor College of Medicine. He returned to the country in 1978, joined Bose Institute and then moved to Indian Institute of Chemical Biology (IICB) as head of Biophysics Division in 1979. Later he became the Director of the Institute in 1995 and remained in this position till he passed in July, 1998. He was also a Visiting Professor at the University of Rochester, USA.

Prof. Das demonstrated the DNA repair mechanism in *Vibrio cholerae* and identified genes involved in these processes. He elucidated the intracellular replication of cholera phages and investigated the mechanism of biotype differentiation by these phages along with the analysis of the cell surface architecture of *Vibrio cholerae* and developed an oral vaccine strain for cholera. His group contributed significantly to the elucidation of *V. cholerae* genome structure, identification of genomic rearrangements and diversity, and went on to construct first a physical map of the *V cholerae* genome. Prof. Das had a desire to understand the physical basis of life, which prompted him to start the Theoretical Biology Group at IICB, Calcutta. Recognizing the needs of the time, he set up a division of human genome research at IICB.

In addition to being chosen as the UGC National Lecturer in 1985, Jyotirmoy Das also received the Ranbaxy Research Foundation Award in 1989 and the Om Prakash Bhasin Award in 1990; INSA Golden Jubilee Commemoration Medal (1998). He was a Fellow of the Indian Academy of Sciences, Bangalore and the National Academy of Sciences (India), Allahabad. He was a member of the editorial board of the *Indian Journal of Biochemistry and Biophysics*.

Since its inception there has been nine memorial lectures by eminent scientists during All India Cell Biology Conferences, viz., Profs. P. Balaram (2001), M.R.S. Rao (2003), P.P.Majumdar (2006), V. Nagaraja (2007), A Surolia (2009), M.Vijayan (2011), Kanury V S Rao (2013), J S Tyagi (2015) and Dr. Alok Bhattacharya (2018).

The Society feels privileged and proud to have Prof. Subramaniam Ganesh, Dean of Research & Development at the Indian Institute of Technology, Kanpur as the speaker of the Tenth Prof. J Das Memorial Lecture today at XLIII AICBC 2019, IISER, Mohali, Chandigarh.

Prof. Subramaniam Ganesh, IIT, Kanpur

Prof. Subramaniam Ganesh is well-known as a teacher, mentor and researcher who has made significant contributions to the field of Lafora disease biology. Dr. Ganesh completed his master's degree in zoology from the University of Madras and then joined the lab of Prof. Rajiva Raman at the Banaras Hindu University (BHU), Varanasi for his Ph. D. Degree. During his Ph.D. he deciphered the genetic basis of sex determination in reptiles while characterizing the functional homologs of the human Y chromosome-linked genes. Then he joined the group of Prof. Samir K. Brahmachari at the Indian Institute Science, Bangalore as a postdoctoral fellow and worked on the neuropsychiatric genes. Later he moved to the RIKEN Brain Science Institute, Japan, to join the group of Prof. Kazuhiro Yamakawa and discovered genes contributing to the familial forms of epilepsies.

Dr. Ganesh joined the Indian Institute of Technology, Kanpur in early 2002 and played a major role in establishing the academic and research programs of the Department of Biological Sciences and Bioengineering. He established a vibrant research group to work in the area of "Human Molecular Genetics". Through collaborations with leading neurologists in the country, Dr. Ganesh deciphered the genetic determinants of Lafora disease (LD), an autosomal recessive and fatal form of teenage onset epileptic disorder, in the Indian population. Using cellular and animal models of Lafora disease, his group demonstrated that the LD products play critical roles in protein quality control and redefined a very old concept that LD is a metabolic disorder. His group established the molecular basis for Lafora disease and developed avenues for therapeutic interventions. Besides LD, his group works to understand the molecular basis of

the neuronal stress response and to identify common pathways of neurodegenerative disorders. Dr. Ganesh is also a popular teacher. Thousands of students across the country have benefitted from his Massive Open Online Courses (MOOCs) on the National Programming on Technology Enhanced Learning (NPTEL) platform, and his MOOCs have also been included for the Faculty Development Programmes of the All India Council for Technical Education (AICTE).

Dr. S. Ganesh is an elected Fellow of the National Academy of Sciences, India and the Indian Academy of Sciences, Bangalore. His other honours include the "SCOPUS Outstanding Young Scientist Award" of the Elsevier South Asia, "National Bioscience Award" of the Department of Biotechnology, "Birla Science Prize" of the B. M. Birla Science Centre, "Outstanding Research Investigator Award" of the Department of Atomic Energy, "CDRI Award for Excellence in Drug Research" of the Central Drug Research Institute, Lucknow, "Rajib Goyal Prize in Life Sciences" of the Goyal Foundation, Kurukshetra University, "OPPI Scientist Award" of the Organisation of Pharmaceutical Producers of India, and the "Basanti Devi Amir Chand Prize" of the Indian Council of Medical Research. Dr. Ganesh is also a recipient of the Ramanna Fellowship of the Department of Sciences & Technology, and the Tata Innovation Fellowship of the Department of Biotechnology.

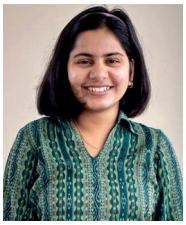
From carbohydrate to cognition: lessons learned from a rare form of neurodegenerative disorder

A disorder that affects lesser than 50 people per 100,000 population is often referred to as a rare disorder. While a given disease is rare, rare disorders together account for up to 5% of the population. The rare disorders are often neglected by the pharma companies for want of a market. Nonetheless, studies on such conditions offer better insight into the genetic basis of physiological processes that often shed light on the common pathophysiological mechanisms affecting the larger population. Our group has been working on a rare and familial form of progressive myoclonus epilepsy known as the Lafora disease (LD). Besides the onset of epileptic seizures in early teenage, patients with LD also display cognitive deficits, hallucinations, muscle wasting and respiratory failure leading to death within ten years of the disease onset. At least two genes have been identified for LD, defects in any one of them resulting in a clinically identical condition. One of the genes codes for a protein phosphatase named laforin and the other gene codes for an E3 ubiquitin ligase named malin. Using cellular and animal models, our group has demonstrated that laforin and malin function, as non-redundant partners, in multiple cellular pathways. These include glycogen metabolism, proteolytic processes, heat shock, and endoplasmic reticulum stress response and post-translation processes. This talk would provide an overview of these findings, discuss how defects in this process lead to specific symptoms seen in LD, and how LD could possibly be treated. This talk would also highlight the common pathways leading to neurodegeneration in a diverse set of disorders.

AWARD WINNERS AMONG STUDENT'S PRESENTATIONS

Eight doctoral students were given awards for their presentations in poster and oral sessions. Short write-ups of their work in their own words are collated below.

Rutambhara Purohit: Prof. A. S. Mukherjee Memorial award for oral presentation



Indian Institute of Technology, Madras, Chennai- 600036

Pannexin: a modulator of $P2X_7$ receptor-mediated calcium influx. Pannexin1 is a channel in the plasma membrane whose major function is to release intracellular ATP to the extracellular side. The released ATP molecules bind and activate purinergic P2X and P2Y receptors on the membrane. The opening of the $P2X_7$ receptor causes an influx of Ca^{2+}, which in turn opens Panx1. This interdependency of $P2X_7R$ and Panx1 requires spatial proximity for efficient and fast functional coupling. Earlier reports have suggested physical interaction between Panx1 and $P2X_7R$. Here the effect of Panx1 on $P2X_7R$-mediated intracellular Ca^{2+} rise is studied. **Methods:** $P2X_7R$ was activated with BzATP, and intracellular Ca^{2+} rise was monitored using Fura-2. Different mutants of rat Panx1 were generated by PCR subcloning. Cells were transfected transiently with different cDNA constructs using Lipofectamine-2000. **Results:** Overexpression of Panx1 was found to attenuate $P2X_7R$-mediated intracellular Ca^{2+} rise in CHO-K1 and HEK-293 cells. On the other hand, Panx1 knockdown in rat cortical astrocytes exhibited significantly higher Ca^{2+} influx through $P2X_7R$, which complements our findings. To identify the specific region of Panx1 associated with $P2X_7R$-inhibition, four truncation mutants were generated, having serial deletions from the C-terminal end. Studies with these mutants indicate that the stretch from Leu350 to Cys426 is crucial for inhibiting $P2X_7R$. The inhibitory effect of C-terminus is independent of the other regions of the channel as the expression of the just the C-terminus was able to attenuate $P2X_7R$, similar to full-length Panx1. **Conclusion:** Panx1 attenuates calcium influx through the $P2X_7$ receptor by its C-terminal domain. The

inhibitory modulation of Panx1 on $P2X_7R$ possibly plays an essential role in physiology and pathophysiology.

Mr. Aniketh Bishnu: Prof. V. C. Sah award for oral presentation

Imaging Cell Signalling & Therapeutics Lab, ACRTEC, Navi Mumbai

Hyper activated ERK1/2 kinase drives autophagy to promote survival of ovarian cancer cells at the onset of chemoresistance. Autophagy, which is regulated by several kinases including ERK1/2, plays critical role towards acquirement of chemoresistance in cancer cells. However, when and how autophagy assists cancer cells to overcome the therapeutic stress during the dynamic development of resistance has not been elucidated well. This study investigates the interplay between ERK1/2 activation and autophagy at different stages of chemoresistance in ovarian cancer (OC) cells. Autophagy flux was estimated by LC3I-II, p62 and ERK1/2 immunoblotting across different stages in OC cellular models of cisplatin-paclitaxel resistance. Transmission electron microscopy (TEM) and confocal microscopy were used to quantitate the number and morphology of autophagic structures. Increased LC3I-II conversion and p62 degradation was observed in early resistant (ER) cells with little or no p62 degradation in sensitive and late resistant (LR) cells after Cisplatin-paclitaxel treatment. The ER cells were characterized with high basal level of activated ERK1/2. TEM analysis revealed increased autophagosome (2-fold) in sensitive cells, increased autophagosome (1.8-fold) and autophagolysosome (2-fold) in ER cells and very low number of autophagic structures in LR cells. In comparison to sensitive or LR cells, a 3-fold higher LC3-$LAMP1^{+ve}$ fusion was observed in the ER cells. ERK1/2 inhibition through chemical and genetic approaches led to reduced p62 degradation and autophagolysosome/ LC3-LAMP1+ve vesicle formation with increased LC3I-II conversion and autophagosome accumulation in ER cells. ERK1/2 inhibitor treatment specifically sensitized ER cells to cisplatin-paclitaxel. Altogether our data suggest that hyper-activated ERK1/2 regulates the autophagosome-lysosome fusion step of autophagy to promote cellular survival during therapeutic stress at the onset of chemoresistance.

Dr. Akhouri Kishore Raghawan: Prof. S R V Rao Award for poster presentation

CSIR-Centre for Cellular and Molecular Biology (CCMB), Hyderabad

HSC70 regulates cold-induced caspase-1 hyperactivation by an autoinflammation-causing mutant of cytoplasmic immune receptor NLRC4. NLRC4 is an innate immune receptor, which, upon detection of certain pathogens or internal distress signals, initiates caspase-1–mediated interleukin-1β maturation and an inflammatory response. A gain-of-function mutation, H443P in NLRC4, causes familial cold autoinflammatory syndrome (FCAS) characterized by cold-induced hyperactivation of caspase-1, enhanced interleukin-1β maturation, and inflammation. Although the H443P mutant shows constitutive activity, the mechanism involved in hyperactivation of caspase-1 by NLRC4-H443P upon exposure of cells to lower temperature is unknown. Here, we show that heat shock cognate protein 70 (HSC70) complexes with NLRC4 and negatively regulates caspase-1 activation by NLRC4-H443P in human cells. Compared with NLRC4, the structurally altered NLRC4-H443P shows enhanced interaction with HSC70. Knockdown of HSC70 enhances inflammasome formation and caspase-1 activation by NLRC4-H443P. Exposure to subnormal temperature results in reduced interaction of NLRC4-H443P with HSC70, and an increase in its ability to form inflammasomes and activate caspase-1. Unlike the NLRC4-H443P mutant, another constitutively active mutant NLRC4-V341A, associated with autoinflammatory diseases, but not FCAS, shows neither enhanced interaction with HSC70 nor an increase in inflammasome formation upon exposure to subnormal temperature. Our results identify HSC70 as a negative regulator of caspase-1 activation by the NLRC4-H443P mutant. We also show that low-temperature–induced hyperactivation of caspase-1 by NLRC4-H443P is due to loss of inhibition by HSC70. We provide a molecular mechanism for exacerbation of inflammation induced by cold temperature in individuals carrying the NLRC4-H443P mutation, which might have broader implications for temperature regulation of FCAS-causing mutations of other receptors.

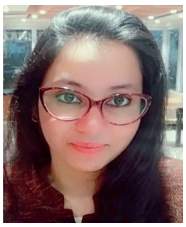
Mr. P. Vineeth Daniel: Prof. B R Sheshachar Memorial Award for poster presentation

School of Basic Sciences, Indian Institute of Technology Mandi, Mandi-175001

Chronic exposure to Pb^{2+} perturbs ChREBP transactivation and coerces hepatic dyslipidemia. Dysregulated hepatic de novo lipogenesis contributes

to the pathogenesis of nonalcoholic fatty liver disease in both humans and rodents. Clinical evidence suggests fatty liver to have a positive correlation with serum lead (Pb^{2+}) levels. However, an exact mechanism of Pb^{2+} induced fatty liver progression is still unknown. Here, we show that exposure to Pb^{2+} regulates ChREBP dependent hepatic lipogenesis. Presence of Pb^{2+} ions within the hepatocytes reduces transcript and protein levels of sorcin, a cytosolic adaptor partner of ChREBP. Adenovirus-mediated overexpression of sorcin in Pb^{2+} exposed hepatocytes and an in vivo mouse model ameliorates liver steatosis and hepatotoxicity. Hereby, we present Pb^{2+} exposure to be a lethal disruptor of lipid metabolism in hepatocytes and highlight sorcin as a novel therapeutic target against Pb^{2+} induced hepatic dyslipidemia.

Ms. Prerna Aggarwal: Dr. Manasi Ram Memorial Award for poster presentation



Department of Genetics, University of Delhi, South Campus, New Delhi.

Role of globin1 in the development and maintenance of the nervous system in Drosophila. Neurogenesis is driven by spatially and temporally regulated proliferation of neuronal progenitor cells that generates enormous number of assorted neurons to drive the complex behavior of an organism. Recent studies have linked globins to various biological and physiological processes. The present study attempts to investigate the role(s) of *globin1* (*glob1*) in the development and maintenance of the nervous system in *Drosophila*. We noted robust expression of Glob1 in the developing neuronal tissues with enhanced concentration throughout the Ventral Nerve Cord (VNC) which harbors numerous types of progenitor cells that finally differentiate into specific neurons. Also, profound expression of Glob1 was noted in the actively dividing cells of outer proliferation center of larval brain and photoreceptor axons of optic stalk. Ubiquitous or pan-neuronal down regulation of *glob1* causes partial lethality and mis-positioning of various neural-progenitor cells present in the embryonic midline cell clusters. In addition, the overall arrangement of photoreceptor axons and stereotype positioning of neuroblast cells present in the central region of the brain were severely affected due to reduced expression of *glob1*. Such larvae and surviving adults develop significant neuro-muscular disabilities and fitness impairments. Our study demonstrates a novel role of *glob1* in development and maintenance of the nervous system in *Drosophila*.

Gautam Chandra Sarkar: Indian Society of Cell Biology (ISCB) award for poster presentation

Molecular Aging Laboratory, National Institute of Immunology, New Delhi

Investigating the role of a novel CDK-like kinase in aging and germline development under conditions of low insulin signalling. The insulin-IGF-1 signaling (IIS) is a conserved signal transduction pathway that regulates important biological processes. In mammals, deregulation of this pathway result in type II diabetes and cancer. Extensive research has established the role of IIS pathway in regulation of metabolism, dauer diapause, stress resistance as well as life span in *C. elegans.* Studies have also indicated that IIS pathway controls germline development and oocyte quality, but the mechanisms are less known. In our lab, we are interested in understanding the undefined roles of protein kinases in regulation of the IIS pathway. In an RNAi screen, we have identified a CDK-like kinase that genetically interacts with the IIS pathway. Knocking down (KD) the kinase using RNAi increases dauer formation in the IIS mutant, *daf-2(e1370).* Preliminary data shows that the KD of the CDK-like kinase leads to increased nuclear localization of the FOXO transcription factor DAF-16, an indication that the IIS pathway may be de-regulated. Interestingly, we also found that KD of the kinase, specifically in the *daf-2(e1370)* mutant, leads to arrest of its germ cells at the pachytene stage of meiosis, in a FOXO/DAF-16-dependent manner. This pachytene arrest is rescued by a gain of function mutant *let-60(ga89),* which suggest that this CDK-like kinase is required for MAPK activation under lowered insulin signaling condition leading to pachytene exit of germ cells in *C. elegans*. We are currently investigating the nature of the interaction of this kinase with the IIS pathway.

Ms. Shivangi Gupta: Indian Society of Cell Biology (ISCB) award for poster presentation

Indian Institute of Science Education and Research (IISER) Mohali

Investigating the role of Pten/ Akt during zebrafish retina regeneration. Upon an acute injury, the differentiated Muller glia (MG) cells of zebrafish retina, attain stem cell-like features and get reprogrammed to Muller glia derived progenitor cells

(MGPCs), which proliferate and increase their number to accomplish retina regeneration, process absent in mammals. But there exists a possible check on the uncontrolled cell proliferation by cell cycle checks or tumor suppressors. In our work, we have tried to investigate the role of a phosphatase/tumor suppressor protein, Phosphatase and Tensin Homolog (Pten), which has not yet been explored during retina regeneration in zebrafish. We report an exclusion of Pten and a colocalization of Akt from the actively proliferating cells as marked by PCNA, observed by Immunohistochemistry. We found an increase in the number of MGPCs upon knockdown and blockade of Pten, which might be due to an upregulation of *ascl1a* or a down regulation of *her4.1* as seen by mRNA in situ hybridization and PCR, and is also supported by an enhanced level of total Akt protein. This increase in proliferation continues even after the proliferation phase abates which leads to the formation of few of the retinal cell types in 30dpi. Pten regulates the levels of an oncogenic marker/matrix metalloproteinase *mmp9*, thereby controlling cell proliferation. Upon *mmp9* knockdown and blockade of mmp9 activity along with Pten blockade, we observed a decrease in the number of MGPCs, owing to reduced Ascl1a levels. Our studies will shed light on the role and interplay of a phosphatase with other regeneration-associated genes during this process of retina regeneration.

Prerna Budakoti: Indian Society of Cell Biology (ISCB) award for poster presentation

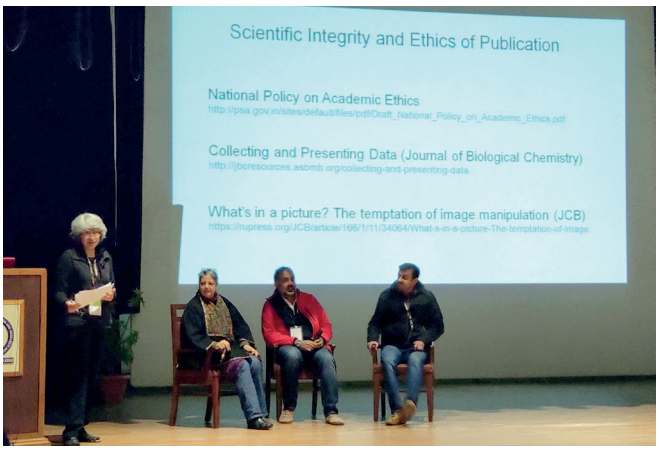
Molecular Cell and Developmental Biology Laboratory, IISER, Mohali

Induction of preleukemic condition in the larval lymph gland of Drosophila, by ectopic expression of human oncogeneic fusion protein AML1-ETO induces changes in the hematopoietic niche. Long-term maintenance of tissue homeostasis relies on cross communication among different cell types constituting the tissue. In stem cell biology, communication between the niche cells that create the microenvironment to support the stem cells plays a prominent role in maintaining the state and fate of stem cells. Disorders like cancer, disrupts the tissue homeostasis and is often induced by the cancer cells. For instance, in hematopoietic malignancies the bone marrow microenvironment gets altered in such a manner that it supports the growth of leukemic cells at the expense of the normal hemocytes. Our understanding about how these changes in the niche are induced by leukemic cells is rudimentary. Given that the vertebrate hematopoietic niche is very complex in nature, we resorted to the relatively simpler and genetically tractable organism, Drosophila, to address this question. Lymph gland, the larval hematopoietic organ in Drosophila, consists of progenitors, differentiating cells and the cells of the posterior signaling centre (PSC) that acts as a niche. We created a pre-leukemic condition in the lymph gland by ectopic expression of the human oncogeneic fusion protein AML1-ETO. Over expression of AML1-ETO results in excessive proliferation and induces hyperplasia of the hematopoietic organ. Reduction in progenitor population and a concomitant increase in differentiated cells exhibit a differentiation bias. Most importantly, despite the fact that ectopic AML1-ETO is expressed in the differentiating blood cells, the size of the PSC is markedly reduced. The mechanistic basis of this non-autonomous effect of pre-leukemic cells on the niche cells will be discussed.

Glimpses of AICBC 2019 Conference at IISER, Mohali, Mohali campus

AICBC group photograph

Ongoing session in AICBC 2019 conference



Panel discussion in the conference

Prof J Das Memorial Award lecture delivered by Prof S Ganesh, IIT, Kanpur

Student awardees with ISCB executive members.

44th All India Cell Biology Conference,

September 2nd-3rd, 2022

Department of Biochemistry, University of Kashmir

The 44th All India Cell Biology Conference (annual meeting of the Indian Society of Cell Biology, ISCB) hosted by the Department of Biochemistry at University of Kashmir commenced from 2nd-3rd September, 2022. This conference was held at the Convocation Complex of the University and was very well attended with over 600 registered participants. Over 200 PhD students and post doctoral fellows presented their work in the form of oral and poster presentations. In addition to felicitation of three scientists for their outstanding work in the field of cell biology, there was panel of distinguished cell biologists who gave an overview of their research work and experiences. The two days conference gave a lot of information on recent advances, new trends and opportunities in cell biology. It also provided a wonderful opportunity to the participants to interact with one another and forge collaborations.

Day 1:

The conference started at 9.15am with the beautiful Kashmir University tarana. The proceedings of the entire conference were conducted by Dr. Misbah Shah of the University of Kashmir. The conference started with a welcome address by Dr Shajrul Amin, Head, Department of Biochemistry, University of Kashmir and the convenor of the conference. She talked about the importance of science in combating diseases and public health. She also briefed about the research activities and funding at the Department of Biochemistry, University of Kashmir. Prof. Pradeep K Burma, Vice President, ISCB, appreciated the efforts and enthusiasm of the Department of Biochemistry, UoK for organizing the academic feast while soaking in the scenic beauty of the campus. Dr. Nisar Ahmad, Registrar, University of Kashmir, appreciated the efforts of the Department of Biochemistry, University of Kashmir and ISCB for holding this event and also impressed upon contribution of the Science departments in enahancing the standard of University of Kashmir, as is reflected by the NIRF and NAAC rankings. Dean Research, Uiversity of Kashmir, Prof. Irshad Ahmad Nawchoo highlighted that cell: is the backbone of all biological sciences and emphasized that scientists have to be futuristic to sustain and to stay relevant in the society. Dean Academic Affairs, University of Kashmir, Prof. Farooq Ahmad Masoodi, talked about importance of basic sciences and the significance of multidisciplinary research.

Prof. Neelofar Khan, Vice Chancellor, University of Kashmir, applauded the efforts of Department of Biochemistry, University of Kashmir and ISCB for convening this conference and taking care of all the logistics. She also talked about the importance of women in science and that the University of Kashmir is always available for conducting such academic and research related events. She highlighted that the theme of this conference *"Molecular and Cellular Insights of Human Diseases"* was particularly relevant in the present times. Subsequently, the guests of honour also inaugurated the Souvenir and Abstract book that was compiled for this conference.

The opening ceremony was concluded by a vote of thanks by organizing secretary of the conference, Dr. Shaida Andrabi. He expressed gratitude and appreciation for the unrelenting support, encouragement and financial assistance from ISCB and the University of Kashmir. He also thanked the funding agencies (India Alliance Wellcome Trust DBT, Jammu and Kashmir State Innovation Council (JKSTIC), sponsors (BD Biosciences, Molecular Devices, Leica Microsystems, Carl Zeiss and ThermoScientific/Biomed Systems) and the organizers.

Technical session-I

The first technical session was chaired by Dr. Bhupendra N Singh, Secretary, ISCB and Prof. Zafar Reshi, Dean Biological Sciences, University of Kashmir. This session had one plenary award lecture and 5 invited talks.

Prof. Chandrima Saha, IICB, Kolkatta and President INSA, was given the XVIII Prof. S P Ray-Chaudhuri Endowment Lecture Award. She was introduced by Prof. Bhupendra N Singh, Secretry ISCB. Her research presentation focused on intracellular parasitism and evolution of human immune system in race against pathogens. In his impressive talk, Prof. Sanjeev Galande, from the Shiv Nader University, Delhi-NCR, discussed that how their research group has repurposed statins for colorectal cancer therapy. He also described SATB family chromatin organizers as master regulators of tumor progression. Dr. Pritha Ray, ACTREC, Mumbai delivered a talk focusing on key molecular structures responsible for chemo resistance. Dr. Rashna Bhandari, CDFD Hyderabad, illustrated her

findings on protein pyrophosphorylation, a concept that was literally novel to everyone in the audience. Her presentation showed how molecular insights into cell provide platform for disease treatment. This session was concluded by the presentation of Prof. V. Radha, CCMB, Hyderabad, who delivered a lecture on importance of signaling molecules like C3G for identification and correction of developmental defects.

Technical session-II

The second technical session was chaired by Prof. Jagat K Roy, BHU, Varanasi and Prof M A Zargar, Central University, Kashmir. This session started with a lecture in memory of late Prof. Jyotirmoy Das who was the first scientist to start a theoretical biology lab at IISCB, Kolkatta.

Prof. Umesh Varshney, IISC, Banglore, was presented with The Eleventh Prof. J Das Memorial Lecture Award. He was introduced by Prof. Jagat K Roy. This was followed by a plenary award lecture in which Prof. Umesh Varshney highlighted the significance of formyl methionine as a link between protein translation and cellular metabolism in *E.coli.* Prof. K. I. Andrabi, University of Kashmir, discussed about implications of mTOR signaling pathway for cell survival and growth. The first online lecture of the session was given by Prof. Jonathan Higgins from the Newcastle University, United Kingdom, who delved into cellular mitosis and the role of histones, H3T3PH and H3S10PH. While Dr. Nazir Ahmad Dar spoke about attenuation of p53 mediated control on G6PD by NF-κβ, Dr. Sagar Sengupata talked about entry of Poly YA into mitochondria and its negative regulation by MITOL dependent ubiquitylation.

Technical Session-3

Prof. V Radha, CCMB, Hyderabad and Dr M. Ashraf Dar, University of Kashmir chaired the last scientific session of Day I. The session started with the Third Prof. Rita Mulherkar Award that was presented to Dr. M. Imtaiyaz Hassan, Jamia Milia, New Delhi. Dr Imtaiyaz Hassan was introduced by Dr. Ritu Trivedi, joint secretary, ISCB. In his plenary award lecture, Dr. Imtaiyaz Hassan focused on targeting microtubule dynamics to control cancer. Dr. Ayub Qadri from the Islamic University of Science and Technology, Kashmir, talked about host pathogen interactions, focusing on conversations that Salmonella engages in, with host cells. The last talk of the day was delivered by Dr. Aamir Nazir from CDRI. In his excellent presentation, Dr Aamir Nazir talked about enrichment of PTR-10 in glial cells and exploiting functional genomics and epigenetic modifications for protective and repair functions in ailments like Parkinson's disease.

Day 2:

The second day of the conference started with the fourth and last technical session of the two day scientific feast. This session started at 9.30 am and was chaired by Prof. Pradeep K Burma, University of Delhi, and Dr. Shaida Andrabi, University of Kashmir. The first talk of the session was an online presentation by Professor Arshad Desai, University of California, San Diego. He presented his work regarding the regulation of mitosis, histone modifications, its regulation by p53 complex and also how targeting this pathway could be exploited to combat cancer.

Prof. L. S. Shashidhara, Ashoka University, Sonepat, delivered his talk on modulation of wing development in *Drosophila melangoster* by *ubx*. He showed how activation of Yki and AKT promote haltere to wing transformation in Drosophila.

Soon after, one of the most awaited and thrilling events of the conference – oral presentations by the research students began. This session was started with two sponsor talks: BD Biosciences and Molecular Devices who introduced their recent products which are used for advanced molecular imaging that could be used by cell biologists. This was immediately followed by the student oral presentations. Out of a total of >100 students who had applied for oral presentations, only seven students were selected by the selection committee. All seven students presented their work in this session and these presentations were evaluated by a panel of 5 judges. Each student was given a time of 8 minutes. In concurrence with the presentations of invited speakers, students also gave excellent presentations with loads of enthusiasm and emotions. Importantly, they all completed their presentations in time and defended all the questions that they were asked.

Poster session

The last scientific session was that of poster presentations. About 200 offline and 60 online posters were presented by students and postdoctoral fellows. Keeping in view this large number, a considerable amount of time was dedicated for poster presentation session. This session attracted a lot of attendees and was lively with scientific debates, arguments, and recommendations. Two judges examined each poster after seeing it in person and evaluated them. This session lasted for >2 hours. Students from diverse backgrounds, reputed universities and institutes presented their work as posters. This event was held in the upper gallery of the Convocation Complex, which was fully packed.

Valedictory session

The valedictory session of the conference was

chaired by Prof. Zafar A Reshi, Dean School of Biological Sciences, University of Kashmir, Prof. Pradeep K. Burma, Vice President ISCB (UDSC, Delhi) and Prof. Bhupendra N Singh, Secretary ISCB (CSIR-CDRI). Prof. Akbar Masood, Vice Chancellor, Baha Ghulam Shah Badshah University, Rajouri, was the chief guest of the session. The highlights of the two days conference were briefed by Dr. M. Ashraf Dar, University of Kashmir. In the closing ceremony, Prof Zafar Reshi mentioned that this conference was a grand success and congratulated the Department of Biochemistry, University of Kashmir and the ISCB for holding this memorable event. Prof. Pradeep K Burma and Prof. Bhupendra N Singh appreciated the organizers, the University of Kashmir officials, sponsors, participants and the volunteers for organizing a successful conference and participating in the event with great enthusiasm. Prof. Akbar Masood expressed his appreciation and gratitude for the Department of Biochemistry, University of Kashmir as well as the ISCB and everyone involved in organizing the conference.

This was followed by prize distribution ceremony. Prizes, in the form of certificates and cash were given to the students for both oral and poster presentations. 3 awards were given in oral presentation while 10 awards were given for poster presentations. While Hilal Ahmad Reshi (CDFD, Hyderabad), Riffat Khanum (Presidency University, Kolkatta) and Debabrata Jana (CCMB, Hyderabad) received the awards for oral presentations, poster awards were given to Nusrat Nabi (University of Kashmir), Avinash (CDRI, Lucknow), Raj bahadur (CCMB, Hyderabad), Younis Ahmad Bhat (University of Kashmir), Ekta Gupta (IISER, Pune), Devanshi Gupta (CDFD), Arpita Singh (CDFD), Mudassar Ali (Shiv Nadar Univ), Sugata Chaudhuri (IISER, Mohali), Afruja Khan (IISER, Mohali) were the winners in poster presentation category.

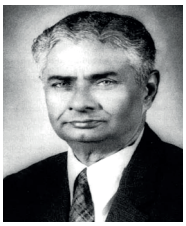
With this event, the 44thAll India Cell Biology Conferencecame to a wonderful conclusion after a long hiatus due to pandemic, in the splendid campus at University of Kashmir.

At the end, Dr. Nazir Ahmad Dar from the Department of Biochemistry, University of Kashmir presented the final vote of thanks. The conference ultimately came to closure at 6.00 pm.

Award lectures at the 44th All India Cell Biology Conference, 2nd Sept. 2022

The XVIII Prof. S P Ray-Chaudhuri 75TH Birthday Endowment Lecture (Year 2020)

Prof S P Ray-Chaudhuri



The Indian Society of Cell Biology started in 1976 and its first President was Prof. Sachi Prasad Ray-Chaudhuri. Prof. Ray-Chaudhuri was a doyen among the chromosome biologists in the country who pioneered research and teaching of animal genetics in India. Having being trained under the tutelage of Prof. H. J. Muller in Edinburgh, Prof. Ray-Chaudhuri initiated research in areas of radiation genetics and comparative cytogenetics in Calcutta University. In 1961 he moved to Varanasi to adorn the chair of the Head, Department of Zoology of Banaras Hindu University. During his tenure, his inspiring leadership modernized the Zoology Department of BHU, and catapulted it to the most dynamic department of animal science in the country. Besides his own research in various areas of genetics and cytogenetics, Prof. Ray-Chaudhuri actively encouraged and pursued development of other areas such as Physiology and Biochemistry, Reproduction Biology, Ecology in the department. A whole generation of students who worked under him not only did commendable work but succeeded in making dynamic schools of their own. His benign and caring support was not confined to his own Ph.D. students but to anyone who came in contact with him. He had an abiding influence on all those who were even remotely associated with him. After his retirement from Banaras Hindu University in 1971, Prof. Ray-Chaudhuri went back to Calcutta University and continued to work until mid nineteen eighties till his age and ill health prevented him from active lab life. He breathed his last in the year 1994 at 87 years of age.

Indian Society of Cell Biology, most appropriately, created an endowment fund on his 75th Birthday and created a lecture series in 1982 whose first lecture was delivered by the celebrated geneticist, Dr Obaid Siddiqi in the Cell Biology meeting held in Madurai Kamraj University. Since its inception there have been 17 lectures till date including Dr. Obaid Siddiqi (1984), Dr. A T Natarajan (1986), Dr. H Sharat Chandra (1990), Dr. Lalji Singh (1995), Dr. A N Bhisey (2000), Prof. S C Lakhotia (2002), Dr. K VijayRaghavan (2007), Prof. B K Thelma (2018). The complete list is available on the website of the society (http://www.iscb.org.in/).

The Society feels privileged and proud to have Dr. Chandrima Shaha, President, Indian National Science Academy as the speaker of the Eighteenth Prof. S P Ray-Chaudhuri 75th Birthday Endowment Lecture Award for year 2020, today, at XLIV AICBC 2022, University of Kashmir, Srinagar.

Dr. Chandrima Shaha, Indian Institute of Chemical Biology (IICB), Kolkata

Prof. Chandrima Shaha, currently the President of the Indian National Science Academy is well known for her contributions on the significance of death in cells with high division index. Prof. Shaha is also currently holding the responsibility of JC Bose Chair Distinguished Professor of the National Academy of Sciences at the Indian Institute of Chemical Biology. She is one of Lilavati's daughters. Born on 14th October 1952, Prof Shaha received her M. Sc degree from the University of Calcutta in 1974. She spent her doctoral stint at the Indian Institute of Chemical Biology at Kolkata followed by Post doctoral studies at the University of Kansas Medical Center and the Population Council at the Rockefeller University. After her post-doctoral stint, she joined the National Institute of Immunology at New Delhi where she served as Director for five years prior to her engagement as Professor of Eminence.

At the National Institute of Immunology, she established a group interested in host-pathogen interactions. Their focus was to explore how death processes in both the host and the pathogen affected the outcome of infections, as co-evolution of the two has shaped both the assault and the defense systems. The group was the first to show that cell death existed in the early mitochondrial eukaryotes and this played a vital role in the pathogen's defense against the host assault. Overall, the demonstration of an intricate relationship between death processes of a mammalian host and a neglected tropical disease pathogen opened new areas of research on the challenges of developing interventions using these pathways as targets. At the National Institute of Immunology, her interest in bringing in young students with the purpose of creating awareness about scientific issues in young people propelled her to evolve a highly successful program of 'Science Setu' that built a bridge between the young students and the scientists. This model has now been adopted by many institutes. She also established a Center for Molecular Medicine with

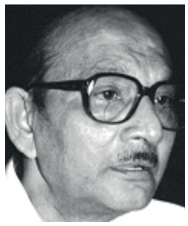
DBT funding at the National Institute of Immunology.

She is an elected fellow of the World Academy of Sciences and fellow of all three Science Academies of India. Notable awards include the Ranbaxy Science Foundation Award for basic sciences; the J.C. Bose Fellowship; Shanti Swarup Bhatnagar Medal of INSA; Om Prakash Bhasin Award; Archana Sharma Memorial Award; Darshan Ranganathan Memorial Award; Chandrakala Hora Memorial Medal and the Shakuntala Amir Chand Prize.

We are pleased to have her as a speaker for the eighteenth **Prof. S. P. Ray-Chaudhuri 75th Birthday Endowment Lecture Award** for year 2020.

The Eleventh Prof. J Das Memorial Lecture Award 2nd September 2022

Prof. Jyotirmoy Das



This lecture is in the memory of late Prof. Jyotirmoy Das who made pioneering contributions in *Vibrio cholerae* research. He worked on elucidation of genetics of cholera phages, physical and genetic maps, repair processes and stress biology of *Vibrio cholera*. He did his B.Sc. and M.Sc. in Physics from Calcutta University and Ph.D. in Biophysics from the Baylor College of Medicine. He returned to the country in 1978, joined Bose Institute and then moved to Indian Institute of Chemical Biology (IICB) as head of Biophysics Division in 1979. Later he became the Director of the Institute in 1995 and remained in this position till he passed in July, 1998. He was also a Visiting Professor at the University of Rochester, USA.

Prof. Das demonstrated the DNA repair mechanism in *Vibrio cholerae* and identified genes involved in these processes. He elucidated the intracellular replication of cholera phages and investigated the mechanism of biotype differentiation by these phages along with the analysis of the cell surface architecture of *Vibrio cholerae* and developed an oral vaccine strain for cholera. His group contributed significantly to the elucidation of *V. cholerae* genome structure, identification of genomic rearrangements and diversity, and went on to construct first a physical map of the *V cholerae* genome. Prof. Das had a desire to understand the physical basis of life, which prompted him to start the Theoretical Biology Group at IICB, Calcutta. Recognizing the needs of the time, he set up a division of human genome research at IICB.

In addition to being chosen as the UGC National Lecturer in 1985, Jyotirmoy Das also received the Ranbaxy Research Foundation Award in 1989 and the Om Prakash Bhasin Award in 1990; INSA Golden Jubilee Commemoration Medal (1998). He was a Fellow of the Indian Academy of Sciences, Bangalore and the National Academy of Sciences (India), Allahabad. He was a member of the editorial board of the *Indian Journal of Biochemistry and Biophysics*.

Since its inception there has been nine memorial lectures by eminent scientists during All India Cell Biology Conferences, viz., Profs. P. Balaram (2001), M.R.S. Rao (2003), P.P.Majumdar (2006), V. Nagaraja (2007), A Surolia (2009), M.Vijayan (2011), Kanury V S Rao (2013), J S Tyagi (2015) and Dr. Alok Bhattacharya (2018).

The Society feels privileged to have Prof. Umesh Varshney, J. N. Tata Chair Professor at Department of Microbiology and Cell Biology, IISc, Bangalore as the speaker of the XI Prof. J Das Memorial Lecture Award for year 2021 at XLIV AICBC 2022, University of Kashmir, Srinagar.

Dr. Umesh Varshney, *FNA, FASc, FNASc, FTWAS*, J. N. Tata Chair Professor, Indian Institute of Science, Bangalore, India.

Prof. Umesh Varshney, a Life Member of the Indian Society of Cell Biology, obtained his M.Sc. in Microbiology (with minor in Biochemistry) from G. B. Pant University of Agriculture and Technology, Pantnagar in 1979 and his Ph.D. in Biochemistry from the University of Calgary, Canada in 1985. He carried out his first post-doctoral research at the University of Calgary and then for a second post-doctoral stint, he moved to the Massachusetts Institute of Technology, Cambridge, USAin 1988. He returned to India in 1991 to join as an Assistant Professor at the Indian Institute of Science, Bangalore (IISc). Since then, Prof. Varshney has continued to serve IISc in various capacities and is presently a J N Tata Chair Professor and the Dean of the Faculty of Science. In addition, as a part of his services to the nation, he continues to servein different capacities in various committees of the funding agencies, academic research organizations and Universities.

Prof. Varshney uses biochemical and molecular genetics approaches to investigate mechanistic and evolutionary aspects of protein synthesis and DNA repair in bacteria. The studies are important to discover newer principles to combat antimicrobial resistance and understand ribosome biogenesis. His work on DNA repair focuses on the biochemical and physiological aspects of proteins of the base excision repair, and nucleotide excision repair pathways, and cross-talk between different DNA repair pathways. Prof.

Varshney's research includes studies on *Mycobacterium tuberculosis*, which causes tuberculosis, a global human health problem.

Prof. Varshney has contributed immensely toadvancement of science by mentoring young students and researchers during his tenure at IISc. So far,28 students have received their Ph.D. degrees with him. Many more have received training as post-doctoral fellows or short-term trainees. Prof. Varshney continues to mentor PhD students, post-doctoral researchers and the undergraduate students for their research. He has published over 180 research papers in reputed journals. Importantly, many of the researchers who obtained their Ph.D.degrees or post-doctoral training with him areserving at various research institutions, Universities and, biotechnology organizations of their own in India and abroad.In 2002, Prof. Varshney's efforts were instrumental in bringing together a group of scientists in India interested in RNA research. This forum known as 'RNA Group' meets periodically in different parts of the country.

Prof. Varshney has received several recognitions for his contributions. Among these are the fellowships of the Indian National Science Academy, New Delhi; Indian Academy of Sciences, Bangalore; National Academy of Sciences (India), Allahabad; and The World Academy of Sciences, Trieste. Also, Prof. Varshney has been a recipient of several awards that include the Shanti Swarup Bhatnagar Prize, G. N. Ramachandran Gold Medal for Excellence in Biological Sciences and Technology, J. C. Bose National Fellowship, National Bioscience Career Development Award, Life Sciences Research Award of the Novo Nordisk Education Foundation (India), Ranbaxy Research Award, Goyal Prize, P. S. Sarma Memorial Award of the Society of Biological Chemists (India), J.V. Bhat Endowment Oration, Alumni Award for Excellence in Research for Science, IISc, and an OutstandingAlumnus of the College of Basic Science and Humanities, G. B. Pant University of Agriculture and Technology.

The Third Prof. Rita Mulherkar Lecture Award September 2, 2022

Prof. Rita Mulherkar

Prof. Rita Mulherkar having completed her Ph.D. in Zoology from Calcutta University started her scientific career from Cancer Research Institute, Advanced Centre for Treatment, Research and Education in Cancer and superannuated from the same institute. She worked in the area of oncology with focus on Genetic predisposition to tobacco-related head and neck cancers; association of low penetrance genes with environment in cancer, Gene Therapy for Head and Neck cancers, Genomics of cervical cancer: Search for Biomarkers. She received several Honors and Awards, viz. Prof. K .P. Bhargava Memorial Medal, INSA; Fellow- National Academy of Sciences, India and Maharashtra Academy of Sciences.

Prof. Mulherkar has been a role model for her students and colleagues. She inculcated scientific rigor, and spirit of inquiry which has been of immense help as professionals. As a mentor, she shepherded a varied bunch of students and got the best out of each. There was no competition, healthy or otherwise, as all were there to enjoy the process of discovery, learn the discipline and ethics required to carry out the work honestly, and help each other in doing so. She stood up for all her students and staff alike in difficult situations, encouraged them when low, applauded them when successful, and always stood at the finishing line. Being part of Mulherkar lab was pride for all of us. Mulherkar lab was also engaged in community based projects for which sample collection from outdoor location was an intense and fun filled activity. The newer generation of scientists and women in science in the country need more mentors like her, who will lead by example.

On the occasion of superannuation of Prof. Rita Mulherkar after more than 30 years of dedicated service at Tata Memorial Centre, in January 2014, her students and colleagues decided to institute an award in her honour.

Dr. Md. Imtaiyaz Hassan, Jamia Millia Islamia, New Delhi

Dr. Md. Imtaiyaz Hassan is an Assistant Professor of Biophysics at the Centre for Interdisciplinary Research in Basic Sciences, Jamia Millia Islamia, New Delhi. Dr. Hassan earned his Master's degree in Biotechnology from Aligarh Muslim University, where he was Gold Medallist. Then he joined the Department of Biophysics, All India Institute of Medical Sciences, New Delhi for his Ph.D. in Structural Biology under the mentorship of Professor T. P. Singh. He worked in the innovative area of structural proteomics and received Institute Gold Medal for his seminal contribution to the field. Dr. Hassan moved to the Jamia Millia Islamia as Young Scientist in 2007 and subsequently joined as Assistant Professor of Biophysics in 2009.

He established a research group to work on structure-based drug design and discovery, targeting human kinases. His research has elucidated mechanisms of many signalling processes touching

upon the causative factors and likely mechanisms of amelioration. In addition to that, recently, his group has developed user-friendly molecular docking software, InstaDock, which is freely available. He has published more than 400 papers in high-quality peer-reviewed journals in short duration of his research career proving his scholarly attributes. Recently, he was recommended among the top ten bioinformatician of India and among the top 2% scientists worldwide.

Dr. Md. Imtiyaz Hassan is the recipient of many National and International awards. He has been elected as a Member of the National Academy of Science India, Allahabad and a Fellow of Indo-US Science and Technology Forum, DST India. He has been elected as a Fellow of the Royal Society of Biology (FRSB) and a Fellow of the Royal Society of Chemistry (FRSC), London, UK, for his significant contributions to the advancement of drug discovery. He is associated with different scientific societies in various capacities like Vice-President, General Secretary and Joint Secretary. He serves as Editor of many leading journals of biology, including Journal of Biomolecular Structure and Dynamics, Briefings in Functional Genomics, Plos-ONE, Scientific Reports, Frontiers in Oncology, etc.

He is one of the outstanding present day researchers in the country, a dedicated teacher who is always willing to impart his knowledge with greater enthusiasm and excellent communication skills. He is extremely popular among his students and regarded as a role model for many of them.

The Society feels privileged to have Dr. Md. Imtaiyaz Hassan as speaker of the 3rd Professor Rita Mulherkar Lecture Award for year 2020 at XLIV AICBC 2022, University of Kashmir, Srinagar. We wish him success in all his future endeavours.

Students Awards for presentations in 44th AICBC 2022, Srinagar

Best paper presentation in oral session: Three Awards

1. Prof. S. R. V. Rao Award:

Riffat Khanam

The Emerging Role of Adamts4, A MMP and ECM Molecule as a Novel Cardiac Injury Biomarker with Implications in Patients with Cardiac Injury

Riffat Khanam, Arunima Sengupta, Dipankar Mukherjee, Santanu Chakraborty

Department of Life Sciences, Presidency University, Kolkata-700073, West Bengal

Pathological cardiac remodelling as an aftermath of a severe cardiac injury can lead to ventricular dysfunction and subsequent heart failure. Our study focuses on Adamts4, a matrix metalloproteinase (MMP) and extracellular matrix (ECM) marker following cardiac injury. Our *in-vivo* studies in mice model show widespread prevalence of Adamts4 throughout chamber myocardium in the embryonic stages but that its expression severely wanes and is only restricted to the edge of the Interventricular septum (IVS). However, reactivation of Adamts4 in LV of chamber myocardium post Myocardial Infarction (MI) induction in adult murine model is observed and interestingly, the expression of Adamts4 co-localised with cardiomyocytes as confirmed by MF20 co-labelling study. To further decipher the signalling, Adamts4 induction was induced by hypoxia and ROS stress treatment in H9c2, a rat cardiomyocyte cell line. In response to both the stress conditions, Adamts4 expression along with Tgf-B, a-SMA, Col-III and Periostin was significantly enhanced as validated by Western Blot, IF and qPCR data. Moreover, Tgf-B inhibition by ALKI treatment shows Adamts4 inhibition and thereafter inhibition of the above-mentioned ECM and fibrosis markers. However, Adamts4 loss of function by Adamts4 specific siRNA transfection showed no significant change in the expression of Tgf-B indicating the Tgf-B dependent Adamts4 functioning. Finally, Adamts4 and a-SMA expression was studied in clinical samples with a history of MI (Anterior wall MI, Inferior wall MI) and Dilated cardiomyopathy (DCM) where the expression of Adamts4 was significantly elevated as quantified by Western blot for Adamts4 and a SMA in addition to Adamts4 specific ELISA. Our work for the first time highlights the emerging role of Adamts4 as an alternative cardiac injury marker in addition to the routinely assessed cardiac biomarkers.

2. Prof. V. C. Shah Award

Debabrata Jana

Trophoblast Stem Cells and Blastoids Generation Follow Competing Molecular Trajectories

Debabrata Jana, Priya Singh, Purnima Sailasree, Mansi Srivastava, P Chandrasekhar

Centre for Cellular and Molecular Biology, Hyderabad

Early mammalian development comprises of two major processes: development of epiblast, and development of extraembryonic layers like trophoblast and hypoblast for providing support to the epiblast. I am particularly curious to understand how these very early cells of embryo decide to become foetus or supporting cells, using mouse as a model system. Developmental potential of Embryonic stem cell (ESCs) known to restrict to embryo proper and hypoblast but not trophoblast. However recent reports in the past one year have shown that ESCs cultured in extended potential media can form extraembryonic layers including trophoblast. However, mechanism of such process is still unknown. In this study we have identified one of the major signalling pathways and the molecular players essential for attainment of the extended pluripotent and differentiation to trophoblast lineage. We have also identified a complex interaction between three transcription factors regulated by small molecules leading to trophoblast differentiation of pluripotent cells. In addition, we have derived trophoblast cell lines and also show that the cells can contribute to trophoblast lineage when injected, into 8-cell morula. With these mechanistic insights and few other perturbations, we were able to selforganise preimplantation embryo like structure solely from mouse embryonic stem cells in-vitro. We also show that these embryo-like structures can implant and develop till dpc 7.5.

3. Prof. A. S. Mukherjee Memorial Award

Hilal Reshi

EYA-SCAMP3 Facilitate Wntless Trafficking

Hilal Reshi and Dr Maddika Subba Reddy

Centre for DNA Fingerprinting and Diagnostics, CDFD, Hyderabad

Eyes Absent proteins (EYAs) initially deemed critical for Drosophila eye development, are now well known for a myraid of functions including DNA repair, innate immunity and organogenesis. The highly modulated domain structure of EYA proteins enables them to possess several functions that include phosphatase activity and transcriptional co-activation. However the role of EYA proteins in endocytic trafficking is unknown. Here we demonstrate that besides these actvities, EYA proteins form a complex that facilitates the retrograde trafficking of Wntless cargo from early endosomes to the trans-golgi thus regulating wnt-signalling. Mechanistically, EYA complex interacts with the retromer on the surface of wntless-enriched early-endosomes and coats them with SCAMP3 (Secretory Carrier Membrane Protein 3), a step necessary for their fusion with the Golgi membrane. The SCAMP3 coating on wntless endosomes act as molecular cues that distinguish between the recycling vesicles and the vesicles meant for lysosomal degradation. We also show that disintegration of EYA complex by depletion of any EYA component or SCAMP3 directs the vesicles to lysomes for degradation. This places the EYA complex at an important interface between the early endosomes and trans-golgi where it can both maintain the balance between wntles recycling and degradation and skew it towards any direction as per the cellular need. We also demonstrated that EYA mutations found in people that suffer from progressive sensori-neural hearing loss and craniofacial syndrome form a dysfunctional EYA complex, that either lacks several interactions between individual EYA members or fails to bind the retromer. The dysfunctional EYA complex doesn‟t induce wnt-signalling that is critical for the development of inner ear and other tissues including the lining of intestine and lungs. The dependence of wnt-signalling on EYA complex formation and lack thereoff in hearing-loss patients points to a causal relation between EYA expression and wnt-driven cell polarity in the hair cells of inner ear. The sequence of events, mechanistic details and significance will be discussed.

Best paper presentation in poster session: Five Awards

1. Prof. S. R. V. Rao Award
   Raj Bahadur, CCMB (Abs. no. 255)
2. Prof. V. C. Shah Award
   Ekta Gupta, IISER, PUNE (Abs. no. 307)
3. Prof. B. R. Sheshachar Memorial Award
   Devanshi Gupta, CDFD (Abs. no. 141)
4. Dr. Manasi Ram Memorial Award
   Abinash Swain, CDRI (Abs. no. 326)
5. ISCB award (Online Presentation)
   Arpita Singh, CDFD (Abs. no. 70)
6. ISCB award
   Mudassar Ali, Shiv Nadar Univ (Abs. no. 206)
7. ISCB award
   Nusrat Nabi, Kashmir Univ (Abs. no. 212)
8. ISCB award
   Sugata Chaudhuri, IISER, Mohali (Abs. no. 234)
9. ISCB award
   Younus A Bhat, Kashmir Univ (Abs. no. 268)
10. ISCB award
    Afruja Khan, IISER, Mohali (Abs. no. 351)

Glimpses of 44th AICBC 2022 Conference at University of Kashmir, Srinagar

Inaugural session of the AICBC Conference

Prof. Neelofar Khan, Vice Chancellor, University of Kashmir addressing the audience

Prof. Chandrima Shaha received 18th **Prof. S. P. Ray-Chaudhuri Lecture Award** for Yr. 2020

Prof. Umesh Varshney received XI Prof. J Das Memorial Lecture Award for Yr. 2021

Dr. Md. Imtaiyaz Hassan received 3rd Prof. Rita Mulherkar Lecture Award for Yr. 2020

Faculties interacting with students during poster session

Dr A. J. Rachel giving her remarks on behalf of audience during valedictory session

Prof. Akbar Masood, Vice Chancellor, Baha Ghulam Shah Badshah University, Rajouri, the chief guest of the valedictory session addressing the audience.

Student receiving the award certificate from Dr Shaida Andrabi and Prof. Zafar A. Reshi

Student awardees with faculty members after receiving awards during valedictory session

Photothermal Cell Death: Intricate coordination among unfolded protein response pathway and autophagy signalling in breast cancer cells

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Apart from conventional radiation and chemotherapy, cancer nanomedicine has been grooming an attractive and promising approach for cancer treatment. However, in-depth analysis of the molecular mechanisms involved in the post-treatment perturbation of the cell involved, inducing cytotoxic effects is largely unexplored and thus hinder its clinical translation. Recently, gold nanostructures mediated, near infrared (NIR) light triggered heat generation (Photothermal therapy -PTT) for localized thermolysis of cancer cells has emerged as one such credible approach for cancer ablation [1-2]. This has entered phase-II clinical trials within a decade of its inception. In our group ongoing work on plasmonic photothermal therapy using gold nanoshell with a polymeric core (AuPLGA) has shown dramatic efficacy in terms of tumor ablation in mouse model [3-4]. However, a detailed mechanistic insight on photothermal cell death remains elusive. So, we also aim for identifying the perturbation in the biological pathways post-PTT, in order to develop a knowledge database that will further allow us to tweak the therapy towards low dose making the therapy more appealing for its translation from bench-to-bedside.

Cells harbour complex signalling network which in response to external trigger coordinate to mediate either cell survival or cell death. Unfolded protein response (UPR) pathway and autophagy are two such pathways which go hand-in-hand to induce either cell homeostasis or death depending on the extent of cellular stress [5-6]. Our study for the first time using

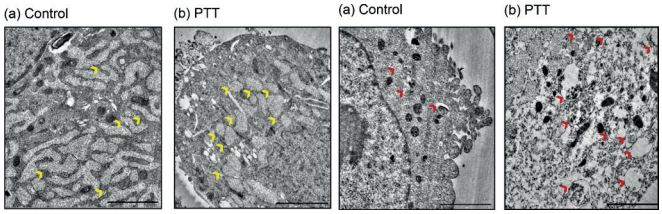

Fig .1. A. TEM analysis for the presence of stressed endoplasmic reticulum (yellow arrows). (a) Control cells showing normal endoplasmic reticulum (b) PTT treated cells showing presence of swollen endoplasmic reticulum. B. TEM analysis for the presence of autophagic bodies (red arrows). (a) Control cell showing basal level of autophagy; few autophagic bodies (b) PTT treated cells showing significant ($p < 0.001$) increase in the number of autophagic bodies per cell.

classical transmission electron microscope (TEM) analysis reveals that upon PTT treatment the presence of swollen endoplasmic reticulum within the cells due to obvious protein denaturation and unfolded protein accumulation, causing endoplasmic reticulum stress (ER stress) and induction of the UPR pathway (Fig.1.A). A significant decrease in the level of UPR chaperone (BiP) and enhanced expression of UPR death mediators (CHOP and Ero1Lα) within PTT treated cells, implicates that the homeostasis is shifted more towards cell death rather than cell survival.

Moreover, induction of UPR also leads to excessive reactive oxygen species (ROS) generation post-PTT. Additionally, a dramatic increase in the number of autophagic bodies/cell was also revealed in TEM analysis showing significant induction of autophagy ($p < 0.001$) post photothermal treatment. Enhanced LC3 I to LC 3 II protein conversion coupled with efficient p62 protein degradation further illustrate the occurrence of appropriate autophagic flux and a state of metabolic crisis in PTT treated cells (Fig.1.B) Further, a drastic increase in the level of activated JNK (a connecting link to UPR and ROS mediated autophagy) and the co-existence of swollen ER and autophagic bodies within the same cell was also observed (Fig 2).

These results clearly suggest that simultaneous induction of both these pathways is crucial for executing cell death via PTT. Our findings provide evidence for how cellular signals can intricately associate in response to cytotoxic trigger to mediate cell death. This paves way for combining the use of inducers or inhibitors of critical cellular pathways along with PTT providing a window

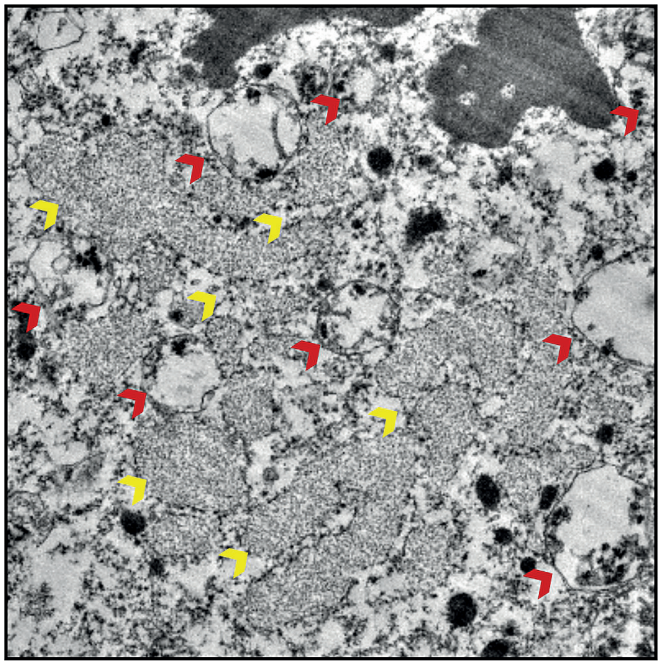

Fig. 2. TEM analysis showing the coexistence of stressed ER (yellow arrows) and autophagic bodies (red arrows) within the same cell.

to operate the therapy even at sub-optimal dose to achieve similar efficacy; further minimizing the therapy side effects if any.

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Drosophila Malpighian tubules: Resilience is thy name!

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The name Malpighi immediately brings into mind several structures, from red blood cells, liver, spleen to plants, and all are credited to Marcellio Malpighi, better known as father of microscopial anatomy, in animals as well as plants. The branch of science that deals with the structure of tissues and paved way to the branch of Anatomy are major contributions from the Marcello Malpighi. In modern day, many glands and tissues, such as the Malpighian bodies of the spleen, the Malpighian corpuscles and pyramids in kidneys are named after him. Marcello Malpighi is also credited to have done precise sketches, a dwindling art, of the observations made by him under the microscope, which have been archived and first published as *Opera Omnia*, his *botanico-medico-anatomicus* in 1687.

The Malpighian tubules of insects, also take their name after Marcello Malpighi (Fig 1), and are essential organs for insects as they maintain the ionic and water balance and are equivalent to human kidneys. *Drosophila* Malpighian tubules comprises of two pairs, the anterior and the posterior and two main cell types, the principal cells and the stellate cells, to carry out the preliminary function of fluid secretion (Sozen et al., 1997). There are multipotent stem cells also, which can differentiate into any of the two cell types in this organ (Singh and Hou, 2009).

Drosophila, being a holometabolous insect passes through pupation, a sedentary stage, bringing an end to the larval life and beginning of the adult stage. It is at pupation that reconstruction of the different organs using imaginal discs and imaginal cells takes place to give the final shape of adult fly. The fate of most of the larval tissues is histolysis by programmed cell death (Jiang et al., 1997; Lee et al., 2000). The interest in Malpighian tubules was ignited when my attention was caught by a non-descriptive statement in the volumes of *Drosophila* development books, describing them as organs that do not undergo histolysis during *Drosophila* development. This stuck a chord in the mind and instantaneously the question clicked as to **HOW** this happens and then next thing was **WHY** this privilege. These aspects of Malpighian tubules were never probed and therefore it proved to be a very fertile ground to tread upon.

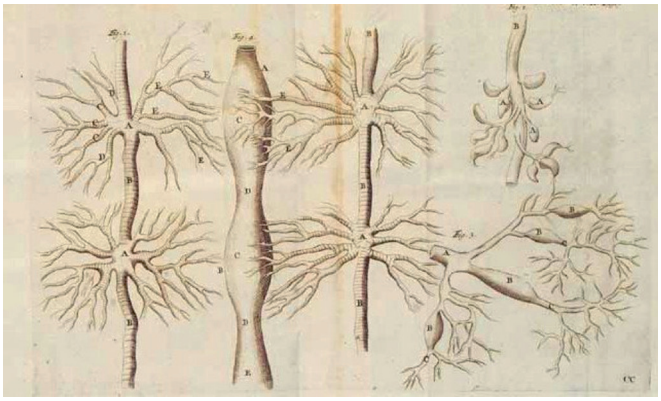

Figure 1. Image from *Opera Omnia* which is on display in two volumes at *Wolfenbüttel's Herzog August Bibliothek*.

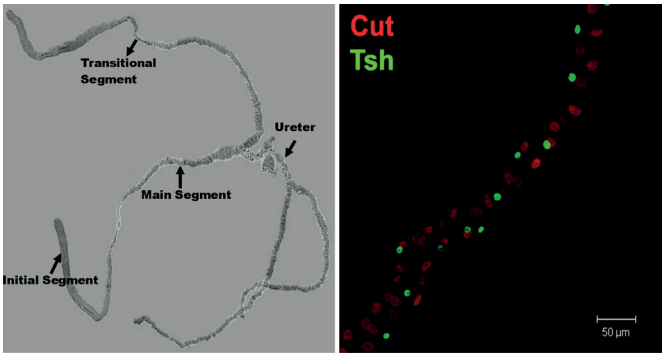

Figure 2. Transition segment and initial segment (Left image). MTs immunostained with cut (red), marker of principal cells and Tsh (Green), marker of stellate cells, showing stellate cells are interspersed with principal cells (Right image).

Apoptotic immunity of these organs was definitely thought provoking and we started to identify the **HOW** part of the question. What was in store was not expected as it was observed that these organs expressed the entire repertoire of apoptotic genes (Tapadia and Gautam, 2011; Ojha and Tapadia, 2019) and still do not undergo cell death, hence one could not fathom why they were not dying. All the more intriguing was the fact that on overexpression of pro-apoptotic genes, *repear, hid* and *grim*, the size and the number of cells were affected along with defects in fluid secretion (Tapadia and Gautam, 2011). Transcriptomic analysis revealed that the primary interplay between ecdysone induced Ecdysone Oxidase, E93 and Forkhead (Ojha and Tapadia, 2019) were some of the key players in the survival of Malpighian tubules. Ectopic expression of *E93* in Malpighian tubules resulted in onset of cell death, but it was because of autophagy and not apoptosis (Ojha and Tapadia, 2019). Further analysis into the role

of caspases revealed that they are important for the proper morphogenesis as well as function of the stellate cells and their absence causes gross morphological defects (manuscript submitted). The identification of non-lethal role of caspases in Malpighian tubules has opened newer avenues for research.

Having partly answered the How part, we started looking for answers as to **WHY** they escape histolysis. Apart from the primary function of osmoregulation, Malpighian tubules also serve the function of providing protection from pathogeneic challenges by production of anti-microbial peptides (AMPs) as the first line of defense (Mcgettingan, 2005; Verma and Tapadia. 2012). So is it possible that the survival of Malpighian tubules has something to do with immune protection? We have a fairly good proof that the reason for these tubules to continue even during pupation is that their role in immunity and particularly during pupation when the fat bodies, which are the immune organs are disintegrated. Two unrelated instances support this hypothesis, first the innate immune response is developmentally regulated and under the control of steroid hormone, ecdysone, and even at high ecdysone titers during pupation the expression of AMPs does not cease (Verma and Tapadia, 2012), unlike *Bombyx mori* (Tian et al., 2010), and second that they exhibit high Forkhead (survival factor) levels during pupation which also regulates immune response (manuscript under preparation). Further analysis may reveal the interplay between the innate immune response and absence of apoptosis.

Deeper insight into the molecular mechanism of caspase regulation may have far reaching consequences in understanding the pathogenesis of many diseases including cancer along with evolutionary importance of protection of certain structures during development. Thus it is important not to discard any information as trivial, because it can be the beginning of a new science. The inconsequential observation about Malpighian tubules proved to be a blessing in disguise.

P.S This is an account of personal satisfaction and the excitement that was experienced in the short journey of science!

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An 'escaping route' for human cells exposed to metal oxide nanoparticles

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Nanotechnology is an exponentially growing field and nanoparticles (NPs having dimensions less than 100nm) with their unique physical and chemical properties are increasingly landing wide range of applications; such as, fields including biomedical, electronic, industrial, cosmetic, paints, food additives etc. Metal oxides particularly, are being developed for diagnostics, electronic sensing and computing. They are the largest class of commercially produced nanomaterials. The enormous flexibility of functionalization and fabrication renders an ever-growing number of tailored applications feasible such as targeted gene therapy and improved bioavailability of pharmaceutical drugs. Enhanced contrast and sensitivity in imaging has also added to the explosion of global demand for nano-scale ingenuity resulting in the ever-increasing use of nano-scale metal oxide particles. As such human exposure becomes inevitable. With the unprecedented human exposure of nanoparticles, risks to human health also increase owing to the high penetrating potential and high reactivity of nanoparticles. High presence of metal oxide nanoparticles at sites surrounding factories as compared to clean areas has been correlated with increase in pulmonary diseases including exacerbation of bronchial asthma. Pulmonary toxicity can manifest into emphysema, edema, fibrosis and oxidant injury often involving cells located in the alveolar septa.

Therefore, in my laboratory we have been working on studying the effect of metal oxide nanoparticles on human alveolar cells (A549). From our recent research work, we have discovered that similar dose-time exposure to TiO_2 NPs confers less lethality as compared to ZnO NP exposure to A549 cells. It is already well established that triggers for migration activate crucial cell surface receptors and induce morphological changes. It is executed by a polarized cell morphology that enables protrusion over a trailing end. Potency for integrin associated attachment to basal lamina is also vital. Together the contraction and release of cytoskeletal structures enable cell movement. Cell movement is ordained by a series of signal transduction pathways that include small GTPases, cytoskeleton-modifying proteins, kinases, lipid second messengers and motor proteins. Cells achieve movement when different signaling cascades are consistently presented in specific locations within the cell while maintaining potency of response to extra cellular triggers. Both epithelial and mesenchymal cells can migrate, although what external cues trigger specific cellular changes to channel directional movement is still under considerable research. However, mesenchymal phenotype has increased migratory and invasive capabilities, combined with a greater resistance to cell death. Epithelial to mesenchymal transition (EMT) thus greatly enables migration and invasiveness, though migration alone does not necessitate EMT. Every toxicological model presents a zone of stress, where in the cell survival is more sensitive than in other zones. Thus, a greater potential for migration such as alteration in surrounding environment or by trans differential processes such as epithelial to mesenchymal transition may enable cells in moving away from this zone of stress, thereby enabling greater tolerance to stress and thus enhanced survival. This phenomenon allows cells to float away from the lamina into a region more conducive for survival. Any number of quorum sensing signals such as dissemination of chemokines and cytokines from the stress affected cells on the lamina may also affect the migrated cells to a lesser degree simply because of a lack of ample access. This hypothesis is especially true with a monolayer tissue system. In our recent work we have demonstrated that epithelial to mesenchymal transition and an increased duration of phosphorylation of eIF2α are crucial mechanisms routing better tolerance to TiO_2 NP treatment over exposure to ZnO. The increased migratory capacity may help cells escape away from the zone of stress. These novel findings could be successfully developed in the future to design relief strategies to alleviate metal oxide nanoparticle mediated stress.

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Cytology to Cellonomics: A Brief History of Trending Times

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Cytology was historically born as a branch of biology dealing with cellular structures using microscope as the primary tool. There is no historical evidence to authenticate who should be credited for coining 'Cytology' or 'Cell Biology' although it was Robert Hooke who introduced the term 'Cell' to biology. It has been a long journey of research and development spanning over four centuries which has transformed cell biology into the present-day multidisciplinary science. Linguistically, the two neologisms in biology, namely 'cytology' and 'cell biology' mean the same, although there exists a contextual difference. Researchers realized the functional complexity of a cell rather than treating it merely as a structural entity. The gradual transformation and acceptance of 'cell biology' as the jargon replacing 'cytology' was part of the historical development of the discipline, which eventually broadened its scope. Cytology thus evolved to cell biology from a 'specialized' to a 'generalized' multidisciplinary science of biology.

The twentieth century witnessed the phenomenal growth of light and electron microscopy in terms of magnification and resolution. Today, subcellular components can be visualized in nanoscale and beyond. For instance, confocal microscopy can produce optical slices to generate a reconstituted three-dimensional profile of a cell. Advanced fluorescence-based detection technology, live imaging techniques, super resolution optical devices aided with advanced computational tools can decipher molecular landscapes of a cell! The last century also witnessed the birth of several sub-disciplines within cell biology to address complex questions such as gene regulatory networks, signalling cascades and so on.

In the post-genomic era, there have been conflicts between the two schools of 'data-driven' versus 'hypothesis-driven' science. The 'omics' technology was a filler to bridge this gap between the two. Despite the advances in high throughput omics technology *viz.* genomics, transcriptomics, proteomics and metabolomics, many well cited studies remained as isolated biological layers. However, there were not many efforts to explore the interconnections. Moreover, the so-called omics mind-set did not percolate much within the broader community of cell biologists. The inertia towards omics was rather transient and in the course of time, the omics technology turned out to be a necessity in cell biology mainly for sample analysis. The huge information gathered during the pre-genomic era threw several challenges for holistic realization of cellular functions and contextualization of biological questions. It was soon realized that the single-level omics would not be able to contribute meaningfully to resolve links between molecular signatures and biological questions. Thus, a multi-omics perspective happened to be the next option and one could notice gradual shift of focus from single-omics to multi-omics approaches in cell biology research *per se*. Initially, many felt optimistic to integrate different omics frameworks for interrogating complex biological phenomena at the cellular level. Such a shift towards multi-omics rapidly captured the attention of many laboratories with a slogan '*when the whole is more than the sum of its parts'*! However, the initial hype of multi-omics did not last long and during the last decade itself non-complementarity of multi-omics data with hypothesis-driven research became a concern amongst cell and system biologists. This was mainly attributed to the diversity in cellular components. The answer to solve this inherent heterogeneity and cell-to-cell variation was only offered by the newly developed single-cell omics technology. At the same time, additional challenges continued to emerge for integrating various omics datasets at the level of single cell which ultimately led us to 'Cellonomics', often referred to as 'single-cell omics'. This was precisely the turning point of cell biology research noticeable in the recent times. If one looks at the evolution of cell biology, the genesis of cellonomics has been the obvious fate.

The story does not end here! Future challenges will include management of huge sources of data generated through single-cell omics, leading us to the premises of 'Bigdata'. Therefore, the community of future cell biologists needs to be equipped for data cleaning, normalization and contextualization. The long journey of 'cytology'-to-'cell biology'- to -'cellonomics' inevitably needs to fulfil the 3i's- integration, interpretation and insights. Only then the big data generated through single-cell omics will turn into a meaningful knowledge for fundamental as well as translational research.

Studying human diseases in tiny worms: How relevant are the findings?

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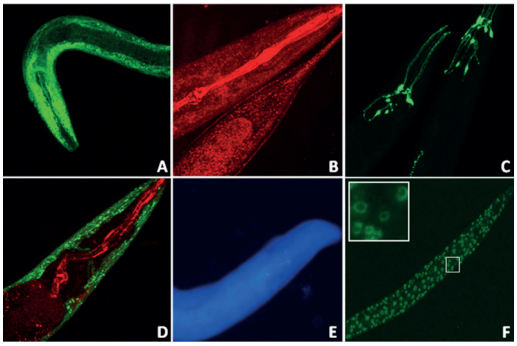
Devising an effective cure for any human disease requires fundamental understanding of the disease process and the associated functions that it impacts. Obtaining such understanding requires use of efficient model systems because of inherent methodological limitations that prevent having such understanding from humans directly. Models as simple as single cell organisms such as yeast and as complex as primates, are employed in research studies aimed at identifying novel ways to cure diseases. Rodents are the most widely used models for multiple reasons including genetic relevance, technical feasibility and ease of studying various critical endpoints. However, non-rodent models such as the soil nematode *Caenorhabditis elegans* (*C. elegans*), fruit fly *Drosophila melanogaster*, zebra fish *Daniorerio* and yeast *Saccharomyces cerevisiae* are considered to be precious models as they possess significant homology of gene sequences with higher mammalians, are easy to rear within laboratory conditions and can easily be genetically manipulated towards addressing scientific questions. Of late, these models have been utilized towards generating a wealth of data from pharmacological, genetics, proteomics, behavioral, developmental and toxicological studies, thus raising a question about the relevance of such data in the context of human functioning. The diversity in phenotype, functioning and even longevity is such that questions arise about how genetic or pharmacologic target from one small organism like a worm or a fly, could act in a similar fashion within higher models, more specifically within the human beings.

The success stories: Researchers working in the field of model organism biology, draw inspiration from many success stories that have proved the utility of genetic conservation and uniformity of many key functions across taxa. We know that the idea of genome sequencing used to appear as a humongous task, but *C. elegans* was the first multi-cellular organism to have its genome sequenced (*C. elegans* sequencing consortium, 1998) which encouraged and paved way for multiple other sequencing projects that were ultimately concluded with success (Nurk et al, 2022). Talking about human diseases, there was a time, when nothing was known about genetic mechanism of Alzheimer's disease (AD) in humans; research employing *C. elegans* identified a gene called *sel-12* that was exhibiting association with amyloid beta plaque formation- a hallmark of AD and this modle has been aiding immensely in understanding of human disease related genes (Apfeld and Alper, 2019). Subsequent research proved that the human orthologue of *sel-12*, called Presenilin-1 (PS-1) was acting similarly in humans. It was not merely the homology of gene sequence, complementation studies wherein *C. elegans* sel-12 was mutated and replaced with human PS-1, restored the functions of lost *sel-12*, thus revealing conservation of function, which means a lot in terms of employing smaller model systems for understanding human processes (Levitan et al 1996; Wittenburg et al 2000). Similarly, the genes involved in the process of apoptosis were first of all identified

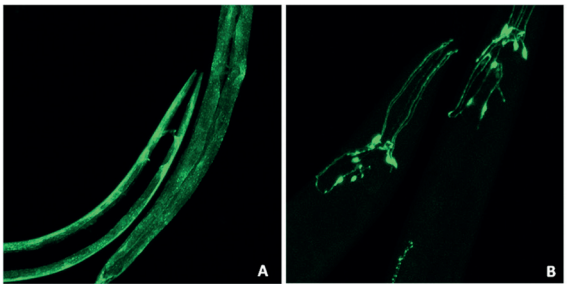


Transgenic *C. elegans* expressing various reporter genes/fluorophores: *C. elegans* expressing "human" alpha synuclein tagged with YEP (A), *C. elegans* stained with lipid specific dye Nile Red (B), Transgenic *C. elegans* strain expressing GFP in subset of neurons (C), *C. elegans* expressing YEP stained with Mirotracker Red (D), *C. elegans* stained with Thioflavin S (E), Transgenic *C. elegans* expressing FOXO Transcription factor Daf-16 tagged with GFP, Specific Cytoplasmic presence of the protein is highlighted in the box (F).

in *C. elegans* and later validated in higher mammalian systems as well (Ellis and Horvitz, 1986). Not only was each member of apoptosis machinery found to function similarly in humans but the entire process was replicated in humans. The knowledge of the process of apoptosis later brought breakthrough changes to the way critical human diseases as cancers were treated.

The experimental advantages: Model organisms not only offer advantages of genetic suitability, but they also provide with systems for technical and experimental feasibility. In particular, their shorter life spans allow us to study their entire life-span or even enable in carrying out multi-generation studies. Such experimental ease provides with an opportunity of carrying out large scale genetic or pharmacologic screens towards excluding huge numbers of irrelevant and functionally

unrelated genes/pharmacological agents; this way high throughput screening proves to be a tremendously useful advantage of such model systems. The genetic amenability also allows for tagging proteins of interest with fluorescent tags/ reporter genes, which enables studies on expression of proteins.



Transgenic *C. elegans* expressing Green Fluorescent Protein (GFP) within muscles (A) and in cholinergic neurons (B)

The whole organismal environment: While studying potential pharmacological agents for the effect on humans, researchers employ simpler tools like *in vitro* cell based systems or mammalian systems as rodents. A major limitation with *in vitro* systems is the lack of "whole organismal environment" thus missing "metabolism" as the potential drug would encounter if recommended for human use. The disadvantage of carrying our certain studies in mammalian systems is the complexity of the models and related functions. Small models like *C. elegans* offer immense advantages as these nematodes offer a whole organismal environment, with intact metabolism and yet presenting with simple anatomy which can be handled with ease. The metabolic outcome is so relevant as the Cytochrome P450 genes of *C. elegans* possess significant homology with their human counterparts. In addition to the genetic and functional similarity, the *C. elegans* model provides technical ease of carrying out multiple biological studies; e.g. in worms, RNAi induced gene silencing can be carried out via simple feeding of worms with genetically engineered bacteria. An RNAi library of approximately 17,000 bacterial clones is available, which aids in carrying out whole-genome or systematic RNAi screens with high efficiency and sensitivity (Kamath et al, 2003). Employing the RNAi induced gene silencing methods, multiple novel molecules and functional aspects relevant to human diseases have been characterized (Haque and Nazir, 2016; Sarkar et al, 2022). The transgenic *C. elegans* models expressing "human" protein have also been employed for screening of potential drug molecules against neurodegenerative and other diseases (Sashidhara et al, 2014).

Limitations: There are certain limitations to use of *C. elegans* in studying human functions; e.g. the lack of fully developed organs and organ systems keeps worms devoid of certain critical functions which can't be studied or compared with humans. For some disease conditions, the low homology of related disease genes poses a challenge and hence cannot be studied with high confidence. However, the advantages far outnumber the limitations as *C. elegans* offers an excellent screening tool thus eliminating need of testing thousands of chemicals or genes in complex mammalian systems. It efficiently helps in reducing the junk and carrying out focused validation studies with the class of chemicals/ genes having likelihood of tending a positive outcome. This reduces the burden of time as well as expenditure while also providing mechanistic cues to key functional outcomes.

Conclusion: We may conclude that, non-mammalian model systems like *C. elegans*, *Drosophila*, yeast and zebra fish offer immense advantages for being employed in studies related to human diseases and related mechanistic aspects. The results and success stories that we have obtained so far encourage further use with confidence as the findings from these lower models are relevant to humans. Evidence exists that these models offer advantages of time efficient, cost efficient and reliable outcome valuable for advanced translation into effective therapies against diseases that burden humankind.

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E3 ligases: The double edge sword in host-pathogen interaction

Varsha Kumari and Niti Kumar

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A wide variety of post translational modifications (PTMs) such as phosphorylation, methylation, acetylation and ubiquitination, play important role in cellular decisions of proliferation and senescence. These PTMs affect the protein stability, protein-protein interactions and localization; thereby regulate cellular processes like protein degradation, DNA repair, transcription, translation, organellar biogenesis and autophagy. Emerging evidence suggests that changes in PTMs landscape can give insights into the cellular homeostasis, disease pathophysiology and infection progression. One of the most abundant post translational modifications is ubiquitination (Ub) which involve modification of ε-amino group of a lysine residue of protein substrate through covalent attachment of ubiquitin (76 amino acid) monomer or multi-ubiquitin molecules(1,2). Protein modification by Ub and Ub-like modifiers is regulated by 3 different enzymes namely; E1 activating enzyme, E2 conjugating enzyme, E3 ubiquitin ligase. Cascade of E1 activating enzyme, E2 conjugating enzymes and E3 ligases helps in attachment of Ub and Ub-like modifiers (SUMO, NEDD8, ATG8, ATG12 etc.) to their substrate proteins. Although, E1 and E2s are quite conserved, E3 ligases are highly diverged with substrate interacting and regulatory domain. Deregulation of any of the components of ubiquitination pathway is associated with many diseases like cancer, neurodegenerative, autoimmune disease etc. E3 ligases have been classified into three different families on the basis of how they transfer Ub from E2 to substrates. RING ((Really interesting new genes), HECT (Homologous to E6-AP C-terminus) and RBR (RING-between-RING) (Figure 1). RING E3 ligases are the largest family with more than 600 in numbers in humans. These ligases work in conjunction with adaptor and scaffolding proteins (cullin RING ligase family) to transfer ubiquitin to substrates. While, 30 proteins have been identified as HECT E3 ligases. In contrast, RBR E3 ligases comprise of 14 proteins, of which PARKIN is one of the comprehensively studied E3 ligase and has been associated with Parkinson disease(2,3). Amongst different components of ubiquitination machinery, proteasome, E3 ligase and deubiquintinating enzymes are being explored as pharmacological targets for pathophysiology associated with cancer, autoimmune disorders and neurodegeneration (3).

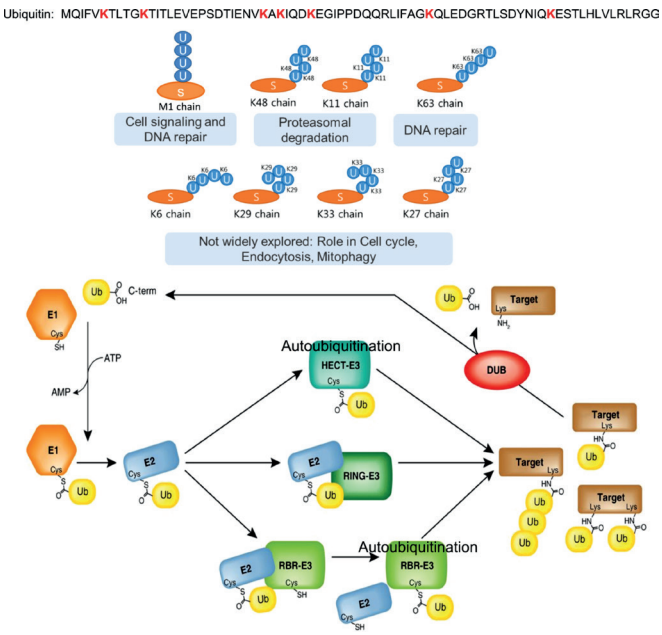

Figure 1: Schematic representation of types of ubiquitination linkages in the substrates mediated by cascade E1 activating enzyme, E2 conjugating enzymes and E3 ligases. (Adapted from Park et al, 2014, BMB Rep. and Smit et al, 2014, EMBO Rep.).

Human ubiquitination machinery is also exploited by pathogensfor their replication and persistence of infection. For instance, bacterial pathogens secrete E3-like effector proteins which specifically hijack host's Ub machinery and perturb the host immune signaling (4).E3-like proteins from pathogen have been broadly divided into three different classes:RING, HECT and NEL (Novel E3 ligases). For example, during *Salmonella* infection, the bacteria secretesSopAprotein which is a HECT-like E3 into the human host and affects inflammatory response in small intestine.Novel E3 ligases identified in*Shigella* is shown to degrade the

NF-κB essential modulator, thereby affecting the NF-κB mediated signaling(4). Recent reports have shown that structural proteins (spike and membrane) of SARS-CoV-2 virus undergoes ubiquitination by host's RING and HECT E3 ligase which affects virion maturation and release(5,6). It is also proposed that selective targeting of host's ubiquitin machinery can inhibit viral egress and can be useful in viral infection management.

In other pathogens, like protozoan parasites, ubiquitination machineryis suggested to finely-regulate their cellular processes for proliferation, differentiation and immune evasion.An interesting example, is human malaria parasite which has a digenetic lifecycle, wherein it survives in different cellular niches inevolutionary distinct hosts (human and mosquito). Another striking feature is that this parasite maintains its metastable aggregation-prone proteome in functional state through ubiquitin-linkages (7). The interest in E3 ligases in parasite was sparked with genome-wide association studies, hinting towards association of artemisinin resistance with mutation in Kelch protein which is an adaptor protein of cullin E3 ligase in human malaria parasite(8). Recent literature has shown human E3 ligases such as MARCH and RNF123 influence the parasite survival during the erythrocytic stage(9,10). However, how E3 ligases affect the parasite survival in the liver stage remains largely unexplored. Even the plasmodial E3 ligases in the erythrocytic stage are not comprehensively explored due to experimental challenges associated with protein expression and purification of plasmodial proteins, biochemical and structural characterization. Few literature reports on plasmodial E3 ligases are based on gene deletions, microscopy experiments and genome-wide association of mapping of resistance. These plasmodial E3 ligases have been suggested to influence the survival, virulence and stage-specific transitions. Our group is trying to understand the role of RBR-E3 ligase which has diverged significantly from its human othrolog. It has a N-terminal canonical RING1 domain which interacts with E2-conjugating enzyme and a non-canonical RING-2 domain which possesses catalytic cysteine for ubiquitination reaction. Our experiment shows that Pf RBR-E3 ligase catalyzes K-6, K-11, K-48 and K-63 mediated ubiquitination and these post-translational modifications may play important role in different biological pathways (such as DNA repair, proteasomal degradation, mitophagy).Through mutational analysis, critical residues in RING1 and RING2 domain which affect its autoubiquitination activity have been identified. Ongoingexperiments are to identify interacting partners and substrates of RBR-E3 ligase to gain insights into probable alternative intervention sites in the malaria parasite.

With limited literature reports across various infection models, the current understanding on how the ubiquitination machinery influences the host-pathogen interaction is still at nascent stage. Additional work is required to gain insights into the double-edged sword role of this machinery in shaping the host-pathogen interface.

Niti Kumar's lab is trying to understand the genome and proteome maintenance pathways in human malaria parasite. The lab is also involved in open-source drug discovery program for identification of hits/lead for drug-resistant malaria.

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A hands-on workshop on "Use of Quantitative PCR in Biomedical Research and Diagnostics" was organized by Dr. Anirudh Singh, Department of Microbiology, All India Institute of Medical Sciences, Bhopal from April 18-20, 2022. The financial support was provided by the Indian Society of Cell Biology.

A total of 14 candidates from different parts of the country participated in the event and included students of MD Microbiology, PhD and M Pharm along with faculties and scientists from several universities and research institutes. The workshop included lectures from faculties from Maulana Azad National Institute of Technology, Bhopal, Bihar Animal Science University, Patna, Agilent Technologies and All India Institute of Medical Sciences, Bhopal. To help facilitate positive learning experiment all the participants were provided with a workshop manual and allowed to perform the experiments themselves. They learned molecular biology techniques likes; 1. Primer designing 2. Nucleic acid isolation and quantification 3. cDNA synthesis 4. Primer efficiency calculation 5. Absolute and relative quantification of nucleic acid using standard curve and 2-ΔΔCt methods 6. Diagnosis of COVID-19 using RT-qPCR.

Other than the participants, trainees and students from the department of Microbiology also actively took part in the workshop and were greatly benefited from it. One of the key highlights of the workshop was the participation of our PhD students as the instructors for the hands-on experiments. Participants were impressed with their dedication and enthusiasm for the workshop.

Unveiling of the manual for the Hands-on Workshop on Use of Quantitative PCR in Biomedical Research and Diagnostics, April 18-20, 2022 held at the Department of Microbiology, All India Institute of Medical Sciences, Bhopal

A lecture on biosafety practices in laboratory during the Hands-on Workshop on Use of Quantitative PCR in Biomedical Research and Diagnostics, April 18-20, 2022 held at the Department of Microbiology, All India Institute of Medical Sciences, Bhopal

Delegates and Participants of the Hands-on Workshop on Use of Quantitative PCR in Biomedical Research and Diagnostics, April 18-20, 2022 held at the Department of Microbiology, All India Institute of Medical Sciences, Bhopal

Participants of the Hands-on Workshop on Use of Quantitative PCR in Biomedical Research and Diagnostics, April 18-20, 2022 held at the Department of Microbiology, All India Institute of Medical Sciences, Bhopal

A session on analysis and interpretation of quantitative PCR data during the Hands-on Workshop on Use of Quantitative PCR in Biomedical Research and Diagnostics, April 18-20, 2022 held at the Department of Microbiology, All India Institute of Medical Sciences, Bhopal

Participants setting up qPCR reactions during the Hands-on Workshop on Use of Quantitative PCR in Biomedical Research and Diagnostics, April 18-20, 2022 held at the Department of Microbiology, All India Institute of Medical Sciences, Bhopal

MOHIT K. SAIGAL & CO
CHARTERED ACCOUNTANTS

B-37/122, SAIGAL HOUSE
MAHMOORGANJ, VARANASI
UTTAR PRADESH-221010
PH.0542-2360015, M.No.7275660055

INDIAN SOCIETY OF CELL BIOLOGY

BALANCE SHEET AS ON 31 MARCH,2021

LIABILITIES	AMOUNT	AMOUNT	ASSETS	AMOUNT	AMOUNT
CAPITAL FUND ACCOUNT:			**FIXED ASSETS**		**22,939.00**
Opening Balance	4,854,144.65				
			INVESTMENTS		**5,284,962.17**
Less: TDS	63,924.00				
Add: Income Tax Refund	87,711.00				
Add: Excess of Income over			**CURRENT ASSETS & LOANS &**		
Expenditure	396,748.00	**5,274,679.65**	**ADVANCES :**		
			TDS ON FDR FY 2020-21	27,773.00	
			XXXVIII AICB Conference	113,159.00	
			Accrued Income	187.00	**141,119.00**
LIFE MEMBERSHIP FEES:					
Opening Balance	1,157,783.00				
Add: during the year	-	**1,157,783.00**			
PROVISION:			**CASH & BANK BALANCES:**		
Audit Fees Payable	35,000.00		SBI, Lucknow	137,121.06	
Legal Fees Payable	10,000.00	**45,000.00**	SBI, Varanasi	891321.42	**1,028,442.48**
TOTAL		**6,477,462.65**	TOTAL		**6,477,462.65**

For INDIAN SOCIETY OF CELL BIOLOGY

Sd/-
MADHU GWALDAS TAPADIA
(SECRETARY)

PLACE : VARANASI
DATE : 01.01.2022

As Per Record Produced Before us
For *MOHIT K. SAIGAL AND CO.*
(CHARTERED ACCOUNTANTS)
FRN: 01553C

MANYA SAIGAL
(PARTNER)
M.NO. 421730

MOHIT K. SAIGAL & CO. VARANASI CHARTERED ACCOUNTANTS

MOHIT K. SAIGAL & CO
CHARTERED ACCOUNTANTS

B-37/122, SAIGAL HOUSE
MAHMOORGANJ, VARANASI
UTTAR PRADESH-221010
PH.0542-2360015, M.No.7275660055

INDIAN SOCIETY OF CELL BIOLOGY

Receipts & Payment A/c for the period 01.04.2020 to 31.03.2021

RECEIPTS		AMOUNT	PAYMENT		AMOUNT
To **Opening Balances:**			By TDS FY 2020-21	27,773.00	**27,773.00**
SBI, Lucknow	441,897.42				
SBI, Varanasi	115,046.06	**556,943.48**			
To Membership Fees	18,650.00	**18,650.00**			
To Interest From HDFC	369,690.00				
To Interest on SB	17,082.00				
To Interest on IT Refund	6,139.00				
To Income Tax Refund	87,711.00	**480,622.00**			
			By **Closing Balances:**		
			SBI, Lucknow	137,121.06	
			SBI, Varanasi	891,321.42	**1,028,442.48**
Total		**1,056,215.48**	Total		**1,056,215.48**

For INDIAN SOCIETY OF CELL BIOLOGY

Sd/-
MADHU GWALDAS TAPADIA
(SECRETARY)

PLACE : VARANASI
DATE : 01.01.2022

As Per Record Produced Before us
For MOHIT K. SAIGAL AND CO.
(CHARTERED ACOUNTANTS)
FRN: 01553C

MANYA SAIGAL
(PARTNER)
M.NO. 421730

MOHIT K. SAIGAL & Co. VARANASI CHARTERED ACCOUNTANTS

MOHIT K. SAIGAL & CO
CHARTERED ACCOUNTANTS

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UTTAR PRADESH-221010
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INDIAN SOCIETY OF CELL BIOLOGY

INCOME AND EXPENDITURE A/c FOR THE PERIOD 01-04-2020 TO 31-03-2021

EXPENDITURE	AMOUNT	INCOME	AMOUNT
To Audit Fees	10,000.00	By Interest From HDFC	369,690.00
To Legal Fees	5,000.00	By Interest on SB	17,269.00
		By Interest on IT Refund	6,139.00
		By Membership Fees	18,650.00
By Excess of Income over Expenditure	**396,748.00**		
Total	**411,748.00**	**Total**	**411,748.00**

For INDIAN SOCIETY OF CELL BIOLOGY

Sd/-
MADHU GWALDAS TAPADIA
(SECRETARY)

As Per Record Produced Before us
For MOHIT K SAIGAL & CO.
(CHARTERED ACCOUNTANTS)
FRN: 01553C

MANYA SAIGAL
(PARTNER)
M.NO. 421730

MOHIT K. SAIGAL & CO. VARANASI CHARTERED ACCOUNTANTS

PLACE : VARANASI
DATE : 01.01.2022

MOHIT K.
SAIGAL & CO.
CHARTERED ACCOUNTANTS

B-37/122,SAIGAL HOUSE
MAHMOORGANJ,VARANASI
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PH.NO.-2360015 MO.NO.-7275660055

INDIAN SICIETY OF CELL BIOLOGY
BALANCE SHEET AS ON 31ST MARCH 2022

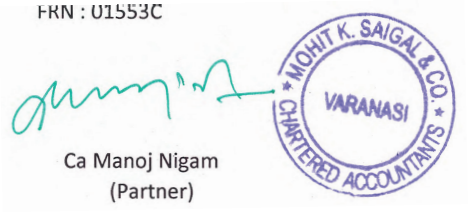
LIABILITIES	AMOUNT	AMOUNT	ASSETS	AMOUNT	AMOUNT
CAPITAL FUND ACCOUNT :-			**FIXED ASSETS**		22,939.00
Opening Balance	5,274,679.65				
Less :-TDS			**INVESTMENT**		
Add : Income Tax Refund			FDR		6,084,962.17
Add :- Excess of Income over Expenditure	313,875.00	5,588,554.65			-
			CURRENT ASSETS & LOANS & ADVANCES:		
LIFE MEMBERSHIP FEES:			TDS ON FDR FY 2020-21	27,773.00	
Opening Balance	1,157,783.00		XXXVIII AICB Conference	113,159.00	
Add: during the year	15,250.00	1,173,033.00	Accrued Income	187.00	141,119.00
PROVISION:			**CASH & BANK BALANCE:**		
Audit Fees Payble	40,000.00		SBI, Lucknow	156,837.06	
Legal Fees Payble	11,000.00	51,000.00	SBI, Varanasi	406,730.42	563,567.48
TOTAL		**6,812,587.65**	**TOTAL**		**6,812,587.65**

For Indian Society of cell biology

Madhu Gwaldas Tapadia
(Secretary)

Place :- Varanasi
Date :- 31-08-2022

As per record produced before us
For Mohit K Saigal & Co.
(Chartered Accountants)
FRN : 01553C



Ca Manoj Nigam
(Partner)

MOHIT K.
SAIGAL & CO.
CHARTERED ACCOUNTANTS

B-37/122,SAIGAL HOUSE
MAHMOORGANJ,VARANASI
UTTAR PRADESH-221010
PH.NO.-2360015 MO.NO.-7275660055

INDIAN SICIETY OF CELL BIOLOGY
INCOME & EXPENDITURE A/C FOR THE PERIOD 01-04-2021 TO 31-03-2022

EXPENDITURE	AMOUNT	INCOME	AMOUNT
To Audit Fees	5,000.00	By Interest from HDFC	315,349.00
To Legal Fees	1,000.00	By Interest on SB	26,976.00
To Miscellaneous Expenses	23,000.00	By Interest on IT Refund	
		By Membership Fees	550.00
To Excess of Income over Expenditure	313,875.00		
Total	342,875.00	**Total**	342,875.00

For Indian Society of cell biology

Madhu Gwaldas Tapadia
(Secretary)

Place :- Varanasi
Date :- 31-08-2022

As per record produced before us
For Mohit K Saigal & Co.
(Chartered Accountants)
FRN : 01553C

Ca Manoj Nigam
(Partner)

MOHIT K. SAIGAL & CO. VARANASI CHARTERED ACCOUNTANTS

Newsletter committee: Sitting (L-R): Dr. Sharad Sharma, Dr. Ritu Trivedi, Dr. Kishore K Srivastava, Dr. Bhpendra N Singh, Standing (L-R): Dr. Prabhat R Mishra, Dr. Raj Kamal Tripathi, Dr. Srikanta K Rath