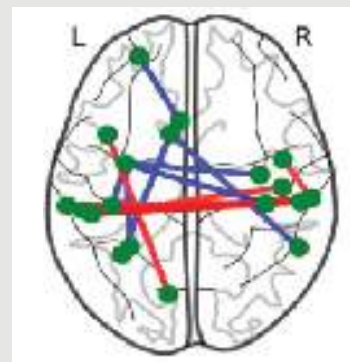
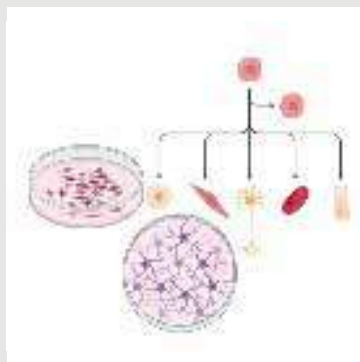
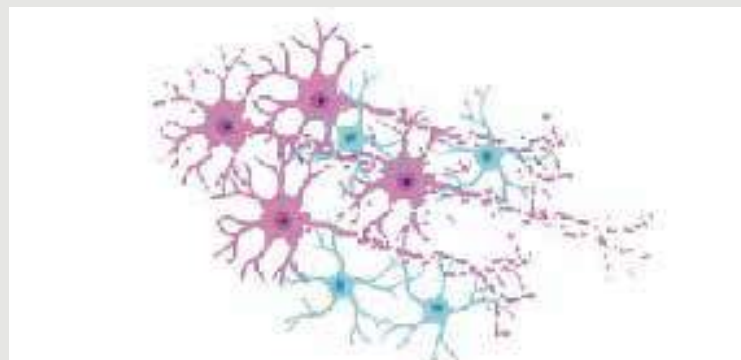
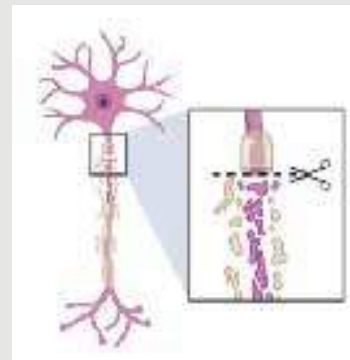


ANNUAL REPORT

2023 2024



An Autonomus Institute of the Department
of Biotechnology Ministry of Science &
Technology Government of India



BIOTECHNOLOGY RESEARCH AND INNOVATION COUNCIL (BRIC)
A DEPARTMENT OF BIOTECHNOLOGY ORGANIZATION

Annual Report

2023-2024



National Brain Research Centre
An Autonomous Institute of the Department of Biotechnology
Ministry of Science & Technology
Government of India



CONTENTS

1. Mandate & Objectives	5	8. Facilities & Resources	93
2. From Director's desk	7	A) Computing Facility	94
3. Scientific Reports	12	B) Animal Facility	98
A) Prof. Anindya Ghosh Roy	13	C) Digital Library	105
B) Prof. Anirban Basu	19	D) DBT's Electronic Library Consortium (DeLCON)	107
C) Prof. Arpan Banerjee	24	E) National Neuroimaging Facility	113
D) Dr. Bhavani Shankar Sahu	29	F) Magnetoencephalography Resource Facility	116
E) Prof. Ellora Sen	38		
F) Dr. Mayanghlambam Dhruba Singh	41	9. Events & Outreach	125
G) Prof. Pankaj Seth	44	10. General & Academic Administration	135
H) Prof. Pravat Kumar Mandal	47		
I) Prof. Ranjit Kumar Giri	52	11. Structure & Governance	142
J) Prof. Soumya Iyengar	57	12. Annual Financial Statements	167
K) Prof. Sourav Banerjee	61	13. Acknowledgment	205
L) Dr. Swagata Dey (India Alliance Early career fellow)	67		
4. Publications Presentations	78		
5. Extramural Research Projects	87		
6. Honors & Awards	90		
7. Academic Programs	92		
A) Ph.D. in Neuroscience			
B) M.Sc. in Neuroscience			
C) Summer Training			
D) Short-Term Programs			

MANDATE & OBJECTIVES

MANDATE

The mandate of National Brain Research Centre (NBRC) is to:

- Pursue research to understand brain function in health and diseases.
- Generate trained human resources with the capability to carry out interdisciplinary research in neuroscience.
- Promote neuroscience in India through networking among institutions across the country.

OBJECTIVES

- Undertaking high calibre research in neuroscience
- Connecting various national & international scientific research institutions, agencies & laboratories working in the field of brain sciences
- To become a national apex centre in the field of neuroscience and grow into a world-class institute with state-of-the-art facilities and reservoir of smart databases for brain research.
- To boost socio-economic impact factor on society with fundamental research and findings.
- Establishing one or more satellite centres to serve different regions of the country.



From Director's Desk...



Prof. Krishanu Ray

Director, NBRC

(Professor-H, TIFR, Mumbai)

National Brain Research Centre – Mission, Vision and Achievements



The study of brain function is essential for understanding the human society. Simply put, the idea of social exchange, as observed in the contexts of economy, commerce, anthropomorphic expressions through art and language, and most importantly, in politics, is a creation of the human mind. The rapid innovation in brain function mapping technology and psychometric tools for higher-order human behaviour have enabled the connectome mapping and elucidation of the underlying neurobiology of human expressions in art, language, decision-making, learning and memory, and many other contexts. The basic understating of neurobiology in turn deeply impacts the diagnosis and disease therapy.

National Brain Research Centre (NBRC) is mandated to pursue research in understanding the functioning of the brain in health and diseases, generate trained human resources with the capability to carry out interdisciplinary research in neuroscience and promote neuroscience in India through networking among institutions across the country. The institute is dedicated to basic and clinical brain research unique in the country with its cutting-edge infrastructural laboratories and facilities at par with any other international institution of similar nature. NBRC, recognised as an Institute of Excellence by the Government and a deemed-to-be University imparts neuroscience degrees at Master's and PhD levels and enjoys scientific leadership in neurosciences. NBRC has a multidisciplinary group of scientists with research activities encompassing all aspects of neuroscience - from molecules to mind, and various *in vivo* and *in vitro* model systems



to humans, for understanding brain function in normal and disease conditions under multiple sub-disciplines – Cellular and Molecular, Systems, Cognitive, Computational, and Translation Research.

The institute has world-class cell and molecular biology labs and non-human primate research facilities and collaborates with clinicians at local hospitals. It has positioned itself with leadership capabilities to study brain functions from molecules to the mind level in healthy and diseased conditions. It is the only S&T institute in the country with the aforementioned capabilities for brain research under one roof that could positively impact the quality of life and society. NBRC scientists have developed technologies to understand and detect the early signs of dementia through non-invasive techniques such as Magnetic Resonance Spectroscopy (MRS) of the adult brain, behavioural assessment tools for Dyslexia in Indian languages (DALI), drugs to alleviate the severity of brain inflammation due to viral infections, and many more. NBRC's research motivation has always been guided by societal necessity and aims to develop technologies for the future.

Scientific Achievements and Outreach Activities of NBRC

In the past one year, NBRC researchers made significant progress in diverse areas. We graduated 18 PhD/Int. PhD students and 11 MSc students who moved on to postdoctoral research and PhD in Universities in India and abroad. The NBRC students and scientists presented their work at national and international conferences. NBRC also published more than 30 research articles in reputed journals. The individual group-wise achievements are listed below.

Dr Arpan Banerjee's group has made notable contributions in explaining how multisensory integration ability, e.g. auditory, visual, tactile etc., into a coherent percept emerges from large-scale brain network interactions using electroencephalogram (EEG), functional magnetic resonance imaging (fMRI) of human subjects. They reported a novel inter-brain regional communication in EEG alpha frequencies (at ~10Hz) could define the individual-specific perceptual experience. Using fMRI and EEG experiments Dr Banerjee's group reformulated the understanding of visual and auditory information processing and teased out the intricate roles of ventral attention networks in shaping saliency processing for visual stimuli. Lately, his group have employed big data analytic tools to characterize neurophysiological patterns of healthy lifespan ageing and explained such patterns using biophysically realistic computational models. Dr Banerjee acquired international visibility on whole-brain computational modelling, applying concepts from Physics like metastability to Neuroscience and tool development for signal analysis in EEG/ fMRI data. He served on the editorial board of the journal NeuroImage and is currently an EB member in Imaging Neuroscience and Associate Editor in Frontiers in Computational Neuroscience.



Dr Anindya Ghosh Roy's laboratory studied the molecular mechanism of nerve regeneration using *C. elegans* as a model system. They identified an integrative role of AMPK signalling in exercise-mediated enhanced functional recovery in muscles and neuron regeneration. They also used the RNAi screening using a new neurite injury protocol to reveal an effect of FGFR receptor tyrosine kinase signalling underlying dendrite regeneration.

Dr Sourav Banerjee's laboratory highlighted how dysregulation of RNA-based mechanisms following early life stress or sleep loss impacts memory storage. The laboratory has initiated a research programme to study the implications of RNA-based mechanisms of synaptic homeostasis and how disruption of this process leads to behavioural deficits observed in autism. The laboratory is funded by the Department of Biotechnology and Science & Engineering Research Boards as well as the Indo-French Center for the Promotion of Advanced Research. Dr Banerjee has organized EMBO meetings as well as IBRO Schools and taken several outreach initiatives including brain awareness week for school children and teachers.

Dr Mayanglambam Dhruba Singh's lab is focused on finding molecular and genetic targets of Human neurodegenerative disorders. He uses *Drosophila* disease models for his study. He recently identified that the knockdown of Phosphatase and tensin homolog (Pten) reduces the abnormal poly(Q) aggregates in Huntington's disease (HD). The reduction of aggregate causes reduced cell death and improves the motor abilities of the flies. Similarly, feeding of Pten inhibitor reduces the poly(Q) aggregates and reduces the cell death in *Drosophila* and HD cell lines. The study suggests that targeting Pten has a potential therapeutic application for HD. The study is currently available in bioRxiv and in revision in Molecular Neurobiology.

Dr Soumya Iyengar's laboratory studies brain regions underlying cognition in house crows, a species of corvids known for their cognitive abilities. A recent publication from her lab has shown that house crows could be trained on a visual discrimination task, involving selecting one of two shapes based on a food reward. Histological methods were used to demonstrate that there was an increase in neural activity in various subdivisions of NC, (caudal nidopallium, the avian analogue of the prefrontal cortex), accompanied by increases in neurogenesis and an increase in the complexity of dopaminoceptive neurons in different parts of the NC in trained birds versus controls. Their findings suggest cognitive tasks involving reinforcement learning in house crows depend on a combination of neural plasticity and neurogenesis (Parishar et al., 2024).

Dr Bhavani S. Sahu's laboratory is engaged in understanding the cellular basis of brain function and disease. They have recently published one of the first major discoveries in understanding the inter-organellar cross-talk between ER stress and its contribution to neuronal secretion. Dr. Sahu and his collaborators elaborated on how a physiological neuropeptide can affect the membrane properties enabling an understanding of



the basic aspects of membrane/ peptide interactions and expanding the horizon in designing better peptides for peptide drug discovery. Their research has also unleashed an evidence-based approach to the traditional Ayurvedic medicinal plant-based therapy and its role in neuroprotection in Huntington's, a neurodegenerative disorder.

Dr Ranjit K. Giri's Laboratory showed how Multiple death receptors are involved in a mouse model of prion disease. Ligands for these death-inducing death-receptors are overexpressed in mice brains afflicted with the prion-disease indicating the activation of death receptors. The death receptor adaptors known to induce anti-apoptotic response are inhibited whereas receptor-interacting proteins are increasingly cleaved suggesting diminished anti-apoptotic activity or enhanced pro-apoptotic activity in prion-diseased mice brains. Collectively, multiple novel cell death factors are discovered to take part in a mouse model of prion disease, similar to the neuropathology of human prion diseases.

Dr Anirban Basu's Laboratory showed how the gut-brain axis plays a pivotal role in the physiological state of an organism. Gut microbiota-derived metabolites are known to play a role in brain disorders including neuroviral infections. They have recently shown short-chain fatty acids abrogate Japanese encephalitis virus-induced inflammation in microglial cells via miRNA. They have also shown prophylactic administration of gut microbiome metabolites abrogated microglial activation and subsequent neuroinflammation in an experimental model of Japanese encephalitis. These findings have a direct application potential.

Dr Ellora Sen's Laboratory identified that mutation in the metabolic gene isocitrate dehydrogenase (IDH1) is associated with better prognosis in glioma through altering the epigenetic landscape. Using IDH1 WT and MT patient tumours and IDH mutant glioma cells as the backdrop, they demonstrated how this mutation could alter the cytoskeletal organization and nuclear architecture to affect telomere function. These results highlighted how distinct molecular signatures having predictive and prognostic values in gliomas influence tumour progression. The work has a direct implication in developing new therapeutic approach to glioma.

Dr Pankaj Seth' lab at NBRC explores the cellular and molecular mechanisms of Zika virus and SARS-CoV-2 mediated damage to human brain cells and their impact on human health. Seth's lab investigated how Zika virus, known for causing birth defects, might influence glial cells for disruption of the blood-brain barrier (BBB). He has identified novel miRNAs responsible for impacting the integrity of BBB following exposure to Zika virus envelope protein. During this period Dr Seth's laboratory has also identified the molecular pathways of neuronal damage following exposure to SARS-CoV2 viral proteins and concluded that human neuronal death occurs through necrotic cell death pathways, these have been validated in postmortem brain sections from COVID-19 patients. He performed these experiments in human neurons and with live SARS-CoV2 virus. Dr Seth is currently engaged in establishing human cerebral brain organoids and plans to use



them as a model system for understanding health and diseased brain functions.

At the same time, NBRC has been engaged in strong outreach activities by organizing public events on and off campus. NBRC scientists participated in the IISF and other major national events and conducted programs at the NBRC campus. These include periodic visits by school and college students to NBRC, Public lectures by invited speakers and open discussions on topics of public interest.

Vision

NBRC now envisions developing an academic programme, a) to study the nexus between brain and body functions e.g., heart, gut, kidney, etc., and b) to map the functional networks of emotion and decision-making using a combination of a non-invasive cognitive neuroscience approach in humans and invasive affective neuroscience study in model animals. One of the most important issues to address would be how conscious and subconscious emotions control the heart, gut, liver, etc., and vice versa. Also, how physiological states and brain structure-function status control the decision-making process in various contexts, particularly in stressful conditions. The critical areas to focus on are – a) The heart-brain and gut-brain nexus correlating to heart disease, diabetes, obesity, and several mental conditions; b) Neurobiology of cognitive learning & communications; connections of the decision-making process; and, c) Predicting the cognitive influence on body functions. For example, the assessment of the learning needs of individuals, predisposition to certain disorders affecting normative functions through omics-based approaches, and enhancing the synergy between brain and body through a rigorous analysis of cognitive function and physiological signals.

In this context, some of the key approaches to be adopted are: i) developing assessment methodologies using non-invasive cognitive function measurement tools for effective learning, ii) estimating the correlation between brain development and environment, iii) using focused stimulation strategies to map the higher-order brain functions involved in creative thinking, emotions, and body function management.

We expect some of the key technological innovations through this approach. For instance,

- 1) the development of non-invasive cognitive function measurement tools using high-resolution EEG, MEG, fMRI, and AI-based brain-mapping strategies,
- 2) portable behaviour and neurological assessment tools through wearable devices and mobile phones,
- 3) the use of standard animal models for specific behaviour-body function correlation analysis for drug development, and
- 4) brain-machine interface devices.

Thus, we would aim to study this nexus for evolving effective lifestyle management policies for a better society.



Scientific Reports





Anindya Ghosh Roy

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Project Assitants: Kavi Nila, and Mydhiy Vasudevan

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Development and repair of neural circuit in *C. elegans* ▶▶▶

Background

The goal of our research team is to understand how neurons and neuronal circuits develop and maintain normal function and regenerate.

We are using a variety of approaches to study the development and function of neural circuits in vivo, including genetics, genomics, sub-cellular imaging, laser neurosurgery and optogenetics. Since *C. elegans* is transparent and has a simple nervous system, we can manipulate and observe individual neurons in a living animal.

We are interested in understanding how neurons are polarized during the initial stages of development, how neural circuits respond to injury in adulthood; and how molecular mechanisms such as cytoskeleton dynamics, RNA based mechanisms, and intracellular signalling affect these processes. One major focus is axon regeneration.

1) Regulation of neuronal microtubule cytoskeleton

Microtubules play an important role in the development and maintenance of the nervous



Microtubules play an important role in the development and maintenance of the nervous system. It is not clear how microtubules are differentially organized in the dendritic and the axonal compartments of the neuron. We are using *C. elegans* mechanosensory neurons to address these questions. We previously showed that loss of KLP-7, a kinesin-13 family microtubule depolymerizing protein, leads to ectopic neurite extensions in touch neurons, which can be suppressed by the pharmacological destabilization of microtubules (Puri et al., 2021).

Since touch neuron does not have proper axon-dendrite polarity, we investigated the function of KLP-7 in axon-dendrite compartmentalization

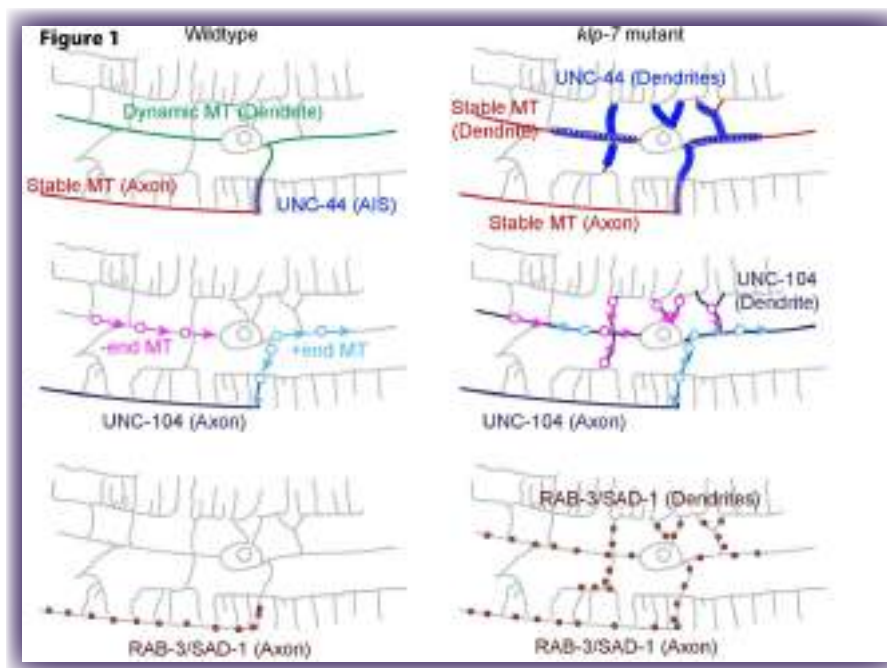


Figure 1: The absence of microtubule depolymerization in the *klp-7(0)*, decreases the microtubule dynamics in dendrites as well as axons leading to ectopic enrichment of UNC-44. Enrichment of UNC-44 in the axon initial segment (AIS) of wildtype or dendrites of *klp-7(0)* directs the axonal traffic of plus end microtubules and axonal motor, UNC-104, and presynaptic cargoes (RAB-3 and SAD-1) in these compartments.

using PVD neuron. Loss of KLP-7 caused a mislocalization of axonal proteins including RAB-3, SAD-1, and their motor UNC-104 to dendrites. This is rescued by cell-autonomous expression of the KLP-7 or colchicine treatment indicating the involvement of KLP-7-dependent microtubule depolymerization. High mobility of KLP-7 is correlated to increased microtubule dynamics in the dendrites which restricts the enrichment of UNC-44, an integral component of Axon Initial Segment (AIS) in these processes. Due to the loss of KLP-7, ectopic enrichment of UNC-44 in the dendrite potentially redirects axonal traffic into dendrites that includes plus-end out microtubules, axonal motors, and cargoes.

These observations indicate that KLP-7-mediated depolymerization defines the microtubule dynamics conducive to the specific enrichment of AIS components in dendrites. This further compartmentalizes dendritic and axonal microtubules, motors, and cargoes thereby influencing neuronal polarity (Dey et al 2024/under revision/ doi: <https://doi.org/10.1101/2023.08.24.554630>).

2) Neuronal Regeneration

Study of Dendrite Regeneration using PVD neuron as Model

The information-receiving units of a neuron, dendrites are equally vulnerable to physical insults. However, less is known about dendrite regeneration (Ramón y Cajal, 1991, Thompson-Peer et al., 2016). To understand the mechanisms of dendrite regeneration, we used PVD neurons, having branched dendrites. The PVD neurons are responsible for harsh touch sensation. After the primary dendrite was severed near the cell body, we observed that the regrowth started from the injured tip and continued following a similar trajectory with complex branching patterns. We found that neither initiation of regrowth nor branching is affected by the axon injury pathways. Surprisingly, we found that a small GTPase CED-10 (RAC) and an upstream GEF TIAM-1 is essential for dendrite regeneration. Our work provides a framework for understanding the cellular mechanism of dendrite regeneration using PVD neuron (Brar et al., 2022, PLOS-Genetics).

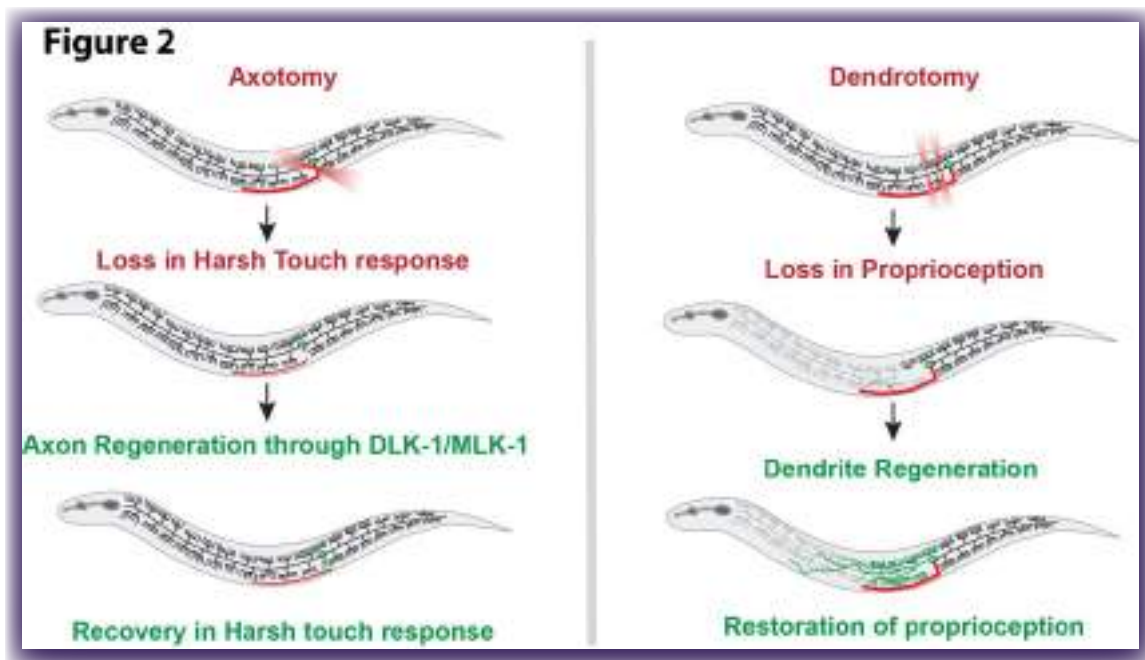


Figure 2: Differential regulation of sensory modalities by the axon and dendrites of PVD neuron. Loss of axon and dendrite cause a decline in harsh touch and posture, respectively which is regained when these compartments regenerate.

PVD neuron operates in multiple sensory modalities including the control of both nociceptive touch sensation and body posture. Although both the axon and dendrites of this neuron show a regeneration response following laser-assisted injury, it is rather unclear how the behavior associated with this neuron is affected by the loss of these structures. It is also unclear whether neurite regrowth would lead to functional restoration in these neurons. Upon axotomy, using a femtosecond laser, we saw that harsh touch response was specifically affected leaving the body posture unperturbed. Subsequently, recovery in the touch response is highly correlated to the axon regrowth, which was dependent on DLK-1/MLK-1 MAP Kinase. Dendrotomy of both major and minor primary dendrites affected the wavelength and amplitude of sinusoidal movement without any apparent effect on harsh touch response. We further correlated the recovery in posture behavior to the type of dendrite regeneration events. We found that dendrite regeneration through the fusion and reconnection between the proximal and distal branches of the injured dendrite corresponded to improved recovery in posture. Our data revealed that the axons and dendrites of PVD neurons regulate the nociception and proprioception in worms respectively. It also revealed that dendrite and axon regeneration lead to the restoration of these differential sensory modalities (**Brar et al 2024 eNeuro/ DOI: <https://doi.org/10.1523/ENEURO.0292-23.2024>**).

Use of ultrafast laser in the past decades have allowed researchers to perform precise injuries to neurites in various model systems (Yanik et al., 2004) for studying the mechanism of neurite regeneration. However, these methods are labor intensive and expensive creating bottlenecks for high throughput assays like RNAi screening for discovering unanticipated pathways.

In this study, we have devised an inexpensive and efficient method to injure the PVD dendrites precisely without causing any major health hazards. In this method, a drawn capillary is placed and rolled posterior to the vulva causing a twist and break in the major dendrite of PVD neurons. More than 90% of worms survive this injury and other health parameters like reproductive health and locomotion are unaffected. This protocol allows breaking the primary dendrites of PVD neuron at specific sites with 90% success at the rate of 4-5 worms per minute on the NGM plate itself. This overall speed up the injury protocol more than ten times in comparison to dendrotomy with lasers. The outcome of twist injury is comparable to laser-induced dendrotomy in terms of length of longest regenerated neurite, branching and self-fusion events. Dendrite regeneration following the twist injury is dependent on pathways such as CED-10 RAC GTPase and AFF-1 as discovered using laser-assisted protocol before (Oren-Suissa et al., 2017, Brar et al., 2022). This validated our protocol for studying dendrite regeneration using genetic screening.



Figure 3

Twist injury protocol with glass capillary



Candidate genes screened using RNAi

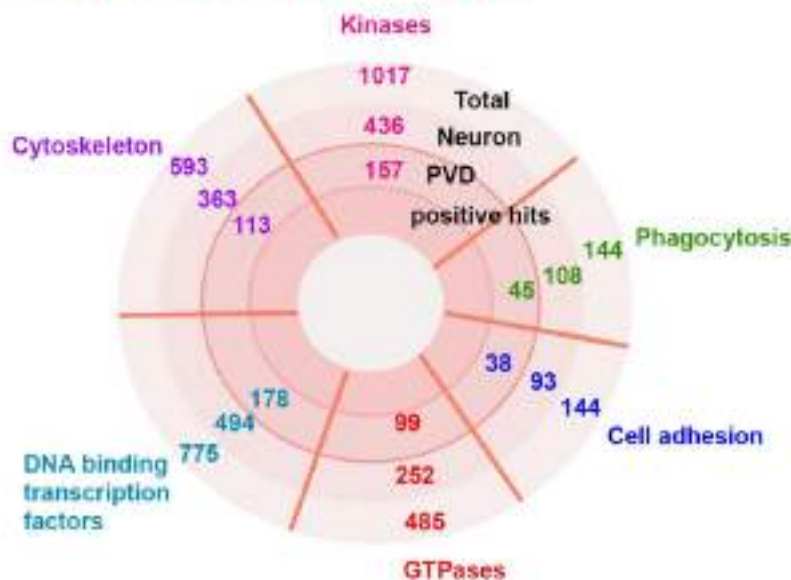


Figure 3: The illustration of new neurite injury protocol and RNAi screening for genes regulating dendrite regeneration in PVD neuron.

To screen for the genes controlling dendrite regeneration, we developed a RNAi sensitive strain overexpressing *sid-1* in PVD (Singh et al 2024, PLOS-One/ <https://doi.org/10.1371/journal.pone.0298766>). This injury protocol combined with RNAi allowed screening of 1500 genes in 8 months. Using this approach, we have identified dendrite regrowth promoting molecules involving Kinases, Transcription factors, small GTPase and cytoskeleton factors (Figure 3/ Singh et al 2024, Manuscript in preparation).

This study not only opened up avenue for high throughput neurite injury, but also revealed new mechanisms controlling dendrite regeneration.

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Doctoral work from the CSIR-Indian Institute of Chemical Biology (IICB), Kolkata & post-doctoral work from Pennsylvania State University College of Medicine, Hershey, Pennsylvania. Dr. Basu's research is primarily directed towards discovering therapeutic strategies to develop disease-modifying therapy by abrogating inflammation in CNS disorders.

Molecular approaches to understand the pathophysiology and pharmacology of infection and inflammatory disorders of Central Nervous System ▶▶

Japanese Encephalitis Virus (JEV) entry into the host is followed by viral replication in the periphery which in turn is accompanied by activation of innate and adaptive arms of immune system. Our lab

been working on deciphering the molecular details of various steps of viral life cycle thus contributing significantly to the field of host-virus interactions.

JEV, a member of the flaviviruses, is the most common cause of arthropod borne human encephalitis in Asia. The primary sites for JEV multiplication are likely to be in either myeloid and lymphoid cells or vascular endothelial cells. Viral persistence in the human nervous system has been reported in approximately 5% of patients with JEV associated encephalitis. This suggests that, following the acute infection phase persistent JEV infection might also be responsible, in part, for the neural sequelae occurring in



approximately 70% of survivors. The host response to infection is central to the effective control and ultimate clearance of invading pathogens. A detailed understanding of the disease pathogenesis is therefore crucial for the prevention of the neurological sequel mediated by JEV in human beings.

Infection of the central nervous system by the Japanese encephalitis virus (JEV) is characterized by extensive neuronal cell death and neuroinflammation. Several protein-coding genes and microRNAs are implicated in JEV-induced neuronal cell death. Central nervous system infection by flaviviruses such as Japanese encephalitis virus, Dengue virus, and West Nile virus results in neuroinflammation and neuronal damage. However, little is known about the role of long non-coding RNAs (lncRNAs) in flavivirus-induced neuroinflammation and neuronal cell death. Here, we profiled the transcriptome of the JEV-infected neuronal cell line and identified several lncRNAs whose expression is altered during JEV infection. We functionally characterized a lncRNA named JINR1 (JEV-induced non-coding RNA 1), which is evolutionarily conserved in primates. JINR1 induction during JEV infection is regulated by nuclear factor-kappa B (NF- κ B). Depletion of JINR1 during infection reduces flavivirus replication, neuronal cell death, and the expression of genes involved in ER stress and neuroinflammation. Interestingly, GRP78 overexpression prevents the decrease in flavivirus replication due to JINR1 knockdown. JINR1 interacts with RBM10 and NF- κ B to regulate the transcription of virus-induced genes. In addition, RBM10 and JINR1 form a feed-forward loop to reciprocally promote each other's expression by regulating NF- κ B activity. Our results suggest the role of JINR1 in promoting flavivirus replication and flavivirus-induced neuronal cell death.

Here, we characterized the role of a flavivirus-induced lncRNA named JINR1 during the infection of neuronal cells. Depletion of JINR1 during virus infection reduces viral replication and cell death. An increase in GRP78 expression by JINR1 is responsible for promoting virus replication. Flavivirus infection induces the expression of a cellular protein RBM10, which interacts with JINR1. RBM10 and JINR1 promote the proinflammatory transcription factor NF- κ B activity, which is detrimental to cell survival.

Japanese Encephalitis Virus (JEV) and West Nile Viruses (WNV) are neurotropic flaviviruses which cause neuronal death and exaggerated glial activation in the central nervous system. Role of host long non coding RNAs in shaping microglial inflammation upon flavivirus infections has been unexplored. This study attempted to decipher the role of lncRNA Gm20559 in regulating microglial inflammatory response in context of flaviviruses. Antisense oligonucleotide LNA Gapmers designed against lncRNA Gm20559 and non-specific site (negative control) were used for Gm20559 knockdown in JEV and WNV-infected N9 microglial cells. Upon establishing successful Gm20559 knockdown, expression of various proinflammatory cytokines, chemokines, interferon-stimulated genes (ISGs) and RIG-I were checked by qRT-PCR and cytometric bead array. Western Blotting was done to analyse the phosphorylation level of various inflammatory markers



and viral non-structural protein expression. Plaque Assays were employed to quantify viral titres in microglial supernatant upon knocking down Gm20559. Effect of microglial supernatant on HT22 neuronal cells was assessed by checking expression of apoptotic protein and viral non-structural protein by Western Blotting. Upregulation in Gm20559 expression was observed in BALB/c pup brains, primary microglia as well as N9 microglia cell line upon both JEV and WNV infection. Knockdown of Gm20559 in JEV and WNV-infected N9 cell led to the reduction of major proinflammatory cytokines – IL-1 β , IL-6, IP-10 and IFN- β . Inhibition of Gm20559 upon JEV infection in N9 microglia also led to downregulation of RIG-I and OAS-2, which was not the case in WNV-infected N9 microglia. Phosphorylation level of P38 MAPK was reduced in case of JEV-infected N9 microglia and not WNV-infected N9 microglia. Whereas phosphorylation of NF- κ B pathway was unchanged upon Gm20559 knockdown in both JEV and WNV-infected N9 microglia. However, treating HT22 cells with JEV and WNV-infected microglial supernatant with and

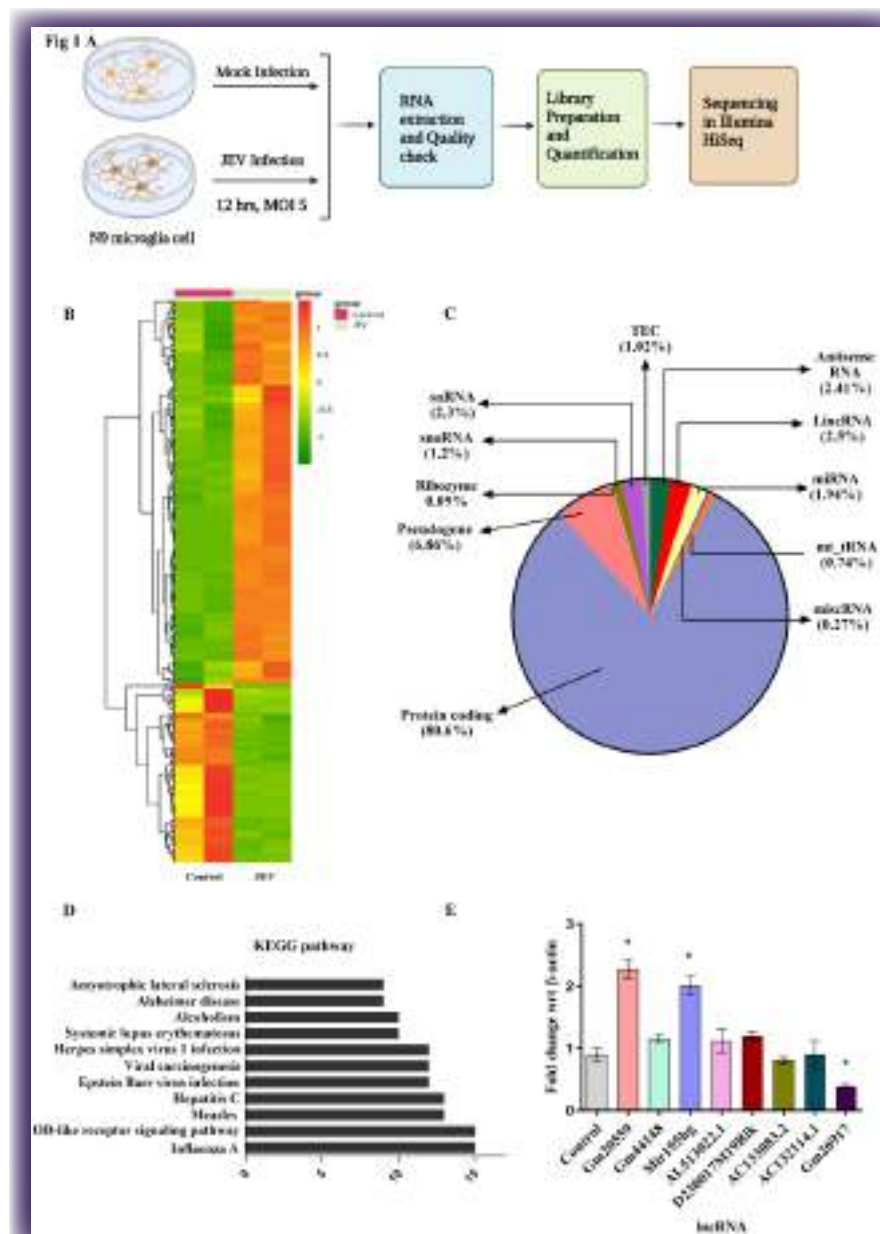


Figure 1: Identification of lncRNAs regulated by JEV in murine microglial cell line N9. (A) Schematic diagram of the experimental paradigm – LncRNA sequencing of mock-infected versus JEV-infected N9 cell line after 12 hrs of infection. (B) Heat Map of DEGs in mock-infected versus JEV-infected N9 cell line after 12hrs of infection. DEGs ($n = 2$; $p < 0.05$ and $\log_2$ fold change ≥ 1.5) (C) Pie Chart depicting various classes of DEGs ($p < 0.05$) as obtained from the whole transcriptome sequencing of N9 cells at 12 hrs. (D) Pathway analysis of DEGs by KEGG – y-axis represents pathways associated with DEGs; x-axis represents fold enrichment. (E) Validation of DE lncRNAs by qRT-PCR analysis of RNA obtained from mock-infected and JEV-infected N9 cells with an MOI of 3, for 12 hrs. β -actin was used as reference. Data in the figures are represented as mean $\pm$ SD, of three independent experiments. Statistical comparisons were done by Unpaired Student's t-test. (* $p < 0.05$, ** $p < 0.01$). Fig. 1A was made using Biorender.com

without Gm20559 could not trigger cell death or influence viral replication. Knockdown studies on lncRNA Gm20559 suggests its pivotal role in maintaining the inflammatory milieu of microglia in flaviviral infection by modulating the expression of various pro-inflammatory cytokines. However, Gm20559-induced increased microglial proinflammatory response upon flavivirus infection fails to trigger neuronal death.

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Austin Varghese

Neuro-cognitive network mechanisms using multimodal neuroimaging ▶▶

Cognitive Brain Dynamics Lab (CBDL) is engaged in basic and translational research using non-invasive neuroimaging tools EEG, MEG, TMS & fMRI. We have primarily two themes of research: 1) Exploring and innovating novel research designs and analysis tools for MEG/ EEG & fMRI recordings and 2) Studying the relationships between in structure and function in the context of whole brain macroscopic neural networks related to various cognitive functions using empirical and theoretical approaches. Here we outline the major project updates from the period April, 2023 - March, 2024. The overarching goal of these projects is to develop and implement network neuroscience approaches for mechanistic understanding of structure and function that may be useful for developing brain stimulation protocols, understanding stroke recovery and empirical observations such as hemispheric lateralization.

Project 1: Effective networks mediate right hemispheric dominance of human 40 Hz auditory steady-state response.

Auditory steady-state responses (ASSR) are induced



from the brainstem to the neocortex when humans hear periodic amplitude-modulated tonal signals. ASSRs have been argued to be a key marker of auditory temporal processing and pathological reorganization of ASSR - a biomarker of neurodegenerative disorders. However, most of the earlier studies reporting the neural basis of ASSRs were focused on looking at individual brain regions. Here, we seek to characterize the large-scale directed information flow among cortical sources of ASSR entrained by 40 Hz external signals. Entrained brain rhythms with power peaking at 40 Hz were generated using both monaural and binaural tonal stimulation. First, we confirm the presence of ASSRs and their well-known right hemispheric dominance during binaural and both monaural conditions. Thereafter, reconstruction of source activity employing individual anatomy of the participant and subsequent network analysis revealed that while the sources are common among different stimulation conditions, differential levels of source activation and differential patterns of directed information flow among sources underlie processing of binaurally and monaurally presented tones. Particularly, we show bidirectional interactions involving the right superior temporal gyrus and inferior frontal gyrus underlie right hemispheric dominance of 40 Hz ASSR during both monaural and binaural conditions. On the other hand, for monaural conditions, the strength of inter-hemispheric flow from left primary auditory areas to right superior temporal areas followed a pattern that comply with the generally observed contralateral dominance of sensory signal processing (Figure 1).

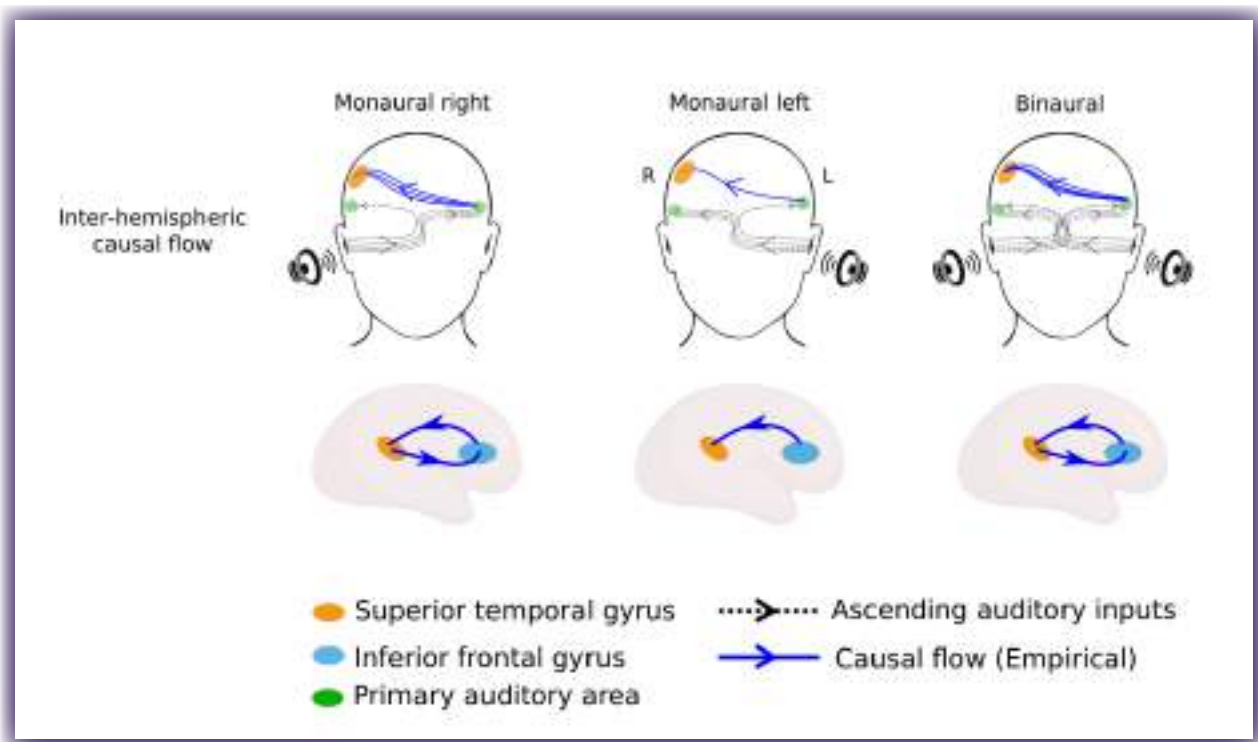


Figure 1: Hemispheric lateralization of auditory steady state rhythms are facilitated by interhemispheric causal information flow from left primary cortical areas to right superior temporal areas at varying degrees of magnitude, lowest for monaural left, followed by monaural right and binaural hearing conditions.

Project 2: Metastability indexes global changes in the dynamic working point of the brain following brain stimulation

Several studies have shown that coordination among neural ensembles is a key to understand human cognition. A well charted path is to identify coordination states associated with cognitive functions from spectral changes in the oscillations of EEG or MEG. A growing number of studies suggest that the tendency to switch between coordination states, sculpts the dynamic repertoire of the brain and can be indexed by a measure known as metastability. In this article, we characterize perturbations in the metastability of global brain network dynamics following Transcranial Magnetic Stimulation that could quantify the duration for which information processing is altered. Thus allowing researchers to understand the network effects of brain stimulation, standardize stimulation protocols and design experimental tasks. We demonstrate the effect empirically using publicly available datasets and use a digital twin (a whole brain connectome model) to understand the dynamic principles that generate such observations. We observed a significant reduction in metastability, concurrent with an increase in coherence following single-pulse TMS reflecting the existence of a window where neural coordination is altered. The reduction in complexity was validated by an additional measure based on the Lempel-Ziv complexity of microstate labeled EEG data. Interestingly, higher frequencies in the EEG signal showed faster recovery in metastability than lower frequencies. The digital twin shed light on how the phase resetting introduced by the single-pulse TMS in local cortical networks can propagate globally, giving rise to changes in metastability and coherence (Figure 2).

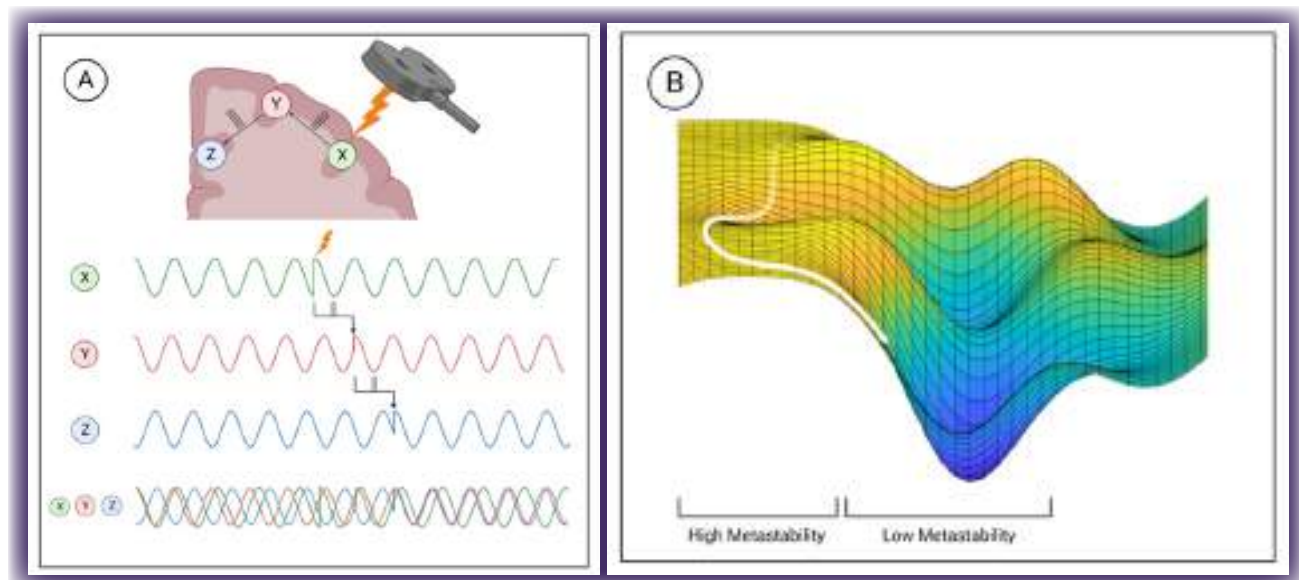


Figure 2: A) Phase resetting of neural oscillations are facilitated by transcranial magnetic stimulation (TMS). B) The phase resetting leads to a rise in synchronous activity that can be quantified by fewer number of dynamical states for the system to reside in, and hence a reduction of metastability in the the global energy landscape. Thus, tracking of temporal window of lower metsatbility can quantify the effective stimulation period.

Project 3: Structural-and-dynamical similarity predicts compensatory brain areas driving the post-lesion functional recovery mechanism.

The focal lesion alters the excitation–inhibition (E–I) balance and healthy functional connectivity patterns, which may recover over time. One possible mechanism for the brain to counter the insult is global reshaping functional connectivity alterations. However, the operational principles by which this can be achieved remain unknown. We propose a novel equivalence principle based on structural and dynamic similarity analysis to predict whether specific compensatory areas initiate lost E–I regulation after lesion. We hypothesize that similar structural areas (SSAs) and dynamically similar areas (DSAs) corresponding to a lesioned site are the crucial dynamical units to restore lost homeostatic balance within the surviving cortical brain regions. SSAs and DSAs are independent measures, one based on structural similarity properties measured by Jaccard Index and the other based on post-lesion recovery time. We unravel the relationship between SSA and DSA by simulating a whole brain mean field model deployed on top of a virtually lesioned structural connectome from human neuroimaging data to characterize global brain dynamics and functional connectivity at the level of individual subjects. Our results suggest that wiring proximity and similarity are the 2 major guiding principles of compensation-related utilization of hemisphere in the post-lesion functional connectivity re-organization process.

Recognition and Service

- 2023- Visiting Professor at Ashoka University (Spring semester),
Taught Mind & Behavior
- 2023 - Editorial board member, Imaging neuroscience
- 2023- Convener, International Electrotechnical Commission (IEC)/ ISO Working Group on Brain Computer Interfaces JTC 1/SC 43/WG 2 Applications (Elected position among Permanent member countries of IEC/ ISO)
- 2022- Member, Bureau of Indian Standards (BIS) Panel 7 on Brain computer interfaces
- 2021-2023 Editorial Board Member, NeuroImage (Elsevier)
- 2021- Associate Editor, Frontiers in Computational Neuroscience
- 2021- Review Editor, Frontiers in Network Physiology & Frontiers in Brain Imaging Methods

Funding

Early diagnosis and structural and functional decline of the brain and associated injuries in professional athletes playing contact sports. Department of Sports, Ministry of Youth



Affairs & Sports, Feb 2023- Feb 2026 67 lakh INR) on no cost extension.

AI tools in low MRI settings. Bill and Melina Gates Foundation, Aug 1, 2023 – Aug 2025, 113922 US\$).

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Vesicular trafficking pathways in neurons / neuroendocrine cells and their physiological consequences ►►

AIM:

To elucidate the mechanistic aspect of impeded regulated Secretion in ER-stressed cells.

OBJECTIVES:

1. To model ER stress in PC12 neuroendocrine cells using Thapsigargin (Tg) and rescue using Docosahexaenoic acid (DHA).
2. To study the effect of ER stress in regulated Secretion in PC12 cells.
3. To understand the molecular basis for altered Secretion in ER-stressed cells. them in response to

BACKGROUND:

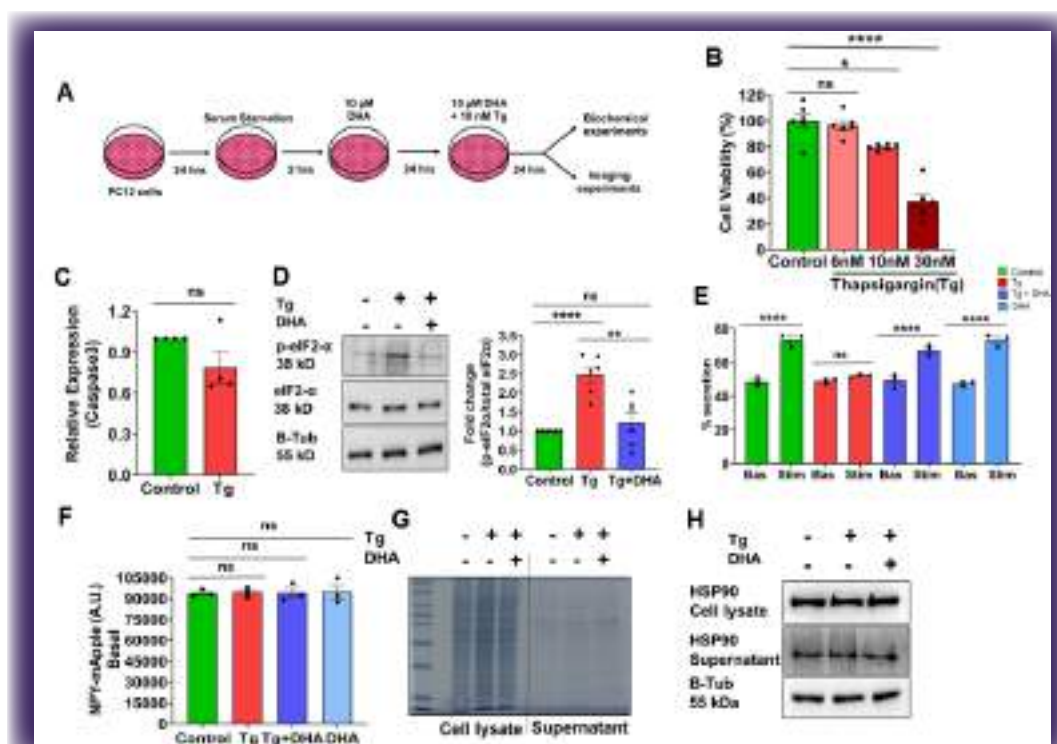
Disruption of protein homeostasis and vesicle trafficking is linked to various neurological and metabolic disorders, primarily associated with endoplasmic reticulum (ER) stress. The ER, responsible for protein synthesis and sorting, activates the Unfolded Protein Response (UPR) through pathways like IRE1- α , PERK, and ATF6 to manage cellular stress. This involves regulating gene expression, enhancing protein folding, and determining cell fate. ER stress induces changes in spliced XBP1 levels via IRE1- α activation, leading to increased chaperone expression. ATF6 contributes to UPR, influencing cell fate through micro autophagy. PERK activation reduces global translation, potentially triggering apoptosis under prolonged stress. Dense core vesicles (DCVs) in neurons and neuroendocrine cells store peptides, hormones, and neurotransmitters, releasing



them in response to physiological stimuli. DCV functionality relies on matrix proteins (e.g., CGA, CGB, SCGII) that regulate cargo release through exocytosis. The release of neurotransmitters and hormones from DCVs is tightly regulated by V-SNARES (VAMP1) and T-SNARES (SNAP25). Altered levels of these substances are associated with various diseases. Misfolded proteins, like mutated alpha-synuclein, are linked to disrupted fusion pore formation, neurodegeneration, and ER stress, suggesting potential crosstalk between ER stress and DCV trafficking. However, the impact of ER stress on DCV and synaptic-like vesicle (SLV) exocytosis remains unexplored. Using PC12 cells, a widely studied model, we induced ER stress and observed compromised DCV exocytosis. Co-treatment with ER stress attenuators like Docosahexaenoic Acid (DHA) restored regulated Secretion. ER stress did not alter DCV size or number but affected their spatial localization, as revealed by transmission electron microscopy (TEM). Molecular analysis indicated expression switching of key proteins associated with exocytosis. Our findings provide evidence that ER stress impairs DCV exocytosis, potentially influenced by expression switching of key proteins through the CREB/ATF4/eIF2 α axis.

RESULTS:

Tg impairs regulated Secretion in PC12 cells rescued by DHA: In PC12 cell lines, Tg induces ER stress, evident from increased p-eIF2 α /t-eIF2 α without causing apoptosis (Fig 1B, 1C), while DHA attenuates ER stress (Fig. 1D). DHA not only inhibits ER stress but also restores impaired regulated exocytosis of DCVs in Tg-treated cells (Fig. 1E, 1F). Coomassie staining and HSP-90 immunoblotting rule out hindrances in the constitutive secretory pathway (Fig. 1G, 1H).



Furthermore, ER-stressed cells exhibit reduced DCV secretion, altered exocytosis dynamics and reduced responsiveness to KCl stimulation, which is normalized by DHA co-treatment (Fig. 1(I-M)). These experiments highlight the specificity of ER stress-associated perturbations in regulated exocytosis and flag the therapeutic potential of DHA in addressing compromised exocytosis induced by ER stress.

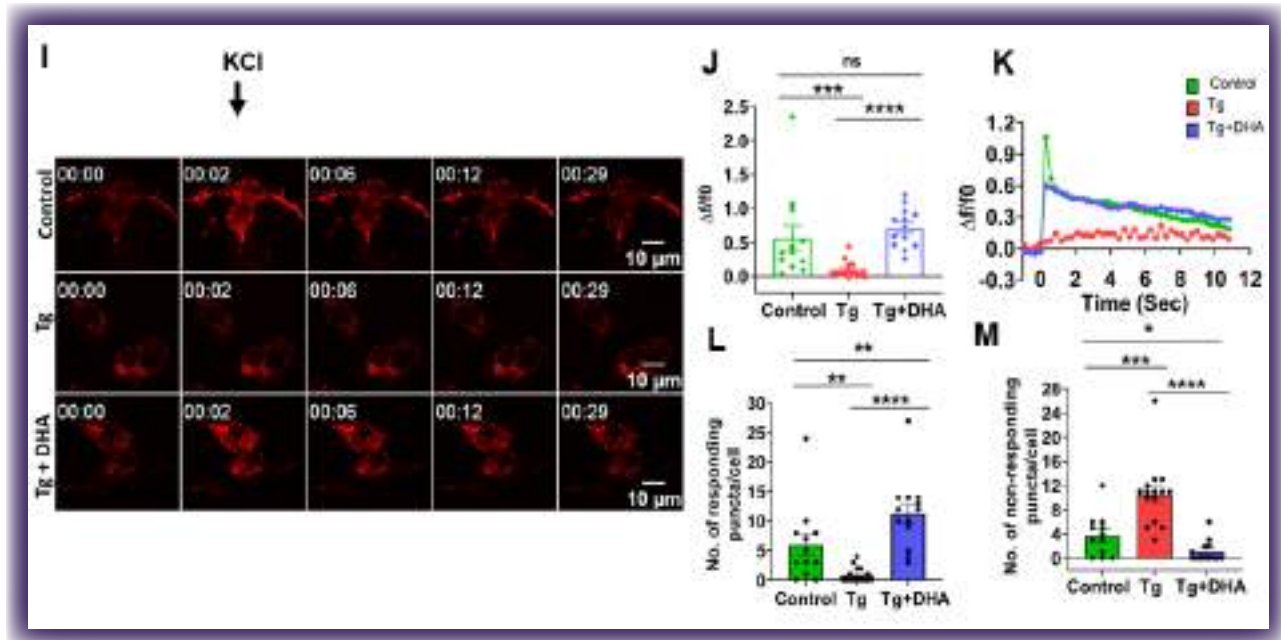


Figure 1: DHA rescues the phenotype by reducing ER stress in dense-core vesicles. **A** Treatment protocol. **B** MTT assay showing cell viability. **C** Transcript levels of Caspase3. **D** immunoblots and quantification of p-eIF2 α /t-eIF2 α . B-Tub and EF2 served as loading control. **E** Quantitative measure of NPY-mApple fluorescence intensities in cells transfected with NPY-mApple showing % secretion. **F** Basal NPY-mApple intensities. **G** Coomassie staining for constitutive Secretion. **H** Immunoblot image of constitutive secretion marker Hsp-90. **I** Representative NPY-pHTomato (red) images in PC12 cells showing basal and KCl stimulated conditions. **J** Quantitative analysis of the maximum $\Delta f/f_0$. **K** Representative time traces of mean NPY-pHTomato fluorescence intensities. **(L-M)** No. of responding and non responding puncta/cell.

Tg treatment does not change the Size and Number but alters the spatial localization of DCV: In light of severe disruption in DCV exocytosis, we wanted to estimate the size and distribution of the vesicles. TEM experiments followed by morphometry suggested non-significant differences in the DCV size and number (Fig. 2 (A-D)) across all treatment. However, DCVs were significantly closer to the plasma membrane in the control cells than the ER-stressed counterpart (Fig 2E), which is mildly reversed upon DHA treatment.

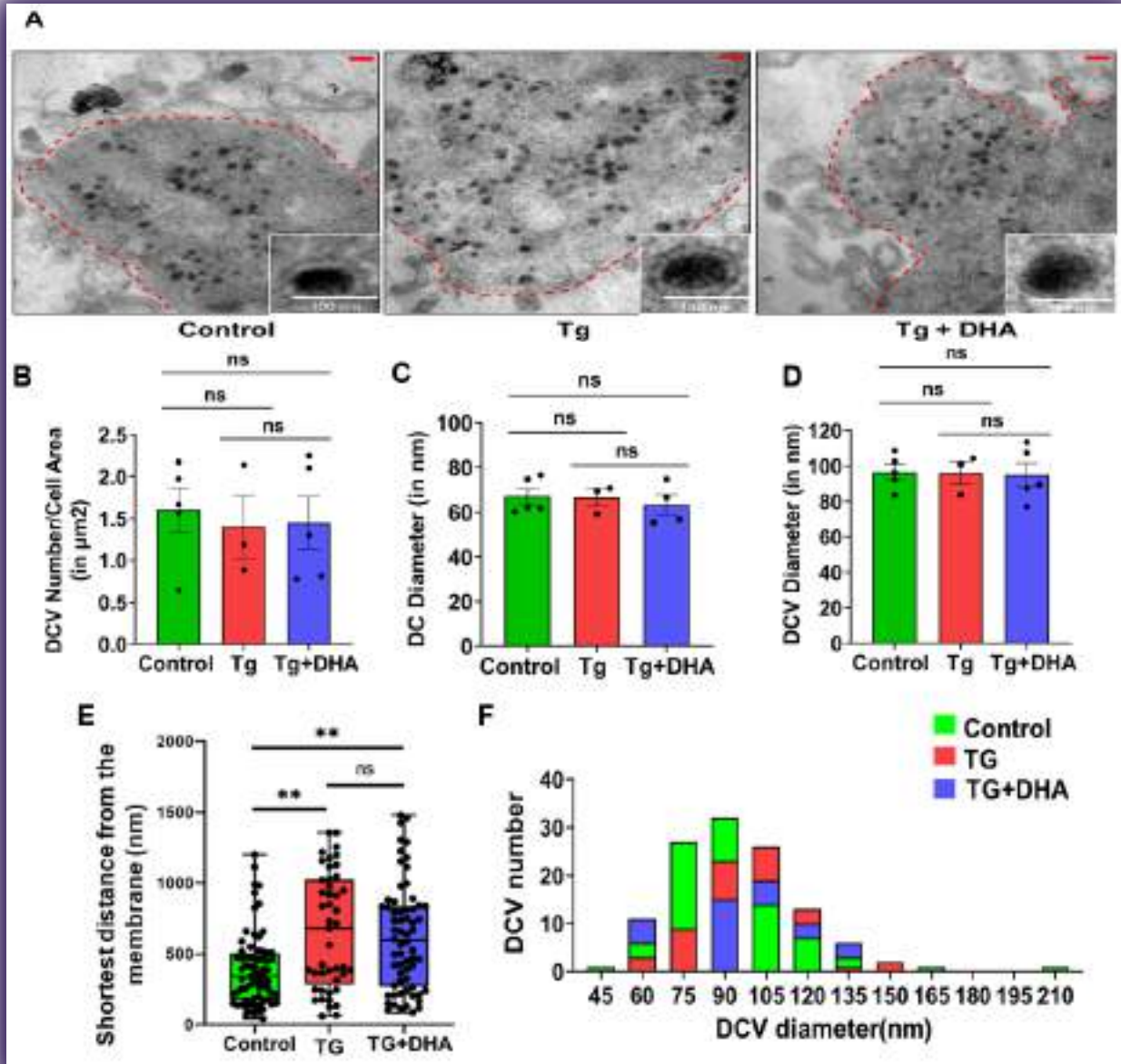


Figure 2: Transmission electron microscopy of PC12 cells. **A** Representative TEM showing electron-dense vesicles. **B** Histogram of DCV Diameter Distribution **C** Quantification of DC diameter. **D** Quantification of DCV diameter showing no significant difference. **E** Quantification of DCV distance from the nearest plasma membrane. **F** Quantification of The number of DCVs/ μM^2 of the cell surface area.

DHA reverses impediment to regulated Secretion in SLVs: In line with our previous observation, we checked for the effect of DHA co-treatment in the exocytosis of SLVs in ER-stressed cells. Although there was a subtle restoration of SLVs secretion in DHA co-treated ER-stressed cells, the magnitude of restoration was not in the order of DCV exocytosis (Fig. 3 (A-E)). We report that DHA treatment as an inhibitor of ER stress also causes a mild reversion in SLV dynamics towards normal or wild-type conditions along with the reversal of the impaired regulated secretion phenotype as seen in the ER-stressed cells.

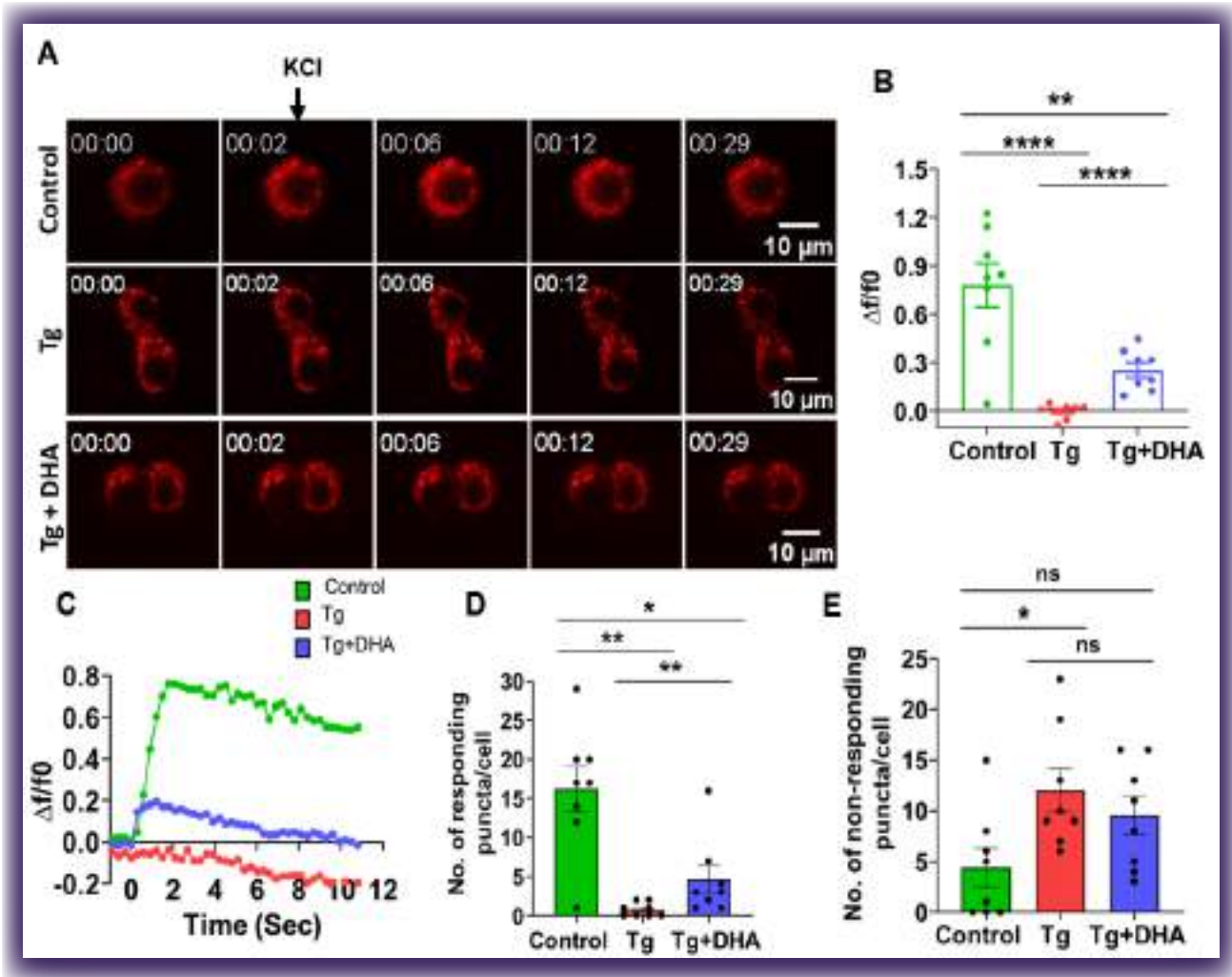


Figure 3: DHA rescues the phenotype by reducing ER stress in synaptic like vesicles. A Representative Synaptophysin-pHTomato (red) images showing basal and KCl stimulated conditions. **B** Quantitative analysis of the maximum $\Delta f/f_0$. **C** Representative time traces of mean Synaptophysin-pHTomato fluorescence intensities. **(D-E)** No. of responding and non responding puncta/cell.

ER stress alters the abundance of signature genes associated with exocytosis: Investigating impaired exocytosis induced by Tg triggered ER stress, we focused on SNAP25 expression. Tg-treated cells exhibited reduced SNAP25 levels, restored by DHA co-treatment. (Fig. 4A, 4B) Surprisingly, increased CREB activity persisted with DHA co-treatment (Fig. 4C). We further explored *atf4*, an UPR pro gene and a CREB inhibitor, observing elevated ATF4 levels in ER-stressed cells, returning to control levels with DHA co-treatment (Fig. 4E, 4F). ATF4 inhibition using ISRIB restored SNAP25 levels (Fig. 4G) Further, overexpressing SNAP25 in ER stressed cells restored DCV exocytosis to near control levels (Fig. 4(J-N)). These results suggest a negative modulatory role of ATF4 on SNAP25 expression by interfering with CREB activity.

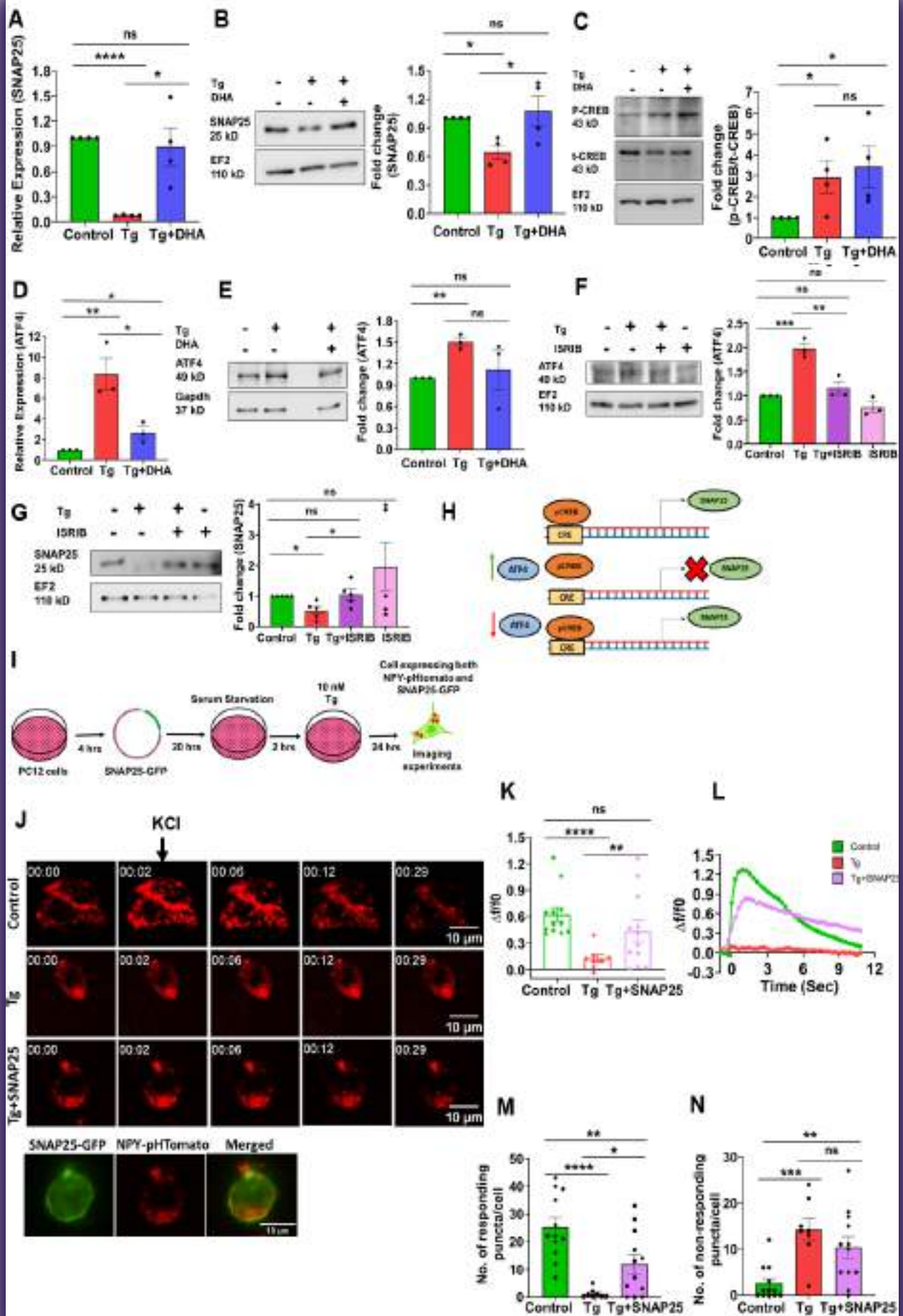
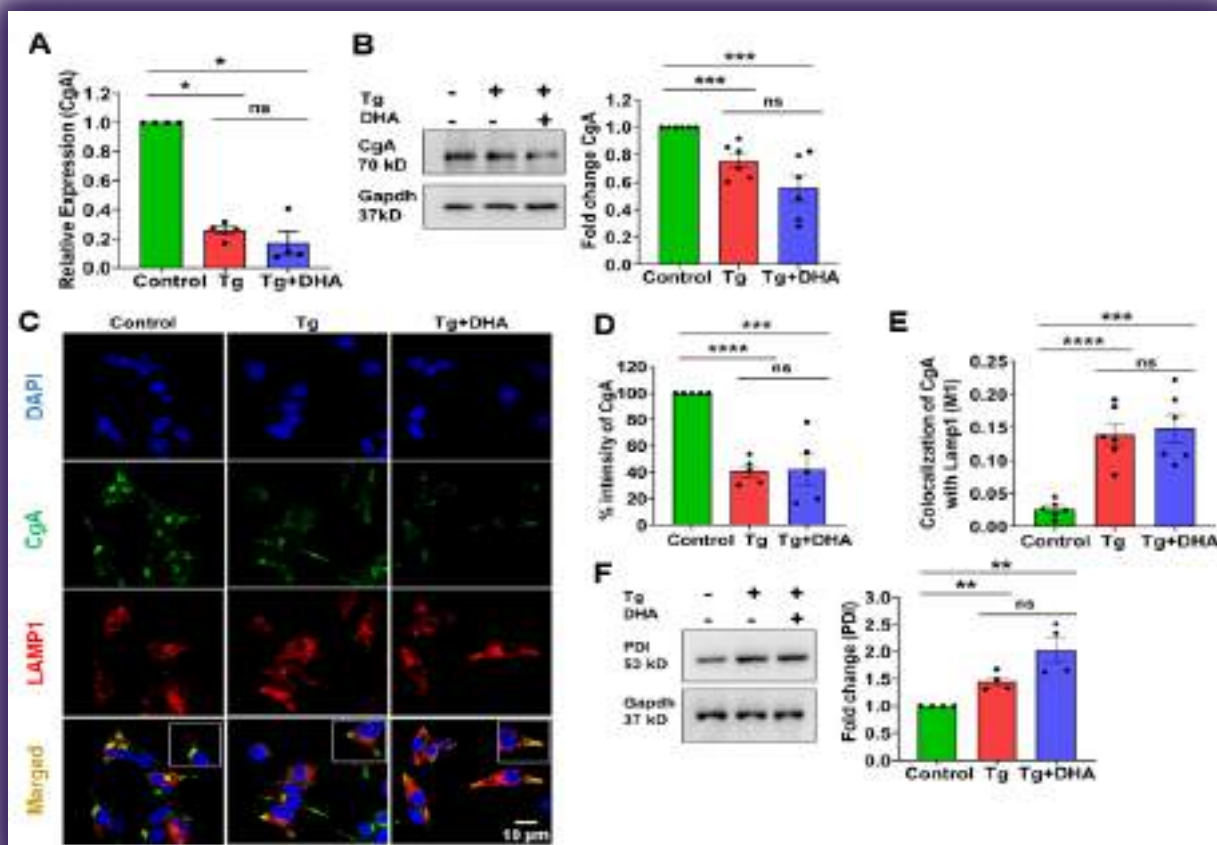


Figure 4: Change in the expression of exocytosis regulating gene SNAP25: **A** Quantification showing transcript levels of Snap25. **B** Representative blots and quantification of SNAP25. EF-2 served as a loading control. **C** Representative blots and quantification of p-CREB and t-CREB. EF-2 served as a loading control. **D** Atf4 transcript levels in Q-RT PCR. **E** Representative blots and quantification of ATF4. GAPDH served as a loading control. **F-G** Representative blots and quantification of ATF4 (F) and SNAP25 (G) upon treating them with ISRIB. **H** Schematic showing the transcriptional regulation of SNAP25 by CREB and ATF4. **I** Schematic of experimental paradigm for overexpression. **J** Representative NPY-pHTomato images in chosen cell expressing both NPY and SNAP25. **K** Quantitative analysis of the maximum $\Delta f/f_0$. **L** Representative time traces of mean NPY-pHTomato fluorescence intensities. **(M-N)** No. of responding and non-responding puncta/cell.

ER, stress lowers the abundance of key granulogenic signatures: Examining the granulogenic potential of chromogranin A (CGA) in ER-stressed cells, we observed a significant decrease in their mRNA and protein levels, a trend unchanged with DHA co-treatment (Fig. 5A, 5B). Colocalization of CGA with the lysosomal marker Lamp1 increased in Tg-treated cells, persisting with DHA co-treatment (Fig. 5 (C-E)). Elevated levels of Protein Disulfide Isomerase (PDI) supported enhanced lysosomal localization (Fig. 5F). Ectopic introduction of CgA partially reversed the secretory impairment, although less effectively than SNAP25 (Fig. 5 (H-L)). Overall, these findings suggest a depletion of granins, coupled with increased lysosomal localization, contributing to a severe impediment in regulated DCV exocytosis in ER-stressed conditions.



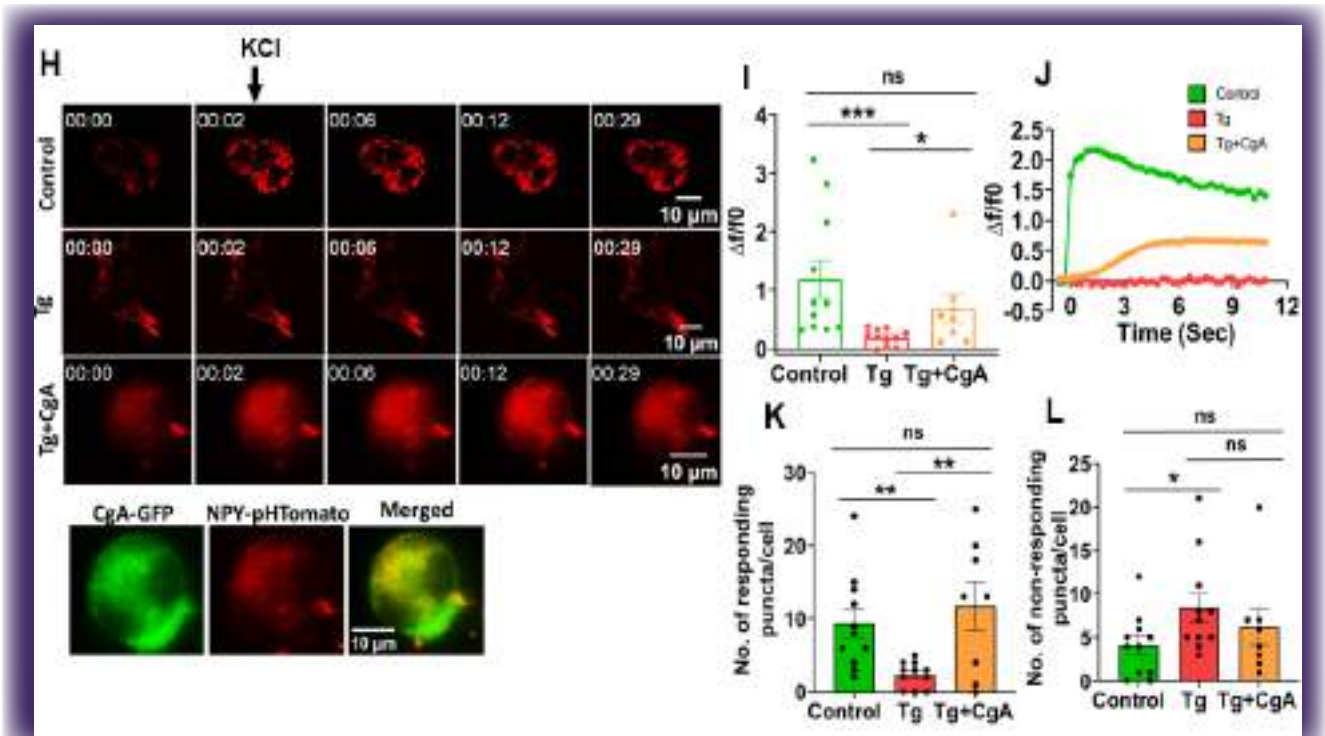


Figure 5: Change in DCV matrix proteins. **A** Transcript level of CGA in Q-RT PCR. **B** Representative blots and quantification of CGA. **C** Representative immunofluorescence images showing DAPI (blue) and CGA (green) immunostaining in mCherry-Lyso20 (red) transfected PC12 cells, scale bar=10μm (2048*2048 pixels) **D** Quantification of CGA fluorescence intensity levels. **E** Quantification showing Mander's correlation coefficient between CGA and LAMP1. **F** Representative immunoblot and quantification of PDI. **H** Representative NPY-pHTomato images in PC12 cells and chosen cell expressing both NPY and CgA. **I** Quantitative analysis of the maximum $\Delta f/f_0$. **J** Representative time traces of mean NPY-pHTomato fluorescence intensities. **(K-L)** No. of responding and non-responding puncta/cell.

CONCLUSION:

Tg-induced ER stress impeded regulated DCV and SLV content secretion in PC12 cells while retaining normal constitutive release. The response was rescued to near control levels by co-treating them with an ER stress inhibitor DHA. Molecular analysis revealed the role of SNAP25 and CGA in dictating the secretory phenotype in ER stressed cells. ATF4, an UPR activating gene also showed a negative modulatory role on SNAP25 expression by interfering with CREB activity. Re-routing of CGA to lysosomes potentially by PDI also hindered DCV cargo (NPY) release. Overall, this study shed light on the negative effect of ER stress on regulated exocytosis by altering exocytotic and Granulogenic switches.

FUTURE DIRECTION:

We are currently conducting RNAi experiments to study the effect of ATF4 knockdown in ER-stressed cells and their role in regulating SNAP25 levels. We aim to look at the PDI-lysosome interaction to understand the CGA re-routing better. We also plan to investigate the trafficking status of DCV cargo using the RUSH system. Furthermore, we

also aim to explore this phenomenon in pathological ER stress conditions.

Funding:

DBT RLS, SERB SRG, IBRO, ICGEB

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Degrees Awarded MSc (PhD):

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Ellora Sen

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MSc Student: Aastha Vartak

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Dr. Ellora Sen's group investigates how deregulated metabolism and aberrant inflammation modulates epigenetic landscape to affect genes associated with survival responses in glioblastoma- the most malignant of brain tumors. Using multipronged approaches (epigenetics, biochemistry, and cell biology), their studies demonstrated the existence of distinct metabolic-epigenetic landscape in gliomas harboring different molecular signatures that have prognostic values. With insights gained from analysis of such interactions between these crucial components, their group has been able to suggest

mechanisms that affect chemosensitivity and provide novel insights into design of effective anti-glioma therapeutic strategies.

Targeting epigenetic-metabolic landscape of gliomas: Implications in chemotherapy ▶▶▶

Background and significance

Tumors are characterized by dysregulated metabolism, aberrant inflammation and altered epigenetic landscape. Several studies have delineated the molecular circuitry linking specific genetic alterations with distinct metabolic phenotypes. The effect is bidirectional as metabolic reprogramming results in modification of the cancer genome through epigenetic, transcriptional and posttranslational modifications. Overlap of metabolic and epigenetic pathways in the tumor microenvironment affects genes



associated with resistance to chemotherapeutics in Glioblastoma multiforme (GBM) - the most malignant of brain tumors.

1. Our recent finding provided evidence of a feed-forward loop between tumor metabolite lactate, pro-inflammatory cytokine IL-1 β and circadian CLOCK/BMAL1 in glioma. To elucidate the implication of this complex interplay, we developed a mathematical model that quantitatively describes this LDHA-IL-1 β -CLOCK circuit and integrates experimental data to predict potential therapeutic targets. The study employed a multi-start optimization strategy and profile likelihood estimations for parameter estimation and assessing identifiability. The simulations are in reasonable agreement with the experimental data. Sensitivity analysis found lactate dehydrogenase (LDHA) and IL-1 β as potential therapeutic points. Mathematical models described here can provide insights to understand the complex interrelationship between metabolism, inflammation and circadian rhythm, and in identifying effective therapeutic targets. **(Sharma et al., Integrative Biology).**
2. Mutation in the metabolic gene isocitrate dehydrogenase (IDH1)-R132H is recognized as a prognostic marker in glioma. Gliomas harboring IDH1 MT are characterized by the accumulation of metabolite D-2-hydroxyglutarate (D-2-HG), and greater sensitivity to chemotherapeutics. These mutants also exhibit diminished levels of transcriptional coactivator YAP1 (Yes-associated protein 1)- an important regulator of mitochondrial dynamics and redox homeostasis. Enhanced DNA damage observed in glioma cells harboring IDH1 mutant was accompanied by reduced expression of folate receptor 1 (FOLR1). TCGA data analysis demonstrated better patient survival with reduced FOLR1 expression. Depletion of FOLR1 rendered IDH1 wild-type gliomas more susceptible to temozolomide-mediated death. By identifying the influence of YAP1-FOLR1 link in DNA damage, our findings suggest that simultaneous depletion of both could amplify the potency of DNA damaging agents while concomitantly reducing the release of inflammatory mediators. Given the involvement of FOLR1 depletion in augmenting the efficacy of temozolomide, this study highlights the novel role of FOLR1 as a probable prognostic marker in predicting responsiveness of gliomas to temozolomide and other DNA damaging agents **(Patrick et al., Cellular Signaling).**
3. Increasing evidence suggests the oncogenic role of missense mutation (AKT1-E17K) of *AKT1* gene in meningiomas. Elevated levels of glycolytic enzyme hexokinase 2 (HK2) was observed in meningioma patients with AKT1-E17K, as well as in meningioma cell line harboring the mutant AKT1. Treatment with the conventional drug of choice AZD5363 (a pan AKT inhibitor) enhanced cell death and diminished HK2 levels in AKT1 mutants. Sensitivity of AKT1-E17K cells to AZD5363 was heightened in the presence of HK2 inhibitor Lonidamine. Increased levels of inflammatory mediators observed in AKT1-E17K cells, was also dampened in the presence of AKT or HK2 inhibitors. Our findings suggest that targeting AKT-HK2 cross-talk could be exploited as novel therapeutic approach in meningiomas. **(Singh et al., Cytokine).**



Funding:

SERB-POWER (Promoting Opportunities for Women in Exploratory Research) **Fellowship**, 2022

Centre for Advanced Research (CAR) by Indian Council of Medical Research (ICMR) sanctioned project "Transforming CNS Tumor Care: Harnessing Histopathological Data, Multi-omics, and Artificial Intelligence for Precision Diagnosis and Effective Management" March 2024 (5 years). Ellora Sen Co-PI in collaboration with Vaishali Suri (PI) AIIMS, Delhi

Collaborator

Dr. Vaishali Suri, Professor, Neurosciences Centre, All India Institute of Medical Sciences, New Delhi

Degrees awarded (PhD)

Kirti Lathoria: Role of Pentraxin 3 in regulating survival responses in glioma

Shalini Sharma: Lactate-dependent inflammatory response in glioma





Mayanghlambam Dhruba Singh

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Athira Sarath, Dipti Chakraborty

Technical Assistant :

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Hari Shankar- Technician A

Project Associate: Akash Gupta

Dr. Dhruba received Ph.D. from the Department of Genetics, University of Delhi, South Campus in 2014. He received his post-doctoral training from the Pennsylvania State University and the University of Florida (2015-2020). Dhruba's lab is interested in studying human neurodegenerative disorders using *Drosophila* disease models.

Identification of novel targets of human neurodegenerative disorders using *Drosophila* models. ▶▶

Our lab uses *Drosophila* model systems to study human neurodegenerative diseases. Our lab is mainly interested in Huntington, disease, Spinocerebellar ataxia-3, Alzheimer's disease, and Myotonic Dystrophy 1. These disorders are mostly adult onset and progressive in nature and cause loss of neurons in the brain. These disorders cause loss of cognition, dementia, and movement disorders. The therapeutic drugs approved so far only improve the symptoms of the disease. However, these drugs do not slow down or cure the progression of the disease. Currently, our lab is focused on poly(Q) disorders such as Huntington's disease and Spinocerebellar Ataxia-3. We utilize a bipartite UAS-Gal4 system to express the mutant poly(Q) protein in a tissue-specific manner. Using eye-specific Gal4, we observed loss of pigmentation and roughness of the eye due to the expression of mutant poly(Q) proteins. The *Drosophila* model system is genetically highly tractable and utilizing this power of genetics, we screen to identify the genetic modifiers that could have therapeutic potential in the treatment of these neurodegenerative disorders. From our genetic screen we identified Pten could ameliorate the disease



phenotype in the *Drosophila* eye model expressing mutant Htt protein. Moreover, Pten suppressed the progressive loss of photoreceptor neurons. To check whether the neurotransmission is improved by Pten, we quantified the synaptotagmin expression which is a marker of synaptic vesicle in *Drosophila* photoreceptor neuron axon projections into the brain with syt-GFP puncta (Fig. A-D). We observed that in mutant Huntingtin-expressing flies the level of syt-GFP was significantly reduced in the axon terminals (Fig. 1C). However, the expression of Pten-RNAi significantly improved the presence of syt-GFP in the axon projections and it was as comparable to the control flies (Fig. 1D). Subsequently, we investigated whether Pten-RNAi improves the degeneration of brain structure in adult flies. We employed Elav-Gal4 which expresses throughout the nervous system. The expression of mutant Htt protein in the brain caused the degeneration of mushroom body neurons which is the learning and memory center of the *Drosophila* brain. In control flies, the mushroom body when stained with the FasII neuropile marker gives a peculiar mushroom-like structure with α , β , and γ lobes (Fig. 1E-F). However, in mutant Htt-expressing flies, truncation and thinning of mushroom body lobes were observed (Fig. 1G). The loss of mushroom body lobes was restored due to the knockdown of Pten (Fig. 1H). Furthermore, we investigated their lifespan by scoring the number of dead flies starting with 100 flies in each genotype. We observed that the control flies live for about 70-80 days. However, the Flies expressing mutant Htt have shortened lifespans and the knockdown of Pten improved the lifespan of these flies (Fig. 1I).

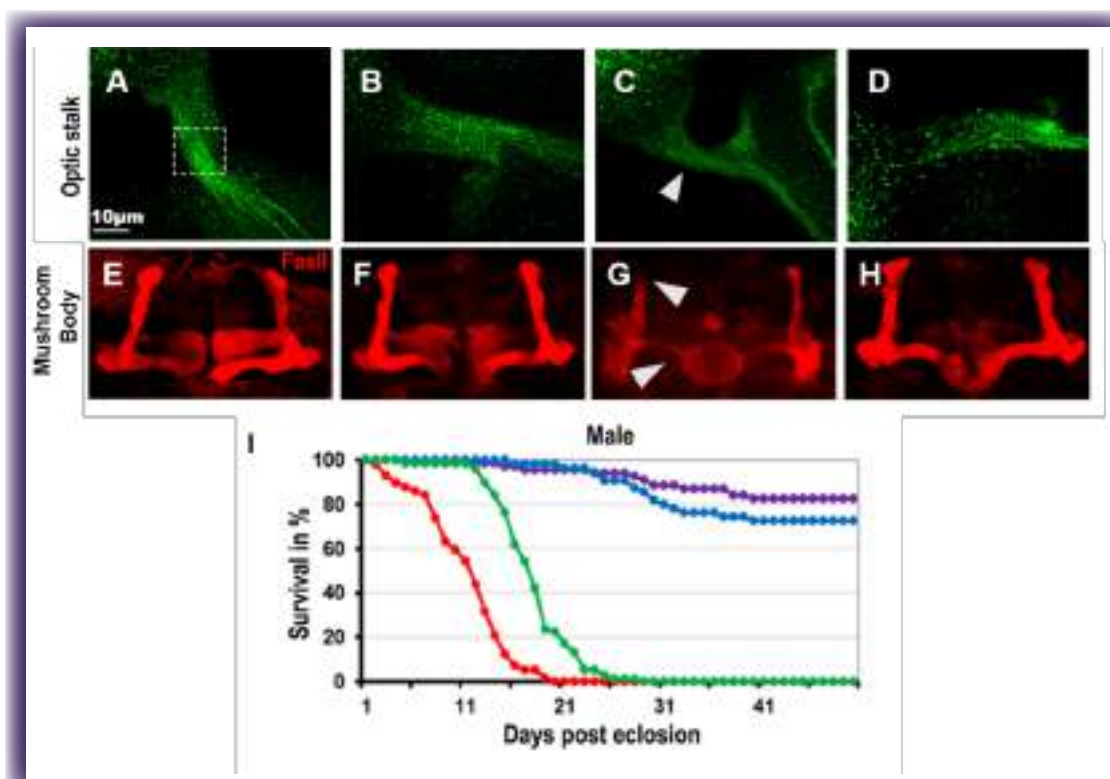


Figure 1. Knockdown of Pten improves the HD phenotype in *Drosophila*. (A-D) *Drosophila* eye disc expressing syt-GFP. (A) Control. (B) GMR-Gal4; Pten-RNAi. (C) Expression of mutant Htt causes loss

of syt-GFP in the axon projection. (D) Pten Knockdown improved the distribution of syt-GFP. (E-H) *Drosophila* brain stained with anti-FasII. (E) Control. (F) Elav-Gal4; Pten-RNAi. (G) Overexpression of mutant Htt caused loss of mushroom body lobes. (H) Knockdown of Pten suppresses the degeneration of the mushroom body. (I) Kaplan-Meier plot indicating the lifespan of flies.

Furthermore, we wanted to investigate how the pharmacological intervention of Pten inhibition could suppress poly(Q) toxicity. We used VO-OHPic which is a potent Pten inhibitor and administered in their food. We observed that feeding of Pten inhibitor has significant improvement in reducing the poly(Q) aggregate in larval eye disc (Fig. 2A-C, D). Moreover, the reduction of aggregate was accompanied by the reduction of apoptosis in the eye disc (Fig 2A-C, E). Our study indicates Pten is a potent genetic modifier with potential therapeutic application for the treatment of Huntington's disease.

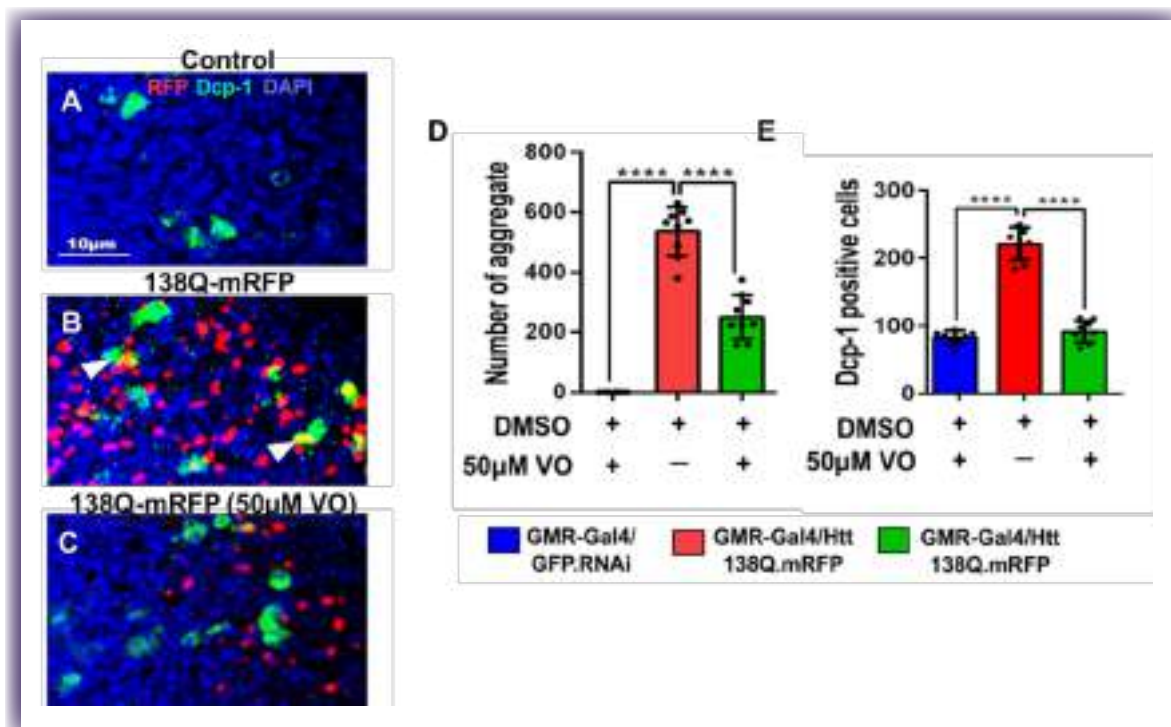


Figure 2. Pten inhibitor VO-OHPic improved the HD phenotype in the Fly model. (A-C) Third instar eye disc stained with anti-Dcp1 antibody. (A) Control. (B) Fly-expressing mutant Htt has a large number of poly(Q) aggregates and increases the level of Dcp-1. (C) Administration of Pten inhibitor reduced the poly(Q) aggregates and Dcp-1 staining in Htt-138Q expressing flies. (D) Bar Graph representing the level of poly(Q) aggregates. (E) Bar graph representing the level of Dcp-1 positive puncta in the eye disc. One-way ANOVA, error bars represent mean $\pm$ SE (* P <0.05, *** P <0.001). Green: Dcp-1, Red: Htt138Q.mRFP and Blue: DAPI.

Funding:

This work is supported by NBRC Core funds and the SERB-EEQ grant (2024-2026).

Collaborator:

Dr. Bhavani Shankar Sahu, NBRC, India.



Pankaj Seth

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Masters Dissertation : Sudipto Chaki

Project Assistants : Ajay Pal, Saumya Rastogi

Technical Assistants : Durgalal Meena and Naushad Alam

Cellular and Molecular Mechanisms of Zika and SARS-CoV2 Virus Induced Neuropathogenesis ▶▶

Our laboratory focuses on understanding the neuropathogenesis that culminate into neurological deficits by viruses such as HIV-1, Zika and the novel corona virus, SARS-CoV2.

The Zika virus (ZIKV) is a pathogen affecting populations in various countries, including India. ZIKV induces a neurodevelopmental disorder called microcephaly, where approximately 7-10% of infants are born with abnormally small sized head to mothers that contract this virus during their first trimester of pregnancy, as most of the head development occurs in the first three

understanding of the pathways leading to microcephaly is crucial for preventing such *in utero* defects during fetal development, and hence we have deeply invested our research efforts into unravelling cellular and molecular mechanisms of Zika virus induced microcephaly.

Using a dual cell model of blood brain barrier (BBB), comprising primary culture of human brain microvascular endothelial cells (hBMECs) and human astrocytes established in a transwell apparatus, we have now unravelled previously unknown mechanisms of virus induced neuropathogenesis, particularly in role of astrocytes and BMECs in Zika virus mediated damage. Findings of our current experiments have revealed that Zika virus E protein increases ER stress pathways in hBMECs. More precisely, we have observed



months after conception. A comprehensive While in increase in the ER stress sensors such as Binding immunoglobulin Protein (BiP) and phospho-protein kinase like-ER kinase (pPERK). depth studies are in progress, we have better understanding of the mechanisms that may alter the levels of the tight junction proteins that results in disruption of the BBB integrity. We also plan to do the small RNA sequencing and study their profile in hBMECs. Furthermore, we observed that the non-structural proteins like NSP4A and -4B alter the properties of human neural stem cells by promoting autophagy and leading to an aberrant neuronal differentiation, which may also contribute to microcephaly in infants.

We employ cutting edge cell and molecular biology tools such as time lapse microscopy, live cell calcium imaging in human brain cells, gene and protein expression studies, lncRNA and miRNA sequencing based assays, as well as *in silico* approaches to address our research questions. In addition to this, we validate the *in vitro* findings from human brain cells in post-mortem brain sections, based on availability of the brain sections.

The other virus that has gathered our research interest is the novel corona virus, SARS-CoV2, that caused the COVID-19 pandemic. Although the pandemic has subsided and mortality rates are far lower now, it has a long-lasting morbidity among most of the COVID-19 survivors. Worldwide, the Post-acute sequelae of COVID-19 (PASC) is a major problem affecting a large population of Long COVID-19 survivors. Majority of these morbidities are related to the nervous system which are also referred to as “Brain Fog”. Several postmortem studies have demonstrated presence of viral particles, viral proteins or viral RNA as well as signatures of inflammation and neuronal damage in different areas of brain. It is critical to recognize that the manifestations of post-acute sequelae impact the quality of life of survivors, and detailed mechanisms for neurological consequences of PASC remain unclear. Considering our interest in understanding the virus induced neuropathogenesis, our lab is well placed to take up the challenges to study how COVID-19 virus may be causing neuronal damage. Currently, we are studying the underlying factors contributing to these long-term symptoms and to develop effective management strategies against PASC related effects. Although various model systems have been employed to investigate post-COVID effects, human-based cell culture systems (2D and 3D models) have gathered highest priorities above others due to their effectiveness in depicting diseases and their closeness to the real-life situations.

An understanding of detailed molecular mechanism of virus-mediated neuronal damage are warranted for designing potential therapies. We screened the structural and non-structural SARS-COV2 viral proteins for cell death, and found that Orf6 protein, an accessory protein of the virus known for inhibiting nuclear export and blocking host cell's interferon response, causes maximum death of primary human neurons. Upon further investigations, we found that in Orf6 expressing neurons, necroptotic cell death pathway was activated among other coronavirus-mediated cell death pathways (apoptosis, pyroptosis and autophagy). To validate this, we infected primary cultures of



human neurons with the whole virus and determined cell death mechanisms. Further we used histological staining techniques for checking the expression of necroptotic cell death markers in the post mortem brain sections of COVID-19 patients. Our findings so far suggest that the neuronal damage following exposure to the virus itself or overexpression of Orf6, leads to a necroptotic cell death of neurons. This has been confirmed in postmortem brain sections of COVID-19 patients. We are continuing to delve deeper into the mechanisms of necroptotic neuronal death to suggest prevention of neuronal injury in long COVID patients.

Our lab maintains its leadership in studying virus induced neuropathogenesis using physiologically relevant experimental models that represent the disease and offer insights into the cellular and molecular mechanisms of disease pathogenesis in cells of human origin. We routinely differentiate human astrocytes, neurons and oligodendrocytes from human Neural stem cells (hNSCs). We are currently focusing on developing human brain organoids from iPSCs to have a 3D culture system.

Our research efforts have successfully addressed numerous challenges in comprehending the molecular mechanisms related to important neurotropic viruses that impact populations.

Funding:

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Dr. Mandal, an expert of Neuroimaging methodology developments and inventor of anti-oxidant glutathione as an early diagnostic biomarker for Alzheimer's disease. His work on development of Indian Brain template, BRAHMA, database, and analytics "SWADESH". His work on brain signal methodology development "KALPANA" was awarded US (2022), European patent (2024). His AI based early prediction of AD methodology is awarded Indian patent in 2024. Accelerated brain volume loss caused by anti-beta-amyloid drugs was also reported, indicating the involvement of other factors responsible for triggering the disease.

The Identification of an Early Biomarker of Alzheimer's Disease Through State-of- the-art Brain Imaging Modalities and Validated through Transgenic Animal Model study.▶▶▶

Alzheimer's disease has created a huge impact on millions of people worldwide due to impairment of activities of daily living. At present, with improved scientific and clinical understanding, the diagnosis of AD has improved but the exact cause of AD is not yet identified. Amyloid plaque formation and tau phosphorylation were two dominant hypotheses for the three decades. Current therapeutic agents for AD temporarily improve symptoms but do not alter the underlying disease modifying course.



Accelerated brain volume loss caused by anti-beta-amyloid drugs was also reported, indicating the involvement of other factors responsible for triggering the disease. One of these factors is described is the oxidative stress (OS) hypothesis, which suggests that the brain remains healthy as long as 'free radicals' that are produced in the course of various biochemical are kept in check by anti-oxidants. The OS hypothesis predates the nearly quarter-century investigation into amyloid-linked causes of AD dementia but of late has gained greater significance with researchers around the world. The brain remains healthy as long as there is a balance between the pro-oxidative molecules and antioxidant to neutralize them. Glutathione (GSH) is the master antioxidant among the available anti-oxidants. The levels of GSH in the hippocampus is an important research focus.

Why Hippocampus:

Hippocampus participates in learning and memory and aids in the generation of episodic memory. MRS studies have found significant depletion of GSH as well as non-significant increase of susceptibility (e.g. iron) in the hippocampal region of MCI patients. However, susceptibility (e.g. iron) level increases significantly in the hippocampal area in AD compared to MCI. Autopsy studies have indicated that GSH level is significantly depleted in the hippocampal area of MCI patients compared to healthy age matched tissue of control subjects.

The comprehensive journey of identification of the biomarker of AD is shown in the Figure 1

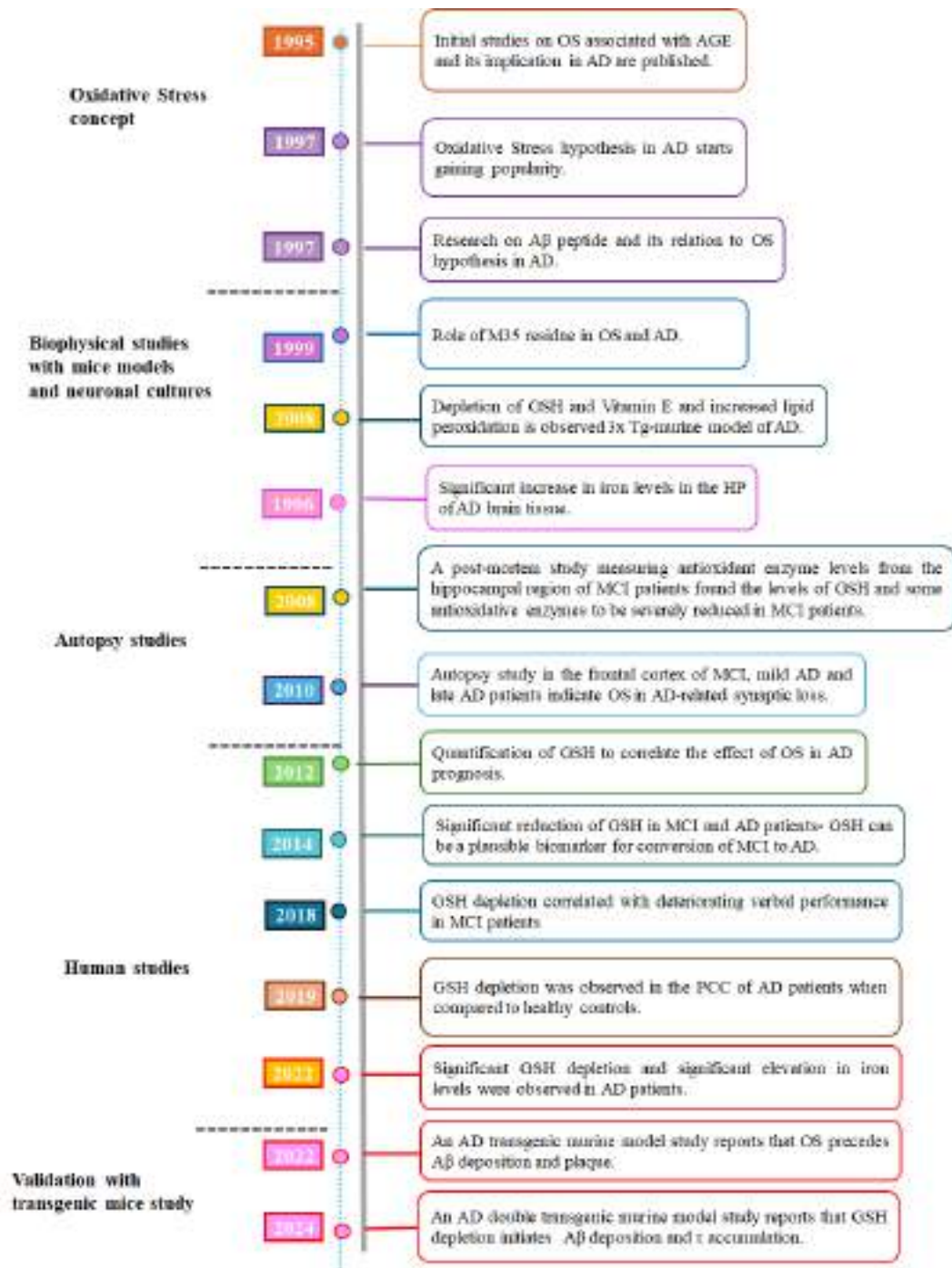


Figure 1: Flowchart illustrating the major chronological development of the OS hypothesis in AD research as mentioned from various research work. A review of the medical literature found initial research dating back to the early 1990s with a focus on advanced glycation end (AGE) products and their role in accelerating the oxidative load on the brain. Subsequently, the OS hypothesis was further advanced. A study with the AD mouse model reported significant reduction in GSH levels and an increase in lipid peroxidation products in the AD mouse brain. Autopsy studies on human AD brains have also reported significant elevation of iron levels or a reduction in GSH levels. GSH depletion is now established as a major phenomenon in AD pathology. Supporting these clinical observations, two animal model studies validated that GSH depletion is early stage in AD prior to plaque formation and tau phosphorylation.

The possible cause of AD is proposed in Figure 2

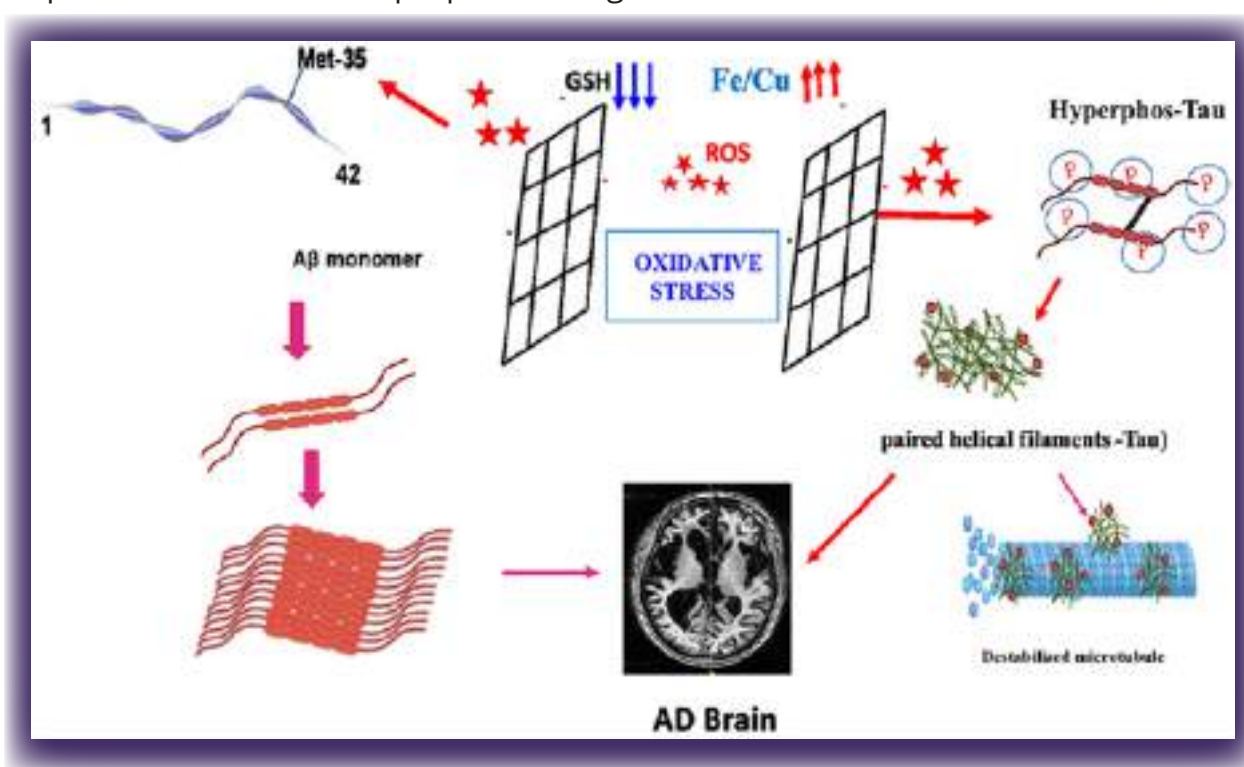


Figure 2: Diagrammatic presentation of AD disease process based on oxidative stress.

Validation of GSH depletion as an early process in AD from two independent studies from other centers and our work is cited in those animal model studies listed below.

1. Pontrello, C. G.; McWhirt, J. M.; Glabe, C. G.; Brewer, G. J. Age-Related Oxidative Redox and Metabolic Changes Precede Intraneuronal Amyloid-beta Accumulation and Plaque Deposition in a Transgenic Alzheimer's Disease Mouse Model. *J. Alzheimer's Disease* 2022, 90 (4), 1501–1521.
2. Hashim, K.N.B, Matsuba, Y, Takahashi, M., Kamano, N., Tooyama, Saido, T.C. and Hashimoto, S., Neuronal glutathione depletion elevates the Ab42/Ab40 ratio and tau aggregation in Alzheimer's disease mice, *FEBS Letters* 2024 doi:10.1002/1873-3468.14895

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Ph.D. from Institute of Life Science, Bhubaneswar, Odisha & post-doctoral positions from National Institute of Immunology, New Delhi (1997-1999)/ University of Southern California, U.S. (1999-2003)/ McLaughlin Research Institute, Great Falls, U.S. (2003-2007). Dr. Giri's research is focused on understanding the mechanisms underlying the neuropathology of Alzheimer's and prion disease using animal and CNS stem/progenitor cell-based models.

Differential expression of death receptors, their ligands, and adaptors as molecular signatures of neuropathology in a mouse model of prion disease ▶▶▶

Prion diseases are transmissible and fatal neurodegenerative diseases characterized by accumulation of a misfolded form of a host-encoded protein (PrP^{Sc}), astrogliosis, microgliosis, spongiosis, and extensive neuron loss. How PrP^{Sc} accumulation regulates cellular pathways leading to neuronal cell death is unclear. The misfolding of PrP^{C} to PrP^{Sc} is reported to produce neurotoxic stimuli, potentially through oligomerization of $\text{PrP}^{\text{C/Sc}}$ on the cell surface and interacting with unknown transducing partners. Moreover, PrP^{Sc} aggregate has been implicated in the activation of caspase-8 and caspase-3, resulting in apoptosis. Collectively, the synthesis of proinflammatory cytokines by activated microglia, oligomerization of $\text{PrP}^{\text{C/Sc}}$ on cell surface and activation of caspase-8 hint towards the activation of death receptor (DR) pathway/s in prion diseases. expresses seven DRs, which are TNFR1, Fas, DR3, DR5, DR6, p75NTR, and EDAR. Upon the ligation of DRs,



the fate of a cell is regulated by the levels of four major DR adaptors, such as, TRADD, TRAF2, FADD, and RIPK1. Currently, no report is available on DR3, DR5, DR6, TL1A (ligand of DR3), TRAIL (ligand of DR5) and all 4 major DR adaptors in any prion diseases. This research gap is notable and investigated in this study.

To understand this complex and multifaceted nature of neuropathology of prion disease, I dissected the role of 16 death receptor (DR) factors using a C57BL/6J mouse model and significantly advancing our understanding of this complex neurodegenerative disorder. Manifestations of clinical and neuropathological features in this mouse model range from abnormal gait, increased PrP^{Sc} accumulation, and pronounced astrocytosis (Figure 1) are in line with earlier reports.

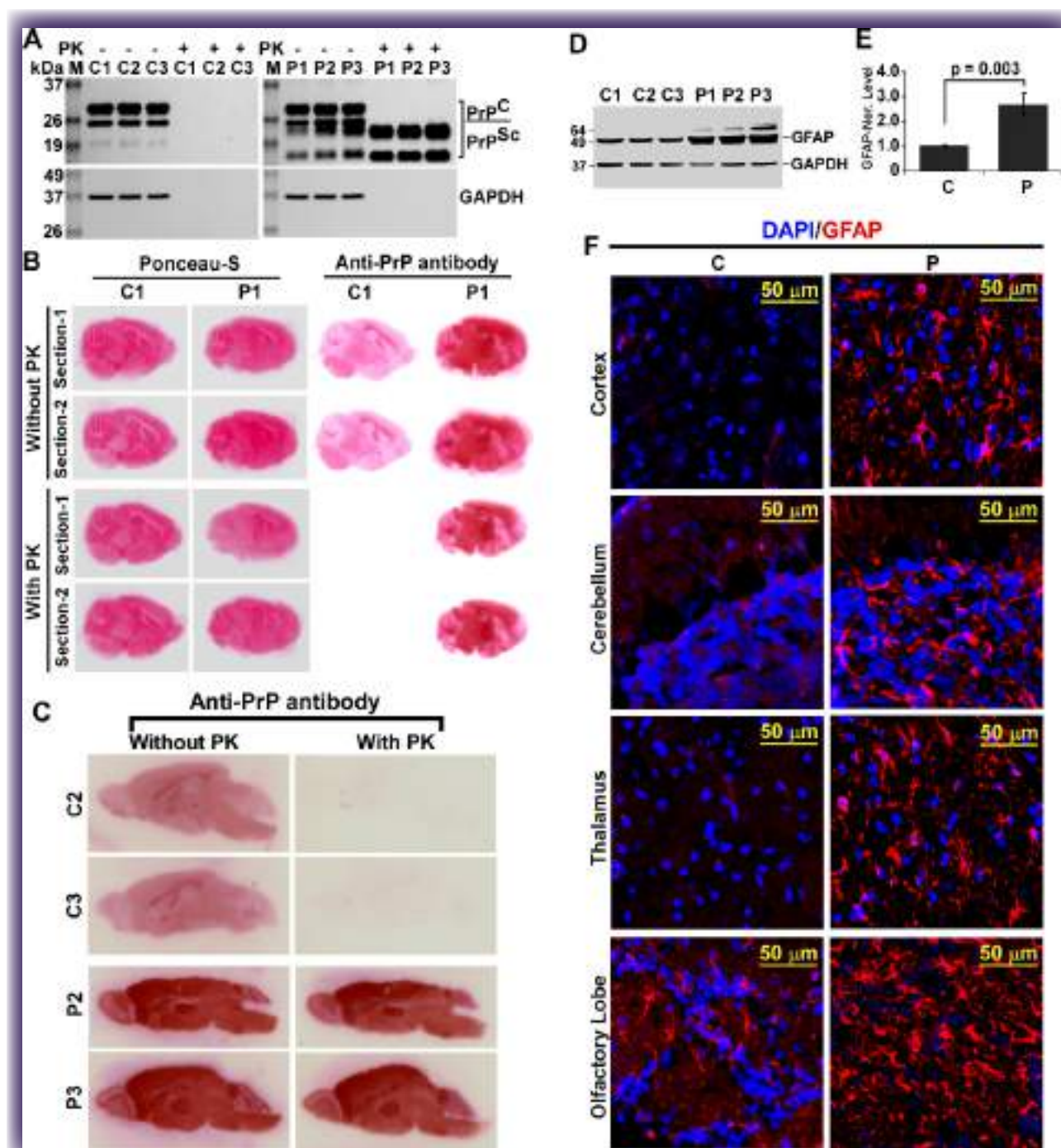


Figure 1: Establishment of neuropathologies in C57BL/6J mouse prion disease. A Western blot analysis of PrP^C/PrP^{Sc} in control (C) and prion-infected (P) mice brain homogenates (MBH). PrP^C and GAPDH are seen in normal MBHs without PK treatment but degraded upon proteinase-K (PK) treatment (Left panel). In right panel, both PrP^{C/Sc} and GAPDH are observed in samples without PK treatment but only PrP^{Sc} but no GAPDH in PK-treated prion MBHs. B, C Brainblot analysis of three control and three prion infected mice brain sections shows increased PK-resistant PrP^{Sc} in all prion-infected but none in any control mice brain sections. D, E Prion-diseased mice brains show increased GFAP level than control mice. Each histogram represents mean $\pm$ standard deviation of a set of three mice brains from control and prion groups. $P \leq 0.05$ is considered statistically significant (two-tailed unpaired t-test). F Immunofluorohistochemical analysis shows increased GFAP +ve cells (astrogliosis) in prion-infected mice brains than controls. DAPI: 4',6-diamidino-2-phenylindole. Collectively prion infected mice exhibit robust neuropathological features of prion diseases.

Moreover, increased expression of caspase-8 and caspase-3 proteolysis underscores its position downstream of caspase-8 activation, strengthening the involvement of DR pathways in this prion disease.

Results from the expression of DR factors suggest possible inhibition of cell survival pathways from TNFR1, DR3, DR6, p75^{NTR}, Fas and partially from DR5 due to marked reduction in TRADD and TRAF2 expression, the susceptibility of DR3 and DR5 expressing cells indicated by TL1A and TRAIL overexpression, and steady FADD levels coupled with elevated FasL, Fas, and caspase-8 pointing towards apoptotic cell demise. Enhanced RIPK1 proteolysis with caspase-8 cleavage signature confirms apoptosis, while high levels of RIPK1 and caspase-8 alongside constant FADD may induce apoptosis through Fas, DR5, TNFR1, DR3 and p75^{NTR} ligation. Overexpressed TNFR1, Fas, FasL, and stable TNF α levels combined with reduced DR5 might promote astrogliosis and microgliosis in a TRADD-independent, RIPK1-dependent manner. Novel glycosylated and oligomeric TNFR1 and FAS isoforms imply enhanced ligand binding, and diminished monomeric, oligomeric, and glycosylated soluble TNFR1 and Fas isoforms indicate impaired neuroprotection, contributing to cell death in prion diseases. Lastly, neurodegeneration in prion diseases appears to be driven not by a single death receptor pathway but by multifactorial, multi-receptor-mediated cell death processes, as evidenced in this mouse model of prion disease (Figure 2:).

The present study narrates a complex network of death receptors, their ligand and adaptor proteins elaborately intertwined into the multifarious nature of prion disease neuropathology, thereby laying a robust foundation for future explorations into their functional association with prion-induced neurodegeneration and potential avenues for targeted therapeutic interventions. Moreover, findings from this research may help in designing and developing decoys for TL1A, TRAIL and FasL to restore DR3, DR5 and Fas

pathways respectively in experimental prion diseases. Similarly restoring RIPK1, TRADD and TRAF2 expression would be direct clinical implications on prion diseases. Notably, deep brain stimulation of cortico-striatal pathway may restore motor deficit in prion patients, which would be another clinical implication of the data obtained from the present mouse model of prion disease.

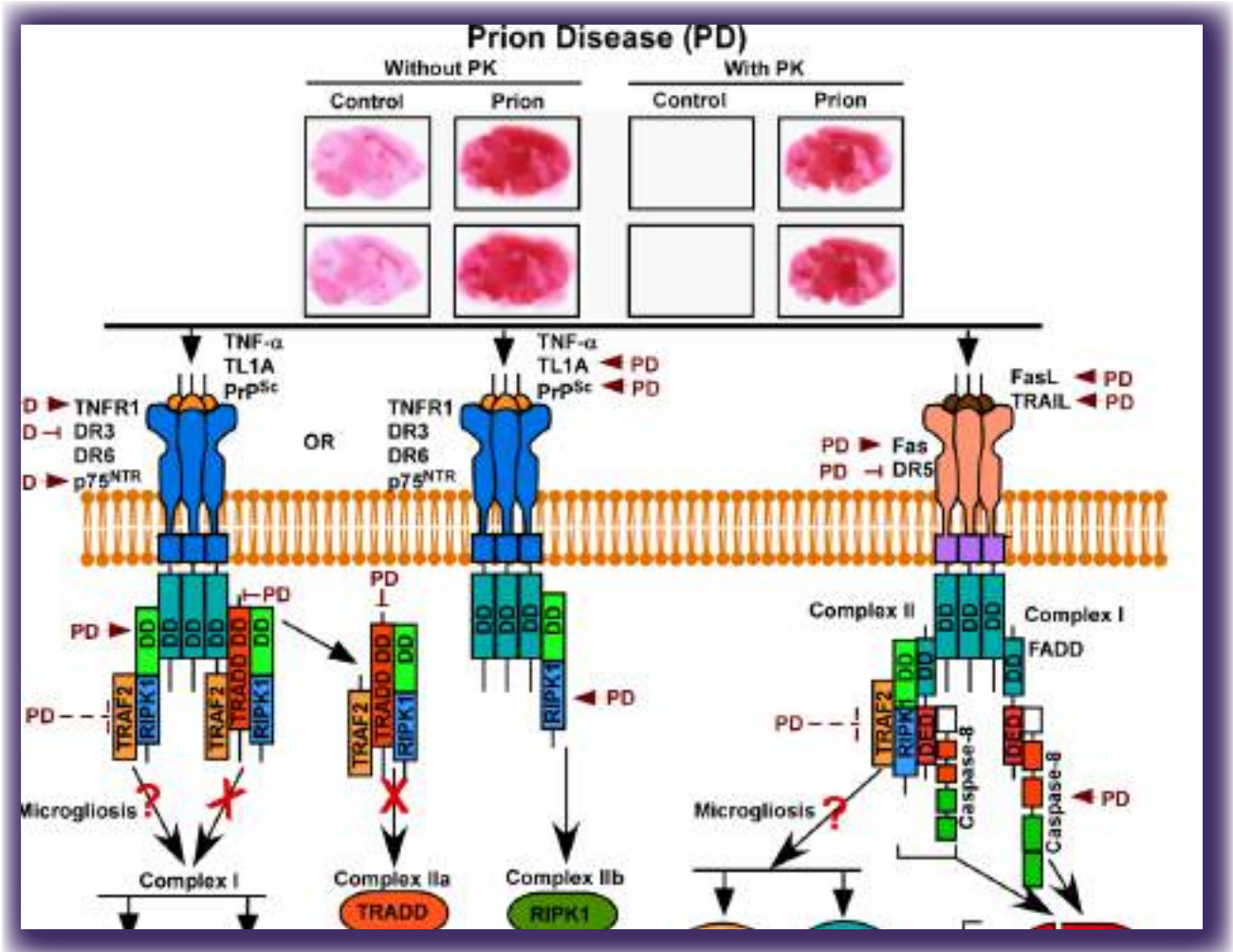


Figure 2: Schematic representation of probable mechanisms regulated by major death receptor pathways, based on altered expression pattern of DRs, their ligands and adaptors in C57BL/6J mouse prion disease. Upon binding to their specific ligands, TNFR1, DR3, DR6, and p75NTR activate primarily NF κ B, MAPK and JNK-mediated proinflammatory and antiapoptotic pathways by recruiting TRADD-RIPK1-TRAF2 or TRADD-RIPK1 or RIPK1-TRAF2 adaptors to form complex-I. Similar complexes can be formed from TRAIL-DR5 ligation. Reduced expression of TRADD and TRAF2 but increased proteolysis of RIPK1 clearly suggests the inhibition of complex-I and -IIa formation, upon the ligation of these DRs. Thus, increased expression of TNF α , TL1A, PrP^{Sc}, full-length RIPK1 along with sustained level of FADD clearly suggest the formation of complex-IIb from TNFR1, DR3 and DR6/p75NTR respectively by forming complex with RIPK1-FADD-caspase-8. Ligation of Fas and DR5 by their specific ligands, mediate mainly apoptotic pathway by forming complex with either FADD-caspase-8 or RIPK1-FADD-caspase-8. Thus, higher levels of FasL and TRAIL in this study suggest activation of apoptosis. Furthermore, increased cleavage of RIPK1

and caspase-3 indicate caspase-8 activation, which are established molecular markers of apoptosis and inhibitors of necroptosis. Increased expression of FasL, Fas and RIPK1 suggests proinflammatory and antiapoptotic response from macrophages (microglia in brain). Similar responses can be achieved from increased expression of TNFR1, RIPK1 along with sustained levels of TNF α in microglia in a TRADD independent manner. Finally, reduced expression of DR5 along with increased expression of TRAIL can be considered as molecular signature of astrocytosis in mouse prion disease. An arrow ($\rightarrow$) represents either increased expression of a DR factor or activation of a specific pathway. A cross road ($\vdash$) suggests either reduced expression of a DR factor or inhibition of a specific pathway. Any factor without any an arrow or cross-road is considered as static expression in prion disease. Finally, the inhibition of a pathway resulting from prion disease is overlayed by a cross in red color (X).

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Collaborator:

Prof. George A Carlson for providing RML scapie prion





Soumya Iyengar

Research Fellow: Anjali

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Development of the Human Auditory Cortex during the Sensitive Period for Speech and Hearing ►►

Research in my laboratory focuses on understanding changes in the brain involved with cognition in corvids.

We also study the effects of the endogenous opioid system on modulation vocalization and vocal learning. A third project focuses on studying structural changes in the developing human auditory cortex.

It is very important to have a detailed knowledge of the sensitive periods of plasticity in the human auditory cortex, given that cochlear implants are not always successful as interventions for deaf children. Understanding how the auditory cortex develops also provides the neural basis of the ability to learn multiple languages. We had earlier demonstrated that excitatory connections, that is, the cortico-cortical synapses appear to undergo pruning by ~7 postnatal years, which correlates with the sensitive period for learning speech, whereas thalamocortical synapses undergo pruning between adolescence and adulthood (Pundir et al., 2016).

Recent studies in our lab have focused on examining the establishment of and structural changes in somatostatin-positive interneurons (SOM+, one of the main subtypes of inhibitory interneurons) across development in the human auditory cortex. Stereological counts of SOM+ neurons (**Figure 1A**) demonstrated that they were present across different layers in all parts of the human auditory cortex (**Figure 1B**). Furthermore, we observed that a number of SOM+ interneurons migrated into the auditory cortex from



the underlying white matter between the third trimester and the first postnatal year. The final pattern of SOM+ neuronal distribution appeared during childhood (4 years), wherein their density was the highest in L2, followed by that in L3. Taken together, our results suggest that the patterns of distribution of SOM+ neurons are established by childhood. After an initial increase in the density of SOM+ interneurons between the third trimester and childhood, there was a significant decrease in this measure between childhood and adolescence.

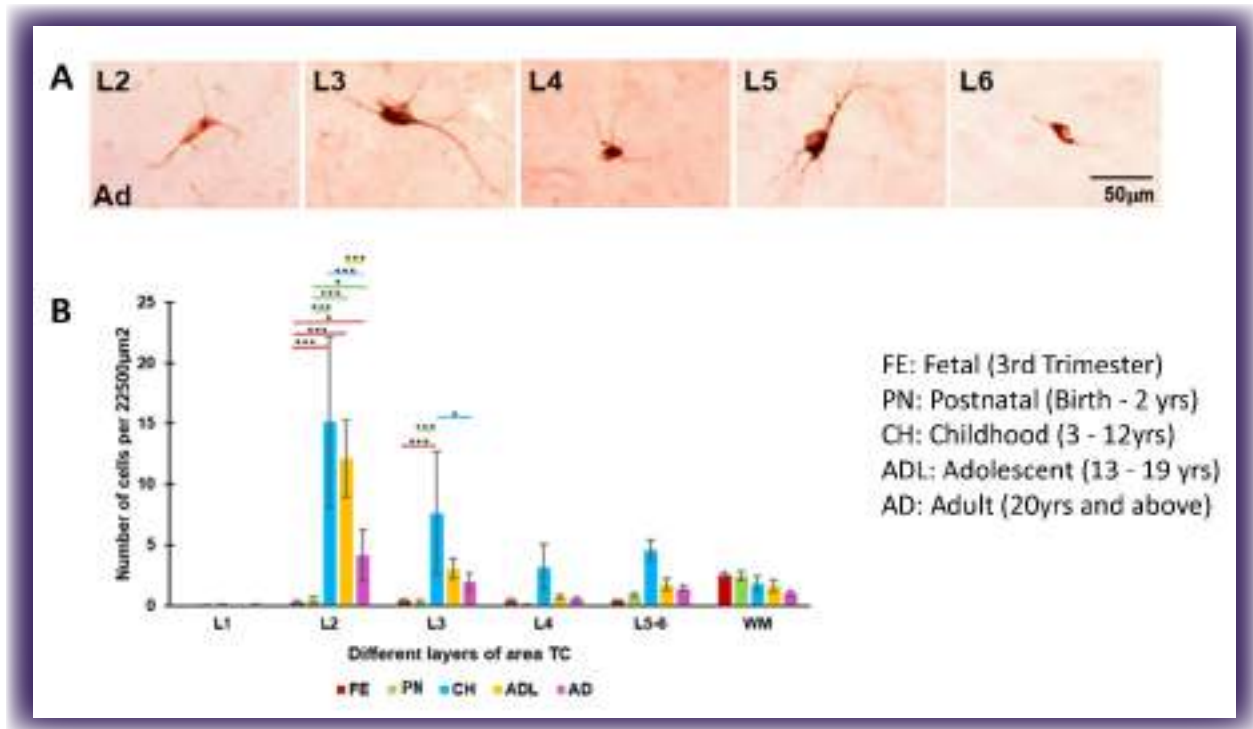


Figure 1: (A) Somatostatin-positive neurons in Layers 2 – 6 of the adult (Ad) auditory cortex, Scale bar, 50µm. (B) There was a significant increase in the number of SOM+ neurons in the human auditory cortex between the third trimester and childhood (3-12 years). The greatest number of SOM+ neurons were present in Layer 2 during childhood, followed by that in adolescence.

Besides quantifying SOM+ neurons in the auditory cortex across development, we also performed three-dimensional reconstructions of these neurons. Our results demonstrated that the number of branches and neurite length of these neurons decreased over the course of development with SOM+ neurons in adults having the least number of branches and lowest neurite length (**Figure 2**). Our findings suggest that SOM+ interneurons undergo pruning and become less complex over the course of development, likely coinciding with the development of specific inhibitory synapses in the auditory cortex.

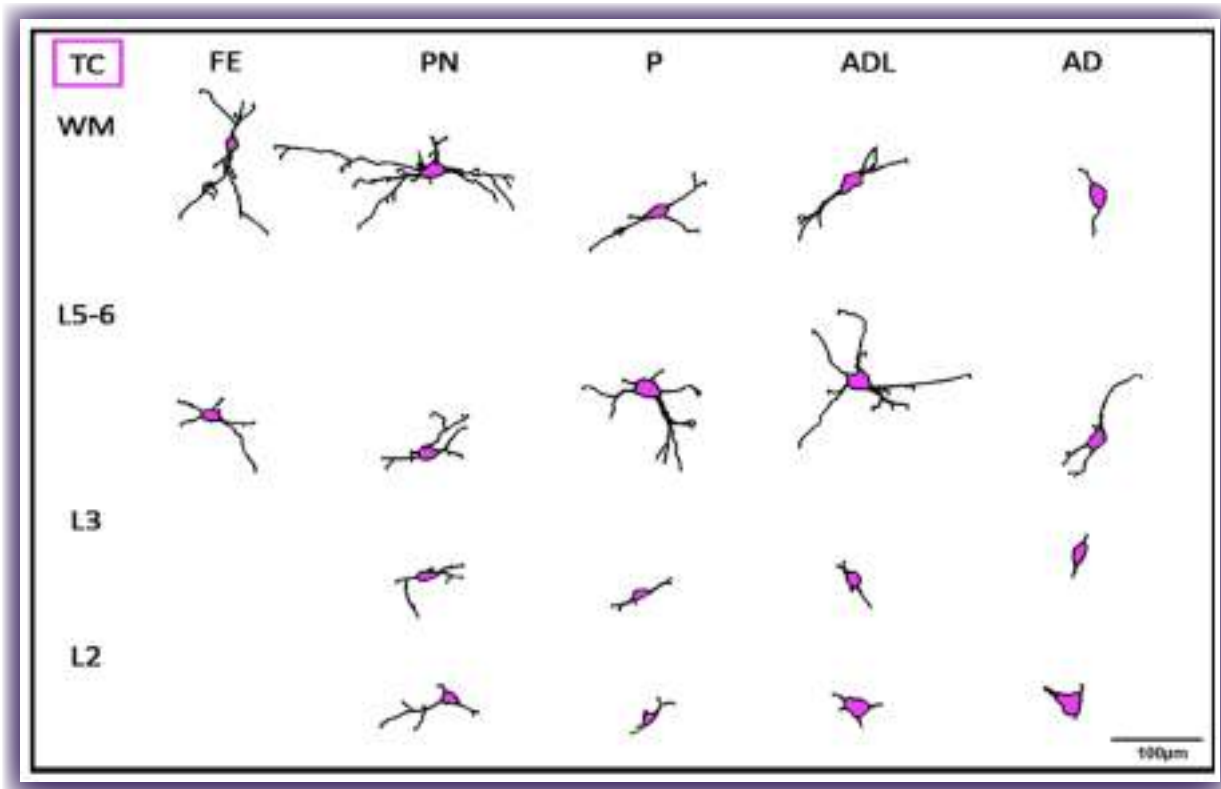


Figure 2: Three-dimensional reconstructions demonstrated that the number of processes of SOM+ neurons increased between the third trimester and early postnatal period, after which neurites underwent pruning. The schematic demonstrates SOM+ neurons in Layers 2-6 and the underlying white matter (WM) in the primary auditory cortex (TC) at different stages of development. Scale bar, 100µm.

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Pooja Parishar (Integrated MSc-PhD student): Awarded MSc and PhD degrees for her dissertation research entitled 'Neural Correlates of Learning and Decision-Making in Corvids', 2023.

Co-supervised Atanu Datta (Integrated MSc-PhD student): Awarded MSc and PhD degrees for his dissertation research entitled 'The effect of dorsal column lesions in the primary somatosensory cortex and medulla of adult rats', 2023.





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M.Sc. dissertation: Shreyas Miraj
(M.Sc. degree awarded in July 2024),
Gauri Shintre

Technical Assistants:

Mr. D. Narender, Mr. Kamlesh Bisht

Obtained his PhD from Jawaharlal Nehru Centre for Advanced Scientific Research (JNCASR), Bangalore, where he worked on fundamental mechanisms of eukaryotic transcription. His postdoctoral research on protein synthesis-dependent control of synaptic plasticity was conducted in Yale University, Connecticut and the University of California, Santa Barbara, USA.

The lab endeavours to identify RNA-dependent mechanisms underlying local protein synthesis at the synapse during synapse development and plasticity. We specifically focus on non-coding RNAs, including miRNAs and long noncoding RNAs to delineate how RNA-driven paradigms modulate neurons during various aspects of cognition, including learning

and memory. We are also working on discovering aberrations in the synaptic noncoding RNA population in neurodevelopmental disorders, such as autism spectrum disorders.

Long non-coding RNAs in memory and anxiety ►►

Research Highlights:

(I) Regulation of synaptic translation by synapse-enriched lncRNAs.

The presence of long non-coding RNAs (lncRNAs) at the synapse led us to explore how synaptic activity influences their distribution in dendrites. We utilized contextual fear



conditioning (CFC), a memory paradigm to assess activity-induced lncRNA distribution following single-trial associative learning. We analyzed the local levels of lncRNAs from synaptoneurosomes (SN) isolated from the dorsal hippocampus after measuring the freezing response three hours post-training, in the temporal window when translation of plasticity-related proteins is known to occur. Our qRT-PCR analysis showed that levels of a novel lncRNA, H16Rik (named as SynLAMP), increased after CFC, while Snhg8 levels decreased in the synapse. However, the overall expression levels of these lncRNAs remained unchanged in the dorsal hippocampus, suggesting that expression changes might occur in specific cell types rather than in CA1 neurons.

The increase in SynLAMP following CFC led us to investigate its role in *de novo* translation processes. We measured the activity-dependent translation in glutamate-stimulated neurons expressing shRNAs against SynLAMP using FUNCAT, along with MAP2 immunostaining to mark dendrites. Control RNAi neurons showed increased translation in both soma and dendrites, while SynLAMP RNAi resulted in decreased translation in both locations.

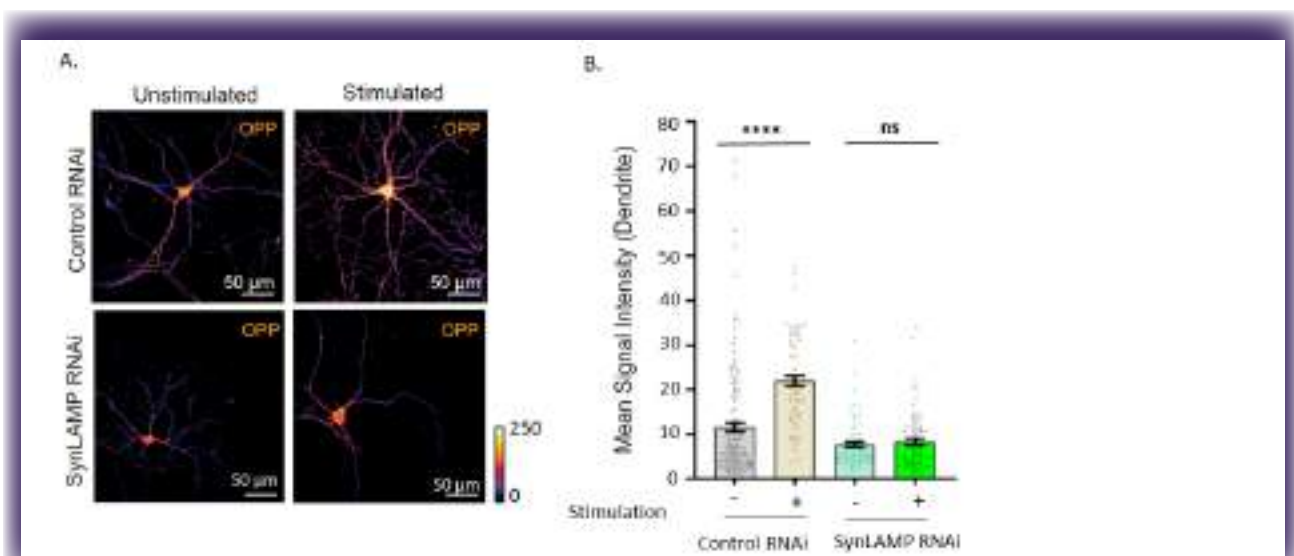


Figure 1: SynLAMP RNAi abrogates dendritic translation. (A) Representative heatmap showing the FUNCAT signal intensity upon glutamate stimulation. Top panel represents Control RNAi neurons and bottom panel represents neurons with SynLAMP RNAi. (B) Histogram of mean FUNCAT signal intensity in neurons with Control RNAi and SynLAMP RNAi respectively. ** $p < 0.005$ and ns = not significant. Obtained from Samaddar et al, 2023, bioRxiv.**

We specifically degraded SynLAMP at the synapse using a CRISPR-based construct (CIRTS) and identified its role in the local translation of one of its targets, CamKIIa, at the synapse, using a combination of puromycin labeling and proximity ligation assay. For long-term memory formation, *de novo* translation at the synapse heavily relies on RNA transport from the soma to dendritic spines. Transcripts associated with RNA-binding proteins (RBPs) and translation regulators are packaged into "RNA granules" that travel

along dendrites. The activity-dependent release of RNA and translation factors from these granules facilitates their transition from a non-translating to an actively translating state. We further find that SynLAMP acts as a sponge for translation repressors, and its regulation of FUS promotes translation from transcripts containing FUS binding sites.

(II) The Role of lncRNAs in Anxiety and Memory Due to Early Life Stress

Early life stress, results in chronic physical and psychological challenges, significantly affecting adult life. Research indicates that such stress can disrupt synaptic functions in the hippocampus, resulting in memory issues and increased anxiety. Based on our ongoing findings that lncRNAs play a role in regulating synaptic gene expression and memory, we hypothesized that early life stress might influence lncRNA-mediated gene expression, contributing to memory deficits and anxiety. To explore this regulatory mechanism, we examined the expression and activity of lncRNAs in a maternal separation model (a recognized early life stress paradigm) and assessed the physiological relevance of lncRNA functions related to spatial memory and anxiety. Our preliminary study revealed that maternal separation increases the expression of the synaptic lncRNA, Gas5, in the hippocampus. Additionally, we found that knocking down Gas5 partially alleviated memory deficits and anxiety caused by maternal separation (Banerjee et al., Journal of Neurochemistry, 2024).

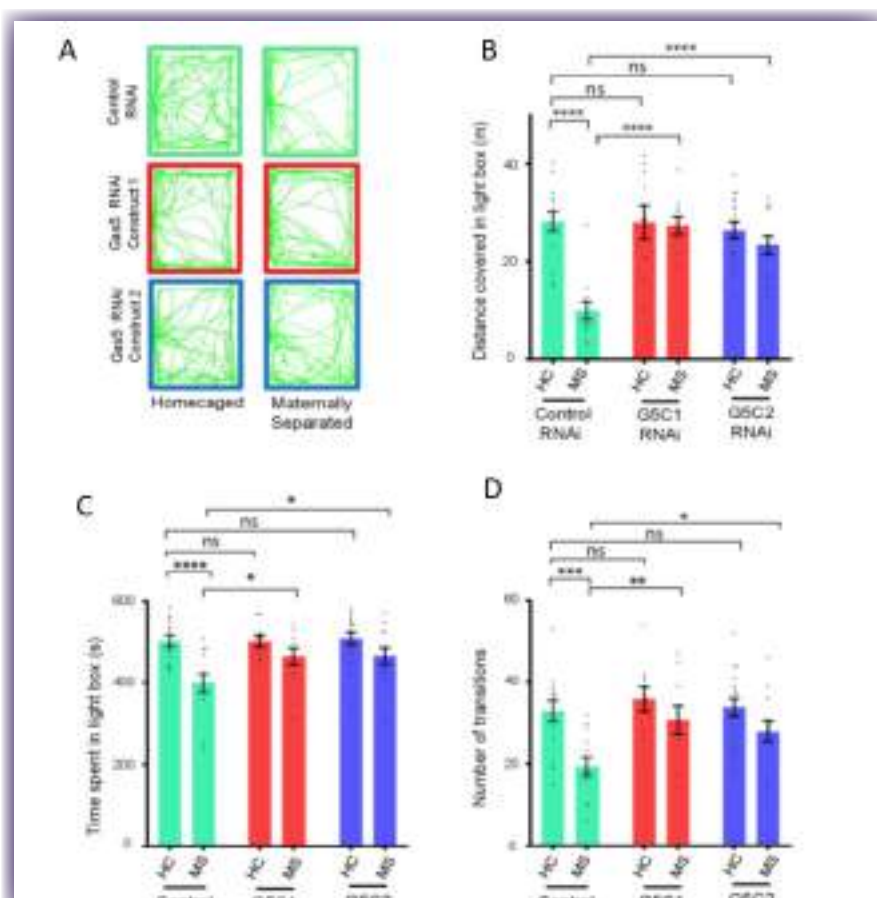


Figure 2: Knockdown of the lncRNA Gas5 reduces anxiety following maternal separation. (A)

Traces showing the movement of mice in the light/dark box. HC = Home caged, MS = Maternally Separated. (B) Distance covered in the light box (in metres). G5C#1 and G5C#2 represent two distinct *Gas5* RNAi constructs. (C) Time spent in the light box (in seconds). (D) Total number of transitions done by HC and MS mice between the lit and dark portions of the light/dark chamber. Data shown as mean $\pm$ SEM, $N = 3-7$, $n = 10-18$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.005$, **** $p < 0.0001$, ns=not significant. Two-way ANOVA with Fisher's LSD test. Obtained from Banerjee, D. et al., *Journal of Neurochemistry* (2024).

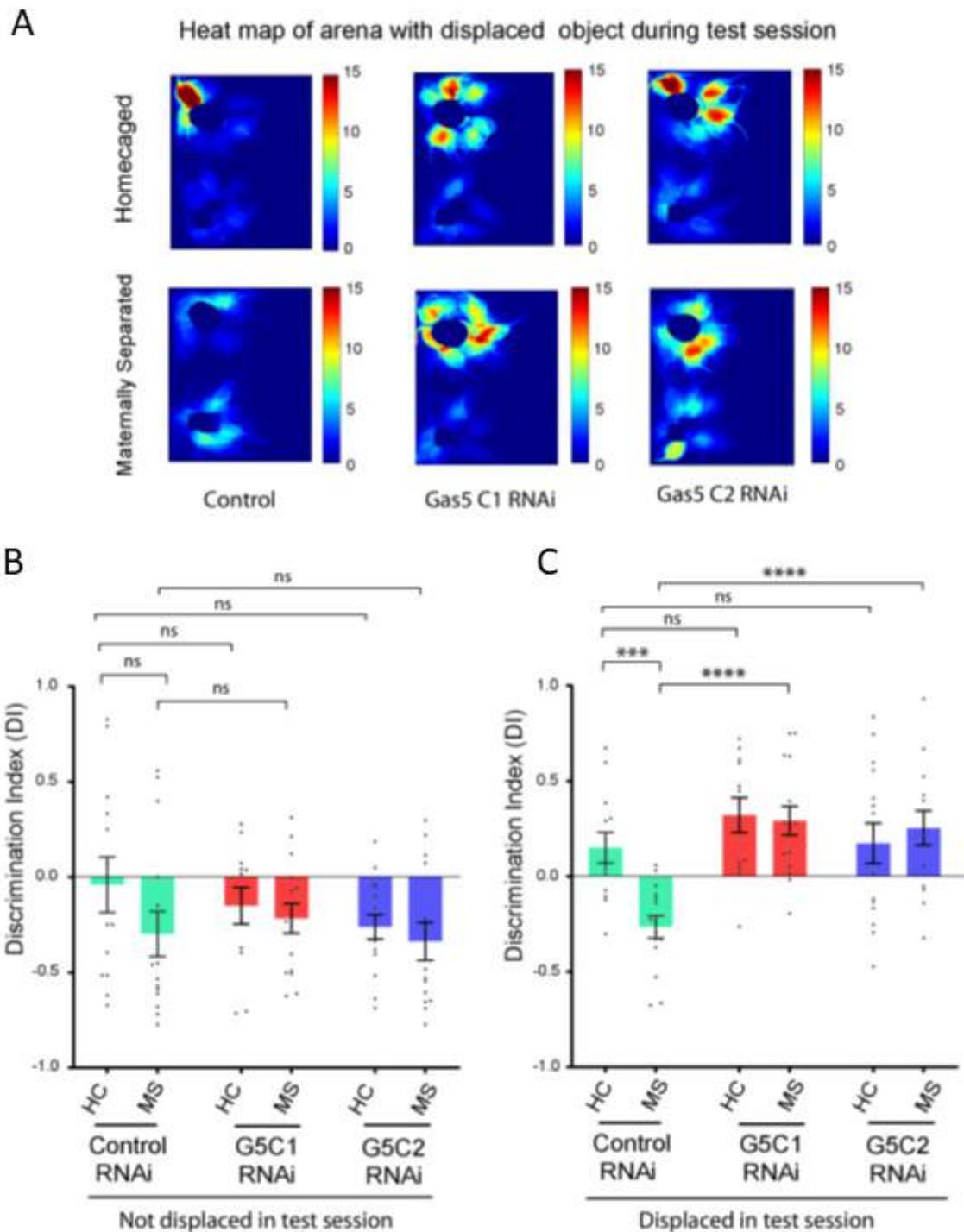


Figure 3: Effects of Gas5 RNAi on memory formation following maternal separation. (A) Representative heatmaps of the arena with the displaced object during test sessions (B) Discrimination index (DI) in the arena with the non-displaced object during the test session. HC = home-caged, MS = maternally separated, G5C1 and G5C2 represent the two distinct RNAi constructs for Gas5. (C) DI in the arena with one displaced object during the test sessions. Data shown as mean $\pm$ SEM. * $p < 0.05$, ** $p < 0.01$, * $p < 0.005$, ns = not significant. Two-way ANOVA with Fisher's LSD test. Obtained from Banerjee, D. et al., *Journal of Neurochemistry* (2024).**

Moreover, our RNA-IP-Seq analysis following maternal separation indicated an increase in m6A modifications in both coding transcripts and lncRNAs (**in collaboration with Tim Bredy, University of Queensland**). We are currently investigating how m6A RNA modifications may contribute to the observed memory deficits and anxiety following maternal separation.

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1. Interactions between FMRP and long non-coding RNAs: characterization and implication in functional synapse development.

Indo-French Center for the promotion of Advanced Research (CEFIPRA)

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2. "Regulation of homeostatic plasticity by long non-coding RNAs."

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3. "Investigating the impact of sleep deprivation on memory deficits involving long non-coding RNAs and its implications in Alzheimer's Disease".

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A cytoskeletal perspective to dendrite remodeling ▶▶▶

A functional nervous system requires intact neuronal compartments and their associated connections. These compartments undergo structural and functional modifications during development, regeneration, and plasticity-related events. Failure to maintain these modifications can lead to the degeneration of specific compartments, loss of functional connectivity with target tissues, and the onset of neurodegenerative symptoms (Figure 1A). Dendrites, as input processes, determine the quality and quantity of information processed by neural circuits. The morphology of dendritic arbors, which is diverse and specific to neuron type and function, is crucial for proper neural function. Abnormal dendrite morphology is a hallmark of various chronic and acute neuropathologies, including Autism, Schizophrenia, Alzheimer's disease, epilepsy, traumatic brain injuries, and the effects of drug abuse.

Unlike other neuronal compartments, dendrites remodel extensively in response to various developmental, sensory, or pathological cues (Figure 1B). For example, during pupa formation, *Drosophila* neurons completely prune their dendritic arbors and



reconstruct them into a new geometry. Similarly, in vertebrates, sensory experiences refine the connections of mitral cells with specific glomeruli. Neurite remodeling has been extensively studied using axon injury models, which involve changes in cytoskeletal architecture, rerouting of polarized transport, and alterations in the transcriptome. For instance, axon injury leads to an increase in intracellular calcium, causing microtubule catastrophe, and elevates levels of cyclic adenosine monophosphate (cAMP), protein kinase A (PKA), and mitogen-activated protein kinase kinase kinase (MAPKKK) such as Dual Leucine Zipper Kinase (DLK-1). DLK-1 downregulates microtubule depolymerization by KLP-7 (Kinesin-13) and promotes the formation of the growth cone through actin turnover mechanisms. Dendrites and axons have distinct molecular constitutions, and conventional axon regeneration pathways are not involved in dendrite regeneration. However, cytoskeletal effectors like AKT, ROR, and Wnt have been implicated in the process.

The dendritic cytoskeleton is primarily composed of microtubules and actin, along with scaffolding proteins such as spectrins and septins. The core machinery for maintaining microtubules includes end-binding proteins like EBP and Patronin, the depolymerizing motor Kinesin-13, assembly factors such as CRMP, and motors like Kinesin-1, which transport most cargoes, including tubulins and microtubule protofilaments. Actin is similarly maintained by polymerization factors like Profilin, depolymerization factors like Cofilin, and branching factors such as Arp-2/3 and WASP/WAVE, which are involved in dendritic arbor formation.

Dendritic arborization also depends on microtubule and actin nucleators, including Golgi outposts, kinetochore proteins, endoplasmic reticulum, and actin blobs, which are enriched at presumptive dendritic branch points. Despite the complexity of dendrites and the lack of comprehensive *in vivo* models, the organization and regulation of the neuronal cytoskeleton for proper dendritic arborization during development or regeneration remain not well understood.

This study, using PVD neurons of roundworms, *Caenorhabditis elegans*, aims to elucidate (Figure 1B):

1. Microtubule Dynamics and the Role of Its Regulators During Dendrite Injury and Regeneration (RNAi Screening of Cytoskeletal Regulators in Dendrite Regeneration).
2. The Role of Actin and Its Regulators in Dendrite Regeneration (Dendrite Remodeling Following Injury Depends on Actin Regulators).
3. Changes in Polarized Transport During Dendrite Development and Regeneration (Dendrite Structure and Composition Defined by Kinesin-13).



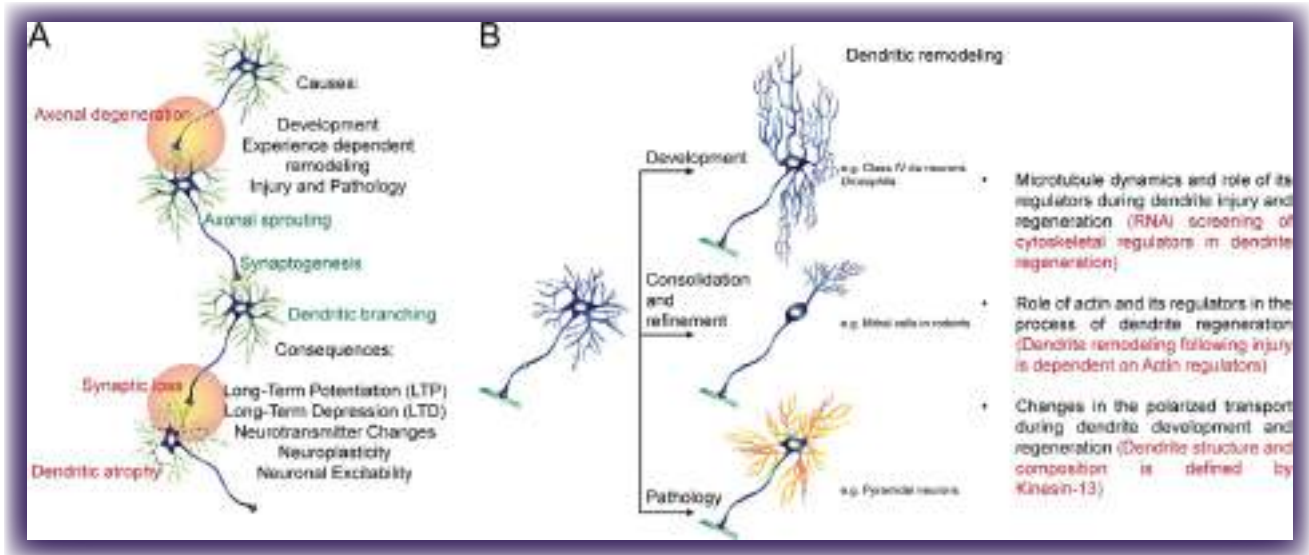


Figure 1: Infographic describing aspects of the study.

- A. The function of the nervous system depends on the structural integrity of the neuronal processes.
- B. Dendrite remodeling occurs frequently throughout the organism's lifetime, especially during development, with respect to experience, injury, and pathological changes. The study aims to understand the cytoskeletal basis of dendrite development and regeneration.

Microtubule dynamics and the role of its regulators during dendrite injury and regeneration

Using Gene Ontology (GO) associations, molecular function analysis, and expression level profiling in PVD neurons (Figure 2A), we have identified a range of candidate genes involved in various processes such as cytoskeleton dynamics, kinase activity, phagocytosis, cell adhesion, GTPase function, and DNA binding (Figure 2B). Our focus has been on cytoskeletal candidates, specifically those related to actin, microtubules, and intermediate filaments (IF). We identified 205 actin-associated, 322 microtubule-associated, and 4 IF-associated genes.

To investigate their roles in dendrite regeneration, we employed RNA interference (RNAi) feeding combined with a high-throughput dendrite injury paradigm. We assessed 113 microtubule-related, 97 actin-related, and 2 IF-related genes, which resulted in the identification of 26 microtubule-related, 34 actin-related, and 1 IF-related positive hits (Figure 2C). RNAi knockdown of these genes revealed defects in the dendrite regeneration of PVD neurons.

In the primary screening phase, positive candidates were identified based on the territory length of the regenerating dendrite, specifically measuring the length of the longest regenerating dendrite. We are now refining these results by filtering out candidates with other types of defects, such as tertiary-tertiary fusion or primary-primary reconnections. Furthermore, candidates that do not show significant defects in dendritic arborization during development are considered exclusively necessary for dendrite regeneration.

Our observations indicate that many of the positive candidates with severe defects in dendrite regeneration upon RNAi knockdown are direct regulators of actin. Interestingly, none of the microtubule-associated candidates identified so far appear to directly regulate microtubules (Figure 2D). This finding suggests that dendrite regeneration, unlike axon regeneration, might be primarily driven by actin-based processes rather than traditional microtubule dynamics.

These results underscore the complexity of dendrite regeneration and highlight the potential centrality of actin regulation in this process. Further investigation is required to fully understand the mechanisms through which actin and its regulators facilitate dendrite regeneration and how microtubule dynamics indirectly support this process.

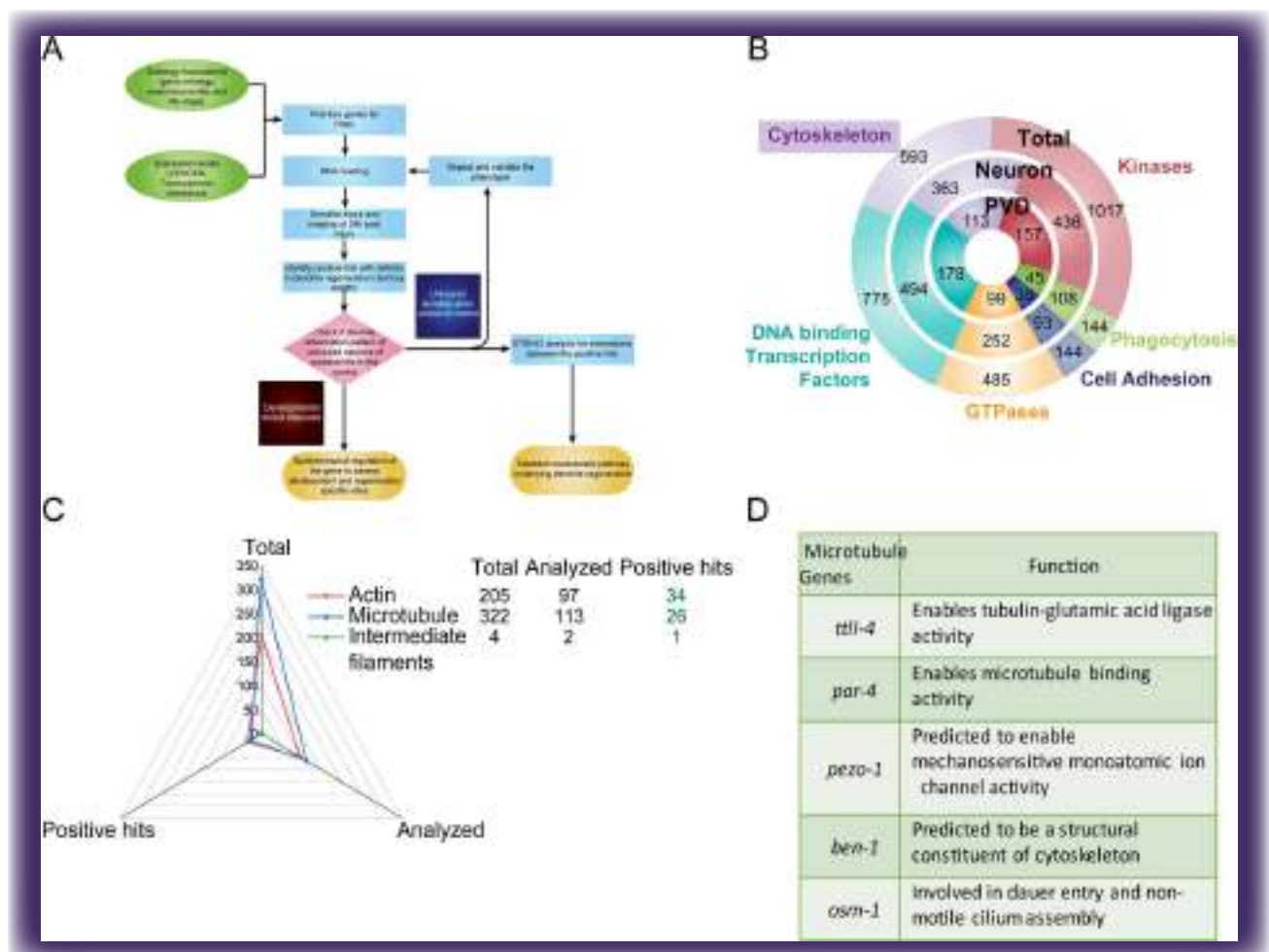


Figure 2: RNAi screening of cytoskeletal regulators in dendrite regeneration.

- A. Flowchart depicting the steps for identification of various regulators of dendrite regeneration.*
- B. The pie chart shows the number of candidates that are identified using Gene Ontology (GO) associations in different categories for primary RNAi screening.*
- C. The radar chart and numbers describe the cytoskeletal candidates identified, analyzed, and positive hits in the dendrite regeneration process.*
- D. The table outlines a few of the microtubule candidates which are the positive hits.*

Role of actin and its regulators in the process of dendrite regeneration

Several actin regulators identified through RNAi screening have been shown to modulate actin polymerization and depolymerization in the cell cortex (Figure 3A). These processes are critical for actin turnover, which we speculate is a key determinant of successful dendrite regeneration like CED- 10 (Figure 3C). The precise regulation of actin dynamics, involving both its assembly and disassembly, appears to be essential for the structural remodeling required during dendrite repair.

To investigate the role of actin and its regulators in dendrite regeneration, I have generated transgenic models expressing the F-Actin reporter GFP::Moesin and established dendrite injury assays (Figure 3B). These models allow for real-time visualization of actin dynamics during the regeneration process (Figure 3B).

The GFP::Moesin reporter, which labels F-Actin, is found to be enriched in higher-order quaternary dendrites (Figure 3B). Following dendritic injury, there is a notable increase in F-Actin intensity in the proximal region of the primary dendrite (Figure 3D0-E). This heightened intensity gradually returns to baseline levels as the dendrite regrows. To validate this observation, we are employing additional actin reporters, including LifeAct, WASP::GFP, and ARX-2::TagRFP.

To test our hypothesis that actin turnover is crucial for dendrite regeneration, we are conducting experiments involving pharmacological perturbation of actin dynamics. By inhibiting or enhancing actin polymerization and depolymerization, we aim to observe the effects on dendrite regrowth. Additionally, we are correlating the observed actin enrichment in the proximal dendritic segment with the genetic perturbation of identified actin regulators using RNAi. This approach will help us understand how specific actin regulators contribute to the remodeling process.

This research highlights the importance of actin turnover in dendrite regeneration and sets the stage for further studies aimed at unraveling the complex interactions between cytoskeletal components during neuronal repair processes. Understanding the role of

actin and its regulators in dendrite regeneration has significant implications for neuronal repair strategies.

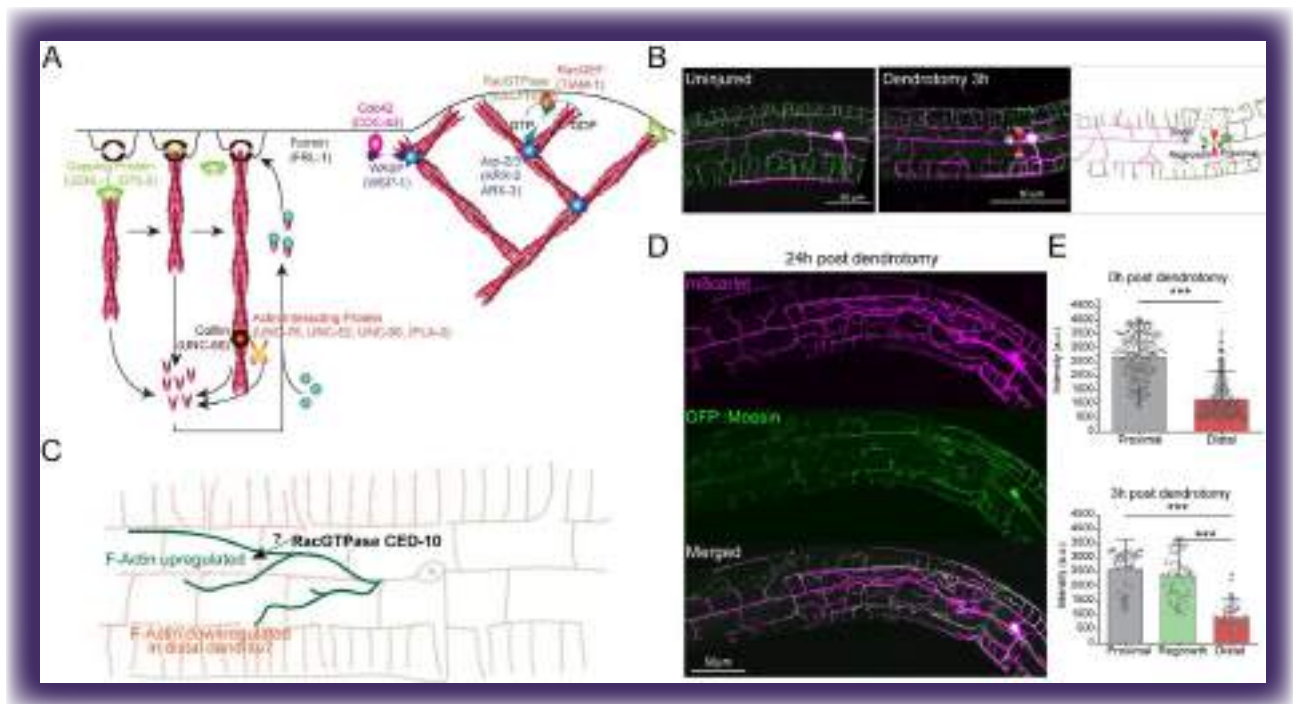


Figure 3: Dendrite remodeling following injury is dependent on Actin regulators.

- The illustration shows different actin regulator candidates identified in the RNAi screening and their possible functions in the cell.
- GFP::Moesin which shows an enrichment in the severed portion of the PVD dendrite at 3 hours after injury.
- Schema exploring the role of CED-10 as an potential upstream regulator of actin turnover for dendrite regeneration
- Representative images showing regrowing dendrites after 0h, and 24h after injury of the PVD neuron expressing GFP::Moesin (green) and mScarlet (magenta).
- Intensities of GFP::Moesin in the proximal and distal portion of severed PVD dendrites at 0h after injury and regrowing dendrites at 3h after dendrotomy.

Changes in polarized transport during dendrite development and regeneration

An intriguing observation during dendrite development and regeneration is the immediate reduction in F-Actin levels in distal dendrites following injury. This decline suggests a disruption in the transport mechanisms responsible for maintaining F-Actin in dendrites post-injury. One putative mechanism involves the transport of DMA-1 and

HPO-30, along with extracellular factors like SAX-7, MNR-1, and LECT-2, which collectively regulate actin turnover in dendrites through TIAM-1. We are currently in the process of generating a reporter for DMA-1, which will provide valuable insights into this process.

Additionally, polarized transport in neurons relies heavily on microtubules. Ongoing studies focusing on KLP-7 (Kinesin-13), a microtubule depolymerization enzyme, have yielded new insights into its role in regulating neuronal polarization. KLP-7 exhibits a differential nature in axons and dendrites, maintaining their characteristic microtubule organization essential for the selective formation of the axon initial segment during neuronal polarization. This process is crucial for compartmentalizing motors in their respective dendritic or axonal compartments.

Loss of KLP-7 caused a mislocalization of axonal proteins including RAB-3 (Figure 4A), SAD-1, and their motor UNC-104 to dendrites (Figure 4B-C). This is rescued by cell-autonomous expression of the KLP-7 or colchicine treatment indicating the involvement of KLP-7-dependent microtubule depolymerization. These observations indicate that KLP-7-mediated depolymerization defines the microtubule dynamics conducive to the specific enrichment of AIS components in dendrites (Figure 4D-E). This further compartmentalizes dendritic and axonal microtubules, motors, and cargoes thereby influencing neuronal polarity.

High mobility of KLP-7 is correlated to increased microtubule dynamics in the dendrites which restricts the enrichment of UNC-44, an integral component of Axon Initial Segment (AIS) in these processes (Figure 4F-G). Due to the loss of KLP-7, ectopic enrichment of UNC-44 in the dendrite (Figure 4H) potentially redirects axonal traffic into dendrites that includes plus-end out microtubules, axonal motors, and cargoes (Figure 4I-J).

Failure of this compartmentalization process can lead to the mislocalization of axonal microtubules, motors, and cargoes into dendrites, rendering them dysfunctional. Such dysregulation in polarized transport mechanisms can disrupt neuronal connectivity and compromise dendrite function, potentially contributing to neurodevelopmental and neurodegenerative disorders.

Further investigation into the intricate mechanisms underlying polarized transport during dendrite development and regeneration is essential. Understanding how various molecules and signaling pathways regulate the transport of crucial components such as F-Actin and microtubules will provide valuable insights into neuronal repair processes. Moreover, elucidating the role of KLP-7 and other molecular motors in maintaining microtubule organization and axonal/dendritic compartmentalization will deepen our understanding of neuronal polarization and its implications for neuronal function and dysfunction.



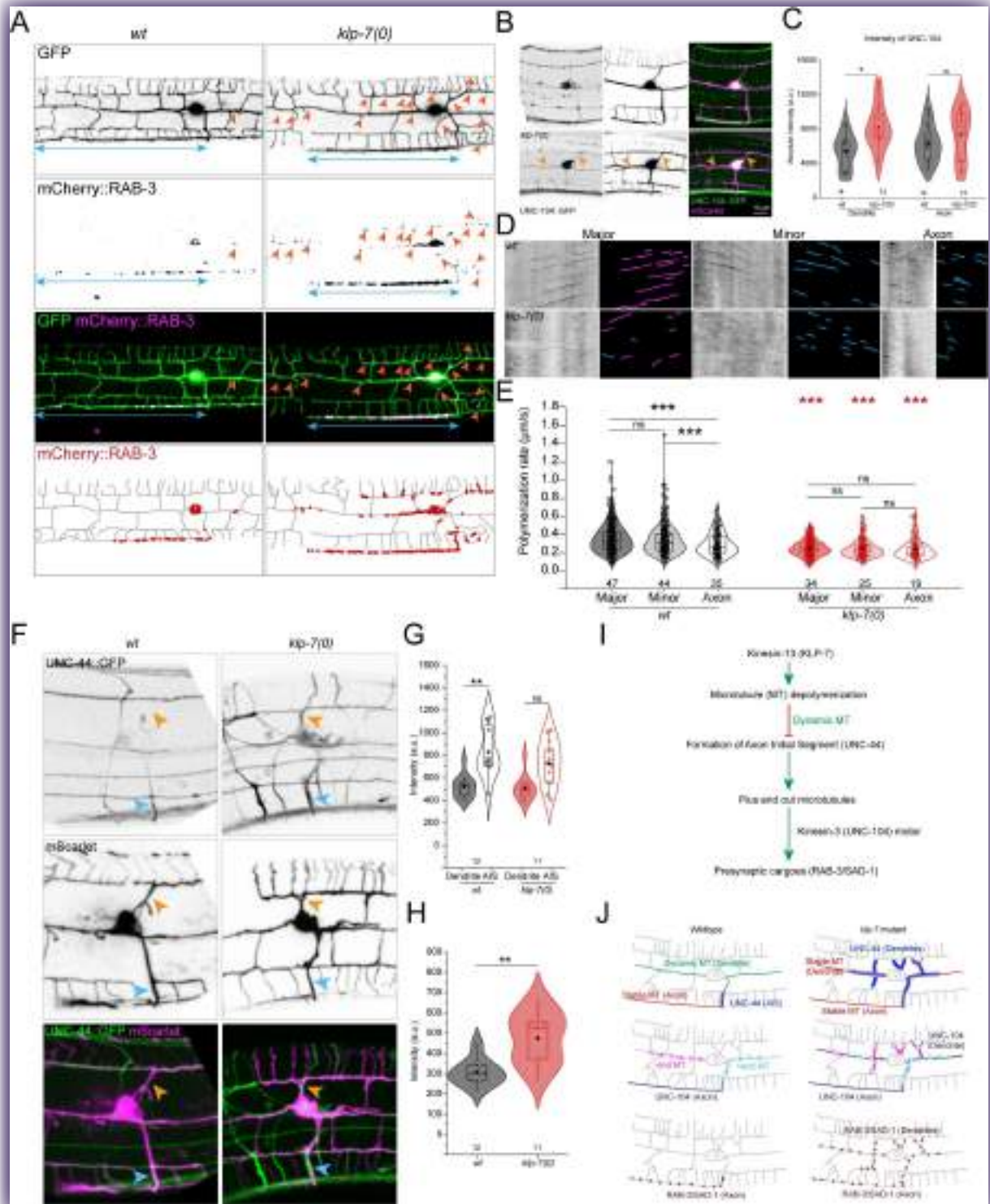


Figure 4: Axon-dendrite compartmentalization is dependent on Kinesin- 13.

- A.** Representative images of the PVD neuron expressing soluble GFP (green) and mCherry::RAB-3 (magenta) in the wildtype (wt), and loss of function mutant of *klp-7(0)*. The axonal and dendritic distributions of mCherry::RAB- 3 in (C) are marked by the blue arrows and yellow arrowheads, respectively. Schematic shows the distribution of mCherry::RAB-3 (red dots) in the dendritic arbor

of various mutants.

- B.** B-C. Representative images (B) and quantification (C) of the distribution of endogenously UNC-104::GFP (green) in the dendrites and axons of PVD neurons expressing mScarlet (magenta). Enrichment of UNC-104::GFP in the dendrites in (B) are marked by yellow arrowheads. Comparison of mean intensities of UNC-104::GFP in the dendrites and axons was done between wildtype (wt), and mutant *klp-7(0)* using ANOVA and Bonferroni Test. $p < 0.05^*$, ns (not significant). Number of worms assessed (n) is mentioned along the X- axis.
- C.** Kymographs with their respective schematics showing the trajectories of EBP-2::GFP comet in the PVD major dendrite, minor dendrite, and the axonal compartments of the wildtype (wt), and mutant *klp-7(0)*. Schematics show the orientation of the comets in the plus-end-out (cyan) and minus- end-out (magenta) directions.
- D.** Rates of polymerization were quantified as the covered distance divided by the duration of the EBP-2::GFP comets in the PVD major dendrite, minor dendrite, and the axon of the wildtype (wt), and mutant *klp-7(0)*. Comparison of means was done using ANOVA and Bonferroni Test. $p < 0.001^{***}$. Number of animals assessed (n) is mentioned along the X-axis.
- E.** Representative images of the PVD neuron in wildtype (wt), and mutant *klp-7(0)* expressing mScarlet and single copy insertion of UNC-44::GFP under its endogenous promoter. Yellow and cyan arrowheads depict the ectopic branches and AIS of the PVD neuron.
- F.** Mean intensities of UNC-44::GFP in the dendrites (major and minor) and AIS of PVD neurons of wildtype (wt), and *klp-7(0)* mutant. Comparison of means was done using ANOVA and Bonferroni Test. $p < 0.01^{**}$, 0.001^{***} , ns (not significant). Number of animals assessed (n) is mentioned along the X- axis.
- G.** Ectopic branches emanating from the cell bodies of wildtype (wt), and *klp- 7(0)* were assessed for their enrichment and quantified as the mean intensity of UNC-44::GFP. Comparison of means was done using ANOVA and Bonferroni Test. $p < 0.01^{**}$, 0.001^{***} , ns (not significant). Number of animals assessed (n) is mentioned along the X-axis.
- H.** Microtubule depolymerization activity of Kinesin-13 (KLP-7) maintains differential microtubule dynamics in the dendrites and axons. The presence of dynamic microtubules due to this activity prevents the enrichment of Ankyrin (UNC-44) in dendrites of wild-type neurons. Specific enrichment of UNC-44 in the AIS ensures trafficking of plus end out microtubules, axonal motor (UNC-104), and presynaptic cargoes into the axon only
- I.** Graphical representation of wildtype and *klp-7(0)* PVD neurons. The absence of microtubule depolymerization in the *klp-7(0)*, decreases the microtubule dynamics in dendrites as well as axons leading to ectopic enrichment of UNC-44. Enrichment of UNC-44 in the axon initial segment (AIS) of wildtype or dendrites of *klp-7(0)* directs the axonal traffic of plus end microtubules and

axonal motor, UNC-104, and presynaptic cargoes (RAB-3 and SAD-1) in these compartments. Accumulation of AIS component UNC-44, plus end-out microtubules, axonal motor UNC-104, and presynaptic cargoes RAB-3/SAD-1 in processes other than axon attributes axon-like identity to such processes.

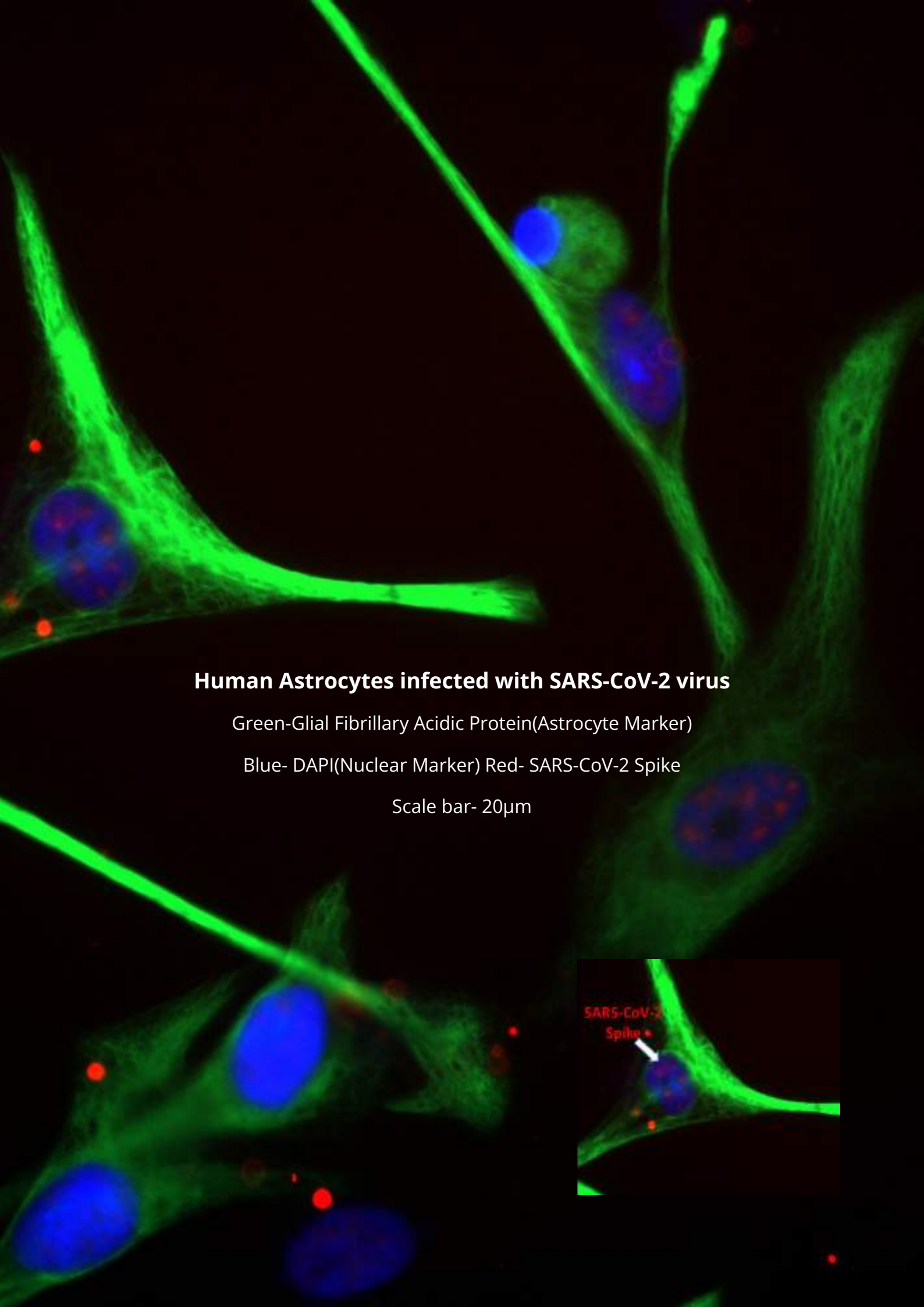
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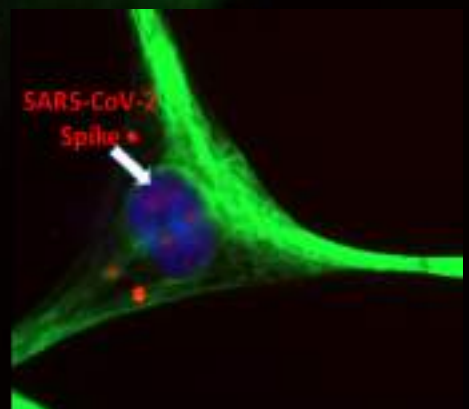


Human Astrocytes infected with SARS-CoV-2 virus

Green-Glial Fibrillary Acidic Protein(Astrocyte Marker)

Blue- DAPI(Nuclear Marker) Red- SARS-CoV-2 Spike

Scale bar- 20 μ m



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Invited Review and Book Chapter (2022-2024)

61. Samaddar S, **Banerjee S** (2024). *Non-Coding RNA and Memory*. In: **Learning and Memory: A Comprehensive Reference, Third Edition** (In production).

Data from 1st April 2023 to 31st March 2024

***The List of Publications includes journals from NBRC Facilities & Faculties.**

Presentations

- 1) **A Ghosh-Roy**, *Study of neurite regeneration using C. elegans*. C. Elegans PI meeting, 27th - 28th September 2023, IIT-Mandi.
- 2) **A Ghosh-Roy**, *Restoration Hardware*. Department of Bioscience and Bioengineering, 21st November 2023, IIT-Mumbai.
- 3) **A Ghosh-Roy**, *Functional rewiring of neural circuits*. SCAN-2022 meeting, 7th–8th January 2024, IIT-Mumbai.
- 4) **A Ghosh-Roy**, *Functional rewiring of neural circuits*. Frontiers in Biology Symposium, 2nd–4th February 2024, School of Biology, IISER Thiruvananthapuram, Kerala.
- 5) **A Ghosh-Roy**, *Functional rewiring of neural circuits*. Physiology to Pathology: Finding the therapeutic roadmap, 8th March 2024, Amity University Kolkata.
- 6) **A Ghosh-Roy**, *Restoration Hardware*. Invited lecture, 11th March 2024, Bose Institute, Kolkata.
- 7) **A Ghosh-Roy**, *Neuroscience*. Invited seminar, 12th March 2024, Lady Brabourne College, Kolkata.
- 8) **A Ghosh-Roy**, *Functional rewiring of neural circuits*. Invited seminar, 26th April 2024, Department of Genetics, Delhi University South Campus.
- 9) **A Basu**, *Infectious Diseases in an era of climate crisis and global conflict*. Department of Mathematics, 17th April 2023, Amity School of Applied Sciences, Gurugram.
- 10) **A Basu**, *Drug repositioning/repurposing: Future drug developmental strategy against viral infections*. DU South Campus, 24th June 2023.
- 11) **A Basu**, *Drug repositioning/repurposing: Future drug developmental strategy against viral infections*. 7th NIPiCON 2024, 7th–9th February 2024, Institute of Pharmacy, Nirma University, Ahmedabad.
- 12) **A Basu**, *Emerging and Re-emerging Viral Diseases: Host Immunity and Therapeutic Interventions*. RKMVERI Colloquium, 1st March 2024, School of Biological Sciences, Ramakrishna Mission Vivekananda Educational and Research Institute (Deemed University), Narendrapur Campus.
- 13) **A Basu**, *Immunomodulatory role of Gut Microbiome metabolites in abrogating microglial activation and subsequent neuroinflammation in an experimental model of Japanese Encephalitis*. BAW-2024: Frontiers in Disease Biology Conference, 27th–31st March 2024, Kalimpong.
- 14) **Banerjee A**, *Invited talk*. SPARC EEG Workshop, January 2024, IIT Chennai.
- 15) **Banerjee A**, *Invited talk*. NSG Gateway Workshop, Society for Neuroscience Satellite Meeting, November 2023, George Washington University, Washington, DC, USA.
- 16) **Sen E**, *Inflammatory Landscape as therapeutic target in Glioma Neuroimmunology: Present Perspectives*. IBRO Symposium, April 2023, Amity University, Noida.
- 17) **Sen E**, *Syncretization of Modern Science*

- and Traditional medicine: Therapeutic implications.* Summer Undergraduate Research Programme (SURP), June 2023, Ambedkar Centre for Biomedical Research (ACBR), Delhi University.
- 18) **Sen E**, *Navigating the metabolic-redox-inflammatory terrain of cancer: Cartography for therapeutics.* NIPiCON, February 2024, Nirma University, Ahmedabad.
 - 19) **Sen E**, *Metabolism-driven nuclear-cytoskeletal architecture in gliomas.* BAW: Frontiers in Disease Biology, March 2024, Kalimpong.
 - 20) **Sen E**, *D-2-hydroxyglutarate accumulation and lamin A depletion affect telomeres in glioma.* International Society for Cancer Metabolism, September 2023, London.
 - 21) **Sen E**, *Forced circumstances and Modes of escape.* Gender Studies, November 2023, Dr. B. R. Ambedkar University, Delhi.
 - 22) **Sen E**, *Consciousness: Perspective from Advaita.* BAW: Frontiers in Disease Biology, March 2024, Kalimpong.
 - 23) **M. Dhruba Singh**, *Downregulation of Pten is neuroprotective in Huntington's disease model in Drosophila.* Regional Young Investigator Meet, 18th–20th January 2024, BITS Pilani. (Oral Presentation)
 - 24) **M. Dhruba Singh**, Nisha, Bhavya Gohil, Sanchi Ahuja, Deepti Thapliyal, *PTEN knockdown ameliorates Huntington's disease in Drosophila model.* 46th All India Cell Biology Conference, 10th–12th January 2024, ACTREC, Mumbai.
 - 25) **M. Dhruba Singh**, Deepti Thapliyal, *Upregulation of PI3K/Akt signalling suppresses Spinocerebellar ataxia-3.* 46th All India Cell Biology Conference, 10th–12th January 2024, ACTREC, Mumbai.
 - 26) **M. Dhruba Singh**, Nisha, Bhavya Gohil, Sanchi Ahuja, *Neuroprotective role of PTEN in suppression of Huntington's disease in Drosophila disease model.* 6th Indian Drosophila Conference (InDRC), 6th–10th December 2023, IISER Thiruvananthapuram.
 - 27) **Seth P.**, *How viruses affect human brain?* National Conference on Recent Advances in Sustainable Approaches for Biological Sciences (RASA-Bio-2024), 29th–31st January 2024, Department of Biotechnology, Veer Narmad South Gujarat University (VNSGU), Surat, India.
 - 28) **Seth P.**, *Novel insights into molecular events following virus-induced neurodegeneration.* 41st Annual Meeting of Indian Academy of Neurosciences (IAN), 3rd–6th October 2023, School of Neurosciences, Jiwaji University, Gwalior.
 - 29) **Seth P.**, *Human Neural Stem cells as a model for studying viral neurotoxicity.* 42nd Annual Meeting of Society for Toxicology (STOX), 23rd–25th November 2023, University of Calicut, Calicut, India.
 - 30) **Seth P.**, *What we knew and what is new in Zika neuropathogenesis.* Science and Technology Block, 20th July 2023, Seton Hall University, South Orange, New Jersey, USA.
 - 31) **Seth P.**, *Molecular insights into Zika virus neuropathogenesis.* Section of Infections of the Nervous System, 14th July 2023, National Institutes of Neurological Disorders and

- Stroke, National Institutes of Health, Bethesda, USA.
- 32) **Seth P.**, *Potential of human neural stem cells as a model to study neurodegeneration*. Bio Analytical Workshop (BAW) 2023, 13th–18th June 2023, Indian Institute of Scientific Education and Research (IISER), Kolkata, India.
 - 33) **Mandal PK**, *Recent development of Imaging-based Research for Alzheimer's disease*. IIT Jammu, 2023.
 - 34) **Mandal PK**, *Recent development of Imaging-based Research for Alzheimer's disease*. IIT Guwahati, 2023.
 - 35) **Giri RK**, *Understanding Biological Processes Underlying Neurodegenerative Diseases*. Fakir Mohan University, 11th October 2013, Balasore.
 - 36) **Iyengar S**, *Establishment of Neural Circuits in the Developing Human Auditory Cortex*. 41st Annual Meeting, IAN, Mini Symposium: Development of the Nervous System, 3rd–6th October 2024, Jiwaji University, Gwalior.
 - 37) **Iyengar S**, *House Crows (Corvus splendens) - Brain and Behaviour*. Nerve-Storming: From Molecules to Behaviour, 8th–9th March 2024, JNCASR, Bengaluru.
 - 38) **Iyengar S**, *Auditory Perception and Developing Human Auditory System*. Alzheimer's Association International Conference (AAIC) Neuroscience Next 2024: India Hybrid Hub Meeting, 22nd–23rd April 2024, NIMHANS, Bengaluru.
 - 39) **Banerjee S**, *Nerve Storming*. Symposium, 2024, Jawaharlal Nehru Center for Advanced Scientific Research, Bangalore.
 - 40) **Banerjee S**, *Non-coding RNA and Epitranscriptomics*. FAOPS Symposium, 2023, South Korea.
 - 41) **Banerjee S**, *Neuroepitranscriptome Regulation: Impact on Brain Development and Physiological Process*. Korea Advanced Institute of Science and Technology Symposium, 2023, South Korea.
 - 42) **Banerjee S**, *Synapse-enriched Long Non-coding RNAs in Memory*. Annual Meeting of Indian Academy of Neuroscience, 2023, Gwalior.
 - 43) **S Dey**, *Axon-Dendrite Compartmentalization in Worm Neurons*. Organellar Biology and Membrane Trafficking Meeting 2023, 2023, organized by National Brain Research Centre, Manesar, October 2023.
 - 44) **S Dey**, *Kinesin-13 Defines the Axon-Dendrite Compartmentalization in PVD Neurons in Caenorhabditis elegans*. India Alliance Conclave 2023, July 2023, Hyderabad, India, organized by DBT/Wellcome Trust India Alliance.
 - 45) **S Dey**, *KLP-7/Kinesin-13 Dependent Microtubule Organization and Dendritic Identity in PVD Neurons*. 24th International Worm Meeting 2023, (Virtual), June 2023, organized by Genetics Society of America.

Extramural Research Projects

(Financial Year 2023-24)

S. No.	Name of P.I.	Project S.No.	Name of Project	Name of the Implementing Agency	Date of Sanction of Project	Actual Sanctioned/Release Amount for F.Y. 2023-24 (Rs. In Lakh)	Original Sanctioned Cost (Rs. In Lakh)	Date of Completion	Sanction No.
1	Prof. Anirban Basu	1	J.C Bose Fellowship	S.E.R.B	09.10.2021	18.00	95.00	08.10.2026	JCB/2020/000037
2	Dr. Ellora Sen	2	Non canonical function of metabolic genes in sculpting glioma tumor microenvironment	S.E.R.B	28.03.2022	12.70	38.10	27.03.2025	PDF/2021/000199
3	Dr Soumya Iyengar	3	Exploring Auditory Perception in House Crows using Functional Magnetic Resonance	D.S.T. (SERB)	20.05.2020	5.00	21.87	20.11.2023	CRG/2019/002672
		4	Autism spectrum disorders, genes and the gut microbiome: Utilizing song birds (Zebra Finches) as a model system	D.S.T.	24.02.2021	13.52	41.45	23.02.2025	DST/CSRI/2017/69
		5	The sensitive period of the human auditory cortex a neuro-anatomical study	ICMR	25.09.2019	0.00	42.10	24.07.2023	51/4/2019-ANA/BMS
4	Dr. Pankaj Seth	6	Effect of hypoxia on different neural cell types in vitro-a model to design therapeutic strategies against cerebral palsy in preterm infants	D.B.T.	16.10.2018	7.15	66.96	14.10.2023	BT/PR21413/MED/122/40/2016
		7	Role of FAM43A, the dyslexia and ADHD gene, in brain development and signaling in the brain	S.E.R.B	01.02.2022	0.00	16.74	31.03.2025	PS/SERB/0222/116
		8	Understanding the molecular mechanisms of SARS-CoV-2 mediated neuronal death in covid-19	D.B.T.	02.11.2022	19.94	48.75	01.11.2023	BT/PR44439/MED/29/1582/2021

S. No.	Name of P.I.	Project S.No.	Name of Project	Name of the Implementing Agency	Date of Sanction of Project	Actual Sanctioned/Release Amount for F.Y. 2023-24 (Rs. In Lakh)	Original Sanctioned Cost (Rs. In Lakh)	Date of Completion	Sanction No.
5	Dr. Pravat Kr. Mandal	9	Unravelling the causes of stroke and cognitive decline in general population A cross-Cultural perspective (DBT Netherland Grant)	D.B.T.	21.04.2016	0.00	73.66	13.08.2023	BT/IN/Netherlands/03/KP/2012
		10	Dementia Science Program- Imaging Studies	D.B.T.	18.12.2017	0.00	35.41	17.06.2025	BT/HRD/Dementia/2017
		11	Demetia Science Programme- Basic Biology Studies (i) Genetic Studies at lab	D.B.T.	18.12.2017	0.00	106.64	17.06.2025	BT/HRD/Dementia/2017
		12	Dementia Science Program- Tissue MRI Studies	D.B.T.	18.12.2017	0.00	35.41	17.06.2025	BT/HRD/Dementia/2017
		13	Artificial intelligence for early predictive diagnosis of Alzheimer's disease using multi model imaging data	Meity	17.09.2019	11.00	59.96	30.06.2023	4(5)/2019-ITEA
6	Dr. Sourav Banarjee	14	CRISPR-Cas13- mediated engineering of endogenous long non-coding RNAs for fluorescent tagging to study RNA dynamics	D.B.T.	29.02.2020	0.00	72.00	27.02.2024	BT/RLF/PR31811/GET/119/285/2019
		15	Regulation of homeostatic plasticity by long Non-coding RNAs	SERB	30.06.2024	28.52	65.19	29.06.2026	CRG/2022/003117
		16	Investigating the impact of sleep deprivation on memory deficits involving long non-coding RNAs and its implications in Alzheimer's Disease	D.B.T.	11.12.2023	48.40	96.08	10.12.2026	BT/PR45525/MED/122/311/2022
7	Director NBRC	17	Magnetencephalography (MEG) Resource Facility	D.B.T.	27.03.2018	1.68	1498.86	26.09.2023	BT/MED/122/SP24580/2018
		18	Delcon (E- Laibray Consortia) Project	D.B.T.	18.03.2009	9659.25	37123.67	31.12.2024	BT/BI/12/053/2012
		19	Demetia Science Programme- Coordination Administration & Management setup at DBT	D.B.T.	18.12.2017	0.00	530.42	17.06.2025	BT/HRD/Dementia/2017

S. No.	Name of P.I.	Project S.No.	Name of Project	Name of the Implementing Agency	Date of Sanction of Project	Actual Sanctioned/Release Amount for F.Y. 2023-24 (Rs. In Lakh)	Original Sanctioned Cost (Rs. In Lakh)	Date of Completion	Sanction No.
8	Dr. Arpan Banerjee	20	Collaborative research and experimentation RMID Only	Gate Foundation	01.03.2023	\$28280.75	\$113122.98	28.02.2025	3905(KCL)
9	Dr. Anindya Gosh Roy	21	Wellcom Trust/DBT Indian Alliance	DBT/WT	01.01.2024	124.16	438.77	31.12.2028	IA/S/221/506243
		22	Study of Neuronal Regeneration after injury using Caenorhabditis Elegans	SERB	20.05.2020	0.00	50.72	19.05.2023	CRG/2019/002194
10	Dr. Bhavani Shankar Sahu	23	Understanding the regulated secretory pathway and its role regulating physio-metabolic functions	D.B.T.	14.11.2018	0.00	103.6	13.11.2023	BT/RLF/Re-entry/38/2016
		24	ICGEB Research Grant	ICGEB	01.12.2020	1500 Euro	4500 Euro	31.12.2024	CRP/IND20-05_EC dt. 01.12.2020
		25	Investigations on the role of Syntaxin 6 in dense-core vesicle biogenesis	SERB	11.10.2022	6.00	31.42	13.10.2024	SRG/2022/000745
11	Dr Dhruva Singh	26	Role of PI3K/Akt Signaling in Spinocerebellar Ataxia-3 in Drosophila Model	SERB	05.01.2024	24.37	46.71	04.01.2024	EEQ/2023/000147
12	Dr. Swagata Dey	27	Wellcom Trust/DBT Indian Alliance	D.B.T.	01.01.2020	23.53	167.73	31.12.2025	IA/E/18/1/504331
13	Dr. Nivethida	28	Modulating effective connectivity in the agency network of humans	D.B.T.	16.08.2021	13.92	60.43	15.08.2024	BT/13/IYBA/2020/12
14	Dr Suman Saha	29	N-PDF	SERB	14.02.2022	8.00	19.2	13.02.2024	CRG/2021/0585

Honors & Awards



Achievements

Awards

1. **A Ghosh-Roy:** Wellcome Trust DBT India Alliance Senior Fellowship
2. **BS Sahu:** Young scientist award, society for neurochemistry, India
3. **Sen E:** Organization of Pharmaceutical Producers of India OPPI Women Scientist of the year, 2023
4. **Seth P.:** Prof SK Shankar Memorial Lecture, National Institute of Mental Health and Neurosciences (NIMHANS), Bangalore, India, September 26, 2023.

Poster Awards

1. **Kirti Iathoria:** Arthur Falek Young Investigator Award for the most outstanding Poster presentation at 27th Scientific Conference of the Society of NeuroImmune Pharmacology (SNIP), New Delhi, 2023
2. **Mohima Mukherjee:** Best Poster Award in the Organellar Biology and Membrane Trafficking (OBMT) Meeting 2023, NBRC, Manesar

Fellowship

Dr. Sushma Dagar: Awarded DBT-RA fellowship, January 2024

Academic Programmes



NBRC was awarded Deemed University status (de-novo category) in 2002 under Section 3 of UGC Act, 1956 (3 of 1956) vide notification No.F.9-52/2001-U.3 dated 20th May, 2002 issued by Ministry of Human Resources Development, Government of India. NBRC is the first autonomous Institution to attain the status of Deemed University among the other Institutes of the Department of Biotechnology. The 'Deemed to be university' status of NBRC has been reviewed by the Committee duly constituted by the UGC and also by an independent Committee constituted by Ministry of HRD, on completion of five years as Deemed University. The committee recommended extension of Deemed University status and placed NBRC under "A" category.

Ph.D. in Neuroscience

NBRC has a Ph.D. Programme in Neuroscience to develop trained manpower having a broad overview of different aspects of Neuroscience.

NBRC provides a fellowship of ₹37,000/- per month for Junior Research Fellows and ₹42,000/- per month for Senior Research Fellows.

M.Sc. in Neuroscience

NBRC is one of the first institutes in the country to develop an integrated multidisciplinary teaching programme in Neurosciences. During the academic year 2015-16, NBRC reintroduced the M.Sc. (Neuroscience) programme to develop trained manpower having a broad overview of different aspects of Neuroscience. M.Sc. (Neuroscience) students are provided with a fellowship of ₹5,000/- per month.

Summer Training and Short-term Programmes

NBRC conducts Summer Training Programme for the Students, recommended through three National Science Academies viz: (1) Indian Academy of Science, Bangalore (2) Indian National Science Academy, New Delhi (3) National Academy of Sciences, Allahabad. The summer training is for a period of eight weeks and the trainees are provided with shared accommodation at NBRC hostels. Summer trainees are encouraged to attend seminars and journal clubs organized at the Institute. The summer training projects provides an exposure to Neuroscience and motivates trainees to consider it as a future career option.



«« Facilities & Resources »»

Computing Facility (CF) ►►

Coordinator

Prof. Arpan Banerjee

Technical Staff

Kedar Singh Bajetha

Seepika

Tarnnum Mansoori

Sachin Kumar

The Computing Facility of the National Brain Research Centre manages the overall Information and Communications infrastructure of the Institute apart from aiding in R&D activities. The followings are summarized as under:

- A) **Campus Converged Network (NBRC-IntraNet)** The NBRC campus network consists of a campus-wide Local Area Network running on a 10Gbps fiber optic backbone with redundant paths over manageable switching fabric which is further integrated with wireless access points managed through a central controller for mobility needs. The redundancy and robustness are built into the network architecture. The network is supplemented with a secure firewall/UTM cluster for network safety, intrusion detection system, gateway level antivirus, VPN facility, managing IT policy and detailed auditing/logging, etc. The campus network is IPv6 compliant and IPv6 services are functional in the dual stack. The wireless network of the institute has further been integrated with Eduroam service by integrating it with National NREN (ERNET-India), the Eduroam service provides visiting scientists and researchers seamless secure wireless access in all participating institutions across the world.

The campus converged network of the institute is integrated with National Knowledge Network (NKN), on a 1 Gbps optical fibre link provided by BSNL that is further supplemented with a 200Mbps backup radio link for redundancy. The NKN linkage is instrumental in the running of several scientific projects for multi-site high-volume data applications like the NBRC-AIIMS data pipeline for MEG as part of the collaborative Centre of Excellence in Epilepsy project funded by DBT.

The campus converged network not only carries data traffic but also the Voice traffic from the IP-PBX system as well as the Video traffic from the IP-CCTV system.

- B) **IP-PBX facility** The telecommunication systems of the institute are running on IP-PBX and the campus network is used to carry the voice traffic along with data traffic,

the endpoints user's IP-Phones are connected to LAN. The facility is running on automatic failover mode on virtualized servers from the institute's data center. The external incoming and outgoing voice traffic is routed on E1-PRI of BSNL. The users are also provided with various facilities like multi-point conferencing, voicemail, directory, call forwarding, etc. over the provided endpoints.

C) **Institute Core and Application Servers** The computing facility manages and maintains the server infrastructure of the institute which is housed and maintained in the data centre facility. In essence, the institute currently has five fully utilized 42U server racks in the data center facility. The various services running on these servers can be classified as follows:

- Web servers for the institute and various web servers related to ongoing computational projects and applications of various scientific groups. The primary web server for the official website is running from the NIC cloud.
- NBRC mail services (nbrc.ac.in) are running from NIC cloud with advanced security features and enabled with 2FA. The email facility is available to the users without any downtime. The mailing system is monitored by the NIC team round the clock and fixed the issues at the earliest if arises. The mailing system also provides distribution facility for sending mails to the groups in one go. NBRC provide email accounts to its core employees, students, post-doctoral fellows, project assistants and project staff as well. All accounts are secured and authenticated by the user's mobile number as per the latest security policy. DNS servers for the nbrc.res.in and neuroscienceacademy.org.in domains are running from NBRC datacenter and we also used a DNS facility of Enet India for the nbrc.ac.in domain.
- Central Storage servers of 400TB have been installed and it is working along with backup servers handling storage requirements of the users and laboratories for online central storage and data processing. Major steps have been taken for upgrading the central storage infrastructure.
- Radius and authentication servers for access, accounting, and authorization of computing resources
- License management servers for managing institutional site/network/concurrent licenses.
- Application servers running on windows and Linux platforms for common computing requirements of the users and also other specialized computing servers for specific data processing requirements of various laboratories.

D) **Other Facilities & Services**

- *NIC Cloud and Email Services* : The CF also manages the Virtual Machines on the NIC Cloud for better availability of web resources (especially the official website

<http://www.nbrc.ac.in> and public DNS). Similarly, users having GOV.IN email ids on NIC platform for better availability.

- *Central Documentation Facility:* The central documentation facility provides round-the-clock availability to users for various computational needs like a facility for printing, scanning, poster-printing, etc. apart from providing data-processing computational nodes.
- *ICT Support & Service:* The computing facility also provides support and manages maintenance activities for the entire computing infrastructure of the institute which also includes user endpoints like computers, peripherals, software etc. Computing Facility has increased its wireless facility in the student's hostel to improve internet connectivity. We have added 4 new Meraki outdoor access points with 10-year license.
- *CCTV Monitoring and Management:* More cameras are added to the surveillance system to enhance coverage of the campus. The installed IP Cameras are connected to the core network which is enhanced the security and monitoring of the campus. Most entry/exit points of the buildings are covered with the Central CCTV system.
- *Software Development:* The computing facility also undertakes software development activities in line with the institute's requirements, several scientific and e-Governance applications have been developed in-house. As per the requirement of the research community of NBRC, the computing facility developed a **Central Instrumentation Facility Booking System (CIFBS)** and The system is used for booking scientific equipment that is used by faculty, students and project staff of laboratories. It is very helpful for researchers to book the equipment any time as per the availability and it is very hassle free for researchers to keep record manually. The user may book any equipment as per their research plan prior to 15 days of advance booking. Each user has a maximum time limit to book one piece of equipment to ensure that the equipment is also available for other users. Users can use their booking id to cancel a reservation if they need to. Additionally, the system will automatically cancel the booking if the user fails to mark their attendance after a predetermined amount of time, for which a countdown is shown to the user. The system is also capable of generating various kinds of reports like booking reports user-wise, equipment-wise, cancelled reports user-wise, and equipment-wise
- *Infrastructure Improvement:* The computing facility plans to implement an e-office facility in the NBRC to move the paperless files system as per the instructions of the Director. It will enhance the work and files will be trackable. It will be implemented from the new FY 2024-2025.
- *Video-conferencing:* Computing facility has installed new setup for online audio-

video conferencing in the NBRC Auditorium and Director's Board room. The rooms are well equipped with smart interactive panel, camera and sound system. The department conducted online meetings / interviews / lectures / workshops and online classes events through licensed online meeting tools to do the institute's activity smoothly.



Animal Facilities

NBRC is an autonomous institute of Department of Biotechnology, Govt. of India, with a mandate of carrying out frontline research to understand brain function in health and disease. As part of the infrastructure, NBRC has a state-of-the-art animal facility to meet the requirements of the scientists for advanced neuroscience research.

The Institute recognizes that use of laboratory animals in research is an important privilege accompanied by a great ethical responsibility to ensure humane care and use of these valuable subjects. To ensure appropriate care and use, detailed programs of excellent veterinary and husbandry care, and programs for peer-reviewed evaluation of all activities prior to use of any animal in research are in place. NBRC is committed to the highest standards of research and recognizes that laboratory animals must receive the best possible care, not only to obtain valid research data, but also to ensure the health and safety of animals, researchers, and animal caretakers. Qualified and trained veterinarians oversee all the animal health concerns, and provide all necessary veterinary care to ensure that healthy animals are available for research.

The Animal Facility is registered with the Committee for the Control and Supervision of Experiments on Animals (CCSEA), Ministry of Fisheries, Animal Husbandry and Dairying, Government of India, New Delhi. (Registration number: 464/GO/ReRcBiBt-S/Re-L/01/CPCSEA; initially registered on 24/08/2001. All activities of the Animal Facility are carried out as per standard operating procedures (SOPs). The Animal Facility maintains the records of day-to-day activities as well as breeding, maintenance and experimentation as per the statutory requirement of CCSEA.

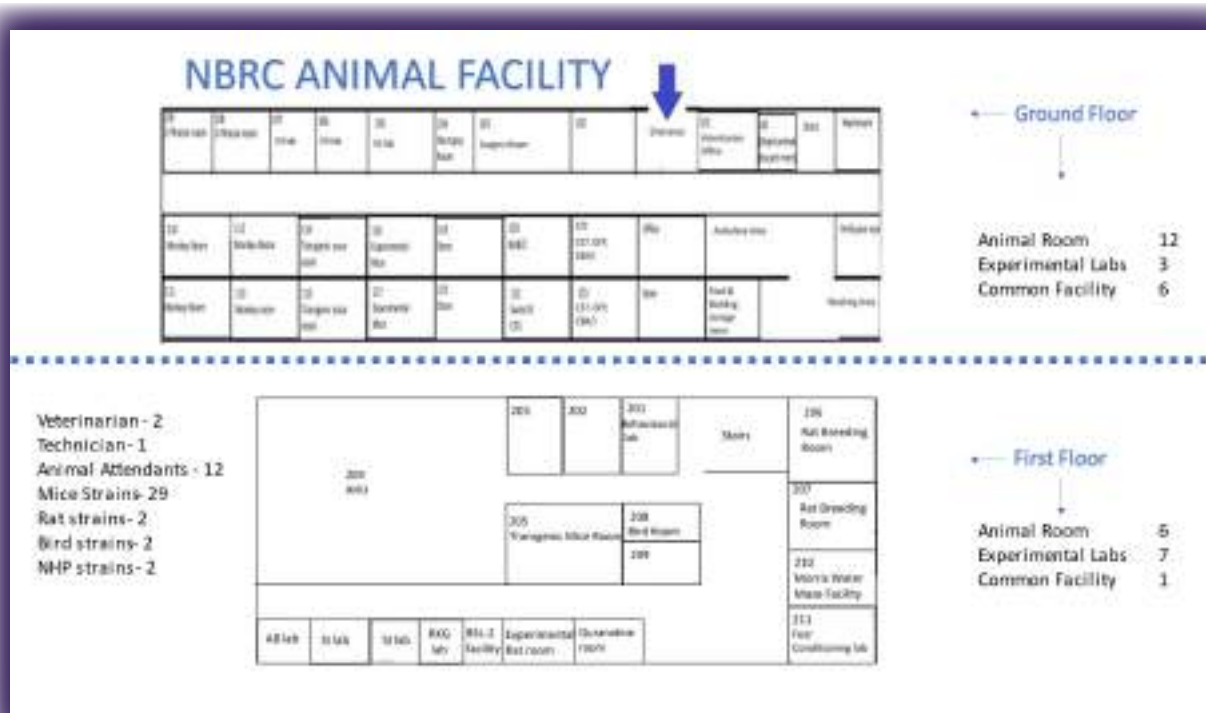


The main activities of Animal Facility are to procure and breed a wide variety of species of laboratory animals and supply quality animals to in-house researchers, which are used as animal models for understanding the human brain in health and disease. A high degree of hygienic conditions are maintained in the animal house by regular cleaning and sterilization of the cages, water bottles, bedding and feed. The animal rooms are also regularly disinfected. Heavy-duty steam autoclaves have been installed for these purposes. A hot vapour jet machine is used for cleaning the large monkey cages. The staff is required to take shower, and change to work-overalls before entering the animal rooms, and again in the evening after finishing the work. All users are required to use appropriate PPE before handling animals.



The facility is spread over two floors and is designed and built for accommodating animal species, which include rats, mice, birds (crow and zebra finches) and non-human primates. The ground floor houses rooms for rodents and non-human primates and transgenic animals, birds and rats are housed on the second floor. The Animal Facility also houses the 2-Photon imaging facility and is connected to the MRI building, making it convenient to move animals for experiments if required.





All the animal species are housed in species appropriate cages, which are designed as per the CCSEA guidelines. The outdoor play area for non-human primates has six large interconnected enclosures that provide a flexible layout for optimising enrichment and social interactions. The transgenic, knock out and mutant mice are housed under germ-free conditions in filter top cages and individually ventilated cages (IVC). Such animals are handled in laminar hoods, and then moved to fresh cages in cage-changing station under hepa-filtered air.



The animals are maintained under controlled environmental conditions as specified in CCSEA guidelines, with temperature maintained between $22 \pm 2^\circ \text{C}$, relative humidity between 45-55%, 12:12 hr light-dark cycle, and 12-15 air changes per hour. The air-handling system uses 100% fresh air for each change.

All animals are procured as per CCSEA guidelines. A health surveillance program for screening incoming animals is carried out to assess animal quality. Animals procured from other places are kept in quarantine to minimize risk for introduction of infection in established colony.

The animal facility has a state-of-art surgical suite equipped with intensity controlled surgical lights, advanced surgical microscopes, gas anesthesia machines, equipment for monitoring the physiological state of the animals, including heart rate monitor, pulse oximeter and rectal thermometer. For cleaning and sterilization of the surgical instruments there is an ultrasonic instrument cleaner, glass bead sterilizer and ethylene oxide gas sterilizer.



The animal facility has a necropsy room, perfusion room with a perfusion hood, deep freezer for carcass storage, and incinerator for disposal of the animal carcass.

The animal facility has been equipped with a card reader security system. The access is restricted to the animal house staff, maintenance staff and the investigators who are listed in the IAEC approved protocols. All the personnel who handle animals are required to have a current tetanus vaccination, and those who handle non-human primates (NHP) are regularly screened for tuberculosis. Everyone handling NHP's is trained in the procedures for the first-aid in case of an injury from an animal bite or scratch.



Close circuit monitoring cameras have been installed at various locations in the facility to help in effective monitoring of the animal facility.

The Veterinary staff of Animal Facility also conducts short-term training for M.Sc. and Ph.D. students, Project Assistants and other scientific staff in the field of laboratory animal science covering ethical and statutory guidelines that regulate scientific experiment on animals, general biology and



reproduction of the laboratory animals, *animal identification techniques, blood collection, injections, anesthesia and monitoring, handling and restraint, husbandry and care, sex differentiation, humane euthanasia, etc*

The animal facility is currently maintaining the following species and strains of laboratory animals.

Mice Strains

- SWISS
- BALB/c
- C57BL/6J
- CD1

Transgenic Mice

- B6C3-Tg (APP695)85DboTg(PSEN1)85Dbo (Alzheimer disease model).
- B6SJLTg(APPSwFILon,PSEN1*M146L*L286V)6799Vas/Mmjax
- B6Sjlf1/j
- NPas4 cre
- NpPas4cr-ER
- UBC-GFP (Green fluorescent protein)
- B6CBA-Tg (Hdaxon1) 62Gpb/3J (Huntington disease model)
- B6 129P2Pvalb< tm1(cre)Arbr>/J
- B6.CgGt(ROSA)26Sor<tm9(CAGtdTomato)
- B6.CgTg(Scnn1acre)3Aibs/J
- STOCK Gad2<tm2(cre)Zjh>/J
- B6.CgTg (Camk2a-cre)T29-1Stl/j
- B6.129-Rp122<tm1.1Psam>/j
- STOCK Tg(Thy1-EGFP)MJrs/J
- B6.Cg-Tg(Thy1-YFP)16Jrs/J
- B6.Cg-Tg(Thy1-YFP)HJrs/J
- B6;129S6-Tg(Camk2a-cre/ERT2)1Aibs/J
- STOCK *Sst*^{tm2.1(cre)Zjh}/J
- B6.Cg-Gt(ROSA)26Sor^{tm6(CAG-ZsGreen1)Hze}/J
- B6:129X1-Gt(ROSA)26Sor<tm(EYFP)Cos>/j

- C57Bl6-Tg(Nes-cre/ERT2)Keise/j
- B6Cg-Tg(camk2a-cre)T29-1stl/j
- B6;129S6-Gt(ROSA)26Sortm96(CAG-GCaMP6s)Hze/J

Rat Strains

- Long Evans
- Sprague Dawley

Non-human primates

- Rhesus Monkeys (*Macaca mulatta*)
- Bonet Monkeys (*Macaca radiata*)

Birds

- Zebra finches (*Taeniopygia guttata*)
- House crows (*Corvus splendens*)

All the mice strains are maintained by inbreeding and the rat strains by out breeding. Zebra finch colonies are maintained by out breeding. The transgenic and knockout mice are maintained under a specialized breeding program after the investigators provide the molecular genotyping of these strains based on presence or absence of the gene of interest.

NBRC has a state-of-the-art animal facility to meet the requirements of the scientists for advanced neuroscience research. We are following all the practices and standard operating protocols required by CCSEA, GOI for the animal facilities.

We receive several requests regarding animal procurement and also about research collaborations related to animal facility from various scientists across India. We always try to cater this type of requests so as to promote biomedical research on animals.

Neuroscience research requires the use of complex and sophisticated technology and equipment, which can be expensive to acquire and maintain by everyone. Contract research can provide a mechanism for researchers to access NBRC's state-of-the-art equipment and expertise, without having to bear the full cost of purchasing and maintaining these resources. This can be a source to generate revenue for NBRC along with a valuable opportunity for collaboration and knowledge exchange. Further, we also receive several requests regarding research collaborations related to animal facility from various scientists across India in the field of neuroscience.

Depending upon today's increasing demand for quality animals and research for commercial purposes, NBRC has finalized the user charges for trading the animals and research for commercial purposes as below listed table-

User Charges: -

Name of the Instrument	Type of Services	User Charges	
		Govt. Academic Institution	Industry / Private Academic Institution
Animal Facility	Trading of small animals	Rat -350 Rs/animal	Rat -450 Rs/animal
		Mice- 200 Rs/animal	Mice- 300 Rs/animal
	Research for commercial purposes	Rat – 26.17 Rs/animal/day	Rat – 39.25 Rs/animal/day
		Mice- 12.30 Rs/animal/day	Mice- 18.45 Rs/animal/day
		Non-Human Primates – 541.52 Rs/animal/day	Non-Human Primates – 812.28 Rs/animal/day

Library ▶▶▶

The NBRC Library plays a vital role in the collection, development and dissemination of scientific and technical information to meet the present and future needs of the Centre and also provides facilities and support to the scientists, researchers, students, staff and NBRC's networked centers. The Library is housed in a spacious two-storey building, with reading room, reference room, video conferencing, online journal access facility, book section, internet access and reprographic facilities etc. The main aim of the NBRC Library staff is to provide excellent services to users in NBRC and all centers associated with the Institute. The NBRC library has a large collection of Journals, books and other relevant research materials on Neuroscience, Biochemistry, Genetics, Molecular Biology, Immunology & Microbiology, Pharmacology and Toxicology, Psychology, Physics, Mathematics, Computer Science and general subjects. The NBRC Library currently subscribes to 985 online journals through the DBT e-Library Consortium (DeLCON), 01 specialized journals, 03 magazines, 122 freely accessible online journals and 01 legal database Manupatra. It also maintains digital archives and news clips about the Centre and subscribes to Newspapers and News Letters. The collection of the NBRC Library is growing day-by-day along with new developments in research and knowledge in the field of Neuroscience and related areas. To provide optimum service to all users, the NBRC library is currently digitizing its list of collections using the LSEASE software, to which all users will have full access. A barcode technology has also been installed for accurate and speedy circulation and the management of all library documents. The new software will also help in efficient library operations viz. administration, acquisition, circulation, serial control, cataloguing and information retrieval. The Library has set up 22 Computers with Internet facility to provide services for use of researchers and students in the NBRC Common room/ Computing Facility and has been providing electronic access to the subscribed journals through the campus portal. The NBRC Library also provides Inter Library Loan Services to NBRC's 48 networked centres all over India. Researchers at different centres send their requirement for research material or journal articles through email to NBRC Library library@nbrc.ac.in or to the Librarian Dr. D. D. Lal, ddlal@nbrc.ac.in which are then downloaded and sent to them free of cost. The library entertains an average of approximately 450 requests for articles and this number is increasing every year. The NBRC Library regularly evaluates its information services to ensure that the Institution's requirements are met. It promotes resource sharing and cooperation activities among libraries by providing an efficient and reliable means of resource sharing, that is, the inter library loan for the maximum use of resources, by providing copies of documents which are not available to researchers at centres outside the institute.



MAIN ACTIVITIES OF NBRC LIBRARY

1. Book Acquisition
2. Periodicals Acquisition
3. Selective Dissemination Information (SDI),
4. Current Awareness Services (CAS)
5. Inter Library Loan
6. Resource Sharing
7. Circulation services
8. Reference Services, Bibliographic services
9. Indexing and Special Services
10. Collects maintains, store and retrieves information and data keeping in the view of evolving needs of its researchers
11. Help to Network Centres.

DBT's Electronic Library Consortium (DeLCON) ►►

DeLCON CONSORTIUM: A NATIONAL LIBRARY CONSORTIUM FOR LIFE SCIENCES & BIOTECHNOLOGY HOSTED AND ADMINISTERED BY NBRC AND SPONSORED BY DEPARTMENT OF BIOTECHNOLOGY (DBT)

The DBT Electronic Library Consortium (DeLCON) is a major initiative of the Department of Biotechnology (DBT) to provide unlimited access to most of the relevant periodicals to the researchers at participating institutions. It was initiated in the year 2008 and finally launched in the month of January 2009 with 10 DBT core member institutions (including DBT H.Q. & ICGEB) enabled with a centralized subscription to a large number of high impact online journals. It is a national initiative for providing access to scholarly electronic resources including full-text and bibliographic databases in all the life sciences disciplines to the DBT institutions.

It facilitates the access to high quality e-resources to the faculties, scientists, research scholars, students and Project Assistants of the DBT research Institutions in the country to improve teaching, learning and research. DeLCON consortium was extended in three phases; and in the second phase 17 DBT Institutions were added, in the year 2010. Subsequently 7 more institutional members were added in the 3rd phase of extension in the year 2011. In the year 2012, DBT merged all the phases and it became a single 'DeLCON Consortium' with 33 members.

In the year in 2019, the DBT added one new Institute i.e. Institute for Stem Cell Science and Regenerative Medicine (InStem) under DeLCON Consortium. Currently DeLCON has a total of 35 members. The 'DeLCON Consortium' provides current (presently 985 online resources) as well as archival access to more than 1176 core peer-reviewed journals in different disciplines from 21 foreign publishers.

The DeLCON comprises the following 35 member institutions:

List of DBT & NORTH EAST REGIONAL (NER) INSTITUTIONS

DBT Institutions

1. Department of Biotechnology (DBT), New Delhi
2. National Brain Research Centre (NBRC), Manesar
3. National Institute of Plant Genome Research (NIPGR), New Delhi
4. National Institute of Immunology (NII), New Delhi
5. National Centre for Cell Science (NCCS), Pune
6. Institute of Life Sciences (ILS), Bhubaneshwar
7. Institute of Bioresources and Sustainable Development (ISBD), Imphal

8. Centre for DNA Fingerprinting and Diagnostics (CDFD), Hyderabad
9. Rajiv Gandhi Centre for Biotechnology (RGCB), Thiruvananthapuram
10. International Centre for Genetics Engineering and Biotechnology (ICGEB), New Delhi
11. National Agri-Food Biotechnology Institute (NABI), Mohali, Punjab
12. National Institute of Biomedical Genomics (NIBMG), Kalyani, Kolkata DBT's Electronic Library Consortium (DeLCON) NBRC Annual Report 2016-17 129
13. National Institute of Animal Biotechnology (NIAB), Hyderabad
14. Regional Centre for Biotechnology (RBC), Faridabad, as a part of NCR Biotech Science Cluster (BSC)
15. Transnational Health Science & Technology Institute (THSTI), Faridabad, as a part of NCR Biotech Science Cluster (BSC)
16. Biotechnology Industry Research Assistance Council (BIRAC), New Delhi
17. Institute for Stem Cell Science and Regenerative Medicine (InStem), Bangalore.

North Eastern Region (NER) Institutions

18. Dibrugarh University, Assam
19. Assam University, Silchar
20. North Eastern Regional Institute of Science & Technology, Arunachal Pradesh
21. North East Institute of Science & Technology, Assam
22. Mizoram University, Mizoram
23. D. M. College of Science (DMC), Manipur*
24. Sikkim University, Gangtok
25. College of Veterinary Science, Assam Agricultural University, Guwahati
26. Guwahati University, Assam
27. Manipur University, Imphal
28. College of Veterinary Science & Animal Husbandry Central Agricultural University, Mizoram
29. Rajiv Gandhi University, Arunachal Pradesh
30. Nagaland University, Nagaland
31. North-Eastern Hill University (NEHU), Shillong
32. St. Anthony's College (SAC), Meghalaya*
33. Indian Institute of Technology Guwahati, Assam
34. Tezpur University, Tezpur, Sonitpur, Assam
35. Sikkim State Council of Science and Technology, Gangtok, Sikkim

(* = DMC is a part of Mizoram University & SAC is a part of NEHU)

In terms of number of users, the DBT's Electronic Library Consortium (DeLCON) is the largest Consortium in India constituted in the area of Biotechnology and Life Sciences with a vision and plan to reach out to all DBT Institutions departments, research institutions, universities and their colleges affiliated to DBT.



The complete list of full-text resources (e-Journals) and bibliographic databases subscribed under the DeLCON Consortium is given below.

LIST OF JOURNALS UNDER DeLCON CONSORTIUM

Name of Publishers Journals Hyperlink of the publishers No. of Journals

- ❖ American Association for Cancer Research (AACR) <http://www.aacr.org> 09 Journals
- ❖ American Society For Microbiology (ASM) <http://www.asm.org/> 16 Journals
- ❖ Cold Spring Harbor Laboratory Press (CSHL) <http://www.cshl.edu> 4 Journals
- ❖ Taylor & Francis (T&F) <http://www.informaworld.com> 39 Journals
- ❖ Nature Publications <http://www.nature.com> 34 Journals
- ❖ Oxford University Press (OUP) <http://www.oxfordjournals.org> 21 Journals
- ❖ Springer India <http://www.springerlink.com> 343 Journals
- ❖ Microbiology Society (MBS) <http://mic.sgmjournals.org> 02 Journals
- ❖ Wiley-Blackwell <http://www3.interscience.wiley.com/cgi-bin/home> 82 Journals
- ❖ Elsevier Science (ScienceDirect) <http://www.sciencedirect.com> 433 Journals
- ❖ American Association of Immunologist (AAI) <http://www.aai.org/> 1 Journal
- ❖ Proceedings of National Academy of Sciences (PNAS) <http://www.pnas.org> 1 Journal

Archives only

- ❖ Lippincott William & Wilkins/Wolter Kluwer/OVID <http://ovidsp.ovid.com> 11 (Only Archives from 2009-2011)
- ❖ Marry ANN Liebert (MAL) <http://www.liebertonline.com> 92 (Only Archives from 2009-2018)
- ❖ American Chemical Society (ACS) <http://pubs.acs.org> 47 Journals (Only Archives from 2009-2016)
- ❖ Annual Reviews (AR) <http://www.annualreviews.org> 23 Journals (Only Archives from 2009-2011)
- ❖ The New England Journal of Medicine (NEJM) <http://www.nejm.org> 1 (Only Archives from 2009-2018)
- ❖ American Association for Advancement of Science (AAAS) <http://www.sciencemag.org> 3 Journals (2009-2019)
- ❖ American Society for Biochemistry and Molecular Biology (ASBMB) <http://www.jbc.org> 2 Journals (2009-2020)
- ❖ American Society for Hematology (ASH) <http://bloodjournals.hematologylibrary.org> 1 Journal (2009-2019)

BENEFITS OF DELCON CONSORTIUM (GENERAL)

The consortia-based subscription to e-resources is a viable solution for increasing the access to electronic resources across DBT institutions at a lower rate of subscription. Major benefits of DeLCON Consortium are:

- ❖ DeLCON acts as a single window service for a large number of DBT Institutions with their diverse research and academic interest.
- ❖ DeLCON with its collective strength of participating institutions, attracts highly discounted rates of subscription with most favourable terms of agreement for a wider range of e-resources. Most of the e-publishers have responded positively to the call of the Consortium. The rates offered to the consortium are lower by 66% to 99% depending upon the category of DBT institutions.
- ❖ DeLCON has triggered remarkable increase in sharing of electronic resources amongst participating DeLCON members
- ❖ The research productivity of DBT institutions has improved with increased access to international full text resources (Journals and database).
- ❖ Users have immediate access to material previously not subscribed to, at no incremental cost for accessing back files.
- ❖ It improves the existing library services and reduced the subscription cost.
- ❖ DeLCON is open so that other DBT institution can also join the DeLCON Consortium.
- ❖ DeLCON offers better terms of agreement for use, archival access and preservation of subscribed electronic resources, which would not have been possible for any single institutions.
- ❖ Members of the DeLCON Consortium have the benefit of cap on the annual increase in the rates of subscription. While the usual increase in price of e- resources is vary from 15% to 20%, but the DeLCON members enjoy a cap on increase in price ranging from 0% to 2%.
- ❖ Since the subscribed resources is accessible online in electronic format, the DBT institutions have less pressure on space requirement for storing and managing print based library resources.

MAJOR ADVANTAGES OF DELCON FOR CONSORTIUM MEMBERS

Some of the important advantages of the DeLCON consortium provides to members as given below:

- ❖ Consortia-based subscription to electronic resources provides access to wider number of electronic resources at substantially lower cost.
- ❖ Optimum utilization of funds.
- ❖ Facilities to build up digital libraries
- ❖ Helpful in providing better library services like CAS and SDI
- ❖ Cost sharing for technical and training support



- ❖ Electronic Journals demand neither library space nor shelving costs
- ❖ The DeLCON consortium has been offered better terms of licenses for use, archival access and preservation of subscribed electronic resources, which would not have been possible for any single institution; and
- ❖ Available 24 hours a day, 7 days a week

SELECTION PROCEDURES OF RESOURCES UNDER DeLCON CONSORTIUM

In order to understand the compilation base in DBT member Institutions, meetings of DBT Directors, & DeLCON Nodal Officers were held and their views and feedback are obtained. The print & online collection base available in DBT research institutions libraries and their needs are surveyed with the aim to recognize and determine e-resources to be subscribed under the DeLCON Consortium. Based on the feedback received from DBT Members, e-resources of various publishers are recognized and evaluated before negotiating licensing arrangements. Keeping in view the multiplicity of research programmes offered by DBT Institutions, every attempt was made to subscribe to e-resources that are multidisciplinary in nature with wide scope and coverage.

All e-resources were evaluated on the criteria as given below:

- i) Qualitative and quantitative contents;
- ii) Coverage;
- iii) Their availability on different platforms and their comparative advantages / disadvantages;
- iv) Rates applicable for these resources to individual institutions as well as to other consortia.

SUBJECT AREAS OF DeLCON CONSORTIUM

The DeLCON Consortium covers all the disciplines and subjects coming under Life Sciences i.e. Biotechnology, Bioinformatics, Biochemistry, Biology, Chemical Biology, Sciences, Immunology, Neuroscience, Plant Genome, Plant Biology, Microbiology, Physiology, Psychology, Physiotherapy, Psychotherapy, Genome, Gene, Genetics, Mathematics, Physics, Chemistry, Radiology, Medicines, Computational Biology, Cell Biology, Cell Sciences, Molecular Biology, Molecular and Cellular Biology, Computational Neuroscience, System Neuroscience etc.

OPERATIONAL FUNCTIONALITY OF DeLCON CONSORTIUM

The DeLCON is fully funded by DBT and has network connectivity among DBT Institutions. Individual Institutions have unique static IP address through which access is given by the publishers. However, the whole programme is administered, monitored and maintained by DeLCON Nodal Centre at NBRC and DeLCON National Steering Committee.

NODAL CENTRE & HEAD QUARTER OF DeLCON CONSORTIUM & ITS ACTIVITIES

The consortium headquarter functions under a National Steering Committee with the responsibilities of ensuring inter-institutional coordination; monitoring licenses for electronic resources, ordering and payment for subscribed services, establishing work groups on different subjects to improve the functioning of consortium as well as to identify new resources and evaluates the existing resources, and propagating the consortium to attract new members in it. The Department of Biotechnology has also setup a National Review Committee that have the overall responsibility of making policies, monitoring the progress, coordinating with Member Institutions for promoting the activities of DeLCON Consortium. The important functions of the consortium headquarter are : to act as nodal agency for increasing the cooperation amongst participating institutions; to coordinate all activities concerned with subscription of e-resources on behalf of consortium; to liaison with electronic publishers to provide training and technical help to participating member institutions to coordinate with DBT and participating institutions for subscription to resources; to organize the meeting of the National Steering Committee and to decide upon the policy issues to maintain a web site for the Consortium for the benefit of its members and to encourage sharing of resources in an online mode; to propagate the consortium with other institutions and enroll new members in the consortium; to organize annual meetings of the consortium members.

National Neuroimaging Facility (NNF)▶▶▶

National Neuroimaging facility, sponsored by the Department of Biotechnology, Govt. of India, came into existence in the year of 2006. The main purpose of this National Facility is to facilitate/support cutting edge brain imaging research undertaken by intramural and extramural laboratories. The facility is equipped with the following equipments:

1. 3 Tesla Magnetic Resonance Imaging (MRI): Philips Achieva 3.0 T scanner
2. Electroencephalography (EEG): Two systems in place
 - A) 64-channel Synamps 2 EEG system, Compumedics Neuroscan, Inc
 - B) 64 channel Brain products ActiChamp system
3. Transcranial magnetic stimulation (TMS): Magventure MagPro

All the instruments are available for research purposes for any user with necessary ethics approvals and paying of user charges.

Magnetic Resonance Imaging (MRI)

MRI provides much greater contrast between the different soft tissues of the body compared to computed tomography (CT), making it especially useful in neurological (brain), musculoskeletal, cardiovascular. Various imaging modalities also play important role providing crucial information which can aid to various diagnostic process. The various imaging modalities which are routinely used in National Neuroimaging facility are:

1. MR Spectroscopy (MRS) which provides non-invasive neurochemical level estimations and enables clinical correlation.
2. Functional MRI (fMRI) which, as the name suggests reveals the changes in brain metabolic activity over time.
3. Structural MRI (or simply MRI) can give us detailed high resolution pictures of brain structures as well as brain connectivity using diffusion weighted images.

The 3 Tesla Phillips whole body MRI scanner at our Facility is equipped with state-of-the-art hardware, software and data processing software required for each imaging modality. The facility is being used daily for performing structural and functional MRI and MRS. In addition to understanding brain function and clinical research, the center also is closely interacting with leading imaging centers within the country and across the globe.

Electroencephalography (EEG) is a test that measures and records the electrical activity of the brain. Special sensors are attached to the scalp (in a similar way as ECG) to detect brain electric activity and mV range and the signals are amplified via an amplifier that communicates and stores the information in a computer. Basic brain functions such

as vision, auditory, somatosensory processing as well as higher order functions like memory, emotion, decision making and brain diseases such as epilepsy, dementia, and narcolepsy (sleeping disorder) can be studied by EEG.

Transcranial magnetic stimulation (TMS): TMS is a non-invasive neurostimulation technique by which researchers can induce a transient change in electric currents in a target brain area by applying very small amounts of external field magnetic field. This changes are completely reversible and the technique gives us a window to study brain information processing with profound insights.

Clinical studies on patients with Alzheimer's Disease, Parkinson's Disease, Autism and Brain Tumours, as well as monitoring of aging in normal healthy brain, are being performed extensively in the National Neuroimaging facility. Understanding the basic neurobiology of various sensory and cognitive functions using non-invasive neuroimaging tools are also undertaken by several labs in NBRC.

The following are the publications having data collected at NNF between April 2023 - March 2024.

Publications

1. Bapat R, Pathak A, and **Banerjee A.** (2024). *Metastability indexes global changes in the dynamic working point of the brain following brain stimulation.* **Frontiers in Neurorobotics**, 18:1336438. doi: 10.3389/fnbot.2024.1336438.
2. Chakraborty P, Saha S, Deco G, **Banerjee A.**, Roy D. (2023). *Structural-and-dynamical similarity predicts compensatory brain areas driving the post-lesion functional recovery mechanism.* **Cerebral Cortex Communications**, 4(3), tgad012.
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 11. **Mandal P K***, Dwivedi D, Joon S, Goel A, Ahasan Z, Maroon J.C., Singh P, Saxena R, Roy R.G. (2023). *Quantitation of Brain and Blood Glutathione and Iron in Healthy Age Groups Using Biophysical and In Vivo MR Spectroscopy: Potential Clinical Application. ACS Chemical Neuroscience*, 14, 2375-84.
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 14. **Mandal PK***, Roy R., Kalyani A. (2023). *Distribution Pattern of Closed and Extended Glutathione in the Human Brain: MR Spectroscopic Study. ACS Chemical Neuroscience*, 14, 270–276.

Magnetoencephalography (MEG) Resource Facility ▶▶▶

(Under DBT-SAHAJ Scheme, Funded by the Department of Biotechnology,
Ministry of Science & Technology, Govt. of India)

Investigators from AIIMS: Prof. Manjari Tripathi
Prof. P. Sarat Chandra
Dr. Jyotirmoy Banerjee

Investigators from NBRC: Director, NBRC

Investigator from ACR: Dr. Aparna Dixit



Magnetoencephalography (MEG) Resource Facility at NBRC is a collaborative project between National Brain Research Centre (NBRC) and All India Institute of Medical Sciences (AIIMS) under the aegis of Department of Biotechnology (Government of India). The facility has been incorporated into the esteemed DBT-SAHAJ infrastructure, with the primary objective of creating a "National Service Facility" offering access to resources.

MEG unit in Magnetic Shielded Room (MSR) -NBRC



Orchestrated with a few facilities across the world, NBRC brings together a premier medical science institute and a dedicated neuroscience research centre to study difficult-to-treat epilepsy and other neurological conditions. The main aim of the centre is to develop a cure for drug-resistant epilepsy by bridging the gap between clinical and basic research, mediated by the close coordination between NBRC & AIIMS. For a comprehensive study, the AIIMS component of the centre is using magnetic resonance imaging (MRI), electroencephalography (EEG), video EEG, as well as functional imaging techniques like positron emission tomography (PET) and single photon emission tomography (SPECT) to locate the epileptogenic area. Meanwhile, the NBRC

component of the centre, uses the non-invasive protocol of magnetoencephalography (MEG) for the localization with epileptogenic focus. We are presenting herewith, a brief report on our findings from the proposed objectives under this project, conducted during 2023-2024.

Establishing a diagnosis of drug resistant epilepsy (DRE) is an important milestone in the treatment of epilepsy as it marks the transition of a patient who is taking medications to control a condition and living a relatively normal life to someone who is at risk of worsening seizures, injuries or even death, and deals with social stigma and economic hardship associated with uncontrolled seizures. Identification of such patients and diagnosing drug refractory epilepsy (DRE) are very important steps in the management of these patients. MEG resource facility, one-of-its-kind in Northern India, proved very much fruitful in managing patients suffering with such conditions. Till March 2024, 2880 patients were evaluated using this facility from all over India. International patients in fact, have been also benefitted, especially patients from the SAARC nations.

Total Number of patients scanned till 31.03.2024 =2880 (Inclusive of 886 paid user)

Revenue Generation under DBT SAHAJ scheme-

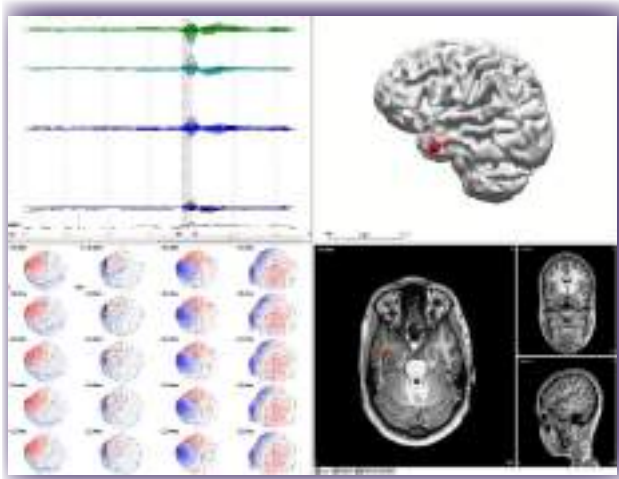
FY 2023-24: INR 11,04,000 (368 Users)

Total Revenue Generated (08.09.2021 to 31.03.2024): INR 26,58,000 (886 paid Users)

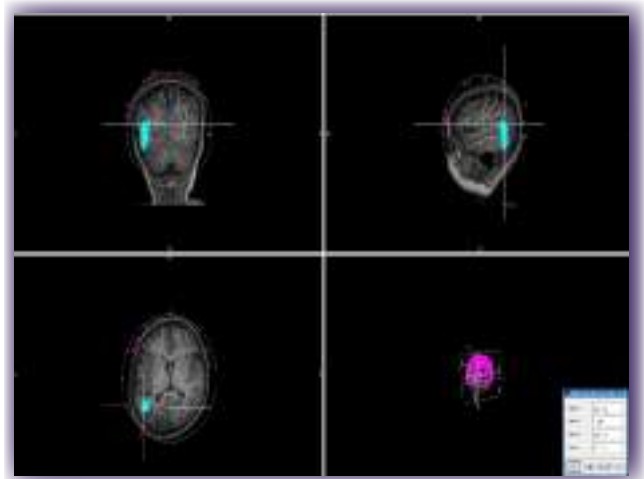
Cutting-Edge Methods for MEG Analysis:

- Ictal High Frequency Oscillations (HFO) are an Indicator of seizure onset areas (80Hz-200Hz).
- Pre-Spike Source Localisation (PSSL) is an indicator of seizure before inter-ictal spikes.
- Standardized Low Resolution Brain Electromagnetic Tomography Method (s-LORETA).
- Inter-ictal high frequency oscillations (80Hz-500Hz) are an indicator seizure onset areas (Working).
- Exploring aberrant functional networks in persons with epilepsy using connectomics.





¹**Epileptic Source Mapping in "CURRY"**



²**Epileptic Source Mapping in "DANA"**

One of the major advantages of this technique over the EEG is the lack of distortion of MEG signals by the skull and intervening soft tissue. In addition, the MEG preferentially records activity from tangential sources thus recording activity predominantly from sulci, which is not contaminated by activity from apical gyral (radial) sources. While the MEG is probably more sensitive than the EEG in detecting interictal spikes, especially in some locations such as the superficial frontal cortex and the lateral temporal neocortex, both techniques are usually complementary to each other. The diagnostic accuracy of MEG source localization is usually better as compared to scalp EEG localization. Functional localization of eloquent cortex is another major application of the MEG. The combination of high spatial and temporal resolution of this technique makes it an extremely helpful tool for accurate localization of visual, somatosensory and auditory cortices as well as complex cognitive functions like language.

Accurate localization of epileptogenic focus is of paramount importance for good seizure-free outcomes following surgery in patients with drug refractory epilepsy. It is also important to know the extent of overlap of the epileptogenic focus with eloquent cortex to avoid post-operative morbidity and prognostication. Invasive EEG is of great help to identify epileptogenic focus and extent of overlap with eloquent cortex. StereoElectroEncephalography (SEEG) implantation is usually done in MRI negative cases, when there is discordance between electro clinical and MRI, overlapping with eloquent cortex and dual pathology cases. Placement of SEEG electrodes is safe and an effective technique to localize epileptogenic zone.

SEEG implantation is currently allowing us to localize the epileptogenic networks accurately and we have been able to develop a paradigm to provide accurately labelled tissues for better cellular electrophysiological and molecular characterization. Such a strategy would allow us to create better models to understand the cellular and molecular mechanisms of epileptogenesis.

- 1 A software to examine epileptic source imaging.
- 2 Neuro-imaging software for epilepsy detection.



We analysed data from our centre, of patients undergoing stereotactic electrode implantation. The following are the situations that required invasive intracranial monitoring in our patients:

1. Seizures that were lateralized, but not localized; or seizures were localized, but not lateralized.
2. Seizures were neither localized nor lateralized.
3. Seizure localization was discordant with other data.
4. Relationship of seizure onset to lesion to be determined (e.g., dual pathology or multiple intracranial lesions).
5. Eloquent motor cortex mapping comparison of fMRI and direct cortical stimulation and NEXSTIM was done in 78 PWE.

SEEG electrodes were implanted with the assistance of stereotactic robotic device. An image guided volumetric T1-weighted MRI with contrast along with FLAIR sequences was used for preoperative planning. Digital Imaging and Communications in Medicine (DICOM) format images were digitally transferred to the robot's native planning software. The proposed targets of SEEG electrodes were decided according to the working hypothesis derived from the non-invasive investigations of the patient. All the proposed trajectories were planned using the planning software. The trajectories were evaluated in all planes (axial, sagittal, and coronal), and also along the reconstructed "probe's eye view", to look for any compromise to the vascular structures. Trajectory was adjusted appropriately without affecting the proposed target area. The procedure was performed under general anaesthesia to ensure the accuracy of registration. Stereotactic registration was carried out using predefined anatomical landmarks. Registration was validated and adjusted accordingly. The desired trajectories were selected on the touch-screen interface. The robotic device used at our institute has an arm with six degrees of freedom, with an adaptor at one end for holding the instruments. After trajectory confirmation, the arm movement was initiated using a foot pedal. The robotic arm automatically locks into position once the position of the selected trajectory was reached. A 2-mm diameter handheld drill (Synthes) was introduced through the adaptor and used to create a skull opening. The dura mater was then opened with a dural perforator after coagulating it with monopolar cautery at low settings. A tract for the electrode was made using a tracker, following which the depth electrode was inserted. The adaptor to target distance was provided by the robotic software. Depth electrodes were inserted using orthogonal or oblique orientation. A guiding bolt was screwed onto the insertion site to hold the electrode in place. The electrode length was decided after subtracting the length of the adaptor and the anchoring bolt. The number and position of the depth electrodes was decided according to the working hypothesis. All patients had post-operative CT scan of the head to ensure proper position of the electrodes with no haemorrhage.

Patients were monitored in the epilepsy monitoring unit. After adequate information was collected regarding the epileptogenic zone, the SEEG evaluations were discussed in-patient management conferences for final decisions. The electrodes were then removed in the under local anaesthesia and sedation.

Twenty-one patients underwent SEEG implantation during the study period at our centre (AllMS). Out of them 17 were males (80.9%). Mean age of patients was 21.7 years (Range- 1.5 years to 44 years). Five patients were less than 18 years of age (35.7%). Two patients (Patient 5 and 17) were not operated due to low frequency of seizures as epileptogenic zone was involving eloquent cortex (visual areas). Two patients (Patient 18 and 20) are yet to be planned for definitive surgery. There were no SEEG implantation related complications in any of the patients. Of the remaining seventeen patients, 13 patients (76.5%) are seizure-free (ILAE Class 1 outcome) at follow up. Mean follow up period is 16.1 months (range-5 months to 36 months). One patient had left hemiplegia in post period and improved to motor power to 3/5 both in upper and lower limbs. There were no other significant post-operative complications. One patient (case 08) continued to have drop attacks in post-operative period and succumbed to death due to head injury. Language showed interesting reorganisation patterns to the other hemisphere/ anterior and superior to pars triangularis. Motor function also shifted to more anterior in 1 and posterior in another.

MEG investigation helped to increase the accuracy of localization of epileptogenic zone as well as the eloquent cortex, thereby enhanced the outcome of surgery. Ictal-MEG source localization added information towards delineating the ictal-onset zone (IOZ) and helped final decision-making in epilepsy-surgery. The findings of the study are published in European Journal of Neurology & Clinical Neurophysiology. *Tripathi M, Kaur K, Ramanujam B, Viswanathan V, Bharti K, Singh G, Singh V, Garg A, Bal CS, Tripathi M, Sharma MC, Pandey R, Dash D, Mandal P, Chandra PS. Diagnostic added value of interictal magnetic source imaging in presurgical evaluation of persons with epilepsy: A prospective blinded study. Eur J Neurol. (2021) 28(9):2940-2951.*

We also compared the diagnostic value and accuracy of ictal SPECT and inter-ictal magnetoencephalography (MEG) in localizing the site for surgery in persons with drug resistant epilepsy. SPECT was found to be non-informative for most patients, but reported better diagnostic output than MEG. It was found that MEG may be a useful alternative for patients in whom SPECT cannot be done or was non-localizing. MEG was useful in indicating sites for SEEG implantation. SEEG implantation was performed in 21 patients with DRE for better surgical outcome. The findings of the study are published in Seizure: European Journal of Epilepsy. *Kaur K, Garg A, Tripathi M, Chandra SP, Singh G, Viswanathan V, Bharti K, Singh V, Ramanujam B, Bal CS, Sharma MC, Pandey R, Vibha D, Singh RK, Mandal PK, Tripathi M. Comparative contribution of magnetoencephalography (MEG) and single-photon emission computed tomography (SPECT) in pre-operative localization for*

epilepsy surgery: A prospective blinded study. Seizure. (2021) 86:181-188.

We invented a new "bloodless" technique for minimally invasive robotic thermocoagulative hemispherectomy (ROTCH). Such a method is being described in the literature for the first time. ROTCH seems to be a safe, feasible, and bloodless procedure, with a very low morbidity rate and promising outcomes. The details of the study are published in *Journal of Neurosurgery: Paediatrics*. Chandra PS, Doddamani R, Girishan S, Samala R, Agrawal M, Garg A, Ramanujam B, Tripathi M, Bal C, Nehra A, Tripathi M. *Robotic thermocoagulative hemispherotomy: concept, feasibility, outcomes, and safety of a new "bloodless" technique. J Neurosurg Pediatr. (2021) 2:1-12. doi: 10.3171/2020.10. PEDS20673.* Due to its significance, this article has been placed on the Journal's cover page.

Reduced endogenous KYNA³ synthesis contributes to enhanced glutamatergic activity in MTLE-HS was demonstrated. Reduced endogenous KYNA synthesis was due to altered levels of KAT II and PLP. This study provides evidence that application of KYNA may have therapeutic potential. The findings of the study are published in *British Journal of Pharmacology*: Dey S, Banerjee J, Dixit A, Tripathi M, Doddamani RS, Sharma MC, Lalwani S, Chandra PS, Banerjee J. *Altered hippocampal kynurenine pathway metabolism contributes to hyperexcitability in human mesial temporal lobe epilepsy-hippocampal sclerosis. Br J Pharmacol. 2021;178(19):3959-3976.*

FCD is a diffuse lesion with poorly defined epileptogenic zones, and poor surgical outcome in FCD is associated with inaccurate localization of the EZ. Hence, identifying novel epileptogenic markers to aid in the localization of EZ in patients with FCD is very much needed. For this purpose, we, for the first time, performed RNA sequencing of surgically resected paired tissue samples obtained from electrocorticographically graded high (MAX) and low spiking (MIN) regions of FCD type II patients with autopsy controls. We identified significant changes in the MAX samples of the FCD type II patients when compared to non-epileptic controls, but not in the case of MIN samples. We found significant enrichment for myelination, oligodendrocyte development and differentiation, neuronal and axon ensheathment, phospholipid metabolism, cell adhesion and cytoskeleton, semaphorins, and ion channels in the MAX region. Through the integration of both MAX vs non-epileptic control and MAX vs MIN RNA sequencing (RNA Seq) data, PLP1, PLLP, UGT8, KLK6, SOX10, MOG, MAG, MOBP, ANLN, ERMN, SPP1, CLDN11, TNC, GPR37, SLC12A2, ABCA2, ABCA8, ASPA, P2RX7, CERS2, MAP4K4, TF, CTGF, Semaphorins, Opalin, FGFs, CALB2, and TNC were identified as potential key regulators of multiple pathways related to FCD type II pathology. We have identified novel epileptogenic marker elements that may contribute to epileptogenicity in patients with FCD and could be possible markers for the localization of EZ. This study has been published in *Molecular Brain*. Srivastava A, Kumar K, Banerjee J, Tripathi M, Dubey V, Sharma D, Yadav N, Sharma MC, Lalwani S, Doddamani R, Chandra PS, Dixit AB. *Transcriptomic profiling of high- and low-spiking regions*

³ Kynurenine acid is a bioactive compound produced along Kynurenine pathway during disintegration of tryptophan.

reveals novel epileptogenic mechanisms in focal cortical dysplasia type II patients. Mol Brain. 2021;14(1):120. doi: 10.1186/s13041-021-00832-4.

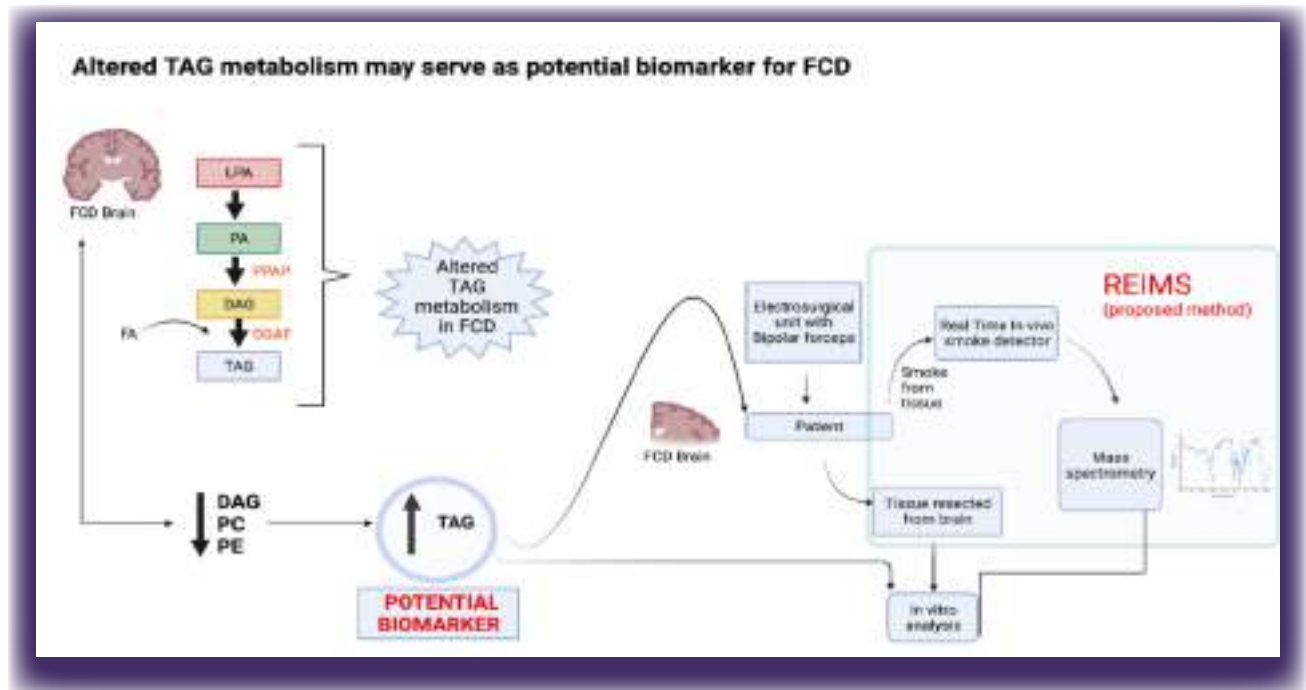
Role of HDACs and its sub-cellular distribution was studied in MTLE patients with hippocampal sclerosis. Knowledge regarding expression pattern and sub-cellular distribution of HDACs may help to devise specific HDACi therapy for epilepsy. Proteomic studies on MTLE patients have been conducted. The findings of the present study are published in Cellular & Molecular Neurobiology. Srivastava A, Banerjee J, Dubey V, Tripathi M, Chandra PS, Sharma MC, Lalwani S, Siraj F, Doddamani R, Dixit AB. Role of Altered Expression, Activity and Sub-cellular Distribution of Various Histone Deacetylases (HDACs) in Mesial Temporal Lobe Epilepsy with Hippocampal Sclerosis. Cell Mol Neurobiol. 2022;42(4):1049-1064.

Alteration in the TGF β signalling, CK2, Cdk5, cPLA2 enzyme, Fibronectin-integrin-Src pathway and MMPs were demonstrated in MTLE-HS patients. These molecules may represent new potential therapeutic target for treatment of MTLE. Bera A, Srivastava A, Dubey V, Dixit AB, Tripathi M, Sharma MC, Lalwani S, Chandra PS, Banerjee J. Altered hippocampal expression and function of cytosolic phospholipase A2 (cPLA2) in temporal lobe epilepsy (TLE). Neurol Res. 2022;44(8):748-753. Paul D, Dixit AB, Srivastava A, Banerjee J, Tripathi M, Suman P, Doddamani R, Lalwani S, Siraj F, Sharma MC, Chandra PS, Singh RK. Altered expression of activating transcription factor 3 in the hippocampus of patients with mesial temporal lobe epilepsy-hippocampal sclerosis (MTLE-HS). Int J Neurosci. 2022; 21:1-7. Paul D, Dixit AB, Srivastava A, Banerjee J, Tripathi M, Suman P, Doddamani R, Lalwani S, Siraj F, Sharma MC, Chandra PS, Singh RK. Altered expression of activating transcription factor 3 in the hippocampus of patients with mesial temporal lobe epilepsy-hippocampal sclerosis (MTLE-HS). Int J Neurosci. 2022; 21:1-7.

Our studies demonstrated that Cdk5 differentially regulates excitatory synaptic activity in the hippocampal and ATL region of patients with MTLE-HS. We also demonstrated that it might have a potential role in increasing the stability of gephyrin-dependent γ 2 subunit containing GABAA receptor clusters in FCD. Part of this study has been published in Neuroscience Letters. Banerjee J, Srivastava A, Sharma D, Dey S, Manjari Tripathi, Sharma MC, Sarat Chandra P, Banerjee Dixit A. Differential regulation of excitatory synaptic transmission in the hippocampus and anterior temporal lobe by cyclin dependent kinase 5 (Cdk5) in mesial temporal lobe epilepsy with hippocampal sclerosis (MTLE-HS). NeurosciLett. (2021):136096. doi: 10.1016/j.neulet.2021.136096.

We utilize liquid chromatography and tandem mass spectrometry to identify altered lipids in resected brain specimens from FCD patients compared to non-epileptic controls. Based on these results, we propose that a similar approach utilizing unique lipid mass spectra can be used for defining the EZs in FCD. The observed distinct lipid mass spectra of cortical tissues from FCD patients could be used for real-time guidance during surgery as well as for ex vivo examination of resected tissues for diagnostic purposes. This study

has been published in Epilepsy Research: Kumar K, Yadav N, Banerjee J, Tripathi M, Sharma MC, Lalwani S, Siraj F, Chandra PS, Sengupta S, Dixit AB. Mass spectrometry-based lipidomic analysis reveals altered lipid profile in brain tissues resected from patients with focal cortical dysplasia (FCD). Epilepsy Res. 2021; 177:106773.



The current findings show association of altered TAG synthesis/metabolism with FCD pathology. Downregulation of DAGs, phosphatidylcholine, and phosphatidylethanolamine in FCD further suggests that degradation and/or decreased synthesis of these structural glycopospholipids and DAGs may contribute to increased TAGs. We propose that increased TAG levels could serve as potential biomarker of FCD and the lipid mass spectra, if validated on a greater number of FCD patients, can aid in real time identification of resection margins akin to intelligent knife (iKnife) approach.

Publications:

1. Kumar K, Dubey V, Zaidi S.S., **Tripathi M.**, Siraj F, Sharma M.C., Chandra P.S., Doddamani R, Dixit A.B., Banerjee J. (2023). *RNA Sequencing of Intraoperative Peritumoral Tissues Reveals Potential Pathways Involved in Glioma-Related Seizures*. **Journal of Molecular Neuroscience**, 73(6):437-447. doi: 10.1007/s12031-023-02125-y. PMID: 37268865.
2. Kumar S, Shanker O.R., Banerjee J, **Tripathi M.**, Chandra P.S., Dixit A.B. (2023). *Epigenetics in Epilepsy*. **Progress in Molecular Biology and Translational Science**, 198:249-269. doi: 10.1016/bs.pmbts.2023.01.005. PMID: 37225322.
3. Shanker O.R., Kumar S, Dixit A.B., Banerjee J., **Tripathi M.**, Sarat Chandra P. (2023). *Epigenetics of Neurological Diseases*. **Progress in Molecular Biology and Translational Science**, 198:165-184. doi: 10.1016/bs.pmbts.2023.01.006. PMID: 37225320.

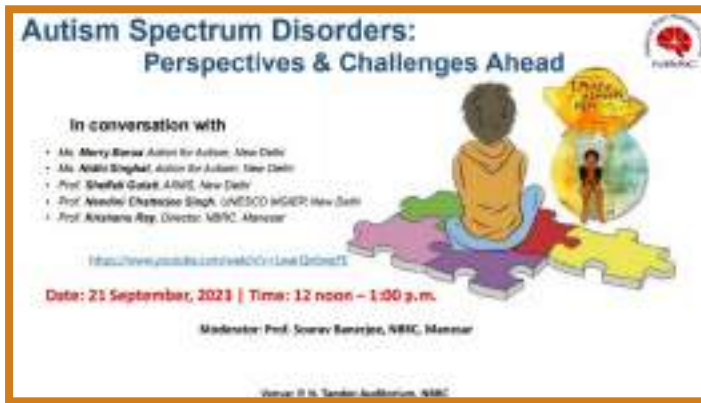
4. Chakraborty A, Dey S, Kumar K, Dixit A.B., **Tripathi M.**, Sharma M.C., Chandra P.S., Banerjee J. (2023). *Novel Variants in GABA_A Receptor Subunits: A Possible Association with Benzodiazepine Resistance in Patients with Drug-Resistant Epilepsy*. **Epilepsy Research**, 189:107056. doi: 10.1016/j.eplesyres.2022.107056. PMID: 36469977.
5. Singh V, Grewal K.S., Vibha D, Singh R.K., Ramanujam B, Nehra A, Chandra S.P., Gaikwad S, Babu I, **Tripathi M.** (2023). *Cortico-limbic Disruption, Material-Specificity, and Deficits in Cognitive-Affective Theory of Mind*. **Brain Communications**, 5(2). doi: 10.1093/braincomms/fcad100. PMID: 37101833; PMCID: PMC10123397.
6. Roth J, Bergman L, Weil A.G., Brunette-Clement T, Weiner H.L., Treiber J.M., Shofty B, Cukiert A., Cukiert C.M., **Tripathi M.**, Sarat Chandra P., Bollo R.J., Machado H.R., Santos M.V., Gaillard W.D., Oluigbo C.O., Ibrahim G.M., Jallo G.I., Shimony N., O'Neill B.R., Budke M., Pérez-Jiménez M.Á., Mangano F.T., Iwasaki M., Iijima K., Gonzalez-Martinez J., Kawai K., Ishishita Y., Elbabaa S.K., Bello-Espinosa L., Fallah A., Maniquis C.A.B., Ben-Zvi I., Tisdall M., Panigrahi M., Jayalakshmi S., Blount J.P., Dorfmueller G., Bulteau C., Stone S.S., Bolton J., Singhal A., Connolly M., Alsowat D., Alotaibi F., Ragheb J., Uliel-Sibony S. (2023). *Added Value of Corpus Callosotomy Following Vagus Nerve Stimulation in Children with Lennox-Gastaut Syndrome: A Multicenter, Multinational Study*. **Epilepsia**, 64(12):3205-3212. doi: 10.1111/epi.17796. PMID: 37823366.

Events & Outreach Activities ►►



Prof. Krishanu Ray, Director, NBRC, interacting with students while explaining #Brain & its #functionality, & how it acts as the main controller of all the #voluntary actions of the #body





On September 21st, 2023, the National Brain Research Centre (NBRC) hosted an enlightening livestreamed discussion on **Autism Spectrum Disorders** (ASD). Streaming live from the NBRC campus in Manesar, Haryana, the session brought together experts who emphasized that "Autism is not a disease, but a condition," aiming to promote understanding and awareness of ASD. The discussion offered valuable insights into the neuroscience behind autism and highlighted approaches to mental wellness and inclusion. This event underscored NBRC's commitment to advancing knowledge in neuroscience and fostering conversations around mental health and neurodiversity.



Organellar Biology and Membrane Trafficking (OBMT) Meeting

October 9-11, 2023 at NBRC, Manesar, Haryana



Global Bio India ▶▶

December 4-6, 2023 at Bharat Mandapam, Pragati Maidan, New Delhi



Foundation Day



On December 16th 2023, NBRC celebrated its Foundation Day with a dynamic program featuring Dr. Rajesh S. Gokhale, Secretary of DBT, as the Chief Guest. The day commenced with interactive lab tours, poster presentations, and an exciting quiz competition for visiting students, culminating in a prize distribution by Dr. Gokhale.

The formal program opened with a welcome by the Director of NBRC, followed by the keynote lecture, "*Newer Frontiers in Psychiatry: Bridging the Brain and Mind*" delivered by Prof. Pratima Murthy, Director of NIMHANS, Bangalore. Open to all, this public lecture offered a valuable opportunity for attendees from diverse backgrounds to gain insights into the evolving connections between neuroscience and mental health. Prof. Murthy's engaging talk shed light on the interplay between brain

and mind, making complex topics accessible to both specialists and the general public.

The event concluded with a valedictory address by Dr. Gokhale, who acknowledged NBRC's advancements in neuroscience research. The day wrapped up with high tea, providing a relaxed space for attendees to connect and reflect on the valuable discussions shared throughout the day.





National Brain Research Centre
Manesar (Gurgaon), Haryana

celebrates
National Science Day
and invites you all to our
Open Day

On February 28, 2024
9:30 AM – 1:00 PM

Witness how a human brain works!

- Lab Visits
- Demonstration of Human Brain
- Brain Imaging Tools
- Panel Discussion: "Identifying Stress and Its management"

Prof. Prasad Sharan, AIIMS, New Delhi
Dr. Rohinddeep Sindhu, Civil Hospital Jaipur

On **National Science Day**, February 28th 2024, NBRC hosted an exciting Open Day, inviting students and the public to explore the wonders of the human brain and the marvels of science. Over 100 enthusiastic students joined the celebration honoring Sir C.V. Raman's legacy. The event featured engaging talks by Prof. Sharan from AIIMS and Dr. Sindhu, who shared valuable insights on stress management, sparking curiosity and inspiring young minds. Attendees enjoyed a hands-on experience and learned about the latest in neuroscience, making it a memorable celebration of science and discovery!



NBRC – JNCASR MINI SYMPOSIUM

January 29-30, 2024



NBRC – JNCASR MINI-SYMPOSIUM

Brain Disorders: Perspectives from Developing and Aging Brain
29th - 30th January, 2024
Venue for all presentations: P.N. Tandon Auditorium, NBRC
Host: Dr Sourav Banerjee, Scientist-VI & Professor

Day 1:- 29th January, 2024

10:30 – 10:40 am:	Opening remarks by Krishanu Ray and Ravi Manjithaya
10:40 – 11:00 am:	Sheeba Vasan Title: A fly model to study the impact of circadian health on developmental and ageing processes
11:00 – 11:20 am:	Mayanglamon Dhruba Singh Title: Downregulation of PTEN provides neuroprotection in Huntington's disease model in <i>Drosophila</i>
11:20 – 11:40 am:	Sourav Banerjee Title: RNA-based mechanisms of neurodevelopmental disorders
12:10 – 12:30 noon:	Kushagra Bansal Title: Thymic development of T cells
12:30 – 12:50 noon:	Sourya Prasad Title: Serpents: A Model System to Study Abnormalities in Learned Vocalizations
12:50 – 1:10 pm:	Bhavana Shrikant Saha Title: Biogenesis of secretory vesicles and investigating the functional status of regulated exocytosis in Alzheimer's disease
2:30 – 2:50 pm:	Tapas Kundu Title: Epigenetic Regulation of Autism
2:50 – 3:20 pm:	Arpan Banerjee Title: TBA
3:20 – 3:50 pm:	Ravi Manjithaya Title: Neuronal autophagy: its regulation and implications in neurodegeneration
3:50 – 4:10 pm:	Pravat Mandal Title: TBA

Day 2:- 30th January, 2024

10:30 – 10:50 am:	Anuraj Anand Title: Human epilepsy genetics: Abundance of opportunities
10:50 – 11:10 am:	Achira Ray Title: Modeling early-onset neurodevelopmental disorders – brain overgrowth, hydrocephalus, and epilepsy
11:10 – 11:30 am:	Anindya Ghosh Ray Title: Functional wiring of neural circuits
11:30 – 11:50 pm:	Krishanu Ray Title: The molecular cell biology of insulin-stimulated axonal transport and its impact on synaptic stability



Symposium on Brain, Behaviour and Society

NBRC organized an inspiring symposium titled *"Brain, Behaviour, and Society"* from December 13-15, 2023. Over three days, leading scientists and young researchers from around the world gathered to discuss the latest discoveries in cognitive neuroscience. The theme, *"Social Brain – from Brain Functions to Society,"* focused on understanding how the brain shapes our behavior and influences society.

The event brought together a range of experts, creating an open, collaborative space for sharing ideas and exploring how neuroscience impacts our daily lives.



India International Science Festival (IISF) 2023

January 17-20, 2024 at DBT-THSTI - RCB Campus, Faridabad, Haryana



General & Academic Administration



General & Academic Administration – A Profile

The Administration of the Institute consists of the following major wings:

1. General Administration is headed by the Chief Administrative Officer, who is responsible for overall Management of Establishment, Personnel & Administration Wing, Stores & Purchase Wing, Import & Project Cell, Finance & Accounts Wing, Estate Management & Engineering Maintenance Wing – Civil, Electrical & Mechanical.
2. Academic Administration is headed by the Registrar, who is responsible for the students' administration, project co-ordination, new students' admissions, course co-ordination etc. The officer is also responsible for administration of all the projects.

During the year under review, the Administration of NBRC observed all the important days as directed by the Government of India such as Republic Day, Independence Day, Hindi Diwas/ Week, Vigilance Awareness week, International Yoga Day etc. The Administration achieved excellence in execution of the following activities at NBRC:

- The annual cultural festival of NBRC, 'TANTRIKA 2024' was organized within the campus which included a variety of cultural and sports events. Students, officers, and staff of NBRC participated in the event. The following talks were organized during TANTRIKA 2023.
 - ❖ A talk by Ms. Shriya Naidu, Program Associate, Foundation for Advancing Science and Technology was organized on 12th March, 2024 at 3.00 pm.
 - ❖ A talk by Ms. Prathyusha Potharaju, Co-Founder, Grailmaker Innovations was organized on 12th March, 2024 at 2.00 pm.

Provided necessary logistics in conducting international and national conferences/ seminars organized in the campus as well outside the campus.

Made major imports from different countries in terms of equipment and other consumables with meticulous planning and adhered to a precise schedule.

The Foundation Day of NBRC was held on 16th day of December, 2023. On this occasion, several programmes were organized within the campus. The daylong celebrations included the poster presentations on ongoing research activities of NBRC. Students from various schools were invited to interact with NBRC scientists and they visited the laboratories.

On this august occasion, a Public Lecture by Prof Pratima Murthy, Director, NIMHANS, Bangalore was delivered to the students and scientific community at NBRC, Manesar.

Implementation of Official Language

NBRC Administration has given due importance for the implementation of Hindi as



the Official Language at this centre and has made full efforts to implement the Official Language in all the administrative jobs such as internal official meetings, interviews, debates, general applications etc.

RTI Act

The provisions of RTI Act are being followed at NBRC in letter and spirit. All RTI applications received during 2023-24 seeking information on various matters concerning NBRC were disposed of within the prescribed time limit. The quarterly reports containing number of requests received with date, details of compliance, amount of charges etc., were sent to CIC and updated on NBRC website.

Women Empowerment

NBRC has a distinct feature of giving equal opportunity to women. The Committees, constituted to do various work of Administration, Academics and scientific activities, have women members on them which ensure fair participation and protection of women. There is a committee for redressal of complaints relating to any sexual harassment of women at NBRC and grievances, if any, from aggrieved girl students/ women employees of NBRC. Any lady/ woman of NBRC, among the Students/ Employees who is subjected to sexual harassment may approach any of the committee members.

Reservations and concessions in Employment & Admissions of Students

NBRC follows reservations & concessions as per rules of Government of India in employment; in the matter of students' admissions, the provision of exemption as provided in Gazette Notification No. 5 dated 4th January, 2007 is applicable.

Vigilance

The Institute has a Chief Vigilance Officer. As per the approval of CVC communicated through DBT, one of the scientists of NBRC has been nominated as part-time Chief Vigilance Officer of the Centre.

Swachh Bharat Special Campaign ▶▶▶



Hindi Pakhwada ▶▶▶



Vigilance Awareness Week



National Unity Day



Structure & Governance



Members of NBRC Society ▶▶▶

S. No.	Name	Designation
1.	Dr. Jitendra Singh, Hon'ble Minister of State (IC) Ministry of S&T, Govt. of India New Delhi	President
2.	Shri. Anil Vij Hon'ble Minister in-charge of the Departments of Haryana Ayush, Health Services, Medical Education, and Science & Technology	Member (Ex-Officio)
3.	Dr. Rajesh S. Gokhale Secretary, Department of Biotechnology Ministry of Science & Technology Government of India New Delhi	Member (Ex-Officio)
4	Dr. Rajiv Bahl Secretary, Department of Health Research and Director General, Indian Council of Medical Research (ICMR), New Delhi	Member (Ex-Officio)
5	Dr. Srivari Chandrasekhar, Secretary, Ministry of Science and Technology Government of India New Delhi	Member (Ex-Officio)
6.	Dr. Ashok Khemka Principal Secretary, Department of Science & Technology, Govt. of Haryana	Member (Ex-Officio)
7.	Shri. Chaitanya Murti Joint Secretary (Admin), Department of Biotechnology, New Delhi	Member (Ex-Officio)
8.	Shri. Vishvajit Sahay Additional Secretary & Financial Advisor, Department of Biotechnology, New Delhi	Member (Ex-Officio)

9.	Prof. Krishanu Ray Director National Brain Research Centre, Manesar, Haryana	Member Secretary (Ex-Officio)
10.	Dr. Ashok Panagariya, Former IMA President, Jaipur	Member
11.	Prof. Vijaylakshmi Ravindranath, Professor, Centre for Neuroscience, Indian Institute of Science (IISc), Bengaluru.	Member
12.	Prof. Prabhu Nath Pandey, Professor & Head, Department of Neurosurgery, Lok Nayak Jai Prakash Narayan Hospital, New Delhi.	Member
13.	Prof. M. V. Padma Srivastava, Professor & Head, Department of Neurology, Chief Neurosciences Centre, AIIMS, New Delhi.	Member
14.	Prof. B. N. Gangadhar, Former Director, National Institute of Mental Health and Neuro-Sciences (NIMHANS), Bengaluru.	Member
15.	Dr. Senapathy 'Kris' Gopalakrishnan, Co-Founder, Infosys Ltd., Bengaluru.	Member



Members of Governing Body ▶▶▶

S. No.	Name	Designation
1.	Dr. Rajesh S. Gokhale Secretary Department of Biotechnology, C.G.O Complex, New Delhi – 110 003	Chairperson
2.	Shri Chaitanya Murti Joint Secretary (Admin), Department of Biotechnology, C.G.O Complex, New Delhi – 110 003	Member (Ex-Officio)
3.	Shri. Vishvajit Sahay Additional Secretary & Financial Advisor, Department of Biotechnology, C.G.O Complex, New Delhi – 110 003	Member (Ex-Officio)
4.	Dr. Sanjay Kr. Mishra Scientist - H Department of Biotechnology, C.G.O Complex, New Delhi – 110 003	Member (Ex-Officio)
5.	Prof. Krishanu Ray Director National Brain Research Centre, Nainwal Road, Manesar – 122 052, Haryana	Member (Ex-Officio)
6.	Prof. Pravat Kr. Mandal Scientist-VII National Brain Research Centre, Nainwal Road, Manesar – 122 052, Haryana	Member (Ex-Officio)

7.	Dr. Suraksha S. Diwan Scientist 'F', Department of Biotechnology, C.G.O Complex, New Delhi – 110 003	Member (Ex-Officio) (Nodal Officer of NBRC)
8.	Shri. Tanmoy Bhattacharya Chief Administrative Officer National Brain Research Centre, Nainwal Road, Manesar – 122 052, Haryana	Member Secretary (Ex-Officio)
9.	Prof. Vidita Vaidya, Professor, Department of Biological Science, Tata Institute of Fundamental Research (TIFR), Mumbai.	Member
10.	Prof. Jayasri Das Sarma, Department of Biological Sciences, Indian Institute of Science Education and Research (IISER), Kolkata.	Member
11.	Prof. M. R. Satyanarayana Rao, Honorary Professor, Chromatin Biology Laboratory, Neuroscience Unit (NSU), Jawaharlal Nehru Centre for Advanced Scientific Research (JNCASR), Bengaluru.	Member
12.	Prof. Kameshwar Prasad, Former Professor, Department of Neurology, All India Institute of Medical Sciences (AIIMS), and Director, RIMS, Ranchi New Delhi	Member

Members of the Finance Committee



S. No.	Name	Designation
1.	Sh. Vishvajit Sahay Additional Secretary & Financial Advisor, Department of Biotechnology, New Delhi – 110003	Chairperson
2.	Dr. Sanjay Kr. Mishra Scientist - H Department of Biotechnology, New Delhi	Member (Ex-Officio)
3.	Prof. Krishanu Ray Director National Brain Research Centre, Nainwal Road, Manesar – 122 052, Haryana	Member (Ex-Officio)
4.	Shri. Tanmoy Bhattacharya Chief Administrative Officer National Brain Research Centre, Nainwal Road, Manesar – 122 052, Haryana	Member (Ex-Officio)
5.	In-Charge F&AO National Brain Research Centre, Nainwal Road, Manesar – 122 052, Haryana	Member Secretary (Ex-Officio)
6.	Prof. Pramod Kumar Garg Executive Director Translational Health Science and Technology Institute (THSTI) NCR Biotech Science Cluster 3 rd Milestone, Faridabad-121001 (Haryana)	Member



7.	Shri Praveen Kumar Bansal, Former Vice President, Income Tax Appellate Tribunal, Government of India	Member
8.	Shri M. Satish Kumar Reddy, IRS, Former Commissioner of Central Goods and Services Tax (CGST) and Customs, Government of India	Member

Members of Scientific Advisory Committee

S. No.	Name	Designation
1.	Prof. Siddharta Roy, J.C. Bose Fellow, Department of Biophysics Centenary Campus, Bose Institute Kolkata	Chairperson
2.	Dr. Sanjay Kumar Mishra Scientist – H, (Scientific coordinator, NBRC) Department of Biotechnology, New Delhi	Member (Ex-Officio)
3.	Prof. Krishanu Ray Director National Brain Research Centre, Nainwal Road, Manesar, Haryana	Member Secretary (Ex-Officio)
4.	Prof. Vidita Vaidya Professor Department of Biological Science, Tata Institute of Fundamental Research (TIFR), Mumbai	Member
5.	Prof. Jayasri Das Sarma Professor Department of Biological Sciences, Indian Institute of Science Education & Research (IISER), Kolkata	Member

6.	Prof. Kameshwar Prasad Director Rajendra Institute of Medical Sciences, Ranchi, Jharkhand	Member
7.	Prof. Venkatasubramanian Ganesan Officer In-Charge Translational Psychiatry Laboratory Cognitive Neurobiology Division, Neurobiology Research Centre, National Institute of Mental Health & Neuro- Sciences (NIMHANS), Bengaluru	Member
8.	Prof. Ashalatha Radhakrishnan Professor Department of Neurology, Sree Chitra Tirunal Institute of Medical Sciences and Tech- nology (SCTIMST), Thiruvananthapuram, Kerala	Member
9.	Prof. Rama Jayasundar Professor & Head Department of Nuclear Magnetic Resonance (NMR), AIIMS, New Delhi	Member
10.	Prof. Vijay Kumar Kuchroo Samuel L. Wasserstrom Professor of Neurology, Harvard Medical School, Boston, USA	Member



BOARD OF STUDIES

Prof. Krishanu Ray Director, National Brain Research Centre, Manesar, Haryana	Dr. Sushil Kumar Jha Associate Professor, Jawaharlal Nehru University, New Delhi
Prof. Kunzang Chosdol Department of Biochemistry, All India Institute of Medical Sciences, New Delhi	All the faculty members of NBRC
Dr. Sushil Kumar Jha Associate Professor, Jawaharlal Nehru University, New Delhi	Offg. Registrar, Ex-Officio Member

Members of the Academic Council

Prof. Krishanu Ray Director, National Brain Research Centre Manesar, Haryana	Prof. K.Natarajan, University of Delhi, External Member
Prof. James Gomes, IIT Delhi, External Members	Prof. Chinmay K.Mukhopadhyay, JNU, New Delhi External Member
And all the faculty members of NBRC	Offg. Registrar, Ex-Officio Member

Members of the Building Committee

S. No.	Name	Designation
1.	Prof. Dinakar M. Salunke Chairperson Director, International Centre for Genetic Engineering and Biotechnology New Delhi	Chairperson
2.	Dr. Amulya K. Panda Director, National Institute of Immunology (NII), New Delhi	Member
3.	Dr. S. K. Gupta Deputy Director (Retired) & Emeritus Scientist National Institute of Immunology (NII), New Delhi	Member
4.	Shri. V. H. Rao Senior Consultant Engineering, Mehrauli - Badarpur Road, Near Batra Hospital, Hamdard Nagar, New Delhi	Member
5.	Shri. M. K. Gupta Engineer-In-Charge (Civil) IUAC New Delhi	Member
6.	Prof. Sidhartha Satpathy HOD, Hospital Administration, AIIMS, New Delhi	Member
7.	Prof. Krishanu Ray, Director (Ex-officio) National Brain Research Centre Manesar, Haryana	Member (Ex-Officio)



Staff at NBRC

Scientific Staff

S. No.	Name	Designation
1.	Prof. Krishanu Ray	Director, NBRC
2.	Prof. Pankaj Seth	Scientist – VII
3.	Prof. Anirban Basu	Scientist – VII
4.	Prof. Pravat Kumar Mandal	Scientist – VII (Retired on 31.12.2023)
5.	Prof. Soumya Iyengar	Scientist – VII
6.	Prof. Nandini C. Singh	Scientist – VI (Currently on deputation to UNESCO-MGIEP, New Delhi)
7.	Prof. Ellora Sen	Scientist – VII
8.	Prof. Ranjit Kumar Giri	Scientist – VI
9.	Prof. Sourav Banerjee	Scientist – VI
10.	Prof. Arpan Banerjee	Scientist – VI
11.	Prof. Anindya Roy Ghosh	Scientist – VI
12.	Dr. Mayanglambam Dhruba Singh	Scientist - III
13.	Dr. Bhavani Shankar Sahu	Scientist - IV
14.	Mr. Mahender Kumar Singh	Information Scientist

Technical Staff

S. NO.	Name of Employee	Designation
1.	Mr. Sanjeev Kr. Choudhary	Assistant Engineer
2.	Dr. D.D. Lal	Technical Officer
3.	Mr. Jitender Ahlawat	Technical Officer – B
4.	Mr. Arvind Singh Pundir	Technical Officer – B
5.	Dr. Inderjeet Yadav	Veterinarian
6.	Mr. Kedar Singh Bajetha	Computer Operator
7.	Ms. Seepika	Computer Operator
8.	Mr. Sachin Kumar	Computer Operator
9.	Ms. Tarnnum Mansoori	Computer Operator
10.	Mr. Sanjeev Bhardwaj	Computer Operator
11.	Dr. Kanhaiya Lal Kumawat	Technician-C
12.	Mr. Shankar Datt Joshi	Technician-C
13.	Mr. Sumit Kumar Sinha Mahapatra	Technician-C
14.	Mr. D. Narender	Technician-C
15.	Mr. Sanjay Kumar	Technician-B
16.	Mr. Mithlesh Kumar Singh	Technician-B
17.	Mr. Ankit Sharma	Technician-B
18.	Mr. Yunish Khan	Technician-B
19.	Mr. Durga Lal Meena	Technician-B
20.	Md. Irshad Alam	Technician-B
21.	Mr. Manish Kumar	Technician-B
22.	Mr. P. Manish	Technician-B
23.	Mr. Dil Bahadur Karki	Technician-A
24.	Mr. Rammehar	Technician-A
25.	Mr. Hari Shankar	Technician-A
26.	Mr. Mahendra Singh	Technician-A
27.	Mr. Sanjay Kumar Singh	Technician-A



Administrative Staff

S. No.	Name of Employee	Designation
1.	Mr. Tanmoy Bhattacharyya	Chief Administrative Officer
2.	Mr. Santosh Kumar Choudhary	Deputy Finance Officer
3.	Ms. Pooja Gosain	Administrative Officer
4.	Ms. Shiwani Tanwar	Administrative Officer (Acad.)
5.	Mr. Ravinder Pal	Store & Purchase Officer
6.	Mr. Suraj Bhan	Office Assistant
7.	Mr. Rakesh Kumar Yadav	Office Assistant
8.	Mr. Himanshu Mal	Office Assistant
9.	Mr. Ajay Kumar Dehariya	Office Assistant
10.	Mr. Parmander Singh Rawat	Office Assistant
11.	Mr. Jitendra Kumar Meena	Office Assistant
12.	Mr. Sanjay Kr. Gupta	Office Assistant
13.	Mr. Bhupender Pal Sharma	Driver
14.	Mr. Satish Kumar	Driver



Project Employees

Project Assistants

S/No	Name and Designation	Till Date
1	Ms. Sruthy Ravivarma, Project Assistant	19-05-2023
2	Ms. Saumya Rastogi, Project Assistant	30-05-2023
3	Ms. Tuhina Sanwal, Project Assistant	N/A
4	Ms. Mydhily Vasudevan, Project Assistant	N/A
5	Mr. Samuel Bekins S, Project Assistant	N/A

Junior Research Fellows

S/No	Name and Designation	Till Date
1	Ms. Risna K. R., Junior Research Fellow	01-09-2023
2	Ms. Aishwarya TMM Iyengar, Junior Research Fellow	28-07-2023
3	Ms. Esha Shiposkar, Junior Research Fellow	N/A
4	Dr. Pooja Parishar, Junior Research Fellow	04-01-2024
5	Ms. Avantika Samkaria, Junior Research Fellow	14-03-2024

Research Associates

S/No	Name and Designation	Till Date
1	Dr. Dimpi, Research Associate-1	N/A
2	Dr. Yashika Arora, Research Associate	31-01-2024
3	Dr. Anupama K.P., Research Associate	08-06-2023
4	Dr. Meenakshi Bhaskar, Research Associate	N/A
5	Dr. Anagh Pathak, Research Associate - III	10-06-2024
6	Ms. Anshita Singh, Research Associate - I	28-03-2024
7	Dr. Yambem Soniya Devi, Research Associate - I	N/A
8	Dr. S Balakumar, Research Associate - I	N/A
9	Dr. Atreye Majumdar, Research Associate - I	17-09-2024
10	Dr. Naorem Tarundas Singh, Research Associate - I	N/A

S/No	Name and Designation	Till Date
11	Dr. Himali Arora, Research Associate - I	11-06-2024
12	Dr. Kirti Lathoria, Research Associate - I	N/A
13	Dr. Puyam Milan Meitei, Research Associate - I	N/A
14	Dr. Suman Saha, Research Associate - III	28-06-2024
15	Dr. Neeraj Kumar, Research Associate - I	N/A
16	Dr. Sibaram Behra, Research Associate - I	N/A
17	Dr. Sushma Dagar, Research Associate (RA-1)	N/A
18	Dr. Bindu, Research Associate - I	N/A

Project Associates

S/No	Name and Designation	Till Date
1	Ms. Reena, Project Associate-1	31.07.2023
2	Ms. Ishanee Bandyopadhyay, Project Associate-1	27-02-2024
3	Ms. Ishanee Bandyopadhyay, Project Associate - I	N/A
4	Mr. Karthick R, Project Associate - I	N/A
5	Mr. Akash Gupta, Project Associate - I	N/A
6	Mr. Neeraj Kasana, Project Associate - II	N/A
7	Ms. Shalini, Project Associate - II	N/A

Project Scientists, Engineers, and Specialists

S/No	Name and Designation	Till Date
1	Dr. Sonia Mor, Clinical Trial Specialist	12-04-2023
2	Mr. Saurav Roy, Scientist	02-06-2023
3	Ms. Aditi Kaushik, Project Scientist-1	07-01-2024
4	Ms. Aanchal Choudhary, Senior R&D Engineer	06-04-2023

MEG Project

S/No	Name and Designation	Till Date
1.	Mr. Manjit, Lab Attendant (MEG Project)	30.09.2023
2.	Mr. Om Prakash Jakhar, Nurse (MEG Project)	30.09.2023
3.	Ms. Priya Shrivastav, Nurse (MEG Project)	30.09.2023
4.	Mr. Gaurav Singh, Technologist (MEG Project)	30.09.2023
5.	Mr. Sachin Kumar, Lab Attendant (MEG Project)	30.09.2023
6.	Mr. Deepak Kumar, Nursing Orderly (MEG Project)	30.09.2023
7.	Ms. Meenu Yadav, Technician (MEG Project)	30.09.2023
8.	Mr. Amit Kumar Srivastava, Nursing Orderly (MEG Project)	30.09.2023
9.	Ms. Divyasree V.M, Nurse (MEG Project)	30.09.2023
10.	Mr. Gaurav Rawar, Technician (MEG Project)	30.09.2023

Other Technicians

S/No	Name and Designation	Till Date
1.	Ms. Mydhily Vasudevan, Technician	N/A

DBT/WT India Alliance Fellow

S/No	Name and Designation	Till Date
1.	Dr. Swagata Dey, DBT/WT India Alliance Fellow	N/A

National Postdoctoral Fellowship

S/No	Name and Designation	Till Date
1.	Dr. Suman Saha, National Postdoctoral Fellowship	09-03-2024



General & Integrated Students ▶▶▶

Ph.D. Degrees Awarded

Sr.No.	Name of the Student
1.	Sarbani Samaddar
2.	Gourav Sharma
3.	S Balakumar
4.	Pooja Parishar
5.	Meenakshi Bhaskar
6.	Atanu Datta
7.	Harjot Kaur Brar
8.	Md. Tipu Khan
9.	Anagh Pathak
10.	Shivangi Sharma
11.	Stuti Mohapatra
12.	Kirti Lathoria
13.	Himali Arora
14.	Neeraj Kumar
15.	Shalini Sharma
16.	Gargi Majumdar
17.	Nisha Chetana Sastry
18.	Sibaram Behra

M.Sc. Degrees Awarded

Sr.No.	Name of the Student
1.	Sudipto Chaki
2.	Triparna Chakraborty
3.	Debadrita Mondal
4.	Tulika Khargonkar
5.	Sreyashi Bhattacharjee
6.	Aninda Sundar Modak
7.	Soumi Sen
8.	Mihir Pradhan
9.	Shivam Malviya
10.	Tanmay Singhal
11.	Rishabh Bapat

Ph.D. Students

Sr.No.	Name of the Student
1.	Ms. Kamakshi Garg
2.	Ms. Partika
3.	Ms. Anjali
4.	Mr. Ankit Yadav
5.	Ms. Archana Mehta
6.	Mr. Chandramouli Mukherjee
7.	Ms. Sonia Balahun Umdor
8.	Mr. Anirudh S
9.	Ms. Archana Panwar
10.	Ms. Athira M Sarath
11.	Ms. Deepti Thapliyal
12.	Mr. Dhyey Vyas
13.	Ms. Krittika Biswas
14.	Ms. P. V. Vinitha
15.	Ms. Sania Sultana
16.	Mr. Shashank Saxena
17.	Ms. Anushka
18.	Ms. Archismita Chatterjee
19.	Ms. Chakshu Mangal
20.	Ms. Dikshalee Bassi
21.	Ms. Dipti Chakraborty
22.	Ms. Pallabi Kisku
23.	Ms. Shafaq Aftab
24.	Mr. Ashish Singh Panchal
25.	Ms. Soumya Golchha
26.	Ms. Banismita Tripathy
27.	Ms. Akasmika Priyadarsani
28.	Ms. Reena
29.	Ms. Lakhuhong Ningchangmi
30.	Ms. Palki Chauksey
31.	Ms. Tamanna Saini



32.	Mr. Maulik Patel
33.	Ms. Ritu Moni Borah
34.	Mr. Azman Akhter
35.	Ms. Kirti Saluja
36.	Ms. Pallavi Singh
37.	Ms. Shashwati Tripathi
38.	Ms. Vinsea AV Singh
39.	Ms. Mohima Mukherjee
40.	Mr. Rishabh V Bapat
41.	Mr. Tanmay Singhal
42.	Mr. Mihir Pradhan
43.	Mr. Shivam Malviya
44.	Ms. Nisha Chetana Sastry (Upto 20th September, 2023)
45.	Ms. Gargi Majumdar (Upto 18th January, 2024)
46.	Ms. Shalini Sharma (Upto 29th February, 2024)
47.	Mr. Neeraj Kumar
48.	Ms. Himali Arora (10th January, 2024)
49.	Ms. Kirti Lathoria (Upto 22nd February, 2024)
50.	Ms. Stuti Mohapatra (Upto 26th December, 2023)
51.	Ms. Shivangi Sharma (Upto 18th December, 2023)
52.	Mr. Anagh Pathak (Upto 03rd July, 2023)
53.	Mr. Md. Tipu Khan (Upto 08th November, 2023)
54.	Ms. Harjot Kaur Brar (Upto 22nd June, 2023)
55.	Ms. Meenakshi Bhaskar (Upto 20th September, 2023)
56.	Ms. Pooja Parishar (Upto 23rd August, 2023)
57.	Mr. S. Balakumar (Upto 17th August, 2023)
58.	Mr. Gourav Sharma (Upto 17th August, 2023)
59.	Ms. Sarbani Samaddar (Upto 22nd August, 2023)



M.Sc. Students

Sr.No.	Name of the Student
1.	Ms. Vibha V
2.	Ms. Aashi
3.	Ms. Gauri Shintre
4.	Mr. Atharva Ashutosh Parolekar
5.	Ms. Sanchari Banerjee
6.	Mr. Austin Varughese
7.	Ms. Ritu Sarkar
8.	Ms. Aarya Mahendraa Patil
9.	Mr. Sudipto Chaki (Upto 31st July, 2023)
10.	Ms. Triparna Chakraborty (Upto 31st July, 2023)
11.	Ms. Debadrita Mondal (Upto 31st July, 2023)
12.	Ms. Tulika Khargonkar (Upto 31st July, 2023)
13.	Ms. Sreyashi Bhattacharjee (Upto 31st July, 2023)
14.	Mr. Aninda Sundar Modak (Upto 31st July, 2023)
15.	Ms. Soumi Sen (Upto 31st July, 2023)
16.	Mr. Arkaprava Bhattacharya
17.	Ms. Shreya Borthakur
18.	Mr. Shreyas Govind Miraj
19.	Ms. Aastha Vartak
20.	Ms. Mahima Elsa Joji
21.	Ms. Ankita Roy

Degree Award Ceremony ▶▶▶



Farewell, neuroscience ninjas! ▶▶▶



Orientation Program ►►



Annual Financial Statements





KUMAR KHARE & CO.

CHARTERED ACCOUNTANTS

S-160, L.G.F. GREATER KAILASH PART-I

NEW DELHI-110048

Phone – 41733110, 9811133110

E-mail ajok@kumarikhareca.com

INDEPENDENT AUDITOR'S REPORT

To the Members

NATIONAL BRAIN RESEARCH CENTRE

NH-8, NAINWAL MORE, MANESAR, GURUGRAM, HARYANA-122052

REPORT ON THE FINANCIAL STATEMENTS

Opinion

We have audited the accompanying financial statements of **National Brain Research Centre** ("the Society"), which comprise the Balance Sheet as of March 31, 2024, the Income & Expenditures Account and the Receipts & Payments Account for the year ended on that date, and a summary of significant accounting policies and other explanatory information (hereinafter referred to as "the Financial Statement").

Basis for Qualified Opinion

We conducted our audit of the Financial Statements in accordance with the Standards on Auditing. Our responsibilities under those Standards are further described in the Auditor's Responsibilities for the Audit of the Financial Statements section of our report. We are independent of the society in accordance with the Code of Ethics issued by the Institute of Chartered Accountants of India (ICAI) together with the independence requirements that are relevant to our audit of the financial statements under the provisions of the Act and the Rules made thereunder, and we have fulfilled our other ethical responsibilities in accordance with these requirements and the ICAI's Code of Ethics. We believe that the audit evidence we have obtained is sufficient and appropriate to provide a basis for our audit opinion on the Financial Statements *except treatment of gratuity and leave encashment which is not as per AS-15. We disagree with the management regarding the treatment of the provision in respect of leave encashment and gratuity. We feel that this is a material misstatement as the surplus for the year is overstated by Rs. 2,73,689/- and Up to the period ending 31.03.2024, the Overall Provision for Gratuity is Less created by Rs. 3,78,82,774/- and Overall Provision of Leave Encashment is Less created by Rs. 4,84,20,158.20 in the books of Accounts of the Society (Refer Point No. 9.1 & 9.2 of Notes to accounts to its financial statements)*

Qualified opinion

In our opinion, except for the possible effects of the matter described in the Basis for qualified opinion paragraph, the financial report presents fairly, in all material respects, the financial position of the society, as at 31st March, 2024 and its financial performance for the year then ended in accordance with the accounting policies. Our opinion in respect of treatment of gratuity and leave encashment as per AS-15 is qualified.

Lucknow Office: GF-2, Kumar Khare House, 193, Wazir Hasan Road, Lucknow-226001,



KUMAR KHARE & CO.

CHARTERED ACCOUNTANTS

S-160, L.G.F. GREATER KAILASH PART-I

NEW DELHI-110048

Phone - 41733110, 9811133110

E-mail ak@kumarkhareca.com

Emphasis of Matter

We draw attention to the following matters in the Notes to the Ind AS financial statements:

- Point No. 3.5 of Notes to accounts to its financial statements in respect of Maintenance of PPE Register and its physical verification.*
- Point No. 4.1 & 4.2 of Notes to accounts to its financial statements in respect of depreciation and its treatment as deferred revenue income as per AS-12*
- Point No. 11.1, 11.2 & 22.1 of Notes to accounts to its financial statements in respect of the balance of advance from the parties under current assets and current liabilities.*
- Point No. 11.3, 15.3 & 15.4 of Notes to accounts to its financial statements in respect of the TDS Refund Receivable and Outstanding Demand Payable as per Income Tax act*

Our opinion is not modified in respect of these matters.

Responsibilities of Management for the Financial Statements

The Society's Board of members are responsible for the preparation and presentation of these financial statements that give a true and fair view of the financial position, financial performance and cash flows of the society in accordance with the AS and other accounting principles generally accepted in India. This responsibility also includes the maintenance of adequate accounting records in accordance with the provisions of the bye laws for safeguarding the assets of the Society and for preventing and detecting the frauds and other irregularities; selection and application of appropriate accounting policies; making judgments and estimates that are reasonable and prudent; and design, implementation and maintenance of adequate internal financial control, that were operating effectively for ensuring the accuracy and completeness of the accounting records, relevant to the preparation and presentation of the financial statements that give a true and fair view and are free from material misstatement, whether due to fraud or error.

In preparing the financial statements, management is responsible for assessing the society's ability to continue as a going concern, disclosing, as applicable, matters related to going concern and using the going concern basis of accounting unless management either intends to liquidate the society or to cease operations, or has no realistic alternative but to do so.

Auditor's Responsibility for the Audit of the Financial Statements

- A) Our objectives are to obtain reasonable assurance about whether the financial statements as a whole are free from material misstatement, whether due to fraud or error, and to issue an auditor's report that includes our opinion. Reasonable assurance is a high level of assurance but is not a guarantee that an audit conducted in accordance with SAs will always detect a material misstatement when it exists. Misstatements can arise



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S-160, L.G.F. GREATER KAILASH PART-I

NEW DELHI-110048

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from fraud or error and are considered material if, individually or in aggregate, they could reasonably be expected to influence the economic decisions of users taken on the basis of these financial statements.

B.) As part of an audit in accordance with SAs, we exercise professional judgment and maintain professional skepticism throughout the audit. We also:

i) Identify and assess the risks of material misstatement of the financial statements, whether due to fraud or error, design and perform audit procedures responsive to those risks, and obtain audit evidence that is sufficient and appropriate to provide a basis for our opinion. The risk of not detecting a material misstatement resulting from fraud is higher than for one resulting from error, as fraud may involve collusion, forgery, intentional omissions, misrepresentations, or the override of internal control.

ii) Obtain an understanding of internal financial controls relevant to the audit in order to design audit procedures that are appropriate in the circumstances but not for the purpose of expressing an opinion on the effectiveness of the Society's internal control.

iii) Evaluate the appropriateness of accounting policies used and the reasonableness of accounting estimates and related disclosures made by management.

iv) Conclude on the appropriateness of management's use of the going concern basis of accounting and, based on the audit evidence obtained, whether a material uncertainty exists related to events or conditions that may cast significant doubt on the Company's ability to continue as a going concern. If we conclude that a material uncertainty exists, we are required to draw attention to our auditor's report to the related disclosures in the Financial Statements or, if such disclosures are inadequate, to modify our opinion. Our conclusions are based on the audit evidence obtained up to the date of our auditor's report. However, future events or conditions may cause the Society to cease to continue as a going concern.

v) Evaluate the overall presentation, structure and content of the Financial Statements, including the disclosures, and whether the Financial Statements represent the underlying transactions and events in a manner that achieves fair presentation.

C.) Materiality is the magnitude of misstatements in the Financial Statements that, individually or in aggregate, makes it probable that the economic decisions of a reasonably knowledgeable user of the Financial Statements may be influenced. We consider quantitative materiality and qualitative factors in (i) planning the scope of our audit work and in evaluating the results of our work; and (ii) to evaluate the effect of any identified misstatements in the Financial Statements.

D.) We communicate with Society's Board of members regarding, among other matters, the planned scope and timing of the audit and significant audit findings, including any significant deficiencies in internal control that we identify during our audit.

E.) We also provide Society's Board of members with a statement that we have complied with relevant ethical requirements regarding independence, and to communicate with them all relationships and other matters that may reasonably be thought to bear on our independence, and where applicable, related safeguards.

(Signature)
19.08.24

Lucknow Office: GF-2, Kumar Khare House, 193, Wazir Hasan Road, Lucknow-226001,



KUMAR KHARE & CO.
CHARTERED ACCOUNTANTS
 S-160, L.G.F. GREATER KAILASH PART-I
 NEW DELHI-110048
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 E-mail alok@kumarkhareca.com

2. We believe that our audit provides a reasonable basis for our opinion:
- We have sought and obtained all the information and explanations which to the best of our knowledge and belief were necessary for the purpose of our audit.
 - in our opinion proper books of account as required by law have been kept by the society so far as it appears from our examination of those books.
 - the Balance Sheet, the Receipts & Payment Account and the Income & Expenditures Account dealt with by this Report are in agreement with the books of account.
3. In our opinion and to the best of our information and according to the explanations given to us, the financial statements give a true and fair view in conformity with the accounting principles generally accepted in India.
- As it relates to Balance sheet, of the state of affairs of the society as at 31st March, 2024;
 - As it related to Income and Expenditures Account, of the Surplus over Income for the year ended on that date.
 - As it related to the Receipts & Payments Account, of the receipts and payment for the year ended on that date.

For Kumar Khare & Co.
 Chartered Accountants
 Firm Regn. No.: 006740C
 UDIN: 24546026BKAAUH5820



Sunil Kumar
 Partner
 Membership No. 546026

Place: New Delhi
 Date: 19th August, 2024

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM, HARYANA
BALANCE SHEET AS AT MARCH 31, 2024

		[Amount-Rs.]	
	Schedule	Current Year	Previous Year
CORPUS / CAPITAL FUND AND LIABILITIES			
Corpus/Capital Fund	1	72,08,89,785.16	1,48,10,43,000.00
Reserve and Surplus	2	24,12,69,079.34	(46,43,94,801.66)
Earmarked/Endowment Funds	3	27,44,03,832.09	27,43,44,842.99
Secured Loans and Borrowings	4	0.00	0.00
Unsecured Loans and Borrowings	5	0.00	0.00
Deferred Credit Liabilities	6	0.00	0.00
Current Liabilities and Provisions	7	6,03,09,318.48	28,61,36,115.54
Total (Liabilities)		1,29,68,72,035.07	1,57,71,29,156.87
ASSETS			
Fixed Assets	8	1,00,71,51,152.19	1,08,42,33,436.61
Investments - From Earmarked/Endowment Funds	9	0.00	0.00
Investments- Others	10	1,34,97,238.22	1,48,36,671.22
Current Assets, Loans, Advances etc.	11	27,42,23,624.66	47,80,59,049.04
Miscellaneous Expenditure		0.00	0.00
(to the extent not written off or adjusted)			
Total (Assets)	24	1,29,68,72,035.07	1,57,71,29,156.87
Significant Accounting Policies	24		
Contingent Liabilities and Notes on Accounts			

SOURAV BANERJEE
A.O.C, NBRC

Prof. Krishanu Ray
DIRECTOR
NBRC
National Brain Research Centre
NH-8, NAINWAL MORE-122051
Haryana

For Kumar Khare & Co
Chartered Accountants
FRN-006748C

TANMOY BHATTACHARYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
National Brain Research Centre
NH-8, NAINWAL MORE-122051
Haryana

Sunil Kumar
Partner
Membership No. 546026
Date: 19/08/2024
New Delhi

NATIONAL BRAIN RESEARCH CENTRE NH-B, NAINWAL MORE, MANESAR, GURGRAM, HARYANA INCOME AND EXPENDITURE ACCOUNT FOR THE YEAR ENDED MARCH 31, 2024				(Amount in Rs.)	
	Schedule	Current Year	Previous Year		
INCOME					
Income from Sales/Services	12	22,95,163.00	13,37,685.00		
Grants/ Subsidies (Revenue) from DBT	13	30,48,45,215.00	20,66,97,722.00		
Fees/Subscriptions	14	12,50,348.27	11,12,193.00		
Income from Investments (Income on Invest. From earmarked/endow. Funds transferred to funds)	15	0.00	0.00		
Income from Royalty, Publication etc.	16	0.00	0.00		
Interest Earned	17	78,90,743.00	82,92,713.50		
Other Income	18	36,04,951.30	38,50,044.00		
Increase/(decrease) in stock of finished goods and work-in-progress	19	0.00	0.00		
Deferred taxman Depreciation (corresponding to Schedule B)		11,18,89,025.45	0.00		
Total Income (A)		43,17,83,446.02	28,12,90,357.50		
EXPENDITURE					
Establishment Expenses	20	10,01,87,176.00	8,32,73,435.00		
Other Administrative etc.	21	19,68,63,412.96	21,47,20,365.18		
Expenditure on Grants, Subsidies etc.	22	0.00	0.00		
Interest Paid	23	0.00	0.00		
Depreciation (Net Total at the year-end corresponding to Schedule B)		11,18,89,025.45	9,72,47,477.27		
Total Expenditure (B)		40,89,39,614.41	39,72,41,277.45		
Balance being excess of Income over Expenditure (A-B)		2,28,43,831.61	(11,59,50,919.95)		
Transfer to Special Reserve (Specify each)		0.00	0.00		
Transfer to /from General Reserve		0.00	0.00		
Balance being Surplus/(Deficit) carried to Corpus/Capital Fund		2,28,43,831.61	(11,59,50,919.95)		
Significant Accounting Policies					
Contingent Liabilities and Notes on Accounts					

Dr. Kishan Singh
Prof. Kishan Singh
Director / Director
National Brain Research Centre
Manesar/Haryana

PROF. KISHAN SINGH
DIRECTOR
NBRC

As per our separate report of even date attached

Per Kumar Khare & Co
Chartered Accountants
FBN-000740C

Sanjit Kumar
Partner

Membership No. 546026
Date: 19/08/2024
New Delhi

TANMOY BHATTACHARYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
T. BHATTACHARYA
Joint Administrative Officer
National Brain Research Centre
Manesar/Haryana

NATIONAL BRAIN RESEARCH CENTRE, MANESAR NH-8, NAINWAL MORE, MANESAR, GURGAON, HARYANA									
RECEIPTS AND PAYMENTS FOR THE YEAR ENDED MARCH 31, 2024									
RECEIPTS			CURRENT YEAR		PREVIOUS YEAR		PAYMENTS		
			Amount in (Rs.)		Amount in (Rs.)		Amount in (Rs.)		

NATIONAL BRAIN RESEARCH CENTRE NH-8, NAINIWA MORE, MANESAR, GURGRAM (Amount-Rs.)			
SCHEDULE FORMING PART OF BALANCE SHEET AS AT MARCH 31, 2024			
SCHEDULE 1-CORPUS/CAPITAL FUND:	Current Year		Previous Year
1 Grant-in-Aid - Balance as at the beginning of the year		1,48,10,43,000.00	1,44,50,02,000.00
Add: Contribution towards Corpus/Capital Fund	9,00,00,000.00		9,40,00,000.00
Less: Contribution towards Corpus/Capital Refund	(15,54,44,140)		-5,79,59,000.00
Less: Accumulated depreciation upto 2023-24	79,47,09,074.84	-76,01,53,214.84	3,60,41,000.00
Balance as at the year end		72,08,89,785.16	1,48,10,43,000.00


SOURAV BANERJEE
A.O.C, NBRC


TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
National Brain Research Centre
NH-8, NAINIWA MORE, MANESAR, GURGRAM
GURGRAM/HARYANA


PROF. KRISHANU RAY
DIRECTOR
NBRC
National Brain Research Centre
NH-8, NAINIWA MORE, MANESAR, GURGRAM
GURGRAM/HARYANA

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FRN-006740C


Samir Kumar
Partner

Membership No. 546026
Date: 19/08/2024
New Delhi

NATIONAL BRAIN RESEARCH CENTRE NH-8, NAINIWA MORE, MANESAR, GURGRAM [Amount-Rs.]			
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2024			
SCHEDULE 2 - RESERVES AND SURPLUS			
	Current Year		Previous Year
1 Capital Reserve:			
As per last Account	0.00	0.00	0.00
Addition during the Year	0.00	0.00	0.00
Less: Deductions during the year (deficit)	0.00	0.00	0.00
2 Revaluation Reserve:			
As per last Account	0.00	0.00	0.00
Addition during the Year	0.00	0.00	0.00
Less: Deductions during the year (deficit)	0.00	0.00	0.00
3 Special Reserves:			
As per last Account	0.00	0.00	0.00
Addition during the Year	0.00	0.00	0.00
Less: Deductions during the year (deficit)	0.00	0.00	0.00
4 General Reserve			
As per last Account	(46,43,94,801.66)	(34,84,43,881.71)	
Addition during the Year	2,28,13,831.61	(11,59,50,919.95)	
Add: Surplus during the year (as per I&E A/c)	68,28,20,049.39	24,12,69,079.34	
Add: Accumulated depreciation till FY2022-23			(46,43,94,801.66)
Balance as at the year end		24,12,69,079.34	(46,43,94,801.66)


SOURAV BANERJEE
A.O.C, NBRC


TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
T. BHATTACHARYYA
300, Jyoti Bhawan
National Brain Research Centre
NH-8, Manesar-122031


PROF. KRISHANU RAY
DIRECTOR
NBRC
National Brain Research Centre
NH-8, Manesar-122031

As per our separate report of even date attached
For Kumar Khare & Co
Chartered Accountants
FNN-0097400
Sunil Kumar
Partner
Membership No. 346026
Date: 19/08/2024
New Delhi

NATIONAL BRAIN RESEARCH CENTRE NH-8, NAINWAL MORE, MANESAR, GURGHAM					Amount in (Rs.)	
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2024					TOTALS	
FUND-WISE BREAK UP						
SCHEDULE 3 - EARNINGS/ENDOWMENT FUNDS						
Endowment Fund for Building		Donation	Current Year	Previous Year		
0.00	0.00	0.00	27,43,44,056.11	40,01,94,025.90		
0.00	0.00	0.00	1,50,04,60,408.28	93,34,04,933.48		
0.00	0.00	0.00	0.00	0.00		
0.00	0.00	0.00	15,01,491.65	64,08,940.31		
0.00	0.00	0.00	1,01,50,41,027.83	93,38,13,073.79		
0.00	0.00	0.00	1,20,43,08,704.84	1,34,40,00,699.89		
0.00	0.00	0.00	21,21,333.00	4,05,90,177.00		
0.00	0.00	0.00	0.00	0.00		
0.00	0.00	0.00	85,11,364.00	2,10,45,003.00		
0.00	0.00	0.00	0.00	0.00		
0.00	0.00	0.00	95,25,6645.15	94,22,13,953.30		
0.00	0.00	0.00	3,49,52,681.56	3,75,20,431.60		
0.00	0.00	0.00	99,40,10,490.60	1,80,87,86,306.00		
0.00	0.00	0.00	99,81,40,823.69	1,64,93,84,566.98		
0.00	0.00	0.00	1,18,42,028.26	2,02,70,280.72		
0.00	0.00	0.00	1,18,42,028.26	3,08,79,209.72		
0.00	0.00	0.00	27,44,03,852.69	27,43,44,042.99		


SOURAV BANERJEE
A.D.C. NBRC


TANMAY BHATTACHARYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
T. BHATTACHARYA
CHIEF ADMINISTRATIVE OFFICER
National Brain Research Centre
NH-8-122051 / Manesar-122051
Gurgaon / Haryana


PROF. KRISHANU RAY
DIRECTOR
NBRC
Prof. Krishanu Ray
Fellow / Director
National Brain Research Centre
NH-8-122051 / Manesar-122051
Gurgaon / Haryana

As per our separate report of even date attached:
For Kumar Khare & Co
Chartered Accountants
FIRM-006740C
Sunil Kumar
Partner
Membership No. 545026
Date: 19/08/2024
New Delhi

NATIONAL BRAIN RESEARCH CENTRE NH-8, KHAUNWAL MORE, MANESAR, GURGRAM SCHEDULE FORMING PART OF BALANCE SHEET AS AT MARCH 31, 2024							Amount in (Rs.)		
FUND-WISE BREAK UP									
SCHEDULE 3 - EARNED/ENDOWMENT FUNDS		Project Fund		Fixed Assets Fund (Project)		Contributory Provident Fund		DeLosa E-library Consortium	
a) Opening Balance of Project Fund		3,34,16,767.61		23,18,76,994.07		46,40,882.00		44,02,162.93	
b) Additions to the Fund		4,04,13,698.28		21,21,212.00		0.00		96,59,25,375.00	
i. Donations/grants/aid to fund		0.00		0.00		0.00		0.00	
ii. Income from investments made on account of funds		13,41,832.65		4,12,55,570.93		21,21,333.00		2,19,650.00	
iii. Other additions (Interest Earnings)		3,51,72,298.54		23,39,98,777.07		48,88,541.00		96,59,25,375.00	
Total (a+b)								97,03,27,532.43	
c) Utilization/Expenditure towards objectives of funds									
i. Capital Expenditure		21,21,333.00		0.00		0.00		0.00	
Fixed Assets (net)		0.00		0.00		0.00		0.00	
Others									
Total		21,21,333.00		0.00		0.00		0.00	
ii. Revenue Expenditure		78,27,030.00		0.00		0.00		6,87,526.00	
-Salaries, wages and allowances etc		0.00		0.00		0.00		0.00	
-Rent		1,24,08,568.53		0.00		0.00		94,01,51,476.50	
-Others		0.00		3,49,52,081.56		0.00		0.00	
-Depreciation									
Total		2,02,24,498.53		3,49,52,081.56		0.00		94,01,51,476.50	
Total (c)		2,02,24,498.53		3,49,52,081.56		0.00		94,01,51,476.50	
d) Refund of unspent fund during the year		1,18,42,038.26		3,49,52,081.56		0.00		94,01,51,476.50	
NET BALANCE AS AT THE YEAR-END (a+b-c+d)		4,00,04,530.65		19,90,46,245.51		48,88,541.00		2,04,84,534.93	

As per our separate report of even date attached

For Katar Khare & Co
Chartered Accountants
PRN-006740C

Suall Kumar
Partner
Membership No. 540726
Date: 19/08/2024
New Delhi

Prof. Krishanu Ray
President
Prof. Krishanu Ray
President / Director
National Brain Research Centre
NH-8, Mainwal More, Manesar, Gurgram

TANVOY BRATTACHARYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
National Brain Research Centre
NH-8-122031 / Manesar-122031
Gurgaon / Haryana

SCHEDULE 4 - SECURED LOANS AND BORROWINGS: NIL

SCHEDULE 5 - UNSECURED LOANS AND BORROWINGS: NIL

SCHEDULE 6 - DEFERRED CREDIT LIABILITIES: NIL

NATIONAL BRAIN RESEARCH CENTRE NH-B, NAIKAL MORE, MANESAR, GURGAON				Amount in (Rs.)
SCHEDULE-7 CURRENT LIABILITIES AND PROVISIONS				
	Current Year	Previous Year		
A. Current Liabilities				
1. Accruals				0.00
2. Sundry Creditors				
- For Goods	22,03,750.00	0	71,25,213.00	71,25,213.00
- Others	0		0	98,92,574.22
3. Advances Received (Security Deposit refundable)				
4. Interest accrued but not due on:				
- Secured Loans/borrowings	0.00	0.00	0.00	
- Unsecured loans/borrowings	0.00	0.00	0.00	0.00
5. Statutory Liabilities:				
- Overdue	0.00	0.00	0.00	
- Others (TDS & GST payable)	7,36,760.00	41,23,102.00		41,73,182.00
6. Others current liabilities				
Total (a)	22,03,750.00	41,23,102.00		25,70,26,850.52
B. Provisions				
1. For Taxation				
2. Gratuity				
3. Superannuation/Pension				
4. Accumulated Leave Encashment				
5. Trade Warranties/Claims				
6. Others (Specifc)				
Total (b)	7,36,760.00	41,23,102.00		41,73,182.00
Balance as at the year end (a+b)	29,40,510.00	82,46,204.00		27,02,67,819.74

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FRN-006740C

Sunil Kumar
Partner

Membership No. 546026
Date: 19/08/2024
New Delhi

PROF. KRISHANU RAY
DIRECTOR
NBRC
Prof. Krishanu Ray
Director
National Brain Research Centre
NH-B, Naikol More, Manesar-122013
Gurgaon/Haryana

SUBRAJ BANERJEE
A.O.C. NBRC.

TANUJ BHATTACHARYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
T. BHATTACHARYA
Chief Administrative Officer
National Brain Research Centre
NH-B, Naikol More, Manesar-122013
Gurgaon/Haryana

[illegible]

Investment in - Investment from government funds:

No peer-reviewed publications were identified.

Baron
SOLANGE BARON-VE
D.C. MBE


 PROF. KRISHANU RAY
 DIRECTOR
 MRCB
 डॉ. कृष्ण राय
 Prof. Krishanu Ray
 Director / Director
 मर्सेन्स नेशनल ब्रेन रिसर्च सेंटर
 MRCB
 National Brain Research Center
 100/100/100/100/100
 100/100/100/100/100

For Remy-Kline & Co.
Insurance Associates
1930-308 718 C

East Point, GA
Post Office

Mr. Remy-Kline & Co.
Date: 11/22/2024

[illegible]

NATIONAL BRAIN RESEARCH CENTRE NH-8, NAINWAL MORE, MANESAR, GURGRAM SCHEDULE FORMING PART OF BALANCE SHEET AS AT MARCH 31, 2024 Amount in (Rs.)			
SCHEDULE 10 - INVESTMENTS-OTHERS			
	Current Year	Previous Year	
1. In Government Securities	0.00	0.00	0.00
2. Other approved Securities	0.00	0.00	0.00
3. Shares	0.00	0.00	0.00
4. Debentures and Bonds	0.00	0.00	0.00
5. Subsidiaries and Joint Ventures	0.00	0.00	0.00
6. Others (CPF Fund)	1,54,97,258.22	1,48,36,671.22	
Total	1,54,97,258.22	1,48,36,671.22	


SOURAV BANERJEE
A.O.C, NBRC


Prof. Krishanu Ray
Director / Director
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
Manesar/Manesar-122051
Haryana

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FRN-006740C


Sunil Kumar
Partner

Membership No. 546026
Date: 19/08/2024
New Delhi


TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
Manesar-122051 / Manesar-122051

NATIONAL BRAIN RESEARCH CENTRE NH-0, NAIENWAL MORE, MANESAR, GURGRAM			
SCHEDULE FORMING PART OF BALANCE SHEET AS AT MARCH 31, 2024			
	Current Year	Previous Year	Amount in (Rs.)
SCHEDULE 11 - CURRENT ASSETS, LOANS, ADVANCES ETC.			
A. Current Assets			
1. Inventories:			
a) Stores and Spares	0.00	0.00	
b) Loose Tools	0.00	0.00	
c) Stock-In-Trade	0.00	0.00	
Finished Goods	0.00	0.00	
Work-in-progress	0.00	0.00	
Raw Materials	0.00	0.00	0.00
2. Sundry Debtors:			
a) Debts Outstanding for a period exceeding six months	0.00	0.00	
b) Others	0.00	0.00	0.00
3. Cash balances in hand (including cheques/drafts and interest)	0.00	0.00	0.00
4. Bank Balances:			
a) With Scheduled Banks:			
- On Current Accounts	10,94,94,971.00	10,94,06,408.00	
- On Deposit Accounts (includes margin money)	14,49,62,897.31	34,27,61,948.29	
- On Savings Accounts			
b) With non-Scheduled Banks:			
- On Current Accounts	0.00	0.00	
- On Deposit Accounts	0.00	0.00	
- On Savings Accounts	0.00	0.00	
5. Post Office Savings Accounts			
Total (A)	24,74,57,868.31		45,21,68,356.29

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FRN-0067499

Sunil Kumar
Partner
Membership No. 546026
Date: 19/08/2024
New Delhi

PROF. KRISHNA KAY
DIRECTOR
NBRC
Prof. Krishnakant Ray
Finance / Director
NBRC
National Brain Research Centre
NH-0, NAIENWAL MORE, MANESAR, GURGRAM

SOURAV BANERJEE
A.O.C., NBRC

TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
T. BHATTACHARYYA
Chief Administrative Officer
National Brain Research Centre

NATIONAL BRAIN RESEARCH CENTRE NH-B, NAINWAL MORE, MANESAR, GURGRAM SCHEDULE FORMING PART OF BALANCE SHEET AS AT MARCH 31, 2024				Amount in (Rs.)
SCHEDULE 11 - CURRENT ASSETS, LOANS, ADVANCES ETC. (Contd.)		Current Year	Previous Year	
B. LOANS, ADVANCES AND OTHER ASSETS				
1 Loans:				
a) Staff		52,07,513.87	55,98,105.87	
b) Other Entities engaged in activities/objectives similar to that of the entity		0.00	0.00	
c) Other (Imprest)		0.279.00	24,533.00	56,22,638.87
2 Advances and other amounts recoverable in cash or in kind or for value to be received				
a) On Capital Account		0.00	0.00	
b) Prepayments (Insurance & Expenses)		9,11,985.00	11,19,291.00	
c) Other - Advances to Parties		26,05,140.75	11,77,595.75	
- Other Advances (Security Deposit Paid)		57,62,502.45	57,62,502.45	00,59,349.20
3 Income Accrued:				
a) On Investments from Earmarked/Endowment Funds		0.00	0.00	
b) On Investments-Others (Accrued Int. on CPF)		24,912.00	1,01,368.00	
c) On Loans and Advances		0.00	0.00	
d) Others (SB A/C)		0.00	0.00	1,01,368.00
b) (Includes income due unrealised-Rs.)				
4 Claims Receivable (TDS Receivable) & Income Tax				
Total (B)		1,22,45,422.28		1,21,07,296.68
Total (A+B)		2,67,65,756.35		2,58,90,692.75
		27,42,23,624.66		47,80,59,049.04


SOURAV BANERJEE
A.O.C, NBRC


PROF. KRISHANU RAY
DIRECTOR
NBRC
श्री. कृष्ण राय
Prof. Krishanu Ray
सचिव / Director
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
मानसरोवर-गुरुग्राम

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FIRN-006740C


Sumit Kumar
Partner

Membership No. 546026
Date: 19/08/2024
New Delhi


TANMOY BHATTACHARYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
T. BHATTACHARYA
मुख्य प्रशासनिक अधिकारी
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
मानसरोवर-गुरुग्राम

NATIONAL BRAIN RESEARCH CENTRE NH-B, NAINWAL MORE, MANESAR, GURGRAM SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2024			Amount in (Rs.)
SCHEDULE 12 - INCOME FROM SALES/SERVICES		Current Year	Previous Year
1 Income from Sales			
- Sale of Scrap		5,500.00	-
- Sale of Rat/Mice		79,450.00	-
2) Income from Services			
- Guest House Charges & Rental Income		5,09,833.00	3,34,515.00
- Overhead Charges		11,30,303.00	10,03,170.00
- Others		5,62,077.00	-
Total		22,95,163.00	13,37,685.00


SOURAV BANERJEE
A.O.C, NBRC


PROF. KRISHANU RAY
DIRECTOR
NBRC

Prof. Krishanu Ray
Director
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
गुरुग्राम/गुरुग्राम-140101

As per our separate report of even date attached
For Kumar Khare & Co
Chartered Accountants
FRN-006740C


Sunil Kumar
Partner
Membership No. 546026
Date: 19/08/2024
New Delhi


TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC

T. BHATTACHARYYA
Chief Administrative Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
गुरुग्राम-122055 / Manesar-122053
गुरुग्राम / Haryana

NATIONAL BRAIN RESEARCH CENTRE NH-8, NAINWAL MORE, MANESAR, GURGRAM SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2024 Amount in (Rs.)			
SCHEDULE 13 - GRANTS/SUBSIDIES (Irrevocable Grants & Subsidies Received)		Current Year	Previous Year
1 Central Government (DBT)		30,48,45,215.00	26,66,97,722.00
2 State Government(s)		0.00	0.00
3 Government Agencies		0.00	0.00
4 Institutions/Welfare Bodies		0.00	0.00
5 International Organisations		0.00	0.00
6 Others (Specify)		0.00	0.00
Total		30,48,45,215.00	26,66,97,722.00


SOURAV BANERJEE
A.O.C, NBRC


PROF. KRISHANU RAY
DIRECTOR
NBRC

Prof. Krishanu Ray
Director / Director
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
मानेसर/मानेसर-122051
हरियाणा

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FRN-006740C


Sunil Kumar
Partner
Membership No. 546026
Date: 19/08/2024
New Delhi


TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
TANMOY BHATTACHARYYA
Chief Administrative Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
मानेसर-122051 / मानेसर-122051
हरियाणा

NATIONAL BRAIN RESEARCH CENTRE NH-8, NAINWAL MORE, MANESAR, GURGRAM		
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED March 31, 2024		
Amount in (Rs.)		
SCHEDULE 14 - FEES/ SUBSCRIPTIONS	Current Year	Previous Year
1 Entrance Fees	6,01,848.27	3,48,693.00
2 Annual Fees/Subscriptions	6,56,500.00	7,63,500.00
3 Seminar/Program Fees	0.00	0.00
4 Consultancy Fees	0.00	0.00
5 Others (Fellowship Grants)	0.00	0.00
Total	12,58,348.27	11,12,193.00


SOURAV BANERJEE
A.O.C, NBRC


PROF. KRISHANU RAY
DIRECTOR
NBRC

श्री. प्रशान्त राय
Prof. Krishanu Ray
प्रिंसिपल / डायरेक्टर
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
एनबीआरसी
National Brain Research Centre
नैनवाल/मानेसर-122052
हरियाणा

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FRN-006740C


Sunil Kumar
Partner

Membership No. 546026
Date: 19/08/2024
New Delhi


TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC, BHATTACHARYYA
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
Chief Administrative Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
नैनवाल-122051 /मानेसर-122052
हरियाणा

NATIONAL BRAIN RESEARCH CENTRE NH-8, MAINWAL MORE, MANESAR, GURGRAM					
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2024					
SCHEDULE 15 - INCOME FROM INVESTMENTS (Income on Invest. From Earmarked/Endowment Funds transfer to Funds)	Investment from Earmarked Fund			Investment-Others	
	Current Year	Previous Year	Current Year	Previous Year	Amount in (Rs.)
1. Interest					
a) On Govt. Securities	0.00	0.00	0.00	0.00	0.00
b) Other Bonds/Debentures	0.00	0.00	0.00	0.00	0.00
2. Dividends:					
a) On Shares	0.00	0.00	0.00	0.00	0.00
b) On Mutual Fund Securities	0.00	0.00	0.00	0.00	0.00
3. Rents	0.00	0.00	0.00	0.00	0.00
4. Others (Project Receipts)	0.00	0.00	0.00	0.00	0.00
TOTAL	0.00	0.00	0.00	0.00	0.00

SCHEDULE 16 - INCOME FROM ROYALTY, PUBLICATION ETC.

NIL


SOURAV BANERJEE
A.O.C, NBRC


TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
T. BHATTACHARYYA
and Associates, Chartered
Chief Administrative Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
मनसार-122051 / Manesar-122051
हरियाणा / Haryana


PROF. KRISHANU RAY
DIRECTOR
NBRC
प्रो. कृष्णानु राय
निदेशक / Director
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
मनसार/Manesar-122051
हरियाणा / Haryana

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FBN-006740C


Sunil Kumar
Partner
Membership No. 546026
Date: 19/08/2024
New Delhi

NATIONAL BRAIN RESEARCH CENTRE NH-8, MAINWAL MORE, MANESAR, GURGRAM		
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED MARCH 31, 2024		
Amount in (Rs.)		
SCHEDULE 17 - INTEREST EARNED	Current Year	Previous Year
1. On Term Deposits:		
a) With Scheduled Banks	56,64,165.00	64,67,524.00
b) With Non-Scheduled Banks	0.00	0.00
c) With Institutions	0.00	0.00
d) Others (Int. received on Security Deposit received)	0.00	2,59,241.50
2. On Savings Accounts:	20,86,970.00	14,11,052.00
a) With Scheduled Banks	0.00	0.00
b) With Non-Scheduled Banks	0.00	0.00
c) Post Office Savings Accounts	0.00	0.00
d) others	0.00	0.00
3. On Loans:		
a) Employees/Staff	0.00	0.00
b) Others (Int. on HBA)	1,39,608.00	1,54,896.00
4. Interest on Debtors and Others Receivables	0.00	0.00
Total	78,90,743.00	82,92,713.50

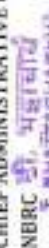

SOURAV BANERJEE
A.O.C, NBRC


PROF. KRISHANU RAY
DIRECTOR
NBRC

As per our separate report of the year ended March 31, 2024
For Kumar Khare & Co. Chartered Accountants
FRN-006740C
National Brain Research Centre
Manesar-122051
Haryana


Sunil Kumar
Partner
Membership No. 546026
Date: 19/08/2024
New Delhi

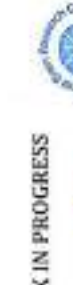

TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC


T. BHATTACHARYYA
For Administrative Officer
National Brain Research Centre
Manesar-122051
Haryana

NATIONAL BRAIN RESEARCH CENTRE NH-8, NAINWAL MORE, MANESAR, GURGRAM		
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED March 31, 2024		
Amount in (Rs.)		
SCHEDULE 18 - OTHER INCOME	Current Year	Previous Year
1 Profit on Sale/disposal of Assets: a) Owned assets b) Assets acquired out of grants, or received free of cost	0.00 0.00	0.00 0.00
2 Export Incentives realized		
3 Fees of Miscellaneous Services	36,04,951.30	38,50,044.00
4 Miscellaneous Income	0.00	0.00
5 Prior Period Income		
Total	36,04,951.30	38,50,044.00

SCHEDULE 19 - INCREASE/(DECREASE) IN STOCK OF FINISHED GOODS & WORK IN PROGRESS


SOURAV BANERJEE
A.O.C, NBRC


Prof. Krishanu Ray
Director / Director
National Brain Research Centre
Manesar/Manesar-121054
gk@nbrc.org
gk@nbrc.org

As per our separate report of even date attached

For Kumar Khare & Co
Chartered Accountants
FRN-006740C


Sunil Kumar
Partner

Membership No. 546026
Date: 19/08/2024
New Delhi


TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
National Brain Research Centre
Manesar-121054 / Manesar, Gurgram



NATIONAL BRAIN RESEARCH CENTRE NH-8, NAINIWAL MORE, MANESAR, GURGRAM		
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED March 31, 2024		
SCHEDULE 20 - ESTABLISHMENT EXPENSES	Current Year	Previous Year
a) Salaries and Wages	7,96,29,356.00	7,16,05,620.00
b) Allowances and Bonus	0.00	0.00
c) Contribution to Provident Fund	0.00	0.00
d) Contribution to Pension Scheme	0.00	0.00
e) Staff Welfare Expenses	3,920.00	35,683.00
f) Expenses on Employees Retirement and Terminal Benefits	0.00	0.00
g) Others - Children education reimbursement	11,07,000.00	12,15,000.00
- Leave encashment	30,36,412.00	5,40,668.00
- Gratuity	25,25,000.00	0.00
- LTC expenses	8,65,581.00	2,08,687.00
- Medical reimbursement	8,54,727.00	14,72,088.00
- NPS (employer subscription)	94,32,211.00	89,17,071.00
- overtime allowance	14,759.00	7,542.00
- Medical Insurance (Staff)	15,59,773.00	11,91,076.00
- Leave Salary	3,48,761.00	0.00
- Pension Contribution	7,94,086.00	0.00
- Staff Honorarium	15,590.00	0.00
Total	10,01,07,176.00	8,52,73,435.00

SOURAV BANERJEE
A.O.C, NBRC

PROF. KRISHANU RAY
DIRECTOR
NBRC

मो. कृषाजु राय
Prof. Krishanu Ray
निदेश / Director
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
As per our separate report of even date attached
प्रमाणित किया जाता है

For Kumar Khare & Co
Chartered Accountants
FRN-096740C

Sunil Kumar
Partner

Membership No. 546026
Date: 19/08/2024
New Delhi

TANMOY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
NBRC

Tanmay Bhattacharyya
Chief Administrative Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
NH-8, Gurgram / Manesar-122051

NATIONAL BRAIN RESEARCH CENTRE NB-R, NABHAWA, MORE, MAHESHWAR, GUJARAT		
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED MARCH 31, 2024		
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES		
	Current Year	Previous Year
1. Electricity and Fuel	3,33,90,501.86	5,65,90,530.35
2. Insurance	51,344.80	77,312.88
3. Repairs and maintenance	3,72,50,325.84	4,40,10,926.72
4. Linoleum	0.00	0.00
5. Rent (office Rent), Rates and Taxes	14,05,277.86	14,05,277.86
6. Vehicles Running and Maintenance	3,53,607.88	3,53,607.88
7. Postage, Telephone and Communication Charges	7,58,164.48	4,20,924.88
8. Printing and Stationery	10,50,562.88	5,61,442.88
9. Traveling and Conveyance Expenses	35,57,205.88	31,28,446.88
10. Expenses on Scholarships/Grants	18,10,735.88	8,22,876.88
11. Subscription Expenses	31,04,700.24	48,22,842.25
12. Insurance on Stock	1,83,164.88	2,24,375.88
13. Auditor Remuneration	2,58,250.88	1,50,300.88
14. Hospitality Expenses	2,17,828.88	2,02,849.88
15. Professional Charges	35,72,368.88	13,40,859.88
16. Immovable Assets Written off	3,88,545.24	2,01,871.92
17. Medical Expenses (Students)	3,10,274.88	4,60,153.88
18. Freight and Forwarding Expenses	0.00	0.00
19. Gas/other charges-Gas	4,15,248.88	3,55,248.88
20. Advertisement and Publicity	15,934.88	4,95,916.88
21. Prior Period Expenses	37,818.88	18,60,809.84
22. Others - Bank charges	38,608.09	53,855.12
- Misc. expenses	4,37,121.88	3,26,250.88
- Books and Periodicals	1,35,648.88	1,10,115.88
- Miscellaneous (Others)	8,57,594.88	4,92,880.88
- Petrol, Diesel & CNG etc.	10,12,493.88	8,73,593.88
- Skilled manpower	2,10,58,227.88	2,72,08,832.88
- Marriages (Householding & Society)	1,92,09,695.88	4,00,03,957.88
- Medicals	36,55,451.88	36,05,107.88
- Training and networking expense	3,71,34,693.88	3,88,58,120.88
- Laboratory & Animal Care charges	2,40,02,203.29	2,55,79,105.88
- Interest on Loan	1,14,159.88	1,64,301.88
- Office expenses	2,13,477.88	65,109.88
23. Appreciation Tax	0.00	0.00
Total	39,68,63,413.96	21,47,20,365.18

SCHEDULE 22 - EXPENDITURE ON GRANTS, SUBSIDIES ETC.

SCHEDULE 23 - INTEREST PAID

Nil
Nil
As per our separate report of even date attached
For Karam Bhare & Co
Chartered Accountants
FIR 006740C


SOURAV BANERJEE
A.O.C. NBRC


Prof. Krishanu Ray
Director / Director
National Brain Research Centre
NBRC
New Delhi
110016


TANMAY BHATTACHARYA
CHIEF ADMINISTRATIVE OFFICER
NBRC
NBRC, NABHAWA, MORE, GUJARAT

NATIONAL BRAIN RESEARCH CENTRE, MANESAR, GURUGRAM

- SIGNIFICANT ACCOUNTING POLICIES & NOTES ON ACCOUNTS FORMING PART OF THE BALANCE SHEET AS AT 31ST MARCH, 2024 AND INCOME & EXPENDITURE ACCOUNT FOR THE YEAR ENDED 31ST MARCH, 2024.

● SIGNIFICANT ACCOUNTING POLICIES & NOTES ON ACCOUNTS

1. Accounting Convention:

- 1.1 The financial statements of National Brain Research Centre (NBRC) are prepared on the basis of historical cost convention and on the accrual basis of accounting.
- 1.2 The NBRC is prepared based on the 'Uniform Format of Accounting' prescribed for the Central Autonomous Bodies by the Ministry of Finance, Govt. of India for preparing the Income & Expenditure Account, Receipts & Payments Account, Balance Sheet & other Schedules thereto.

2. Inventory Valuation:

- 2.1 All purchases of chemicals, glassware, consumables and printing & stationery have been booked/charged to consumption/expenditure at the time of purchases. Inventories had been so booked, based on their purchase cost & other costs incurred in bringing the inventories to their present location & condition.
- 2.2 Further, all entries relating to purchase of consumables/equipment or other fixed assets in accounts are being passed only at the time of submission of satisfactory inspection/installation report irrespective of the date of actual receipt of the supplies/equipment.






3. Property Plant & Equipment & Intangible Assets:

3.1 Property Plant & Equipment & Intangible Assets are stated at written down value. i.e., at their cost of acquisition inclusive of inward freight, duties and taxes & incidental & direct expenses related to the acquisition.

3.2 In respect of projects involving construction, related pre-operational expenses (including interest on loans for specific project prior to its completion), form part of the value of the assets capitalized.

3.3 Property Plant & Equipment & Intangible Assets received by way of non-monetary grants, (other than towards the corpus Fund), are capitalized at values stated, by corresponding credit to Capital Reserve

3.4 Property Plant & Equipment & Intangible Assets have been created mainly out of grants received from the Department of Biotechnology, Ministry of Science and Technology, Government of India.

3.5 The NBRC has maintained the Property Plant & Equipment Register on different Departmental / section basis, however the same has not been maintained on Centralized basis. The Physical Verification of the Property Plant & Equipment has not been done by the NBRC and no reconciliation of the Property Plant & Equipment and its Physical Verification has not been prepared by the Institute.

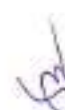
4. Depreciation:

4.1 Depreciation provided for current year on the fixed assets of Project for Rs. 3,49,52,081.56 (previous year Rs. 3,75,29,431.60) and which has been directly debited to the fixed assets funds account. These assets were created through the non-Recurring and project-based grant from the funding agencies.

Depreciation of Core Fixed Assets amounting to Rs. 111,889,025.45 for current financial year (Rs.

97,247,477.27 for previous year) had been debited to Income & Expenditure Account.

NBRC has charged the Depreciation as per Income Tax Act 1961 on W.D.V basis.

Note: - Assets which have been put to use for more than 182 days has been charged in full and for the remaining Assets charged at the rate 50% of the depreciation rates as above

4.2 Upto the FY 2022-23, the Current Year depreciation of the Core was charged as Expense to the Income and Expenditure account and the NBRC was not recording the depreciation of the current year as Deferred Revenue Income in their Income & Expenditure Account.

However, to nullifying the effect of the Depreciation of the Fixed Assets of the Core: Now from the Current financial year for alignment with the recommended practice of recognising Deferred Revenue Income below mentioned adjustment have been done in the current FY 2023-24.

Accumulated depreciation till FY 2022-23 amounting to Rs. 68,28,20,049.39 has been added back to the General Reserve to bring the General Reserve to the level where effect of Depreciation charged till FY 2022-23 is adjusted & the Accumulated Depreciation till 31.03.2024 amounting to Rs. 79,47,09,0974.84 has been deducted from Capital Fund and the Current Year Depreciation of Core Assets amounting to Rs. 111,889,025.45 has been recorded as Deferred Revenue Income.

4.3 Depreciation has been charged during the year of acquisition and no depreciation is provided during the year of asset sold/discarded. In respect of additions to/deductions from fixed assets during the year, depreciation is considered on pro-rata basis.

4.4 Fixed Assets are stated at the cost of acquisition inclusive of inward freight, duties and taxes and incidental and direct expenses related to acquisition.

5. Investments:

5.1 Investments classified as "long term investments" are carried at cost, provision for decline, other than temporary, is made in carrying cost of such investments.






5.2 Investments classified as "Current" are carried at lower of cost and fair value. Provision for shortfall on the value of such investments is made for each investment considered individually and not on a global basis.

5.3 Cost included acquisition expenses like brokerage, transfer stamps.

5.4 Investments in term deposits with banks are valued on cost.

5.5 Interest received on term deposits are accounted for on accrual basis.

6. Government Grants / Subsidies:

6.1 Government grants of the nature of contribution towards capital cost of setting up projects are treated as Capital Reserve/Fund.

6.2 Government grants are accounted for in accordance with the sanctioned terms & on realization basis.

6.3 Interest on Government Grant has been considered under the respective projects in view of the project sanctioned terms, as in the past.

6.4 Grants in respect of specific fixed assets acquired are shown as the deduction from the cost of the related assets

6.5 Recurring grant have been recognized in the Income and Expenditure Account in the year of receipt of grant in aid whereas, non- recurring grants have been treated capital reserve/corpus fund.



7. Foreign Currency Transactions/ Grants:

7.1 Transactions denominated in foreign currency are accounted at the exchange rate prevailing at the date of the transaction.

7.2 Current assets, foreign currency current liabilities are converted at the exchange rate prevailing as at the year end.

8. Lease:

The NBRC is located on the leasehold land at Manesar taken from Indian Vaccine Corporation Ltd. for Rs. 14, 05,777/- per annum lease amount. The annual lease rental being charged against revenue for respective year.

9. Retirement Benefits:

9.1 The NBRC is not registered with the Provident Fund authorities and it maintains a separate Contributory Provident Fund (CPF), which is yet to be recognized and the CPF fund required the separate accounting. At present all the employees have been joined under New Pension Scheme (NPS). The pension of the Government Servant appointed on or after 01.01.2004 is regulated by the new defined contribution pension system (known as National pension system), notified by the Ministry of Finance, Government of India.

9.2. The NBRC has made provision for gratuity and leave encashment of Rs.25,25,000/-in each head on adhoc basis during the financial year 2023-24 as against the requirement of AS-15 issued by ICAI. However, the amount of gratuity and leave encashment to the extent of Rs. 87,62,110.00 and Rs. 43,91,048.00 respectively as on 31st March, 2024, (Rs. 61,62,367.00 and Rs. 17,05,929.00 respectively as on 31st March, 2023) against provision made earlier.



Whereas, as per the Actuarial Valuation Report, the Provision for Gratuity head has to be created upto 31.03.2024 is of Rs.4,66,44,884/- and Provision for Leave Encashment head has to be created upto 31.03.2024 is of Rs.5,28,11,207/-

Hence as per Actuarial Valuation upto 31.03.2024, the Provision for Gratuity is Less created by Rs. 3,78,82,774/- (Including Current year Provision of Rs. 23,66,980/-) and Provision of Leave Encashment is Less created by Rs.4,84,20,158.20 (Including Current year Provision of Rs. 29,56,709/-) in the books of Accounts.

Hence during the financial year 2023-24 as per AS-15, the Provision for Gratuity is excess provided by Rs. 1,58,020& Provision for Earned Leave Encashment is less provided by Rs. 4,31,709/- as per Actuarial Valuation Report. Hence during the year Net Less Provision for Gratuity & Earned Leave Encashment has been made by Rs. 2,73,689/-

10. Taxation:

In view of the tax exemption status the National Brain Research Centre is a Society, registered under Societies Registration Act-1860 at Autonomous bodies. Also, The NBRC has been granted permanent registration under section 12AB vide URN-AAFFN2348KE20217 dated 23.09.2021 and Provisional registration under section 80G (5) vide URN-AAFFN2348KF20231 dated 24.03.2023.

11. Current Assets, Loans & Advances:

11.1 In the opinion of the Management, the current assets, loans and advances have a value on realization in the ordinary course of business, equal at least to the aggregate amount shown in the Balance Sheet.

11.2 The current assets and current liabilities of NBRC includes various old outstanding balances.

11.3 TDS deducted and to be received as refund amounting to Rs. 62,50,153.28 out of which Rs.1,38,125.60 is the current year TDS & TCS. Whereas, Rs.61,12,028/- is still pending since 2007-08 onwards, which needs to be followed up with the Income Tax Department for getting refund and/or needs to be adjusted

6

12. Bank Balance:

All Banks accounts have been reconciled till 31st March, 2024.

13. Fraud/Manipulation of funds encountered by NBRC:

No Fraud was detected during the year.

14. Outstanding Balances of Closed Projects:

The Institute has a policy of incurring expenditure on various projects in accordance with the sanctioned budget under various heads of accounts irrespective of the actual releases during a financial year. Since the actual release of money by the sponsoring agency is subject to various factors, the expenditure on approved heads of accounts is being incurred within the overall sanction of the project.

15. Contingent Liabilities

15.1 Claims against the Entity not acknowledged as debt. Rs. NIL (Previous year Rs. NIL).

15.2 In respect of:

- Bank guarantees given by/on behalf of the entity Rs. NIL (Previous year Rs. NIL).
- Letters of Credit opened by Bank on behalf of the Entity Rs. NIL (Previous year Rs. NIL).
- Bills discounted with banks Rs. NIL (Previous year Rs. NIL).

15.3 Disputed demands in respects of Income tax (TDS) Rs. 22,96,905.00 (Previous year Rs. 24,39,835.00) which is under representation before the concerned authorities.

15.4 There is an outstanding Income Tax Demand for A.Y. 2017-18 amounting to Rs. 2,99,76,345.00 against which appeals to (CIT Appeal- Faceless / Chandigarh has already been filed in prior years. However, Rs.59,95,269/- had been deposited i.e.20% of demand value against IT Notice no. ITBA/COM/F/17/2019-20/1025039709(1) Dtd.15.02.2020 to stay the demand.

88

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16. Capital Commitments

Estimated value of contract remaining to be executed on capital account and not provided for (net of advances) Rs. -Nil (Previous year Rs. NIL).

17. Lease Obligations

Future obligations for rentals under finance lease arrangements for plant and machinery amount to Rs. NIL (Previous year Rs. NIL).

18. Foreign Currency Transactions

18.1 Value of Imports Calculated on C.I.F Basis:

- Purchase of finished Goods Rs. NIL.
- Raw Materials & Components (Including in transit) Rs. NIL.
- Capital Goods Rs. NIL.
- Purchased Consumables / Non-Consumables for Rs. 2,40,90,580.29 (Previous year Rs.2,55,78,305.93)
- Transactions determined in foreign currency are accounted at the exchange rate prevailing on the date of transaction.

18.2 Expenditure in foreign currency:

- a) Travelling charges Rs. 11,88,164.00 (Previous year Rs.13,70,248/-)
- b) Remittances and Interest payment to Financial Institutions/ Banks in Foreign Currency Rs. NIL.
- c) Other expenditure:-
 - Commission on Sales Rs. NIL.
 - Legal and Professional Expenses Rs. NIL.
 - Miscellaneous Expenses Rs. NIL.

18.3 Receipt:

Value of Grant for Project from foreign entity-Rs. 23,35,878 (Previous year Rs.23,17,772/-).

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19. Remuneration to Statutory auditor:

- As Auditors Rs. 1,35,000/- (Previous year Rs. 1,35,000.00).
Audit fee 70000, other Taxation Matter Fee 65,000/-)

20. Receipt & Payment Accounts:

The Receipt & Payment Account has been prepared using direct method presenting all receipts & payments during the year under major heads, in the interest of better disclosure.

21. GST Reconciliation:

There are Input Credit balances in GST ledger in GST portal which required reconciliation and rectification in the Annual Return.

22. Others:

22.1 The Balance in the name of various parties under the head Current Liabilities reflecting the total amount payable to Sundry Creditors is Rs. 22,03,750.00 (previous year Rs. 71,75,213.00).

22.2 Schedules 1 to 23 along with Annexures 1 to 74 are annexed to and form an integral part of the Balance Sheet as at 31st March, 2024 and the Income and Expenditure Account for the year ended on that date.

22.3 Accounting policies not referred to otherwise be consistent with Generally Accepted Accounting Principles (GAAP).



Dr. R. K. R.

22.4 Corresponding to the previous year figures have been regrouped/rearranged wherever considered necessary in conformity with the current year's presentation of the accounts of the institute.

For and on behalf of
National Brain Research Centre


SOURAV BANERJEE
(A.O.C., NBRC)


Tanmoy Bhattacharyya
(Chief Administrative Officer)


T. BHATTACHARYYA
Administrative Officer
Place: New Delhi
Date: 10/08/2024
National Brain Research Centre
New Delhi-110051
Phone: 22051 / Manesar-122051
Gurgaon / Haryana

As per our separate report
of even date attached.
For Kumar Khare & Co,

(Chartered Accountants)
(FRN-006740C)


(CA Sunil Kumar)
Partner
M.No.546026


Krishanu Ray
(Director)
Prof. Krishanu Ray
Director
National Brain Research Centre
Manesar-122051
Gurgaon/Haryana

NATIONAL BRAIN RESEARCH CENTRE, NII-B, MAINWAL VIHIL, MANESAR GURGAON ANNEXURE OF PROJECT GRANTS AND EXPENDITURE FOR THE YEAR 2023-24										
S No./ Annex No.	NAME OF PROJECT	Opening Balance as on 01.04.2023	Grants received during the year 2023-24	Interest/other income earned during the year 2023-24	Capital Exp. during the year 2023-24	Revenue Expenditure during the year 2023-24			Refund of Unspent Balance/Interest	Closing Balance as on 31.03.2024
						Manpower	Others	Total Expenditure		
2	CSIR (apex) Encapsulations - Dr. Anil Kumar Bhatia	10.04	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3	Studying the role of long non-coding RNAs (lncRNAs) (Dr. Arif Khan)	130.226	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	MicroRNA mediated Reg. of Neural stem (Dr. Arif Khan Bhatia)	24,654.62	0.00	0.00	0.00	0.00	14,034.62	14,034.62	0.00	0.00
5	Total innovation fellowship award - Dr. Arif Khan Bhatia	13,275.53	0.00	5,775.53	0.00	0.00	0.00	0.00	0.00	0.00
6	IC BOSE FELLOWSHIP (Dr. Arif Khan Bhatia)	46,712.00	38,00,00,00.00	18,207.00	0.00	0.00	13,67,384.00	13,67,384.00	0.00	1,85,538.00
7	Early Disruption of structural and functional - Dr. Arif Khan	2,67,421.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	2,67,421.00
8	Dementia Tissue MRI studies (Dr. Arif Khan Bhatia)	17,76,098.00	0.00	17,850.00	0.00	0.00	67,090.00	67,090.00	5,17,090.00	8,59,008.00
9	Neurological network dynamics DDT (Dr. Arif Khan Bhatia)	81,793.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	81,793.00
10	Neural stem cell biology (Dr. Arif Khan Bhatia)	18,114.37	0.00	0.00	0.00	0.00	0.00	0.00	0.00	18,114.37
11	National Bioscience Award - Dr. Arif Khan Bhatia	26,402.29	0.00	0.00	0.00	0.00	0.00	0.00	0.00	26,402.29
12	NON-CARCINOGENIC BUNDOLOXIMES - Dr. ELIJAH SEN	33,716.00	12,70,000.00	0.00	0.00	0.00	13,62,130.00	12,62,130.00	0.00	41,506.00
13	Neurobiology of Dyslexia Brain & Behavior - Dr. Arif Khan Bhatia	6,14,948.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6,14,948.00
14	Post neural stem cell to oligodendrocytes (Dr. Parul Sethi)	5,14,522.00	0.00	0.00	0.00	0.00	3,49,580.00	3,49,580.00	0.00	1,64,942.00
15	Cytosine Linked RNA (Dr. Parul Sethi)	7,97,811.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	7,97,811.50
16	Effect of ligands on different Neural-Dr. Parul Sethi	10,03,555.13	7,14,038.00	0.00	0.00	3,46,000.00	4,64,525.00	13,10,525.00	0.00	4,04,860.13
17	Hypoxia and Change in Blood Brain-Dr. Parul Sethi	(0.00)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	A CROSS-CULTURE PERSPECTIVE ON NEURODEGENERATION - Dr. PRAVAT MANDAL	3,84,531.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3,84,531.00
19	Characterizing biomarkers of Alzheimer's disease - Dr. Pravat Mandal	(10,01,120)	10,01,120	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	Dementia Imaging studies (Dr. Pravat Kumar Mandal)	1,71,984.00	0.00	0.00	0.00	0.00	1,29,044.00	1,29,044.00	4,350.00	38,590.00
21	Neural Imaging Disruption Info-Aus grant (Dr. Pravat Kumar Mandal)	4,50,462.00	0.00	0.00	0.00	0.00	0.00	0.00	4,50,462.00	0.00
22	SPECIFIC BRAIN TEMPLATE DST - PRAVAT K MANDAL	18,67,110.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	18,67,110.50
23	Total innovation fellowship Award - Dr. Pravat Mandal	15,21,200	0.00	3,212.00	0.00	0.00	0.00	0.00	0.00	0.00
24	Crypt System - Dr. Sourav Banerjee	17,47,793.32	0.00	0.00	0.00	0.00	0.00	0.00	0.00	17,47,793.32
25	DEFT Mima Medicate Control - Dr. Sourav Banerjee	1,80,009.97	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1,80,009.97
26	Mechanism of Combinational Cognition (DEFT) - Dr. Sourav	1,83,567.00	0.00	0.00	0.00	0.00	0.00	0.00	1,83,567.00	0.00
27	Investigating impact of sleep DBF: Dr. Sourav Banerjee	0.00	40,40,343.00	0.00	0.00	0.00	0.00	0.00	0.00	40,40,343.00
28	Regulation of human Placental SERS DBF: Dr. Sourav Banerjee	0.00	20,52,000.00	4,139.10	0.00	0.00	8,64,224.00	9,61,967.00	0.00	19,31,464.00
29	Innovation in Science Funds for Inspire	2,32,009.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	2,32,009.10
30	DEFT-CSI (Dr. Sourav Banerjee)	20,016.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	20,016.10
31	Centre for Excellence for Science (Phase II)	20,80,685.10	1,67,294.00	11,04,050.00	0.00	29,55,104.00	0.00	0.00	0.00	2,30,825.10
32	Dementia Science Programme	45,84,153.49	0.00	0.00	0.00	0.00	34,974.00	34,974.00	12,62,271.00	32,86,906.49
33	Dementia Basic Research (Dr. Sourav Banerjee)	26,27,090.03	0.00	0.00	0.00	0.00	5,22,549.00	5,22,549.00	13,58,200.00	9,46,241.03
34	Pathway Protein of NBRC	52,51,108.30	0.00	0.00	0.00	0.00	0.00	0.00	52,51,108.30	0.00
35	DEFT-SERS-Dr. Sourav Banerjee	1,40,075.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1,40,075.00

S. No./ Annex.	NAME OF PROJECT	Opening Balance as on 01.04.2023	Grants received during the year 2023-24	Interest/other income earned during the year 2023-24	Capital Exp. during the year 2023-24	Revenue Expenditure during the year 2023-24	Closing Balance as on 31.03.2024
						Margover Others Total Expenditure	
36	FOE-SERBI(Soakam Engineering)	26,472.00	0.00	0.00	0.00	0.00	30,472.00
37	DST- Inspired Culture (Giridhar Mahapatra)	4,280.00	0.00	0.00	0.00	0.00	4,280.00
38	TWAS-DST (Sudha Bhatnagar)	11,045.00	0.00	0.00	0.00	0.00	11,045.00
39	IBRO RETURNED HOME START-UP CHARTER SHAWAN SHANKAR SAHU	10,22,796.00	0.00	0.00	0.00	0.00	10,22,796.00
40	UNDERSTANDING THE REGULATED SECRETORY PATHWAY AND ITS ROLE IN REGULATING PHYSIO-METABOLIC FUNCTION- DR BHAVANI SHANKAR SAHU	14,65,692.60	0.00	12,701.00	0.00	4,61,094.00	10,70,109.60
41	ROLE OF EPITHELIAL RECEPTORS IN HIV RESISTANCE- DR PANKAJ SETHI	1,17,832.57	0.00	0.00	0.00	36,800.00	91,832.57
42	NEUROPATHOGENESIS- DR PANKAJ SETHI	7,85,836.00	0.00	0.00	0.00	1,92,836.00	2,46,636.00
43	ARTIFICIAL INTELLIGENCE- DR PRAVAT K. MANDAL	9,12,276.00	0.00	14,315.00	0.00	4,60,678.00	4,61,678.00
44	AN NEUROANATOMICAL STUDY- DR SOUMYA YENGAIR	10,42,996.79	0.00	0.00	0.00	10,73,234.00	3,66,473.29
45	CAUSAL-CAST-3- DR SOUBHAY BANERJEE	4,58,747.00	0.00	0.00	0.00	0.00	3,60,503.79
46	TECH THROUGH EMOTIONAL PRIMING- DR SHUBHAM KUMAR SWAGATA DEB	4,58,747.00	23,53,440.00	16,621.00	0.00	3,87,015.00	6,50,747.00
47	WELLCOME TRUST/DST INDIA ALLIANCE FELLOW- DR NIVETHIDA T.	0.44	0.00	0.00	0.00	0.44	0.00
48	THE WELLCOME TRUST/DST INDIA ALLIANCE EARLY CAREER FELLOW- DR NIVETHIDA T.	11,57,432.26	13,94,326.00	0.00	0.00	0.00	2,35,092.04
49	C V Raman Institute Science Fellow (Dr. Bellard Kiree)	28,542.00	2,75,000.00	0.00	0.00	1,07,756.00	2,05,446.00
50	Reuni- Co-scientist Network India (Dr. Aman Banerjee)	18,430.00	0.00	0.00	0.00	0.00	0.00
51	Vijaya Gokul Sancha Perumal- Dr. Arpan Banerjee	5,05,272.00	0.00	0.00	0.00	0.00	5,05,272.00
52	Audara Behavior and Diffusion Tensor Imaging- Dr. Narasim C. Singh	60,474.00	0.00	0.00	0.00	0.00	60,474.00
53	DST (TPAR Grant)-Dr. Nandini C. Singh	5,26,234.00	0.00	0.00	0.00	0.00	5,26,234.00
54	CSIR- Project Dr. Nihar Banerjee	73,009.50	0.00	0.00	0.00	0.00	73,009.50
55	Tata Innovation Fellowship- Dr. Nihar Banerjee	9,05,758.00	0.00	0.00	0.00	0.00	3,09,758.00
56	INDUS-25 & NIH 301- Dr. Pankaj Sethi	10,31,826.42	0.00	0.00	0.00	0.00	10,31,826.42
57	National Initiative on Data Lab Research Project- Dr. Pankaj Sethi	92,588.71	0.00	0.00	0.00	0.00	92,588.71
58	PDP-SERBI/AMIT NASKAR	11,28,935.00	0.00	0.00	0.00	0.00	11,28,935.00
59	PDP-SERBI/Adak Dasgupta	45,520.00	0.00	0.00	0.00	0.00	45,520.00
60	DST Tata Innovation Fellowship- Dr. P. K. Roy	6,67,424.00	0.00	0.00	0.00	0.00	6,67,424.00
61	Study of Neuroanatomical Reorganization after Injury/Stroke	3,50,959.21	0.00	0.00	0.00	2,21,351.00	1,29,608.21
62	Carotid Artery Electromyography- Dr. Anindya Ghosh Roy	0.00	1,24,35,590.00	11,185.00	0.00	62,812.47	1,23,83,962.53
63	Wadsworth Trust- Dr. Anindya Ghosh Roy	0.00	24,37,000.00	11,513.00	0.00	1,74,054.00	22,74,457.00
64	Role of PL3/4/5/6/7/8/9/10/11/12/13/14/15/16/17/18/19/20/21/22/23/24/25/26/27/28/29/30/31/32/33/34/35/36/37/38/39/40/41/42/43/44/45/46/47/48/49/50/51/52/53/54/55/56/57/58/59/60/61/62/63/64/65/66/67/68/69/70/71/72/73/74/75/76/77/78/79/80/81/82/83/84/85/86/87/88/89/90/91/92/93/94/95/96/97/98/99/100/101/102/103/104/105/106/107/108/109/110/111/112/113/114/115/116/117/118/119/120/121/122/123/124/125/126/127/128/129/130/131/132/133/134/135/136/137/138/139/140/141/142/143/144/145/146/147/148/149/150/151/152/153/154/155/156/157/158/159/160/161/162/163/164/165/166/167/168/169/170/171/172/173/174/175/176/177/178/179/180/181/182/183/184/185/186/187/188/189/190/191/192/193/194/195/196/197/198/199/200/201/202/203/204/205/206/207/208/209/210/211/212/213/214/215/216/217/218/219/220/221/222/223/224/225/226/227/228/229/230/231/232/233/234/235/236/237/238/239/240/241/242/243/244/245/246/247/248/249/250/251/252/253/254/255/256/257/258/259/260/261/262/263/264/265/266/267/268/269/270/271/272/273/274/275/276/277/278/279/280/281/282/283/284/285/286/287/288/289/290/291/292/293/294/295/296/297/298/299/300/301/302/303/304/305/306/307/308/309/310/311/312/313/314/315/316/317/318/319/320/321/322/323/324/325/326/327/328/329/330/331/332/333/334/335/336/337/338/339/340/341/342/343/344/345/346/347/348/349/350/351/352/353/354/355/356/357/358/359/360/361/362/363/364/365/366/367/368/369/370/371/372/373/374/375/376/377/378/379/380/381/382/383/384/385/386/387/388/389/390/391/392/393/394/395/396/397/398/399/400/401/402/403/404/405/406/407/408/409/410/411/412/413/414/415/416/417/418/419/420/421/422/423/424/425/426/427/428/429/430/431/432/433/434/435/436/437/438/439/440/441/442/443/444/445/446/447/448/449/450/451/452/453/454/455/456/457/458/459/460/461/462/463/464/465/466/467/468/469/470/471/472/473/474/475/476/477/478/479/480/481/482/483/484/485/486/487/488/489/490/491/492/493/494/495/496/497/498/499/500/501/502/503/504/505/506/507/508/509/510/511/512/513/514/515/516/517/518/519/520/521/522/523/524/525/526/527/528/529/530/531/532/533/534/535/536/537/538/539/540/541/542/543/544/545/546/547/548/549/550/551/552/553/554/555/556/557/558/559/560/561/562/563/564/565/566/567/568/569/570/571/572/573/574/575/576/577/578/579/580/581/582/583/584/585/586/587/588/589/590/591/592/593/594/595/596/597/598/599/600/601/602/603/604/605/606/607/608/609/610/611/612/613/614/615/616/617/618/619/620/621/622/623/624/625/626/627/628/629/630/631/632/633/634/635/636/637/638/639/640/641/642/643/644/645/646/647/648/649/650/651/652/653/654/655/656/657/658/659/660/661/662/663/664/665/666/667/668/669/670/671/672/673/674/675/676/677/678/679/680/681/682/683/684/685/686/687/688/689/690/691/692/693/694/695/696/697/698/699/700/701/702/703/704/705/706/707/708/709/710/711/712/713/714/715/716/717/718/719/720/721/722/723/724/725/726/727/728/729/730/731/732/733/734/735/736/737/738/739/740/741/742/743/744/745/746/747/748/749/750/751/752/753/754/755/756/757/758/759/760/761/762/763/764/765/766/767/768/769/770/771/772/773/774/775/776/777/778/779/780/781/782/783/784/785/786/787/788/789/790/791/792/793/794/795/796/797/798/799/800/801/802/803/804/805/806/807/808/809/810/811/812/813/814/815/816/817/818/819/820/821/822/823/824/825/826/827/828/829/830/831/832/833/834/835/836/837/838/839/840/841/842/843/844/845/846/847/848/849/850/851/852/853/854/855/856/857/858/859/860/861/862/863/864/865/866/867/868/869/870/871/872/873/874/875/876/877/878/879/880/881/882/883/884/885/886/887/888/889/890/891/892/893/894/895/896/897/898/899/900/901/902/903/904/905/906/907/908/909/910/911/912/913/914/915/916/917/918/919/920/921/922/923/924/925/926/927/928/929/930/931/932/933/934/935/936/937/938/939/940/941/942/943/944/945/946/947/948/949/950/951/952/953/954/955/956/957/958/959/960/961/962/963/964/965/966/967/968/969/970/971/972/973/974/975/976/977/978/979/980/981/982/983/984/985/986/987/988/989/990/991/992/993/994/995/996/997/998/999/1000/1001/1002/1003/1004/1005/1006/1007/1008/1009/1010/1011/1012/1013/1014/1015/1016/1017/1018/1019/1020/1021/1022/1023/1024/1025/1026/1027/1028/1029/1030/1031/1032/1033/1034/1035/1036/1037/1038/1039/1040/1041/1042/1043/1044/1045/1046/1047/1048/1049/1050/1051/1052/1053/1054/1055/1056/1057/1058/1059/1060/1061/1062/1063/1064/1065/1066/1067/1068/1069/1070/1071/1072/1073/1074/1075/1076/1077/1078/1079/1080/1081/1082/1083/1084/1085/1086/1087/1088/1089/1090/1091/1092/1093/1094/1095/1096/1097/1098/1099/1100/1101/1102/1103/1104/1105/1106/1107/1108/1109/1110/1111/1112/1113/1114/1115/1116/1117/1118/1119/1120/1121/1122/1123/1124/1125/1126/1127/1128/1129/1130/1131/1132/1133/1134/1135/1136/1137/1138/1139/1140/1141/1142/1143/1144/1145/1146/1147/1148/1149/1150/1151/1152/1153/1154/1155/1156/1157/1158/1159/1160/1161/1162/1163/1164/1165/1166/1167/1168/1169/1170/1171/1172/1173/1174/1175/1176/1177/1178/1179/1180/1181/1182/1183/1184/1185/1186/1187/1188/1189/1190/1191/1192/1193/1194/1195/1196/1197/1198/1199/1200/1201/1202/1203/1204/1205/1206/1207/1208/1209/1210/1211/1212/1213/1214/1215/1216/1217/1218/1219/1220/1221/1222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S. No./Ann. No.	NAME OF PROJECT	Opening Balance as on 01.04.2023	Grants received during the year 2023-24	Interest/other Income earned during the year 2023-24	Capital Exp. during the year 2023-24	Revenue Expenditure during the year 2023-24	Total Expenditure	Balance of Unspent Balance/Interest	Closing Balance as on 31.03.2024
						Manpower	Others		
68	NPQF-DIL SOHAN SAHA	215,968.00	8,00,000.00	0.00	0.00	6,51,124.00	1,00,000.00	7,51,124.00	0.00
69	DBT RA Nitoye Majumdar	0.00	5,00,203.00	0.00	0.00	35,085.00	4,49,276.00	4,85,311.00	0.00
70	ICMR-Ospali Singh	36,691.00	5,63,837.00	0.00	0.00	5,78,161.00	18,034.00	5,97,195.00	0.00
71	COLLABORATIVE RESEARCH AND EXPERIMENTATION (ICLL-DR. ARPAN BANERJEE)	23,07,895.00	23,35,878.00	0.00	14,48,480.00	0.00	10,09,214.00	10,09,214.00	0.00
72	STUDY OF NEURONAL REGENERATION AFTER INJURY-DL BHAVANI SHANKAR TARI	13,52,892.00	6,00,000.00	16,137.00	6,52,933.00	0.00	7,99,691.00	7,99,691.00	0.00
73	SARS COV2 DIT PANKAJ SETH	2,24,375.00	29,94,858.00	0.00	0.00	91,800.00	5,29,858.00	6,22,658.00	0.00
74	ICMR-Tips Niza	2,72,723.00	0.00	0.00	0.00	2,76,805.00	278.00	2,77,173.00	0.00
	Total (A)	3,34,16,767.61	4,04,13,698.28	13,41,852.65	21,21,333.00	78,23,838.00	1,24,00,508.00	2,02,24,486.63	1,38,42,228.26
1	DELCON FUTURE CONSTITUTION (B)*	44,02,162.43	96,50,25,373.00	0.00	0.00	6,87,526.00	94,01,55,476.50	94,08,43,002.50	0.00
	Grand Total (A+B)	3,78,18,930.04	3,00,63,95,073.28	13,41,852.65	21,21,333.00	85,11,364.00	95,25,55,945.12	96,10,67,499.33	1,38,42,228.26


SOURAV BANERJEE
A.O.C, NBRC


TANMAY BHATTACHARYYA
CHIEF ADMINISTRATIVE OFFICER
National Brain Research Centre
National Institute of Education
National Brain Research Centre
Sector-12/2051 / Mansarovar-12051
Gurgaon / Haryana


Prof. Krishanu Ray
Director / Director
National Brain Research Centre
National Institute of Education
National Brain Research Centre
Sector-12/2051 / Mansarovar-12051
Gurgaon / Haryana

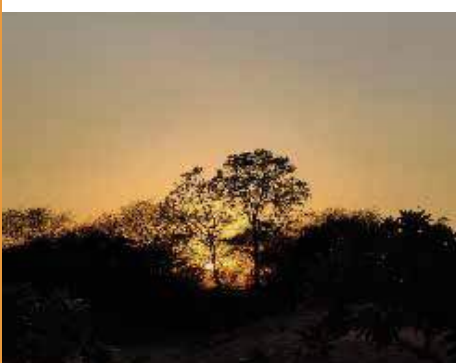
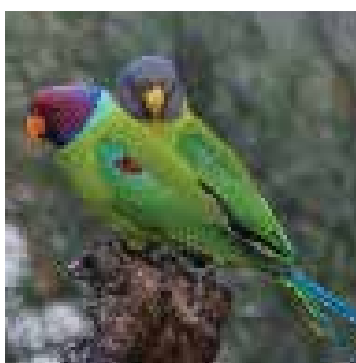
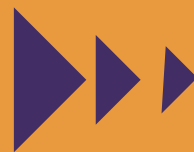
As per our separate report
of even date signed

For Kumar Khare & Co
Chartered Accountants
Firm No. 7406
Date: 19/08/2024
Place: New Delhi

Acknowledgments ►►►

- **Back Cover Image:-** Dr. Sibaram Behera, AGR Lab
- **Front Cover Image:-** Ms. Ritu Moni Borah, CBD Lab & BioRender
- **Image Before Scientific Reports (Page No. 12):-** Dr. Kirti Lathoria, ES Lab
(The image depicts the findings in the form of western India's 'Warli' tribal art.)
- **Image Before Publications (Page No. 77):-** Ms. Archana Mehta, PS Lab
- **Compilation of Annual Report 2023-24:-** Ms. Seepika





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