ANNUAL REPORT

2020–2021

National Brain Research Centre

Manesar, India

Annual Report 2020-21

National Brain Research Centre

Contents

Mandate & Objectives

MANDATE

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

VISION

The vision we have for NBRC is that it would not only grow into a world-class institute for brain research but also create a vibrant active neuroscience community by catalyzing the overall growth of this discipline in India. The desirable outcome of this initiative is the generation of skilled manpower in neuroscience research who would help India achieve an international leadership in this frontier area of science. This initiative would help Indian neuroscientists to participate in global research efforts as equal partners. The knowledge base generated from these efforts would help diagnostic tools and therapeutic strategies for treatment of brain-related disorders. A unique role for NBRC is that it will act as a node with linkages to other centers carrying out neuroscience research in the country, acting in effect as the "hub of the wheel" rather than the wheel itself.

From The Director's Desk

It is an absolute honor for me to be the Director-in-Charge of National Brain Research Center (NBRC). NBRC is devoted to studying brain functions in health and diseases using innovative multidisciplinary approach in basic and translational research. Due to consistent efforts and scientific advancement, NBRC has established itself as an advanced center of global recognition for neuroscience research. Through our M.Sc. and Ph.D. programs, we train personnel with knowledge and expertise to be able to efficiently conquer challenges and conduct interdisciplinary research in neurosciences. Research at NBRC is classified into five divisions, namely Cellular and Molecular, Systems, Cognitive, Computational, and Translational. Nevertheless, faculty collaborate across divisions and other institutions to address their research questions. Owing to multidisciplinary approach, novel model systems, and support of intra- and inter-institutional collaborations, our scientists have made several discoveries during the past year despite the challenges caused by the pandemic. NBRC has conquered outstanding milestones as evidenced by research publications, awards, and honors. It is with sheer joy that I present to you this NBRC's Annual Report (2020–21). The report summarizes our paramount accomplishments and pursuits and highlights as to how NBRC is working toward fulfilling its vision.

NBRC's dedicated research faculty has been delivering high-quality research with technological advancement and major scientific discoveries. Dr. Anirban Basu's lab recently observed that atorvastatin effectively reduces viral load and cell death in the subventricular zone of infected animals and demonstrated importance of the host innate antiviral response in motor functioning deterioration and pathogenesis of flaccid paralysis upon neurotropic virus infection. Using swim-exercise model in nematode *C. elegans*, Dr. Ghosh-Roy's lab demonstrated that exercise session following axonal injury in mechanosensory neuron improves functional restoration through the regeneration of the injured axon. Dr. Bhavani Shankar Sahu's lab plans to study several aspects related to sub-cellular trafficking/secretion in dense core vesicles (DCVs) and understand how DCV proteins regulate neuronal, metabolic, and physiological functions. Prof. Pankaj Seth's lab has made several contributions in deciphering the cellular–molecular mechanisms of viruses, including HIV-1, Zika, and SARS-CoV-2. Soon after the pandemic, NBRC researchers had proposed that along with its respiratory consequences, SARS-CoV-2 potentially affects human brain functions. Prof. Ranjit Kumar Giri's lab works on Alzheimer's and prion disease using mouse and CNS stem/progenitor cell-based models. Prof. Shiv Sharma's laboratory has discovered that a small peptide reduces amyloid pathology and improves memory in an animal model of Alzheimer's disease (AD). Prof. Dhruba's lab aims to understand the mechanism of complex neurodevelopmental and neurodegenerative disorders using Drosophila model system. Prof. Soumya Iyengar's lab studies self-awareness in songbirds by using "mark test." On the other hand, Dr. Sourav Banerjee's Synapse Biology Laboratory elucidated a microRNA-dependent synaptic plasticity mechanism that plays a pivotal role to keep synaptic activity at a threshold point. In my NeuroImaging and NeuroSpectroscopy laboratory, we use multimodal neuroimaging techniques to examine how healthy aging and several dementia forms affect neural cognitive substrates. Our research identifies early diagnostic markers for AD using noninvasive imaging modalities, including MRI and magnetoencephalography.

We assess neurochemical levels and functional performance in AD progression and develop tools and platforms for neuroimaging data processing. Moreover, we recently introduced project SWADESH, a novel initiative for brain research covering comprehensive database and data analytics platforms. SWADESH aims to build India's brain initiative focusing on neuroimaging, neurochemical, neuropsychological data and analytics for brain disorders, including AD.

For institutional development of NBRC, self-study report was submitted on Feb 19, 2021 to the National Assessment Accreditation Council, Bangalore, for the 1st cycle of NAAC accreditation for grading NBRC in the UGC system. The accreditation is expected soon by the yearend. In the last few months, NBRC signed 3 MoUs namely with Florey Institute of Neuroscience and Mental Health (Australia), University of Pittsburgh (Pennsylvania, USA), and HelpAge India (NGO, India). In 2021, NBRC launched Dr. APJ Abdul Kalam Awards for encouraging research efforts of Ph.D. students who accomplish 1st author publication in the first 2 academic years. Since January 2021, 4 students have been awarded. We also initiated Best Employee Awards for encouraging staff members. In February 2021, Green Canopy Committee was constituted for promoting environmental sustainability and nature conservation. In May 2021, new cafeteria Kalpataru was launched for students and staff. NBRC faculty is acutely aware of its responsibility of taking science to the schools. We organize open days and other events for nearby schools. However, considering the impact of the recent viral outbreak, Science Sethu webinar series was conducted as part of DBT's college outreach initiative. Going beyond neurosciences, NBRC has institutionalized its multidisciplinary character by widening its admission criteria to include students with diverse background, such as physics, chemistry, computer sciences, mathematics, and engineering sciences. NBRC conducts symposia, workshops, meetings, and conferences to exchange knowledge and facilitate interactions between students and researchers nationally and internationally. Additionally, state-of-the-art facilities, services, advancing research, and academic programs of NBRC continue to strengthen the foundation.

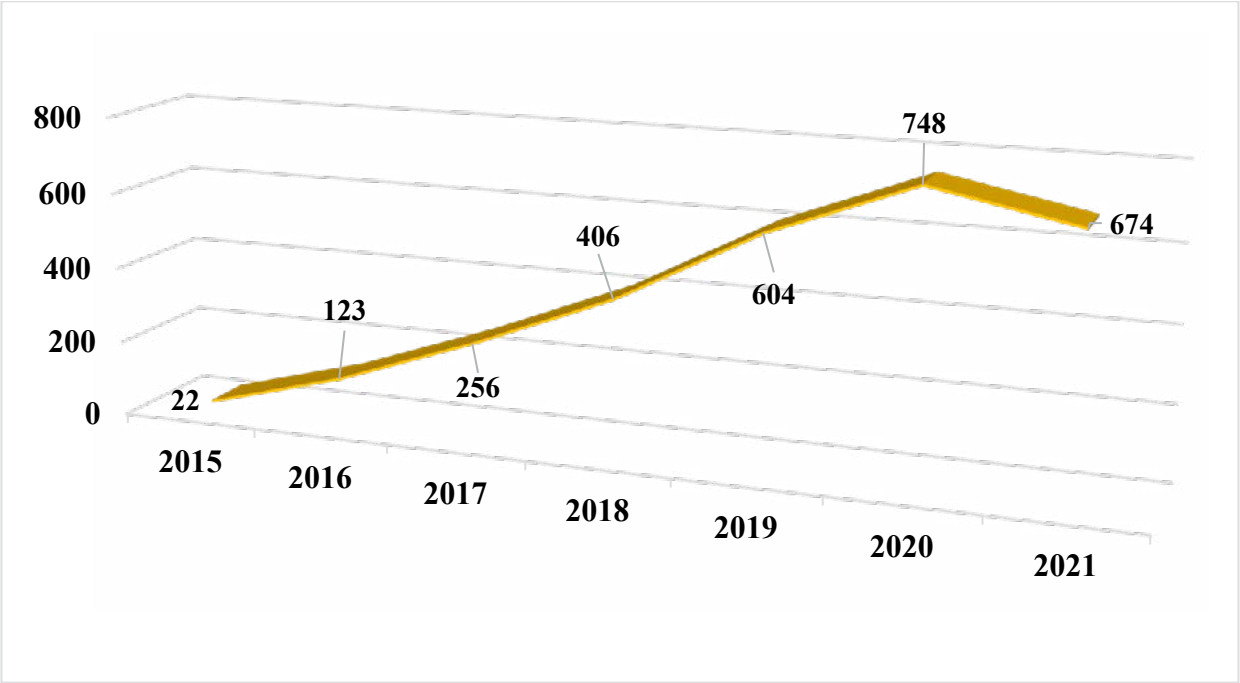
On the occasion of 74th Independence Day, NBRC conducted flag hoisting ceremony on 15th Aug 2021. From 1–15th September 2021, NBRC took up in the national celebration of Hindi Pakhwada and Hindi Diwas on 14th September 2021 by organizing talks lectures and various competitions. Moreover, NBRC has efficiently attended to its societal responsibilities. NBRC offers neurological outpatient department services at Civil Hospital, Gurgaon, to citizens and those from surrounding districts. Patients with epilepsy visit the campus for medical check-ups using the magnetoencephalography facility in collaboration with AIIMS, New Delhi. Patients are treated free of cost. In collaboration with HelpAge India, NBRC conducts awareness programs for senior citizens regarding mental health and neurodegenerative diseases associated with old age. In this pandemic, NBRC collaborated with Civil Hospital to avail vaccination drives to students and employees. With immediate appointment of Resident Medical Officer, we implemented preventive and curative measures at institutional level and is giving special medical attention to students and staff suffering from COVID-19. Our students, faculty, administration, and employees rose to the occasion for fighting COVID-19 crisis.

I end by expressing sincere gratitude to the Department of Biotechnology for its continuous generous support. I am grateful to the Former Directors and pillars of NBRC for their contribution and consistent efforts to take NBRC to greater heights. They enhanced agendas, visions, and goals that NBRC strives to achieve. It has been a steadfast successful growth curve for NBRC, and it continues to rise higher. Special thanks to the members of all the committees of NBRC and those from Indian scientific community who served on various committees, acted as thesis examiners, and advised or collaborated with NBRC. I look forward to this continued support to help NBRC achieve the goals and vision it has set for itself.

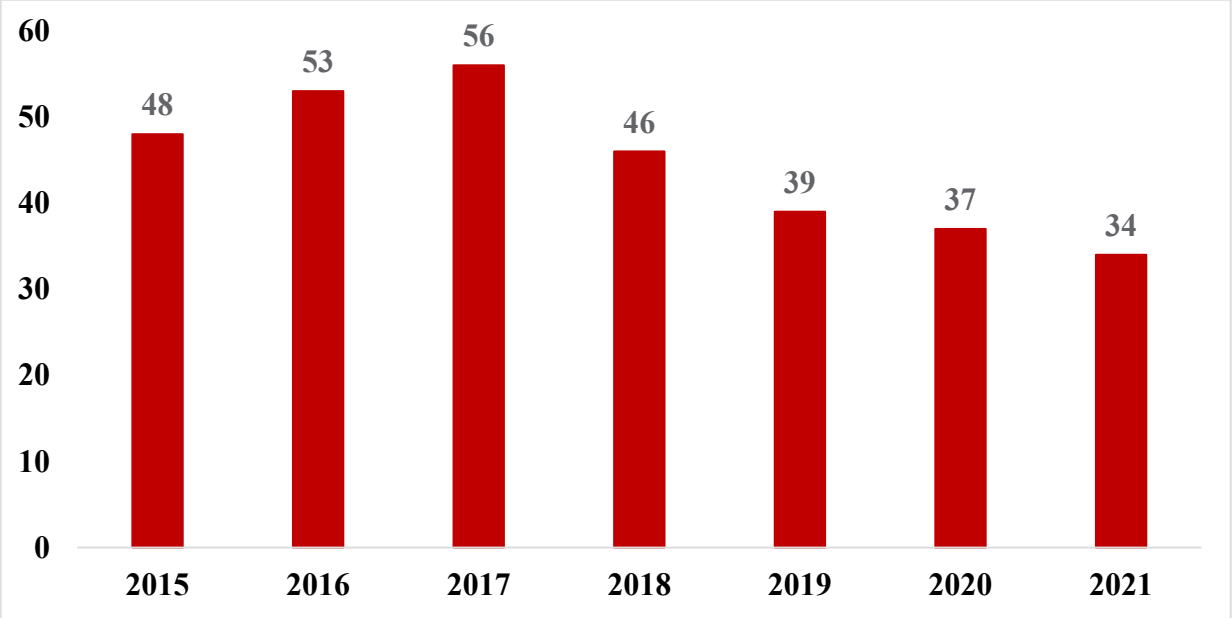
- Prof. Pravat Kumar Mandal
Director-in-Charge,
National Brain Research Centre

NBRC at a glance

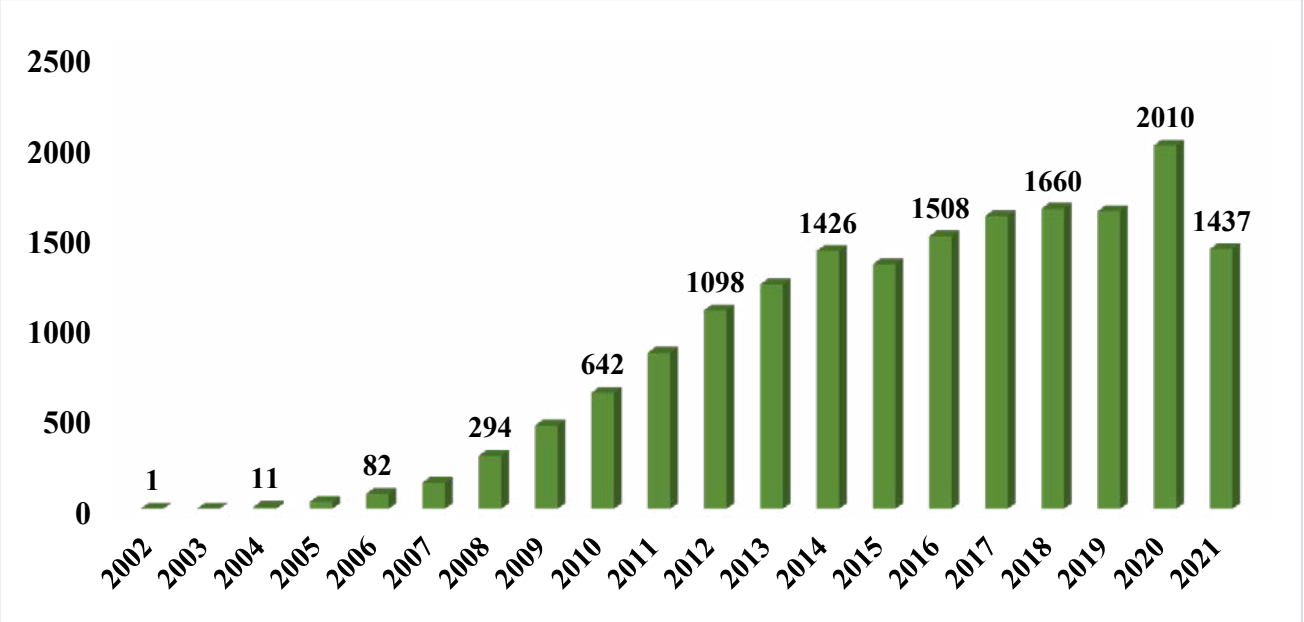
Total no. of citations from NBRC (2015–2021) (year-wise)



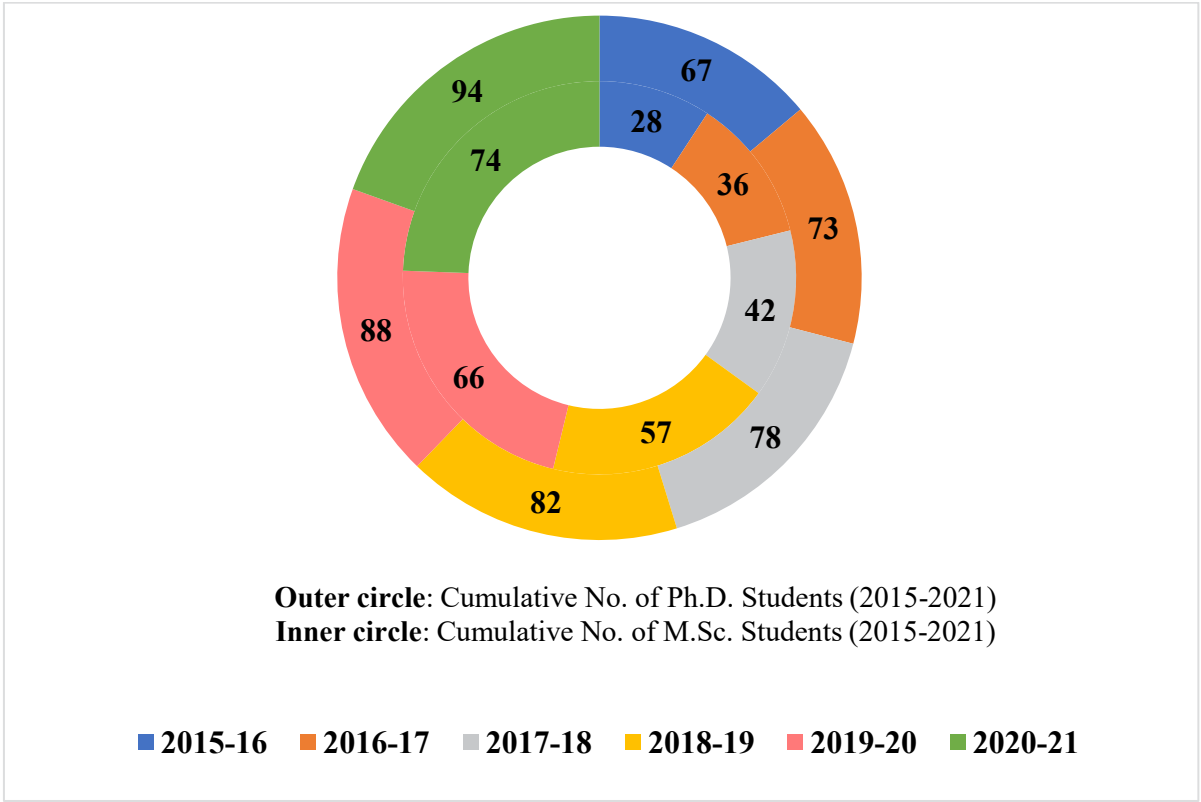
Total no. of publications from NBRC (2015-2021) (yearwise)



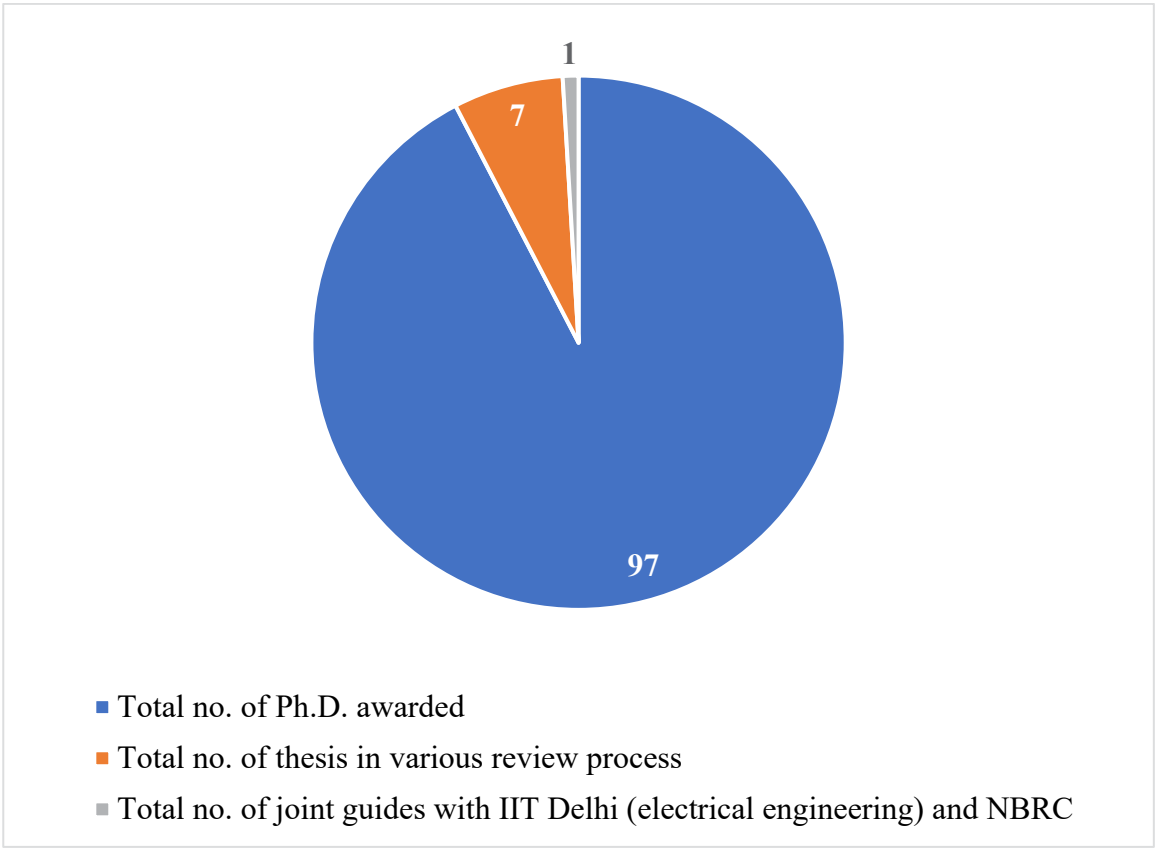
Total no. of citations from NBRC (2002–2021)



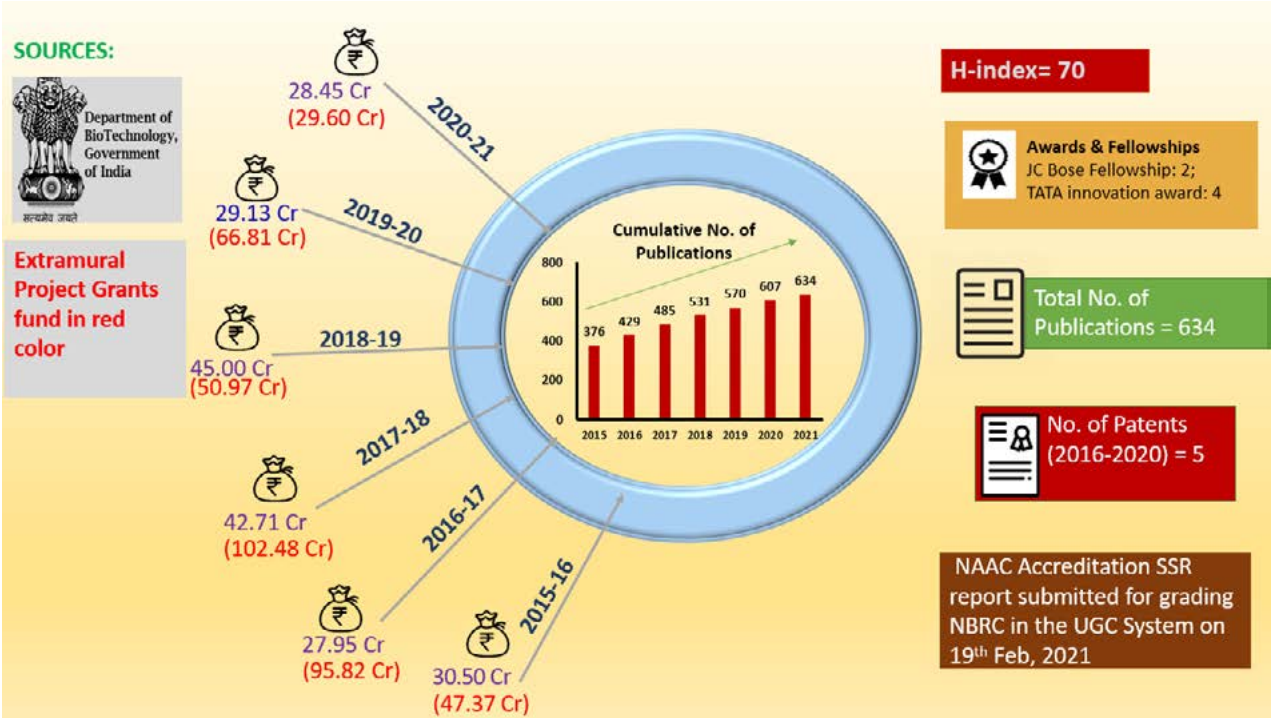
Cumulative No. of Ph.D. Students (2015-2021)



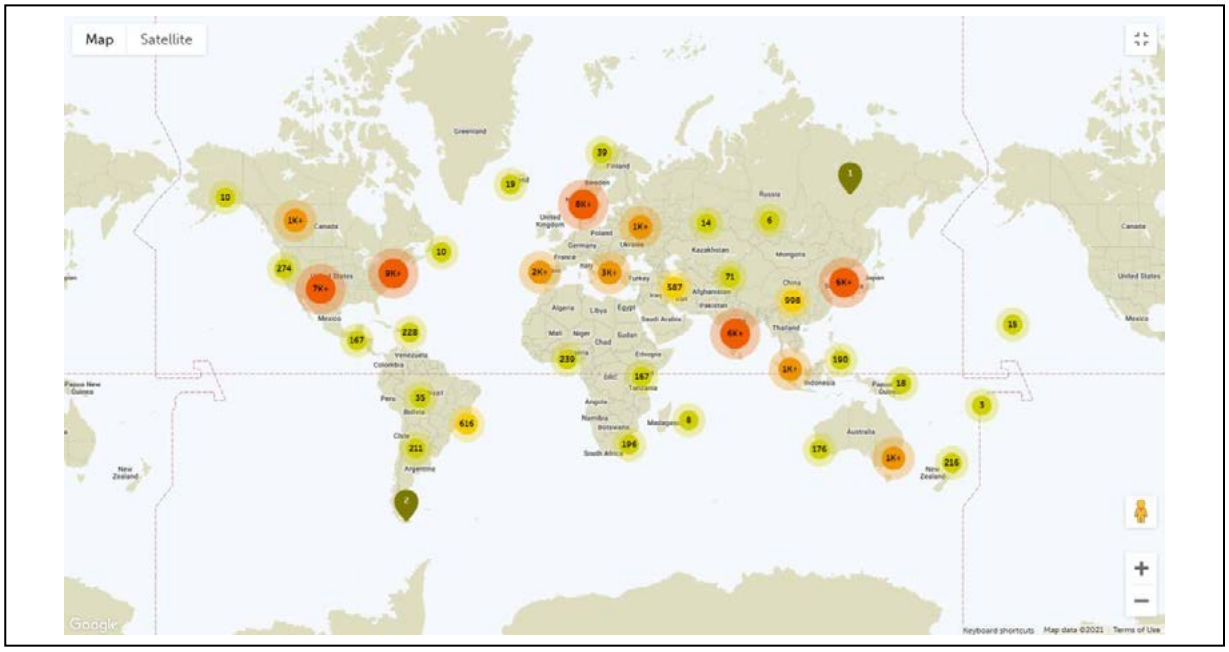
Academic Highlights

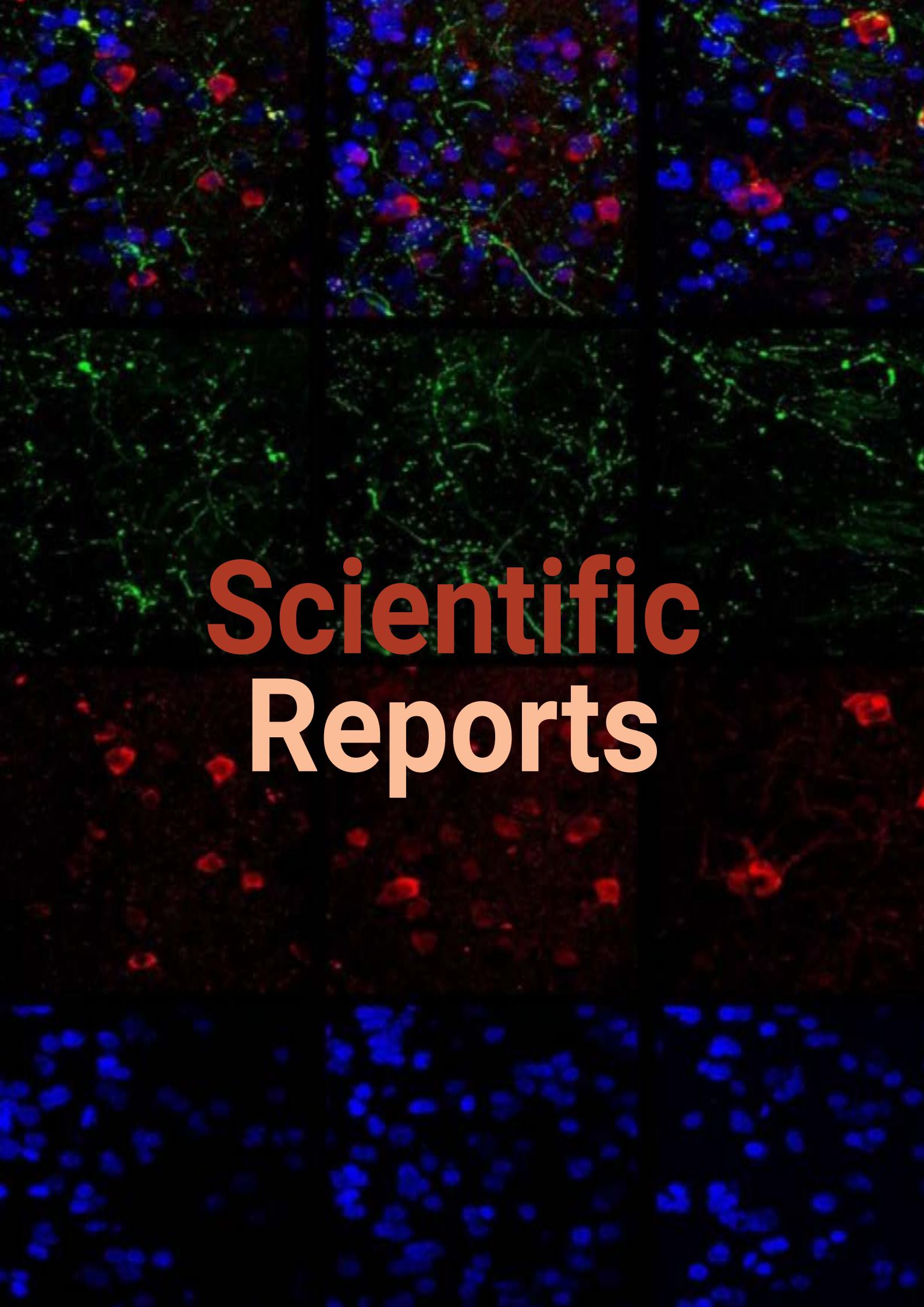


Funding Overview of the last 5 years



NBRC research being recognized worldwide, 2021 demographics for the article entitled "Predictive Imageable Biomarkers for Neurodegenerative and Neurodevelopmental Diseases"




Scientific
Reports

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

Anindya Ghosh Roy

Principal Investigator:

Anindya Ghosh Roy

Research Associate/ Post-doctoral Fellows:

Swagata Dey (India Alliance Early career fellow)

PhD Students:

Dharmendra Puri

Atrayee Basu

Harjot Kaur

Sibaram Behera

Sunanda Sharma

Pallavi Singh

MSc Students:

Debapriya Roy

Project Assitants:

Smriti Bharadwaj

Kavi Nila

Sruthy Ravivarma

Technical Assistant:

Sumit Mahapatra

Yunis Khan

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 intact, living animals. 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 signaling affect these processes. One major focus is axon regeneration.

1) Regulation of neuronal polarity:

Neuronal polarization is defined by the formation of axons with parallel arrays of plus-end-out and dendrites with the non-uniform orientation of microtubules (MTs). In *C. elegans*, Posterior Lateral Microtubule (PLM) neuron is bipolar with its two processes growing along the anterior-posterior axis under the guidance of Wnt signaling. We found that loss of Kinesin-13 family microtubule depolymerizing enzyme KLP-7 leads to ectopic extension of axon-like processes from PLM cell body. Live imaging of microtubules and axonal transport with revealed mixed polarity of MTs in the short posterior process suggesting its dendrite like nature. KLP-7 is positively regulated in the posterior process by Planar Cell Polarity components of Wnt signaling to induce mixed polarity of MTs. Whereas KLP-7 is negatively regulated in the anterior process by the UNC-73/CED-10 cascade to establish uniform MT polarity. Our work elucidated how evolutionary conserved Wnt signaling establishes MT polarity in a neuron through Kinesin-13. (Puri et al 2021, JCB).

To find out novel regulators of microtubule cytoskeleton in neuron, we have screened and identified mutants those suppress the neuronal phenotype of *klp-7* mutant. Mutants affecting many of the microtubule stabilizing factors involving plus or minus end binding proteins, and centrosomal proteins did not suppress *klp-7(0)*. However, the drug Colchicine that destabilizes MTs suppressed the same. Some of the identified genes code for proteins encoding RNA binding protein, beta tubulin, and adaptor for vesicular transport, kinesin.

One of the genes we identified is *muscleblind-1/mbl-1* that encodes for the polypyrimidine tract binding protein. Muscleblind family proteins are known to control RNA splicing. We found that MBL-1 is necessary and sufficient for axon growth of PLM and ALM neuron. Loss of *mbl-1* destabilizes the microtubules and affects axonal transport in PLM neuron leading to short axon phenotype (Figure-1) and improper synapse formation. MBL-1 tagged with GFP is present both in nucleus and axon. To identify the possible targets of MBL-1 in axon development, we performed an interaction analysis between *mbl-1* and genes required for

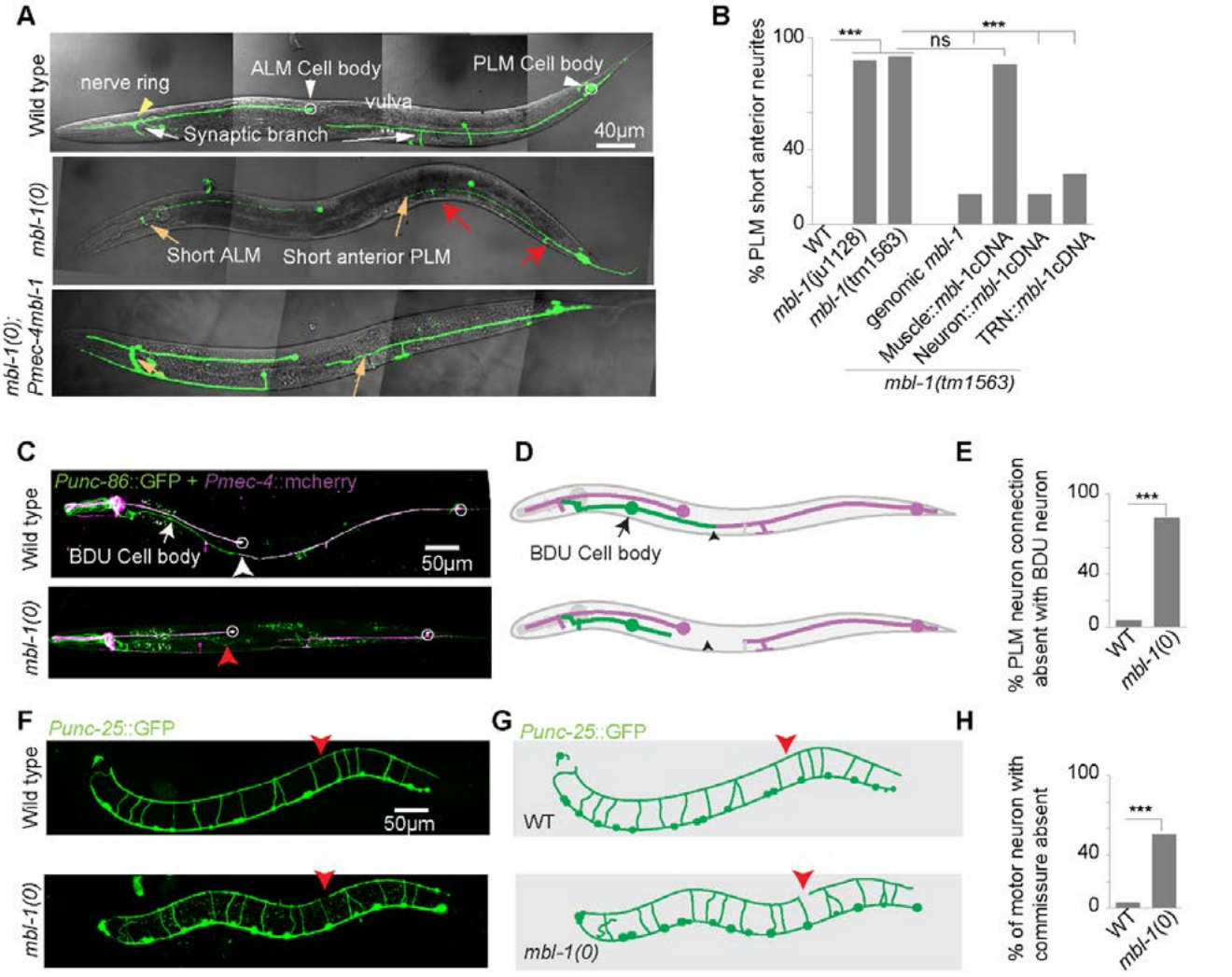


Figure 1: mbl-1 mutant display defect in axon growth. (A) Confocal images of ALM and PLM neurons in WT, mbl-1(0), and mbl-1(0); Pmec-4-mbl-1 background. In mbl-1(0) short anterior process of ALM and PLM is marked in orange arrow. The presence of ectopic synapse in mbl-1(0) is marked in the red arrow. (B) Quantification of short anterior neurite phenotype. Images (C) and schematic (D) of touch neurons (magenta) and BDU neurons (green) in wild-type and mbl-1(0). The presence of physical contact between PLM anterior and BDU neuron is shown in white arrowhead in the wild type background which is lost in mbl-1(0) shown in red arrowhead. The representative images (F) and schematic (G) of motor neurons in the wild-type and mbl-1(0). Red arrow showing defect in the motor neuron in mbl-1(0) background

axon development. We found that *mbl-1* is epistatic to genes encoding MEC-7 (β-tubulin), VAB-8 (Kinesin-11 family motor), and UNC-76 (adaptor for Kinesin-1 mediated transport). We hypothesize that MBL-1 is required for proper splicing of *mec-7*, *vab-8* and *unc-76* mRNA to optimize microtubule growth and transport during axonal growth. Alternatively, MBL-1 might act as an adaptor for these mRNA for their transport (Manuscript under preparation).

2) Neuronal Regeneration

Functional neuronal circuits are subject to injuries during the normal life of an individual. Despite a comprehensive understanding of the mechanisms underlying the development of the nervous system, the pathways that repair damage after injury remain poorly understood. This is a very important from the point of therapeutics as adult nervous system is extremely refractile to accidental damage repair. Our past work has helped develop C. elegans mechanosensory neuron as a model for axon regeneration studies. The conserved Dual Leucine Zipper Kinase (DLK-1) pathway is essential for axon regrowth. Consequent to these discoveries, several efforts have been made using model systems such as worm, fly, and fish to understand the neuron-intrinsic mechanisms of axon regrowth. However, mechanistic aspects of functional recovery during axon regeneration remained unclear.

We have earlier established a neurosurgery protocol with multiple 2-photon lasers. Using this, we have found that the axotomy of Posterior touch neuron PLM leads to a dramatic loss of posterior touch sensation. We found that during the regeneration phase, the fusion between the proximal and distal fragments of an injured axon leads to rapid functional recovery (Basu et al 2017, PNAS). We also discovered that *let-7* miRNA inhibits functional restoration via the fusogen molecule, EFF-1 (Basu et al 2017, PNAS).

Insulin Signalling regulates functional rewiring of injured axon: We have seen that the injured axons which do not show a fusion-like phenomenon give functional recovery in later stages. Particularly, the axons which reach the original target area and accumulate presynaptic proteins in the ventral nerve cord are likely to give functional recovery. Ventral targeting and functional restoration decline with age. We found that loss of Insulin signalling (IIs) receptor DAF-2 promotes ventral targeting in a DAF-16 dependent manner irrespective of age. We further showed that coordinated activities of DAF-16 in neuron, as well as muscle, promote ventral targeting. In response to axotomy, DAF-16 upregulates the expression and localization of the Netrin receptor UNC-40 in the growth cone of the proximal stump. (Basu et al 2021, Development).

Swimming exercise promotes post-injury axon regeneration and functional restoration through AMPK

Pharmacological approaches have not been very successful in alleviating the consequences of nervous

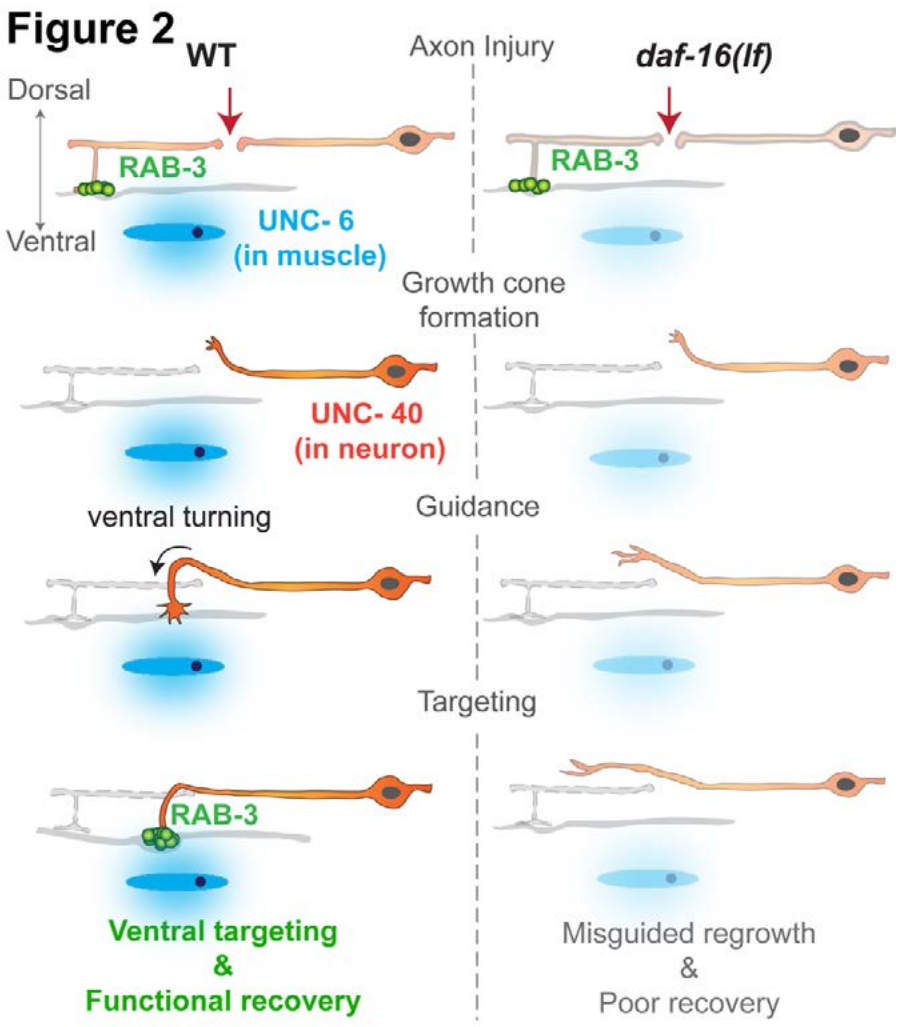


Figure 2: The model explains how the guidance of the injured PLM axon towards the ventral cord is compromised in daf-16 mutant due to lack of expression of UNC-40 and UNC-6 in neuron and muscle, respectively

system injury. On the contrary, physical activity and rehabilitation interventions are often beneficial to improve the health conditions in the patients with neuronal injuries. Using touch neuron circuit of Caenorhabditis elegans, we investigated the role of physical exercise in the improvement of functional restoration after axotomy. We found that a swimming session of 90 minutes following the axotomy of Posterior Lateral Microtubule (PLM) neuron can improve functional recovery in larval and adult stage animals. In older age, multiple exercise sessions were required to enhance the functional recovery. Genetic analysis of axon regeneration mutants showed that exercise-mediated enhancement of functional recovery depends on the ability of axon to regenerate. Exercise promotes early initiation of regrowth, self-fusion of proximal and distal ends, as well as post-regrowth enhancement of function. We further found that the swimming exercise promotes axon regeneration through the activity of cellular energy sensorAAK-2/AMPK in both muscle and neuron. Our study established a paradigm where systemic effects of exercise on functional regeneration could be addressed at the single neuron level. (Kumar et al 2021, eNeuro).

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 (y Cajal,

Figure 3

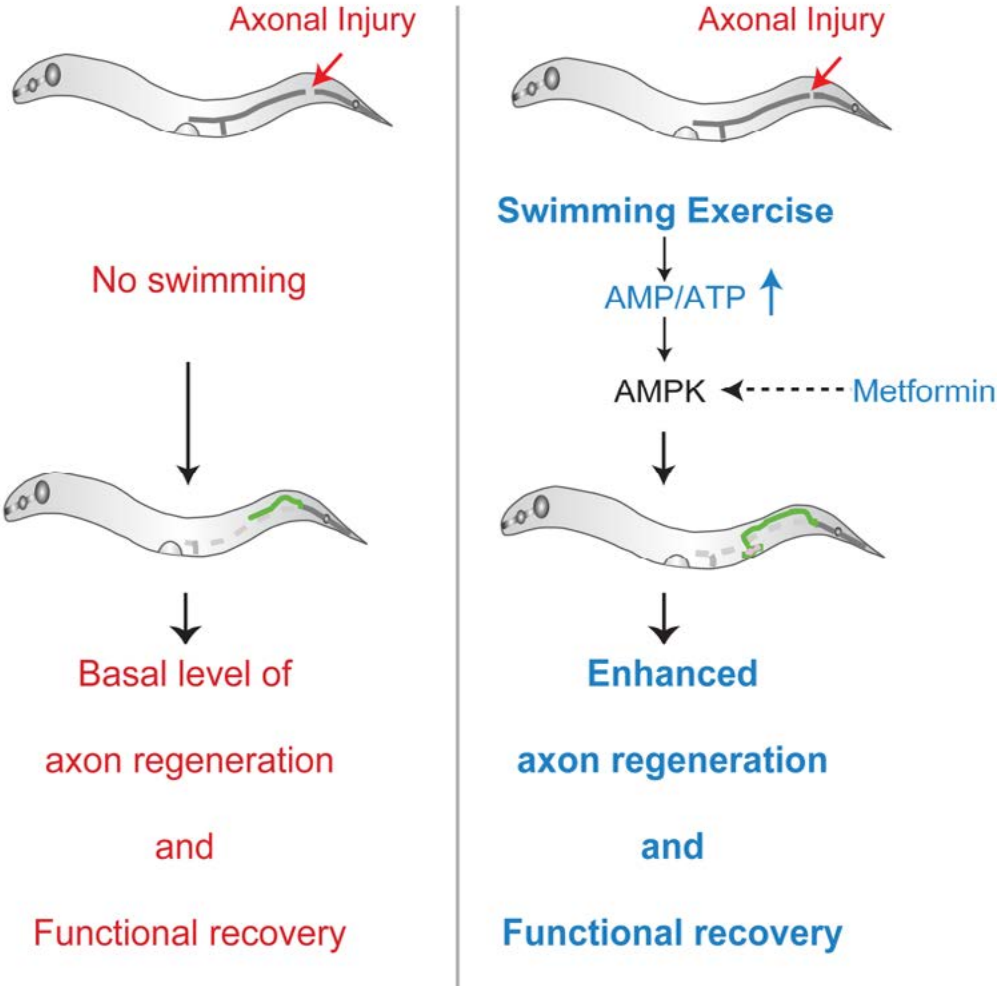


Figure 3: Proposed model illustrating how swimming exercise promotes axon regeneration. Exercise session after axonal injury leads to the consumption of cellular ATP resulting in activation of AMPK. An activated form of AMPK promotes axon regeneration and functional restoration

1991, Thompson-Peer et al., 2016). To understand the mechanisms of dendrite regeneration, we used PVD neurons, which have branched dendrites (Inberg et al., 2019). The PVD neurons are responsible for harsh touch sensation (Tao et al., 2019). 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. To test whether the dendrite regeneration shares the mechanism with that of an axon, we tested the major signalling hubs such as DLK-1, cAMP, let-7 miRNA, Akt-1, Phosphatidylserine (PS) exposure that control axon regeneration. 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 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 (Harjot Kaur Brar, 2021).

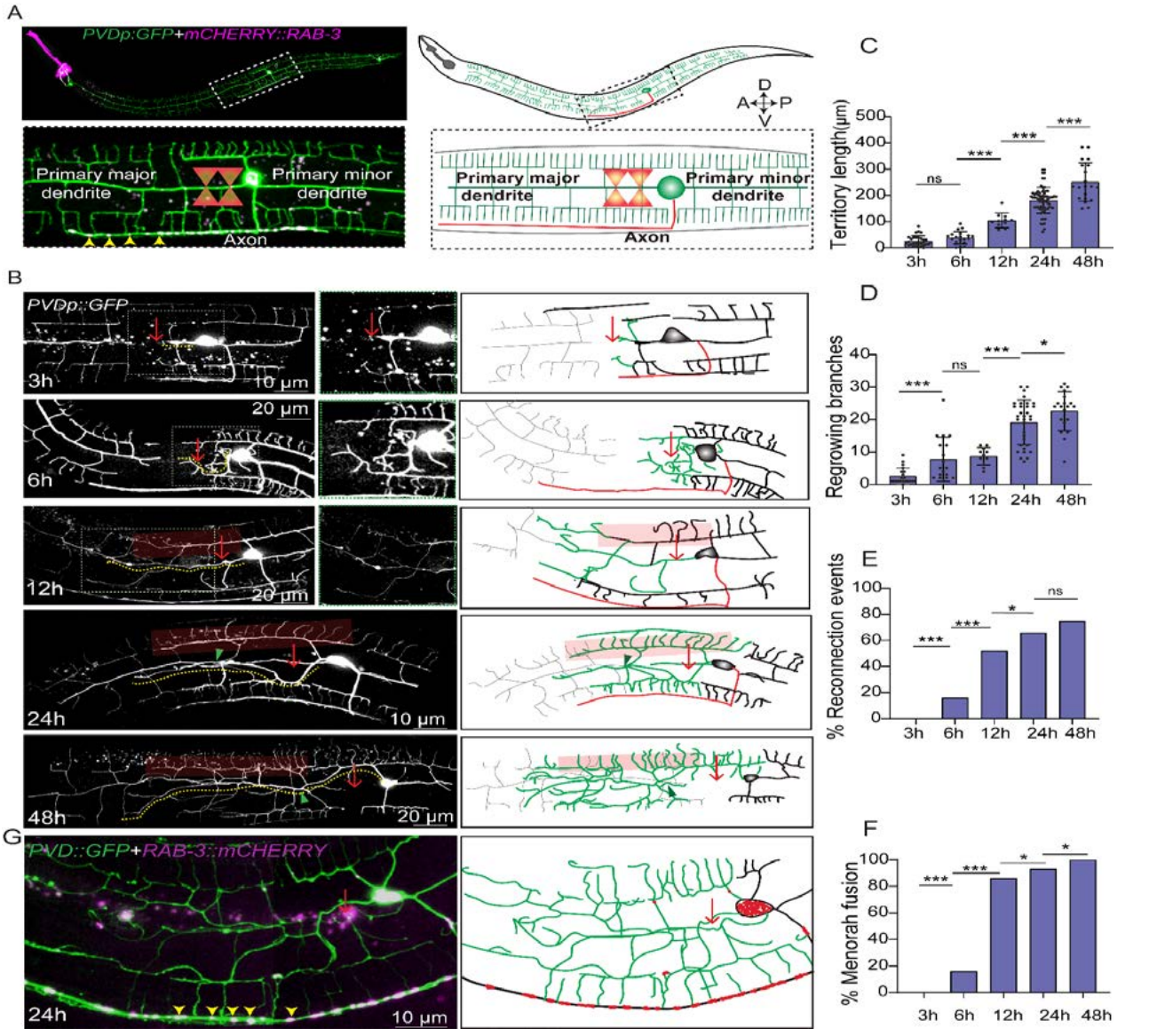


Figure-4: (A) Confocal image and schematics of PVD neuron with branched dendrites. (B) Primary dendrite regrows new processes from the cut dendrite tip. (C-D) Quantification and dendrite regeneration

Publications:

1) NY Kadam, S Behera, S Kumar, **A Ghosh-Roy**, K Babu. The G-protein coupled receptor SRX-97 is required for concentration dependent sensing of Benzaldehyde in Caenorhabditis elegans. eNeuro, 4 January 2021, ***eNEURO*** .0011-20.2020; DOI: https://doi.org/10.1523/ENEURO.0011-20.2020

2) P Pandey, A Singh, H Kaur, **A Ghosh-Roy**, K Babu (2021). Increased dopaminergic neurotransmission results in ethanol dependent sedative behaviors in Caenorhabditis elegans". ***PLOS GENETICS***, February 1, 2021. https://doi.org/10.1371/journal.pgen.1009346

Presentations:

1) IBRO school organized by IGIB, "Regulatory RNAs and the Brain: Development to Disease".

2) Keynote speaker in the Brain Awareness Week on 17th March 2021 organized by Centre for Cognitive and Brain Sciences at the Indian Institute of Technology Gandhinagar, Title of seminar: "C. elegans as a model for nerve regeneration study"

Funding:

NSERB (CRG/2019/00294): 2020-2023

NBRC Core

Collaborators:

Sandhya Koushika, TIFR, Mumbai, India

Sourav Banerjee, NBRC, India

Smarajit Polley, Bose Institute, Kolkata

Kavita Babu, IISER-Mohali

Awards: (if any)

None

Degrees Awarded (Ph.D.):

Akanksha Goyal (MSc degree)

Meetings/Conferences organized:

None

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

Anirban Basu

Department of Cellular & Molecular Neuroscience, Translational Neuroscience

Principal Investigator

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Research Fellow

Surojit Chakrabarty

Meenakshi Bhaskar

Shivangi Sharma

Stuti Mohapatra

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MSc (Neuroscience) student

Ankit Kumar Shah

Technician C

Kanhaiya Lal Kumawat

Technician B

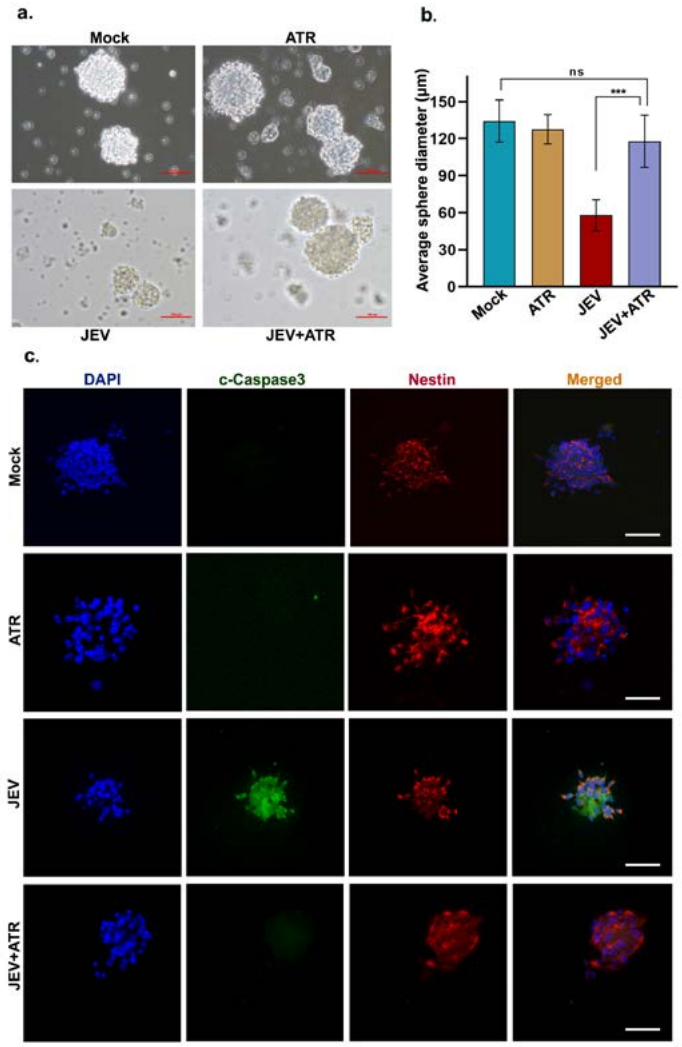
Manish Dogra

Japanese Encephalitis Virus (JEV) entry into the host system is followed by viral replication in the periphery which in turn is accompanied by activation of innate and adaptive arms of immune system. In case of adults, immune system is normally capable of eliminating virus from the circulation thus preventing it from invading central nervous system (CNS). Whereas in children and geriatric patients, owing to weaker immune response against JEV, the latter gains entry into CNS thus initiating a vicious cycle of inflammatory reactions which ultimately lead to neuronal death. This virus-induced encephalitis is considered to be the sole factor resulting in patient mortality in case of Japanese encephalitis Virus (JEV) infections. Virus replication inside host cell is a complex process involving various steps like viral entry, unpacking of viral genomes, genome replication, virus packaging and egress. Each of the aforementioned processes involve activity of a plethora of molecules which acting in concert result in the successful completion of intracellular life-cycle of virus. Our lab has been working on deciphering the molecular details of various steps of viral life cycle thus contributing significantly to the field of JEV-host interactions.

JE virus causes sporadic encephalitis with nearly 25% fatal case reports. JEV infects neural stem/ progenitor cells (NSPCs) and decreases their proliferation. Statin, a commonly used class of cholesterol lowering drug, has been shown to possess potent anti-inflammatory and neuroprotective effects in acute brain injury and chronic neurodegenerative conditions. Here, we aimed to check the efficacy of atorvastatin in alleviating the symptoms of Japanese encephalitis (JE). Using BALB/c mouse model of JEV infection, we observed that atorvastatin effectively reduces viral load in the subventricular zone (SVZ) of infected pups and decreases the resultant cell death. Furthermore, atorvastatin abrogates microglial activation and production of proinflammatory cyto/chemokine production post JEV infection *in vivo*. It also reduced interferon-β response in the neurogenic environs. The neuroprotective role of atorvastatin is again evident from the rescued neurosphere size and decreased cell death *in vitro*. It has also been observed that upon atorvastatin administration, cell cycle regulatory proteins and cell survival proteins are also restored to their respective expression level as observed in uninfected animals. Thus, the antiviral, immunomodulatory and neuroprotective roles of atorvastatin reflect in our experimental observations. Therefore, this drug broadens a path for future therapeutic measures against JEV infection.

Poliomyelitis-like illness is a common manifestation associated with neurotropic virus infections. Functional loss and death of motor neurons in spinal cord often led to reduced muscle tone and paralysis, which subsequently result in clinical symptoms like movement disorders, cognitive impairment and long-term neurological sequelae among survivors. Despite several reports on molecular basis of encephalopathy, the pathogenesis of flaccid paralysis upon viral infection remained largely unknown. The present study elucidates the mechanism responsible for limb paralysis by studying clinical isolates of Japanese encephalitis virus

(JEV) and Chandipura virus (CHPV) causing clinical-AFP (Acute flaccid paralysis) in vast region of south-east Asia including Indian subcontinent. Experimental model for studying virus-induced AFP was generated by intra-peritoneal injection of 10-day old BALB/c mice. Pups were subjected to a series of behavioral tests to assess gait, neurodegeneration and locomotory behavior. Progressive decline in motor performance of infected animals was found when compared with control animals. Paralysis was correlated with death of motor neurons (MN) by studying various cell death-assays both in *in vivo* and *in vitro* settings. Furthermore, this study demonstrates that upon infection, extrinsic apoptotic pathway gets activated in MNs in a RIG-I-dependent fashion via activation of transcription factor IRF-3 and IRF-7. Once activated, this pathway leads to interferon-independent apoptosis of MNs. Both gene-silencing experiments using specific RIG-I-siRNA and in-vivo morpholino abrogated cellular apoptosis, thus validating important role of pattern recognition receptor (PRR) RIG-I in MN death. Hence from our experimental observations, we have demonstrated that host innate antiviral response might play a critical role in deterioration of motor functioning and pathogenesis of flaccid paralysis upon neurotropic virus infections.



Impaired neurosphere formation from JEV infected animals is rescued in atorvastatin treated group.

(**a**) Bright field images of neurospheres cultured from Mock, ATR, JEV and JEV+ATR mice pups. Magnification 10X; scale bar corresponds to 100µm. Images shown in this Fig. are representative of four individual animals from each group (**b**) Average sphere diameter (µm) histogram plotted for the mentioned experimental groups. Values represent mean ± SD (***p<0.001; ns: non-significant). (**c**) Double-immunofluorescence staining images of neurospheres cultured from SVZ region of mentioned groups for Nestin and cleaved-Caspase-3. Magnification 20X; scale bar corresponds to 50µm. Four animals per experimental group were used. Data are representative of three independent experiments.

Publications:

1. Sehrawat, S., Khasa, R., Deb, A., Prajapat, S. K., Mallick, S., **Basu, A.**, ... & Vrati, S. (2021). Valosin-containing protein/p97 plays critical roles in the Japanese encephalitis virus life cycle. *Journal of Virology*, *95*(11), e02336-20.

2. Tripathi, S., Shree, B., Mohapatra, S., **Basu, A.***, & Sharma, V**.** (2021). The Expanding Regulatory Mechanisms and Cellular Functions of Long Non-coding RNAs (lncRNAs) in Neuroinflammation. *Molecular Neurobiology*, 1-24. (Review)

3. Wani, M. A., Mukherjee, S., Mallick, S., Akbar, I., & **Basu, A.** (2020). Atorvastatin ameliorates viral burden and neural stem/progenitor cell (NSPC) death in an experimental model of Japanese encephalitis. *Journal of Biosciences*, *45*, 1-17.

4. Chakraborty, S., & **Basu, A.** (2020). The COVID-19 pandemic: catching up with the cataclysm. *F1000Research*, *9*. (F1000 Faculty Rev):638 (*Review*)

Presentation:

1. A Basu (2021) Host MicroRNA: An important modulator of antiviral immunity in Japanese Encephalitis virus infection. IBRO-APRC Associate school of Neuroscience, CSIR-IGIB, Work shop theme: Regulatory RNAs and the Brain: Development to Disease" Session theme: Neurovirology: Regulation by RNA, 20-26th January, 2021.

2. A Basu (2020) Delivered a lecture on "Brain and it's health", curtain raiser by INSA for IISF-2020. 4th of December, 2020

3. A Basu (2020) Modulation of Neural Stem/ Progenitor Cell response following Japanese Encephalitis Virus infection, Annual meeting of Society for Neurochemistry (SNCI):11-13th December, 2020

4. A Basu (2020) Innate Immunity in the central nervous system: Redefining the relationship between "Immune system" and "Nervous system". KT Shetty Memorial Oration of Indian Academy of Neurosciences (IAN) for the year 2019. Oration delivered in the occasion of XXXVIII Annual Conference of Indian Academy of Neurosciences, October 4 – 7, 2020.

5. A Basu (2020) Modulation of Neural Stem/ Progenitor Cell response following Japanese Encephalitis Virus infection: Webinar entitled: Host-Microbe Interactions: Present and Future Perspectives; School of Biotechnology, Department of Life Sciences, Presidency University, Kolkata, 7th August, 2020

6. **A Basu (2020)** "ACS Science Talks." "Drug repositioning/repurposing: Promising strategy to develop therapy against viral infections." 31st July 2020.

Projects funded by external agency:

1. MicroRNA mediated regulation of neural stem/ progenitor cell fate in neurotropic flaviviral infection [Department of Biotechnology (BT/PR22341/ MED/122/55/2016)] starting from 29/12/2017, for three years. (*Approved for no cost extension for six months*)

2. Understanding the therapeutic role of adult stem cell derived exosome in combating virus induced neurodegenerative disease [Department of Biotechnology (BT/PR15984/MED/31/325/2015) starting from 20/03/2018, for three years.

3. Deciphering Antiviral Properties of Statins against Japanese Encephalitis Virus Infections [Department of Biotechnology BT/PR27796/ MED/29/1301/2018, starting from 26/12/2018, for two years. (*Approved for no cost extension for six months*)

4. Elucidating the role of long non coding RNAs (lncRNAs) in neuronal cell death during Japanese Encephalitis (JE) [Department of Biotechnology (BT/PR126590/MED/122/113/2017) starting from 05/03/2019, for three years.

Collaborations:

Pankaj Seth, Arpan Banerjee, and Dipanjan Roy, NBRC

Sunit Singh, Institute of Medical Sciences, BHU, Varanasi.

Sudhanshu Vrati, Prasenjit Guchhait, Manjula Kalia, and Arup Banerjee

RCB, NCR Biotech Cluster, Faridabad

Krishnan H Harshan, CCMB, Hyderabad

Anita Mahadevan, NIMHANS, Bangalore

Vivek Sharma, Department of Biological Sciences, Birla

Institute of Technology and Science Pilani, Hyderabad Campus, Hyderabad

Award (if any)

K T Shetty Memorial Oration Award (Indian Academy of Neurosciences)-2019 (*awarded in 2020*)

Degree awarded:

Indira S Priya (MSc in Neuroscience)

Meetings/Conferences organized:

Royal Society Yusuf Hamied Workshop for India and the UK, 3-4th March 2021 (virtual platform). Co-Organizer (from India) for the Neuroscience session: Understanding brain structure and function: from molecules to mind.

Neuro-cognitive network mechanisms using multimodal neuroimaging

Arpan Banerjee

Principal Investigator

Arpan Banerjee

Post-doctoral fellows

Dr Amit Naskar

Dr Soibam Shyamchand Singh

Dr Zinkal Shah

PhD Students

Neeraj Kumar

Priyanka Ghosh

Anagh Pathak

Ritu Moni Borah

Nisha Chetna Shashtry

Kirti Saluja

Pratika Siwatch

Shahswati Tripathi

Guncha Bhasin

MSc Students

Darshit Thakkar

Akanksha Gupta

Project Assistants

Varun Madan Mohan

Shubham Singhal

Cognitive Brain Lab (CBL) 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 mental health and investigating various functional brain networks related to speech perception and in particular multisensory integration following the approved objectives of this project. Here we outline the major project updates from the period April, 2020 - March, 2021. The overarching goal of these projects is to develop an understanding for the neurobiological mechanisms of multisensory integration and basic sensory function. The three projects on which we have focussed are related to determining efficient tools for cortical source localization from EEG/ MEG data, investigating the spatiotemporal representational space of neuronal entrainment to tonal rhythmic stimulus and exploring multiscale models of human resting state brain activity.

Project 1: Organization of directed functional connectivity among nodes of ventral attention network reveals the common network mechanisms underlying saliency processing across distinct spatial and spatio-temporal scales

Researchers: Priyanka Ghosh

Collaborator: Dipanjan Roy

Previous neuroimaging studies have extensively evaluated the structural and functional connectivity of the Ventral Attention Network (VAN) and its role in reorienting attention in the presence of a salient (pop-out) stimulus. However, a detailed understanding of the "directed" functional connectivity within the VAN during the process of reorientation remains elusive. Functional magnetic resonance imaging (fMRI) studies have not adequately addressed this issue due to a lack of appropriate temporal resolution required to capture this dynamic process. The present study investigates the neural changes associated with processing salient distractors operating at a slow and a fast time scale using custom-designed experiment involving visual search on static images and dynamic motion tracking, respectively. We recorded high-density scalp electroencephalography (EEG) from healthy human volunteers, obtained saliency-specific behavioral and spectral changes during the tasks, localized the sources underlying the spectral power modulations with individual-specific structural MRI scans, reconstructed the waveforms of the sources and finally, investigated the causal relationships between the sources using spectral Granger Causality (GC). We found that salient stimuli processing, across tasks with varying spatio-temporal complexities, involves a characteristic modulation in the alpha frequency band which is executed primarily by the nodes of the VAN constituting the temporo-parietal junction (TPJ), the insula and the lateral prefrontal cortex (lPFC). The directed functional connectivity

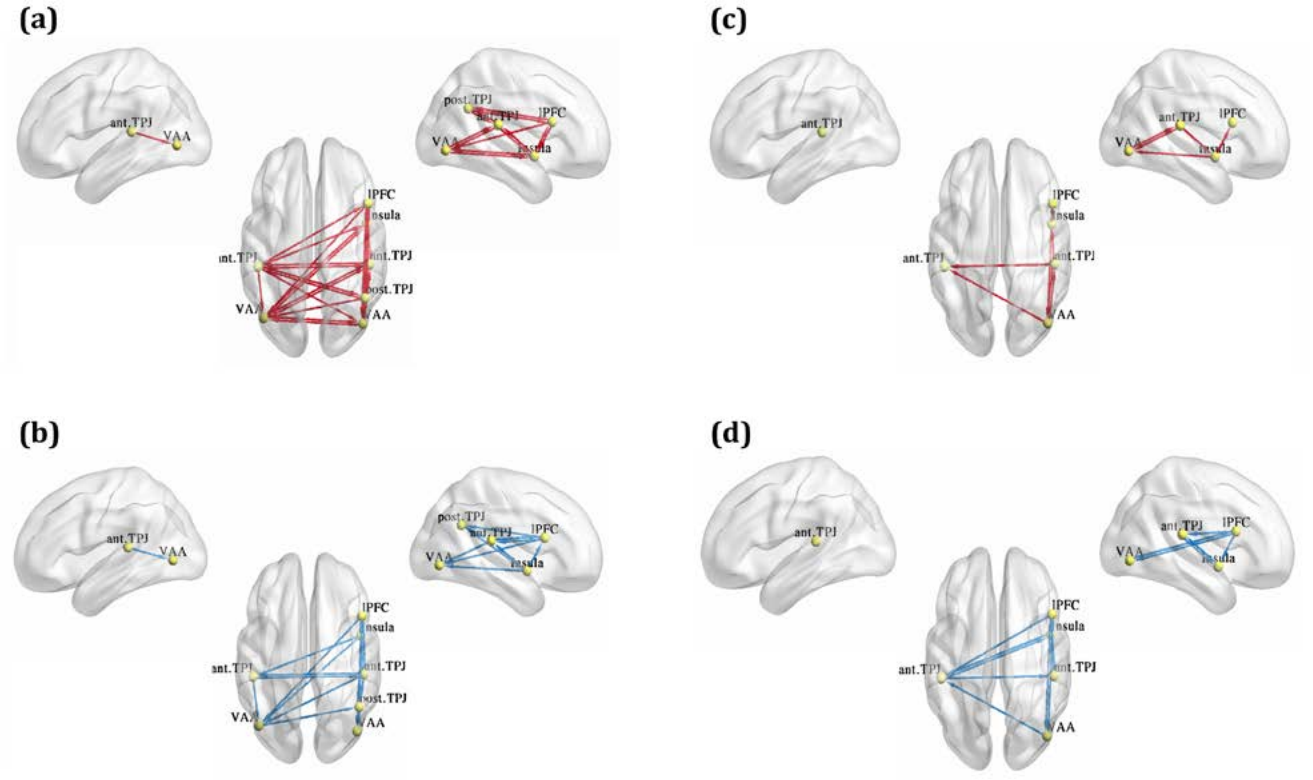


Figure 1: Directed interactions between the nodes of the Ventral Attention Network. The figure represents all significant causal interactions in the spectral domain (8-9 Hz) for node pairs based on their Granger causality scores for with saliency (ST) as well as without saliency (WT) trials in the dynamic (a, b) and the static (c, d) task conditions, respectively. The arrows point from the driver node towards the effector node. Red arrows indicate causations in trials with saliency (ST) whereas the blue arrows indicate causations in trials without saliency (WT)

results further revealed the presence of bidirectional interactions among prominent nodes of right-lateralized VAN, corresponding only to the trials with saliency (Figure 1). Thus, our study elucidates the invariant network mechanisms for processing saliency in visual attention tasks across diverse time-scales.

Project 2: Homeostatic brain network mechanisms facilitate perceptual binding of audio-visual speech

Researchers: Abhishek Mukherjee, Soibam Shyamchand Singh, Dipanjan Ray

Collaborator: Prof Partha Raghunathan

In daily lives, speech perception requires binding of spatiotemporally disjoint auditory and visual cues. Understanding the associated brain information processing requires characterization of the two complementary network mechanisms, functional segregation and integration. Here, we demonstrate using fMRI recordings, during perception of congruent and incongruent (McGurk) speech stimuli, that subjective perceptual experience of multisensory speech stimuli is dependent on a homeostatic balance of segregation and integration mechanisms. We were able to parametrically control the inter-subject variability of illusory perception by introducing temporal lags in the incongruent auditory–visual articulations of speech sounds. Enhancement of activity as a function of propensity to perceive illusion is observed in distributed brain regions, defined as the perceptual binding network whose anatomical locations range from sensory to associative cortices. Reduction in global integration can be inferred from the anticorrelation observed between the seed-based whole-brain functional connectivity computed at each node of the perceptual binding network and the propensity for illusory perception. That enhanced segregation and reduced integration facilitates cross-modal binding, was further validated by the increase in local clustering and modularity, concurrently with lowering in participation coefficient. Further, the perceptual binding network was anti-correlated with other intrinsic functional brain networks, such as dorsal attention and default mode networks during cross-modal perception. The degree of activation and anti-correlation decreased for people who reported lower illusory perception, further strengthened the hypothesis of homeostatic balance.

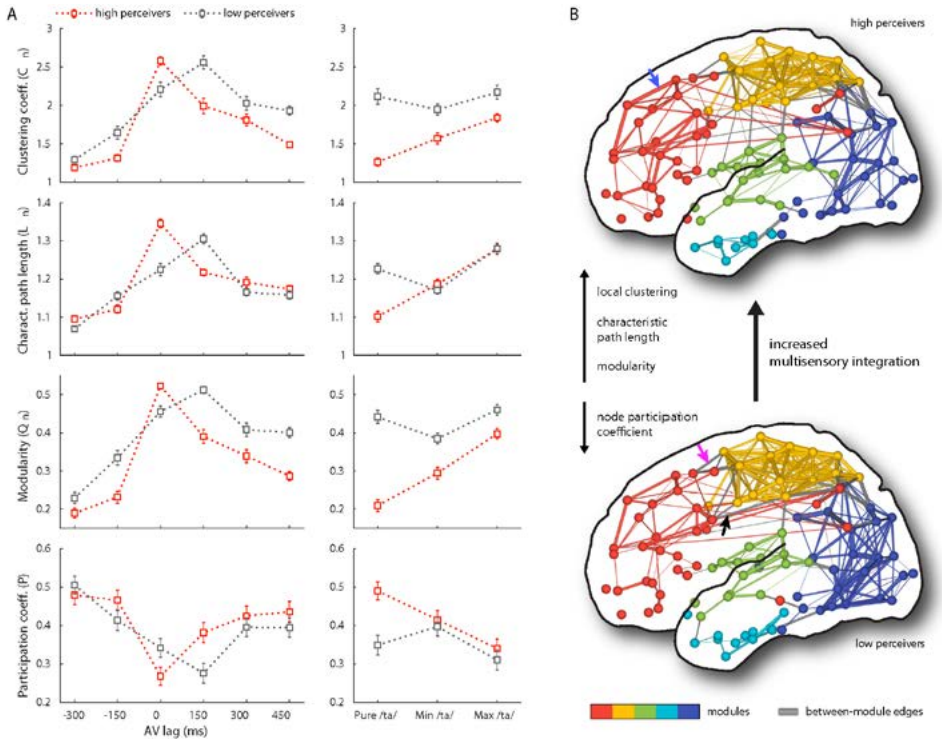


Figure 2: A) Change in the network topological measures for high and low perceivers. B) Left hemispheric brain network differences between high and low perceivers during L0 (synchronous and incongruent) condition

Publications

1. Ghosh, P., Roy, D., & **Banerjee, A.** (2021). Organization of directed functional connectivity among nodes of ventral attention network reveals the common network mechanisms underlying saliency processing across distinct spatial and spatio-temporal scales. *Neuroimage*, *231*, 117869.

2. Das, M., Singh, V., Uddin, L. Q., **Banerjee, A.**, & Roy, D. (2021). Reconfiguration of Directed Functional Connectivity Among Neurocognitive Networks with Aging: Considering the Role of Thalamo-Cortical Interactions. *Cerebral Cortex*, *31*(4), 1970-1986.

3. Sahoo, B., Pathak, A., Deco, G., **Banerjee, A.***, & Roy, D. (2020). Lifespan associated global patterns of coherent neural communication. *Neuroimage*, *216*, 116824.

4. Gupta, D. S., **Banerjee, A.**, Roy, D., & Piras, F. (2020). Temporal Structure of Neural Processes Coupling Sensory, Motor and Cognitive Functions of the Brain. *Frontiers in Computational Neuroscience*, *14*.

5. Kumar, V. G., Dutta, S., Talwar, S., Roy, D., & **Banerjee, A.** (2020). Biophysical mechanisms governing large-scale brain network dynamics underlying individual-specific variability of perception. *European Journal of Neuroscience*, *52*(7), 3746-3762.

Invited talks

1. **March 2021:** Invited speaker at Royal Society Yusuf Hamied Workshop for India and the UK organised by the Royal Society (UK) and the Indian National Science Academy (INSA)

2. **Jun 2020:** Git Commit Show (International Online developer conference) Can data science lead us to the grand unified theory of brain function?

MSc Thesis

Akanksha Gupta: Investigating changes in the functional organisation of brain network during illusionary perception.

Darshit Thakkar: Dynamics of resting state transient spectral alpha bursts with age.

Funding

- NBRC Core
- Department of Sports, Ministry of Youth Affairs & Sports (Feb 2019- Feb 2022) **Early diagnosis and structural and functional decline of the brain and associated injuries in professional athletes playing contact sports.**
- Department of Biotechnology, Ministry of Science & Technology. Flagship programme of NBRC: **Comparative mapping of common mental disorders (CMD) over lifespan.**

Collaborations

Anirban Basu NBRC

Dipanjan Roy NBRC

Ellora Sen NBRC

Partha Raghunathan, NBRC

Beena Koshy, CMC Vellore

How oscillatory brain dynamics and prediction errors link to memory processing

Dipanjan Roy

Principal Investigator:

Dipanjan Roy

Department of Computational Neuroscience, Cognitive Neuroscience, Systems Neuroscience, Translational Neuroscience

Research Associate / Post-doctoral Fellows:

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Dr. Priyanka Chakraborty (NBRC Research Associate)

PhD Students:

Vinsea Singh

Abhishek Singh Narvaria

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Azman Akhtar

Ankit Yadav

Indrajith Nair

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Rudradeep Mukherjee

Project Assistants:

Priyanka Sigar

Fahd Yazin

Vivek Sharma

Kusum Thuwal

Oscillatory band specific alteration in spontaneous activity mediates age associated dynamic perception, attention, and memory processing

Our lab is primary focused in understanding dynamic sensory processing, perception, and attention during spontaneous activity and goal directed task to disentangle the mechanisms how perception and attention give rise to memory. How does these dynamics process and cortex-wide functional connectivity change during late adolescence and in the context of healthy and pathological aging? To address this systematically one of the major focus of our laboratory has been to record and use the sensor and source level EEG/MEG, DTI, fMRI, resting state and behavioural task data during working and episodic memory encoding task. In an earlier work, we tried to understand communication through coherence (interareal synchrony in the brain) at the sensor level to pinpoint age associated alterations in normative brain dyanmics and their relationship with behavioural performance (Sahoo, B., Pathak, A., Deco, G., Banerjee, A.*, & Roy, D.* (2020) Lifespan associated global patterns of coherent neural communications *NeuroImage*, 116824; Also see annual report for 2019-20).

Our recent work with MEG further expanded the above finidngs by providing systematic insight about the accruracy of memory recall and speed of processing performance, cognitive load, and metacognitive awareness during memory task from young, middle age and elderly participants. We show specifically how different oscillatory features and 1/f noisy baseline could link potentially different behavioural measures and brain functions. Our results provide a systematic understanding about developmental, and age associated functional changes in the cortex during perception, attention, and memory processing over the entire lifespan.

Ongoing oscillatory brain dynamics link distinct functional aspects of cognition across adult lifespan

Signal transmission in the brain propagates via distinct oscillatory frequency bands but the coexisting aperiodic component - 1/f activity – has recently become a signal of interest for understanding several aspects of cognition. We used a recently proposed parameterisation model that delimits the oscillatory and aperiodic components of neural dynamics onto lifespan ageing data from large human cohort. Since, healthy ageing underlines an enormous change in local tissue properties, any systematic relationship of 1/f activity would highlight their impact on the self-organized critical functional states. Furthermore, we have used patterns of correlation between aperiodic background and metrics of behaviour, to understand the domain general global effects of 1/f activity. We hypothesized that age-associated change in 1/f baseline, alters the functional critical states of the brain affecting the

global information processing and found that 1/f impact critically all aspects of cognition e.g., metacognitive awareness, speed of retrieval of memory, cognitive load, and accuracy of recall through adult lifespan.

MEG Data acquisition and participant details: We used for this study resting state and behavioural data that were obtained from the Cam-CAN consortium. For all the 700 participants, MEG data were collected by Elekta Neuromag, Helsinki at MRC-CBSU using 306 channels, consisting of 102 magnetometers and 204 orthogonal planar gradiometers. MEG data collection was done in a light magnetically shielded room (MSR). A high pass filter of 0.03 Hz cut off was used to sample the data at 1000Hz. Head-Position Indicator (HPI) coils were used to continuously assess the head position within the MEG helmet. To monitor blinks and eye-movements, two pairs of bipolar electrodes were used to record horizontal and vertical electrooculogram signals. To monitor pulse- related artefacts, one pair of electrodes were used to record electrocardiogram signals. MEG data collected for resting state required the participants to sit still for a minimum of 8mins and 40 sec with their eyes closed. From this subset, 280 participants were included in the present study (70 in each group, namely Young Adult, Middle Elderly, Middle Late, Old Adult).

Visual Short-Term Working Memory Stimulus and task performance across healthy aging: The stimulus design was adapted from Zhang et al., 2008 (**Figure 1**). On each trial, participants were presented with 1,2,3, or 4 coloured discs (mimicking different memory load conditions) for 250ms. Following that, a blank screen was presented for 900ms to hold those colours in memory. One of the original locations was highlighted by a thick black border (acting as a probe for participants to remember the colour at that location), and at the same time, a response colour wheel was presented. Participants had as much time as required to report by touching or clicking, as accurately as possible the remembered hue of the highlighted disc. No feedback was given. After every trial, 830 ms fixation period was there. Participants indicated their lack of confidence in the precision of the colour (metacognitive awareness) by the length of the time they hold down the finger onto the point. Participants complete two blocks of 112 trials, with memory load (1,2,3 or 4) counterbalanced and randomly intermixed. For each set size (memory load), different measures were estimated by fitting the error distribution with a mixture model of Vonmises and uniform distributions.

Meg and behavioural data analysis: We analyzed the MEG and behavioural data in MatLab and python using custom made scripts. We have used Python MNE for preprocessing, standard python libraries including Scipy, Pandas and NumPy for data management and processing, python-matplotlib and seaborn for data

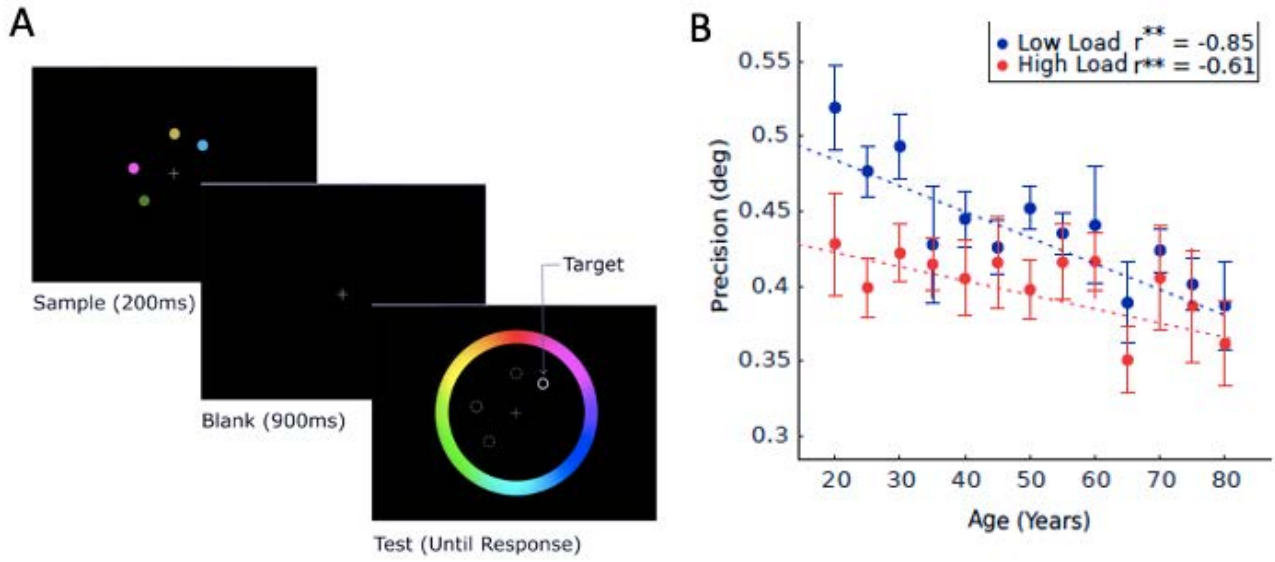

Figure 1. (A) Experimental design of the colour recall VSTM task and (B) Precision in Visual short-term working memory (VSTM) progressively declines with lifespan

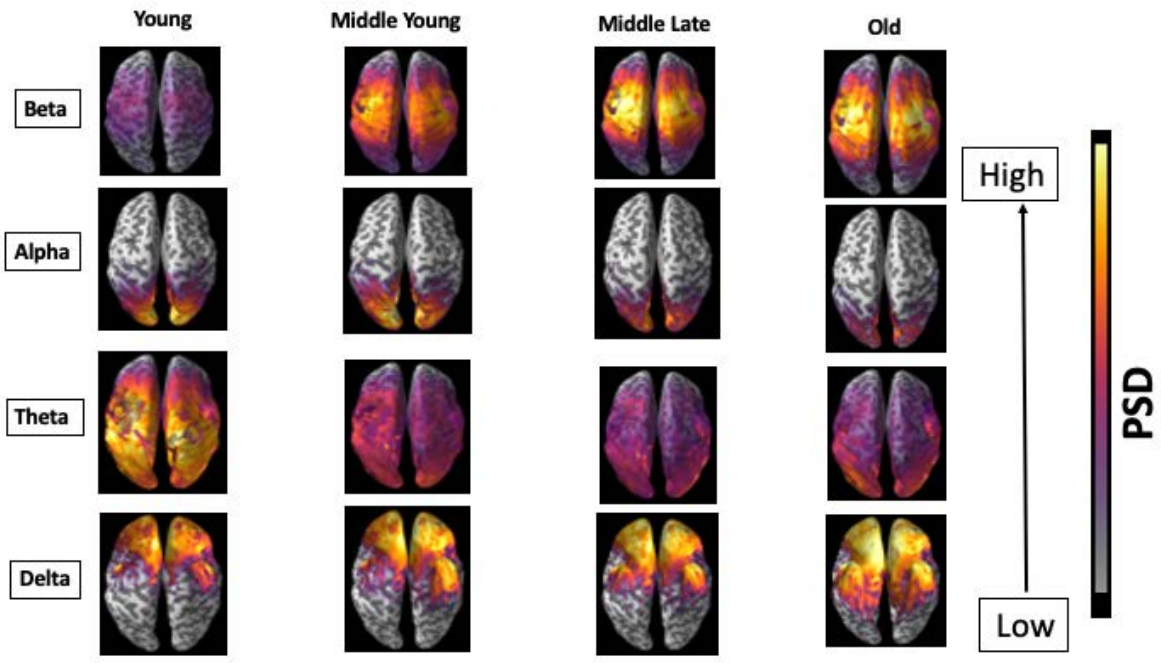

Figure 2. Age associated frequency band specific alterations in PSD across different stages of adult lifespan

visualisation in this study. For all the participants we have estimated Power Spectral Density (PSD) using Welch's periodogram method at the magnetometer sensors as well at the corresponding source level using Desikan Atlas. The results of healthy aging associated changes in PSD across distributed across cerebral cortex at different milestones of adult lifespan is displayed below (**Figure 2**)

Periodic and aperiodic features based on spontaneous MEG narrow and broad band activity: Using the *Fitting Oscillations and One Over F* known as (FOOF) model, we fitted the PSD estimated in the previous step and from the parametrization model fit, all the simulated Gaussian peaks were removed to analyse the background signal. Thereafter, the aperiodic component of the signal was fitted in the log-log space line from which 1/f Slope and offset were (**Figure 3**) extracted for each participant. Periodic features - Central Frequency (CF), Power (PW), Bandwidth (BW)- were estimated using peak parameters from the fitted model (refer to the materials and methods section). To check if the parameterisation using the simulated Fooof model can capture lifespan associated changes, we first simulated the model for young and old adults. The model well captured the established lifespan associated slowing down of Peak alpha frequency (PAF) (**Figure 3**). Original spectrum, aperiodic fit and full model are being depicted in Figure 3(B)(C) for YA and OA group respectively. Our results encapsulate the lifespan associated trend of slowing down of Peak alpha frequency (PAF), decrease in PSD (over the occipital lobe) and flattening of broadband global 1/f slope suggesting an increase with age and a concomitant decreases in the offset parameter with aging. These results suggest that the underlying change in the tissue properties probably affecting broadband criticality and long-range temporal correlations across brain regions.

The domain general and domain specific effect of age-associated aperiodic 1/f activity and oscillatory brain dynamics: In oscillatory dynamics, we observe a significant decline in PAF with age as shown by previous studies. The decrease in PAF was not found to be localised to any specific group of sensors or brain areas, rather a global significant decrease was observed. The speed of alpha is often associated with the speed of information processing therefore, higher alpha speed is needed for optimal performance in cognitive tasks and determine the temporal resolution of visual perceptual integration (Samaha et al., 2015). Figure below shows that the reaction time of the participants or speed of memory retrieval is well predicted by global alpha speed (Figure 4 summarizes all the findings). Hence, higher the speed of alpha, fast is the retrieval, and consequently lesser reaction time for younger adults. As aging is characterized by attentional difficulties, in particular a reduced capability to inhibit irrelevant information, therefore, alpha band power may have important role in determining how accurately older individual's recall the retain and recall memorized items.

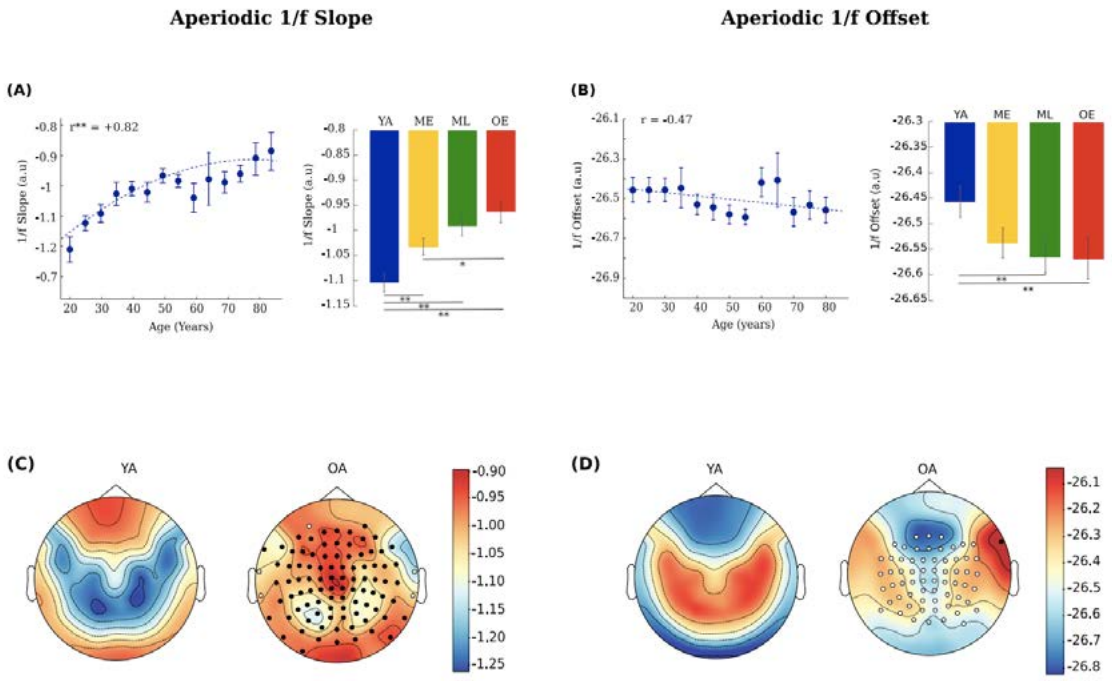

Figure 3. (A) Left: 1/f slope as a function of age. Right: 1/f slope for four age groups. (B) Left: 1/f Offset as a function of age. Right: 1/f Offset for four age groups. 'r' represents the correlation value. The dashed line represents a linear regression fit. Error bar denotes SEM. (C) and (D) Aperiodic 1/f slope and 1/f offset spatial topography for Young (YA) and Old (OA). Clusters of sensors with significant positive and negative differences in 1/fslope and 1/f offset between the OA and YA group are represented with black and white dots, respectively.

Peak frequency, Band-ratio relates to distinct aspects of memory processing

In summary, as Aging is accompanied by the changes in cognitive functions and age itself is a major risk factor for Alzheimer's Disease and other neurological conditions. Our study provides MEG 1/f aperiodic and periodic markers across the healthy adult lifespan and shows that different frequency bands and their spectral features (aperiodic and periodic component) mediate age-related changes across different brain regions, in multiple cognitive and metacognitive domains, which not only provides us with a better understanding of the aging process but also highlights key monitoring markers for management of cognitive impairments. A clear characterization of the association between baseline oscillatory component, 1/f activity, band ratio, healthy aging, and cognition, is established in this study.

Linking prediction errors with sequential Episodic memory processing and recall

Imagine that you see your favourite actor sitting in your chair while entering the office, much to your surprise. The memory of such an event would be harder to forget than other memories in the same office. Due to the low expectation of such events occurring within the given context, the substantial memory consolidation of this event displays the dependency of our day-to-day memories on the underlying context and predictive processes. Episodes or events being the canonical components of episodic memory are marked by a clear beginning and an end with temporal relations. As such, our memories are organized sequentially in contexts that evolve in time. However, whether and how unpredicted events can a#ect this temporal code of our experienced memories is something that surprisingly remains mostly unexplored.

Predictions are a hallmark of episodic memory recall. Therefore, whenever a context is re-experienced, the sequence of episodes is automatically predicted. Prediction errors (PEs) resulting from the violation of these predictions have been shown to influence declarative memory by strengthening incidental encoding, semantic memory acquisition, paired association learning and playing a role in reconsolidation. Despite its widespread effects on declarative memory, its role in episodic memories is only now starting to be uncovered.

In this recent work from our lab by Yazin et al., 2021 we have investigated how prediction errors strengthens or weakens previously consolidated memories time in the face of arrival new and novel sequential episodes.

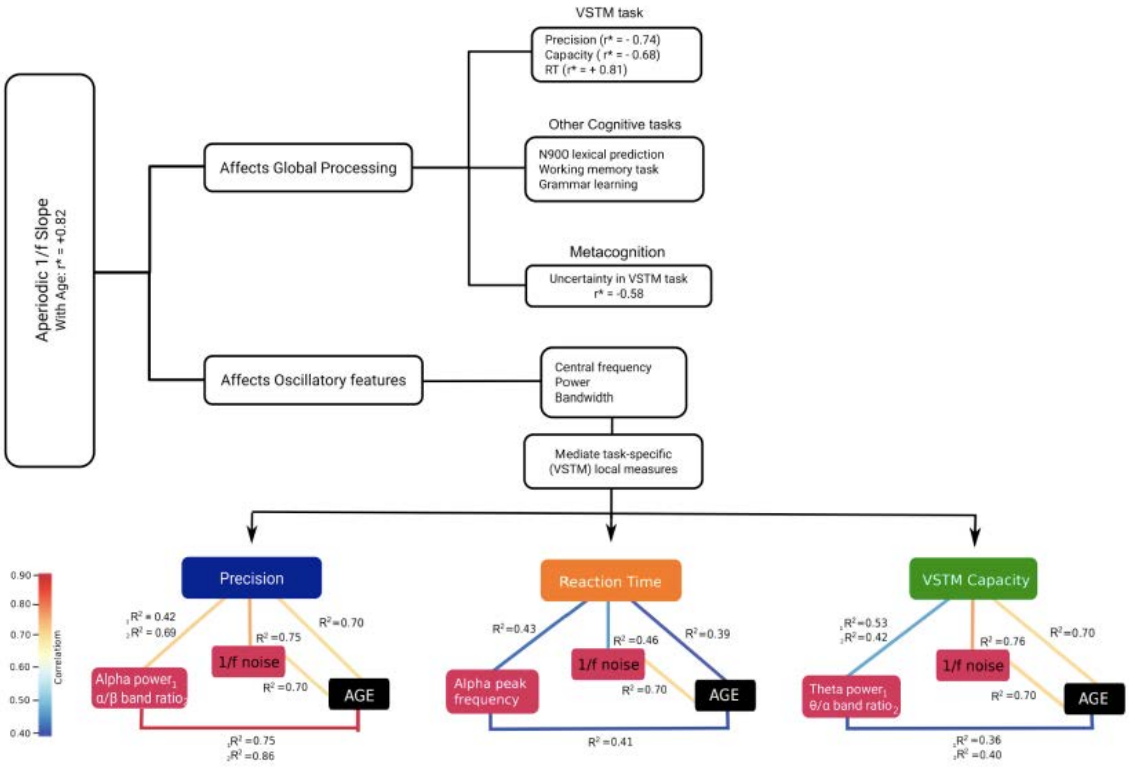

Figure 4. Aperiodic 1/f activity affects global processing and oscillatory features. Aperiodic 1/f slope increases globally which can be observed in performance in different cognitive tasks (affects global processing). It also significantly affects oscillatory features which are more task specific

Additionally, we have looked at Temporal Context Model (TCM) using Drift Diffusion model (DDM) and a multivariate Bayesian Regression model to tease out the effect temporal stability of memory and participant's response time and accuracy of memory recall based on the individual decision threshold.

Stimulus design and experimental paradigm:

We have employed a three-day experimental memory paradigm in this study test the the role of prediction errors to test the overall quality of sequential memory encoding and recall. Participants watched two movies (divided into several different events having multiple segments) on Day1. "The following day (Day2), they saw the same movies in either two conditions—Substitution and Addition. Substitution had another contextually fitting segment substituting a prior encoded segment, while in Addition, the omitted segment is viewed again (after the prediction error). A sequence memory task (2-AFC) of adjacent segments tested participants' temporal order memory for each event on Day3. (a) Example of an event seen on Day1. Each segment is 7s with a blank screen in between (not shown). (b) Day2 conditions. Substitution (top) where participants were predicting a segment (Old) seen the previous day (faded red dots) while the actual segment (New) is a different one which fits with the context. Addition (bottom) which has the Old (predicted) segment re-experienced (hence reactivated) after the new segment induces the prediction error. PrePE-Old temporal sequence memory is taken as old sequence and PrePE-New temporal sequence is taken as new sequence. (c) Schematic of Day3 Sequence memory test block. Each sequence was shown by displaying representative screenshots of those segments involved (in both normal and reverse order). Participants had to choose the correct order of the segments in the order they saw in the movie.

The effects on temporal order memory are specifically because of PEs and not due to novel associations: To truly verify that the memory effects are due to PEs but not other factors like novel associations, we compared the new sequence (PrePE-New) with the subsequent sequence, PostPE-New, which was termed as Novel sequence. The reasoning being, since both these memory associations are encoded on Day2, unless there was a specific effect of PEs, both would justifiably have similar memory encoding. Thus, we hypothesized since the memory strengthening can only be due to PEs, the new sequences would have significantly better memories over novel associations.

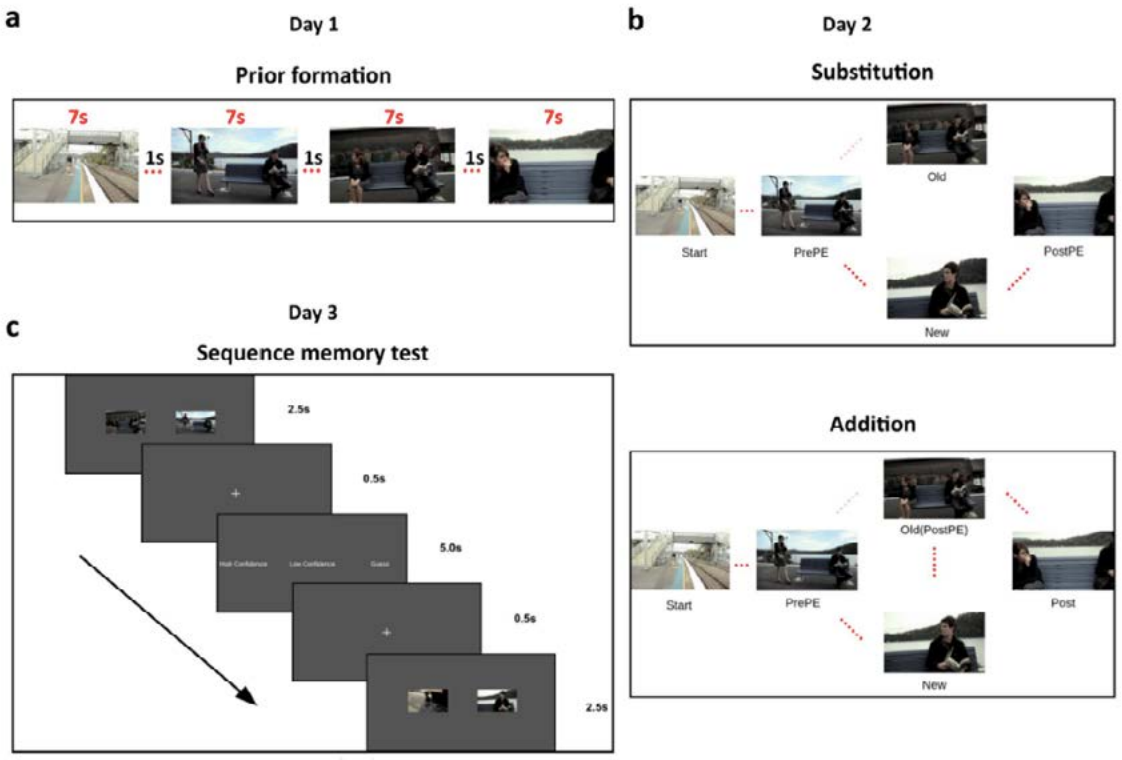

Figure 5: Experiment naturalistic stimulus paradigm to investigate role of prediction error and surprise in sequential episodic memory encoding and recall

In Substitution, there was a significant difference (t (23) = 2.12, p < 0.05, 95% CI [0.002, 0.214], BF = 1.41, d = 0.67) in accuracy between new sequence, (Mean = 58.76, SE = 0.03) compared to the Novel sequence (Mean = 47.88, SE = 0.03) (**Fig.5a**). This difference was significant in response times as well (t (23) = − 3.07, p = 0.0054, 95% CI [− 0.136, − 0.026], BF = 8.18, d = 0.44) between New Sequence (Mean RT = 1517ms, SE = 34ms) and the Novel Sequence (Mean RT = 1600&ms, SE = 41&ms) (**Fig.5b**). This shows that in Substitution, even though both the sequences were seen on the same day, there is a stark difference in memory performance specifically due to PEs and not seen in novel sequences. Next, we have replicated this results in the Addition condition.

Prediction errors reduce the decision threshold during sequence recall of newer memories: The behavioral findings suggested that the response times between New and Old sequences in both conditions show significant differences. To further understand this, we deployed a sequential sampling model to explain the results. In our experiment, we hypothesized that participants would recall the stored temporal sequence upon seeing the two images representing the sequence. In other words, the encoded sequence is reinstated to make the temporal order decision. Modelling the data using a Hierarchical DDM (**Fig.7**), an increased speed for the recall can be due to two reasons- increased drift rate, denoting a faster reinstatement or reduced decision threshold, suggesting a lowered requirement of evidence to decide the temporal order. In addition to the null model, we compared 4 other models. Thus, all the models had drift-rate and boundary set to vary with the Stimulus (Old and New memory sequences). Moreover, we also

assumed the participants' confidence response can also be due to the two parameters. A higher drift rate means the sequences arereinstated rapidly, giving a more subjective feeling of conviction than slowly reinstated ones. Conversely, it can be due to a lowered boundary threshold leading to less cautious response carrying with it more confidence. We adjudicated the models based on the Deviance Information Criteria (DIC) which penalizes more complex models. We set the non-decision time to vary by condition (Substitution and Addition) for all models. This was to know whether there would be any changes in the nondecisional process, owing to the reactivation involved in one.

The main model allowed both drift-rate and boundary parameters to vary with Stimulus. This allowed us to compare which of the two parameters demonstrated the empirical e#ect of PEs, which is the participant's reaction time in choosing the temporal order between

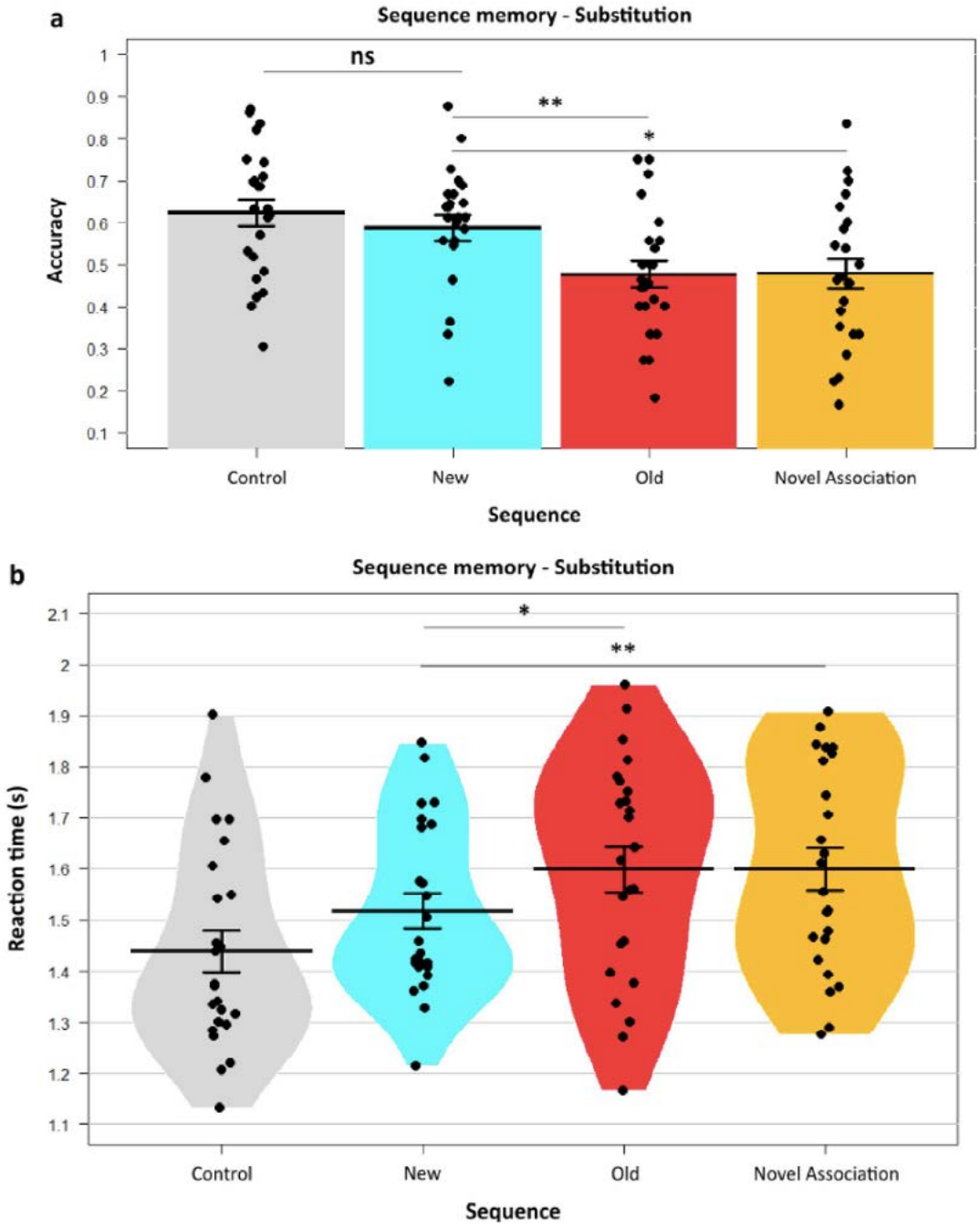

Figure 6. Effect of prediction errors on temporal order memory in substitution. In Substitution, New event is formed after a prediction error (surprise), when participants were expecting a previous sequence (Old). (a) Memory accuracy performance showing a significant difference between new and old sequence for temporal order judgement. No significant difference in accuracy between New and Control sequences was observed. (b) Reaction time data reflecting the main result of accuracy between new and old sequence. Dots represents participants' individual performance (n = 24). Error bars denote SEM

two segments. Drift-rate also varied across Confidence (High Confidence and Low Confidence).

In summary, we test the hypothesis that the contextual prediction errors would fundamentally alter the memorized sequence of events in declarative domain. Specifically, sequences that did not match predictions in a context would be weakened. Concurrently, the newly encoded sequences that were seen instead would be strengthened as a whole. From the perspective of predictive coding new surprising information that violates expectations drives stronger learning. These newly formed sequences would be strengthened over older encoded sequential information to minimize future errors. Our key finding is that contextual prediction

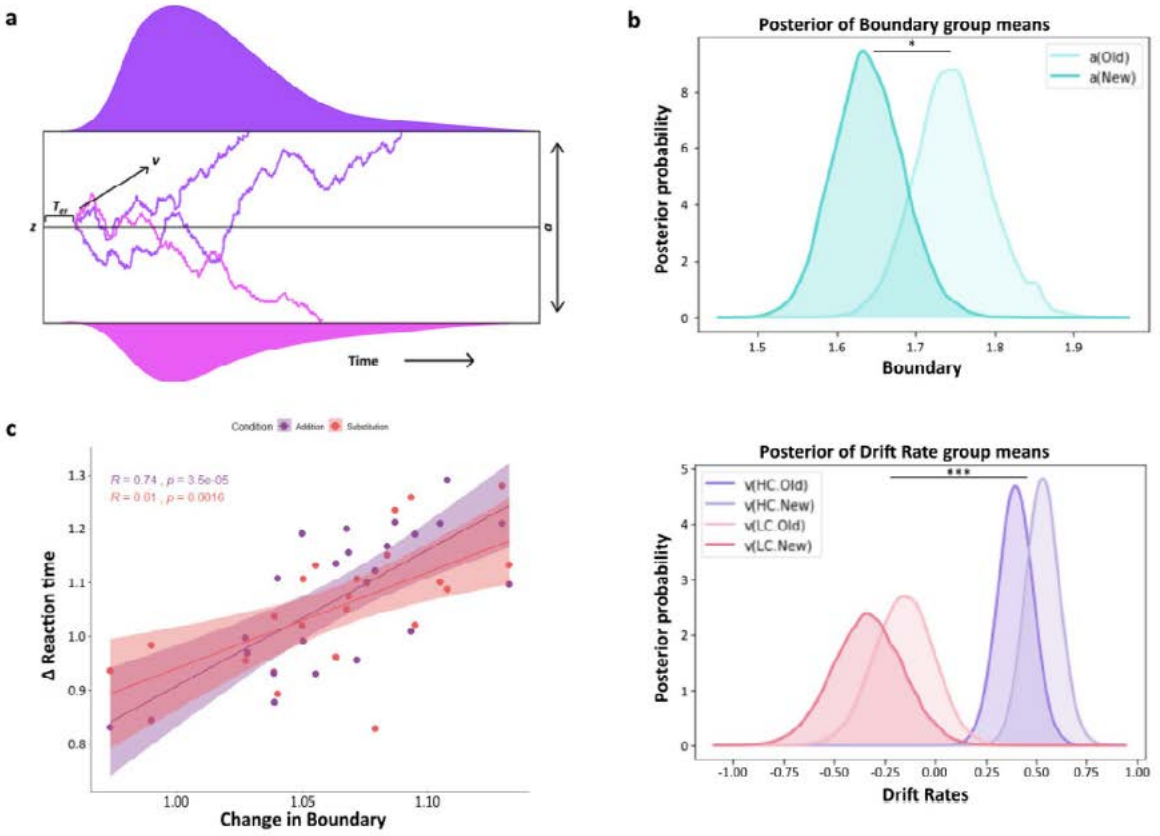

Figure 7. Hierarchical DDM results. (a) An illustration of the DDM. The drift rate v reflects the rate of noisy accumulation of evidence until it reaches either of the two boundaries separated by a parameter a. The process starts at point z, which may or may not have a response bias (not included in the main model) towards either boundary, which results in the model making the response choices. "e response times are a combination of the diffusion process and the non-decision time Ter, which includes no accumulation. Recalling sequences would reinstate the original encoded memories from which a decision is made. Response times for the temporal order judgements can be due to an increased drift rate or decreasedboundary threshold. (b) Posterior density plots of the group mean boundary parameter (top) showing higher evidence requirement for the Old sequences compared to New (p = 0.026). Posterior density plots of the group mean drift-rate parameter (bottom) showing no difference between the Old-and-New sequences but only between High and Low confidence responses (p < 0.001). (c) Subsequent adjustment in decision criteria made by the participants (boundary parameter) for recalling newer memories (compared to old ones) were significantly correlated with their relative change in reaction times in Addition (Purple) r = 0.74, p < .001 and Substitution (Pink) r = 0.61, p = .0016. Dots represent individual participants

errors strengthen the newer memory sequences in time while weakening the order of previously encoded sequences, thereby reorganizing encoded temporal memories. This enhanced performance, reflected by faster reaction times, on subsequent modelling showed that it results from a lower decision threshold while remembering, signifying a more automatic response for the newer sequences. Critically, even the re-exposure of mispredicted segments in an event, later, did not exempt it from getting weakened while recalling. Collectively our findings reveal how prediction errors play a crucial role in determining how episodic memories is organized in time.

Publications:

1. Yazin, F., Das, M., Banerjee, A., & Roy, D. (2021). Contextual prediction errors reorganize naturalistic episodic memories in time. *Scientific reports*, *11*(1), 1-17.

2. Ghosh, P., **Roy, D.**, & Banerjee, A. (2021). Psychophysical data to study the brain network mechanisms involved in reorienting attention to salient events during goal-directed visual discrimination and search tasks. *Data in Brief*, *36*, 107020.

3. **Roy, D.**, & Uddin, L. Q. (2021). Atypical core-periphery brain dynamics in autism. *Network Neuroscience*, *5*(2), 295-321.

4. Ghosh, P., **Roy, D.**, & Banerjee, A. (2021). Organization of directed functional connectivity among nodes of ventral attention network reveals the common network mechanisms underlying saliency processing across distinct spatial and spatio-temporal scales. *Neuroimage*, *231*, 117869.

5. Das, M., Singh, V., Uddin, L. Q., Banerjee, A., & **Roy, D.** (2021). Reconfiguration of Directed Functional Connectivity Among Neurocognitive Networks with Aging: Considering the Role of Thalamo-Cortical Interactions. *Cerebral Cortex*, *31*(4), 1970-1986.

6. Thuwal, K., Banerjee, A., & **Roy, D.** (2021). MEG Oscillatory and Aperiodic neural dynamics contribute to different cognitive aspects of short-term memory decline through the lifespan. *bioRxiv*.

7. Kumar, N., Jaiswal, A. K., **Roy, D.**, & Banerjee, A. (2021). Lateralized functional responses in the cortex arise from the dynamic interactions in the structural connectome. *bioRxiv*, 2020-10.

8. Kumar, V. G., Dutta, S., Talwar, S., **Roy, D.**, & Banerjee, A. (2020). Biophysical mechanisms governing large-scale brain network dynamics underlying individual-specific variability of perception. *European Journal of Neuroscience*, *52*(7), 3746-3762.

9. Sahoo, B., Pathak, A., Deco, G., Banerjee, A., & Roy, D. (2020). Lifespan associated global patterns of coherent neural communication. *Neuroimage*, *216*, 116824.

10. Ray, D., Hajare, N., **Roy, D.**, & Banerjee, A. (2020). Large-scale functional integration, rather than functional dissociation along dorsal and ventral streams, underlies visual perception and action. *Journal of cognitive neuroscience*, *32*(5), 847-861.

11. Gupta, D. S., Banerjee, A., **Roy, D.**, & Piras, F. (2020). Temporal Structure of Neural Processes Coupling Sensory, Motor and Cognitive Functions of the Brain. *Frontiers in Computational Neuroscience*, 14.

12. Triebkorn, P., Zimmermann, J., Stefanovski, L., **Roy, D.**, Solodkin, A., Jirsa, V., ... & Ritter, P. (2021). Identifying optimal working points of individual Virtual Brains: A large-scale brain network modelling study. *BioRxiv*.

Presentations:

1. Invited talk "Tracking cognitive brain dynamics through lifespan" Fifth Brain Mapping & AI workshop IIT Delhi 11-20th March 2021.

2. Invited talk "Tracking Structure-Function relationship and causal dynamics associated with lifespan" Global Neuroscience Partnership meeting NBRC, NIMHANS, and University of Iowa USA Organized by Sourav Banerjee, Ted Abel and Marco Hefti November 13, 2020.

3. Characterizing Variability of Audiovisual Speech Perception Based on Prestimulus Oscillatory Features of Electrophysiological Brain Signals Vinsea A V Singh,Vinodh G. Kumar , Arpan Banerjee, Dipanjan Roy Bernstien Conference 2020, Annual Meeting of the Bernstein Network in Computational Neuroscience, Germany. September 29- October 2, 2020; Virtual Mode.

4. Dynamic Repertoire of Brain During rest and task Nisha Shastry, Dipanjan Roy, Arpan Banerjee, Bernstien Conference 2020, Annual Meeting of the Bernstein Network in Computational Neuroscience, Germany. September 29- October 2, 2020; Virtual Mode.

Funding:

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1. Role of Default Mode Network in Cognitive functions BT/RLF/Re-entry/07/2014 Department of Biotechnology (DBT) Ramalingaswami Re-entry fellowship.

2. Oscillatory Network Dynamics in Perceptual Learning SR/CSRI/21/2016 Department of Science and Technology (DST) Initiated in 2017.

3. BT/MED-III/NBRC/Flagship/Program 2019 Government of India Ministry of Science and Technology, Department of Biotechnology (DBT).

4. NBRC core funding

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- Gustavo Deco Computational Neuroscience Group Instituci Catalana de la Recerc Estudis Avan ats (ICREA), Universitat Pompeu Fabra, Passeig Llus, Companys 23, Barcelona, 08010, Spain

Awards (if any):

NA

Degrees Awarded (Ph.D.):

Vinsea AV Singh (Master's Degree in Neuroscience)

Meetings/Conferences organized:

1. "Brain dynamics of interactions between cognition, emotion, and motivation" by Professor. Luiz Pessoa, Professor, Laboratory of Cognition & Emotion, Department of Psychology, University of Maryland, USA Wed, Nov 25, 2020 6:30 PM - 7:30 PM (IST) Host Faculty: Dr.Dipanjan Roy
2. "Brain dynamics and flexible behaviours: Insights from network neuroscience " by Dr. Lucina Q. Uddin, University of Miami, Department of Psychology, USA Tue, Oct 27, 2020 7:30 PM - 8:30 PM (IST) Host Faculty: Dr.Dipanjan Roy
3. "Mapping the spatiotemporal dynamics of hippocampal-cortical dynamics in health and Alzheimer's disease"- by Dr. Majid H. Mohajerani, assistant professor, the Canadian Centre for Behavioural Neuroscience, the University of Lethbridge, Tue, Dec 22, 2020 7:30 PM - 8:30 PM (IST) Host Faculty: Dr.Dipanjan Roy

Metabolic - Inflammation cross-talk in Glioblastoma: Implications in tumor progression

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Background and significance

Cancer cells reprogram their metabolism for sustaining the increased bioenergetic and biosynthetic demands. The altered cellular energy metabolism in tumor cells characterized by aerobic glycolysis or the "Warburg effect" is regarded as one of the hallmarks of cancer. Importantly, inflammation is an important contributing factor in cancer development, and a dynamic network of metabolic adaptations and inflammatory responses drives tumor progression. By playing critical roles in regulating epigenetics in cancer, metabolism-derived cofactors regulate the metabolism-epigenome axis. Glioblastoma multiforme (GBM) - the most malignant of brain cancers is largely refractory to current therapeutic regimens. The genetic, epigenetic and phenotypic heterogeneity in the same GBM tumor has detrimental implications for diagnostic and therapeutic approaches in the clinic. Our team is focused on understanding the mechanisms underlying deregulated cellular bioenergetics in an inflammatory glioma tumor microenvironment, and how different chromatin modifiers are engaged to govern the expression of genes associated with tumor proliferation and immune evasive responses. (i) Suppressor of cytokine signaling-1 (SOCS1) acts as an important regulator of cytokine responses, and functions as negative regulator of toll-like receptor (TLR) induced inflammatory signaling. As silencing of SOCS1 is concomitant with elevated TLR4 levels in glioblastoma, we investigated the effect of TLR4 inhibition on SOCS1 expression. Genetic manipulation of p53 indicated that SOCS1 expression upon TLR4 inhibition is dependent on p53 mutational status. Increased SOCS1 level was concomitant with diminished nucleosomal occupancy around p53-binding site on SOCS1 promoter. This altered nucleosomal landscape was accompanied by (i) diminished nuclear H3K9me3 and (ii) increased JMJD2A and Brg1 levels. JMJD2A inhibition or ectopic expression of ATPase deficient BRG1 prevented TAK-242 mediated increase in SOCS1 expression. Recruitment of Brg1-p53-JMJD2A complex on p53 binding sites of SOCS1 promoter upon TLR4 inhibition was concomitant with increased SOCS1 expression in p53-mutant cells. Taken together, p53 mutational status regulates transcriptional plasticity of SOCS1 promoter through differential recruitment of chromatin remodelers and epigenetic regulators in response to TLR4 inhibition. Our findings highlighting the involvement of p53 in TLR4 mediated regulation of tumor suppressor SOCS1 has important ramifications from a clinical perspective; as classification of gliomas based on p53 mutational status could determine responsiveness to anti-TLR4 therapy aimed at limiting inflammatory responses.

(ii) Lactate dehydrogenase A (LDHA) – involved in the conversion of pyruvate to lactate is highly expressed in tumor cells, and plays a critical role in tumor progression. Tumor-derived lactate also serves as pro-inflammatory mediator in facilitating tumor development. De-synchronized circadian rhythm in tumors is coincident with aberrant

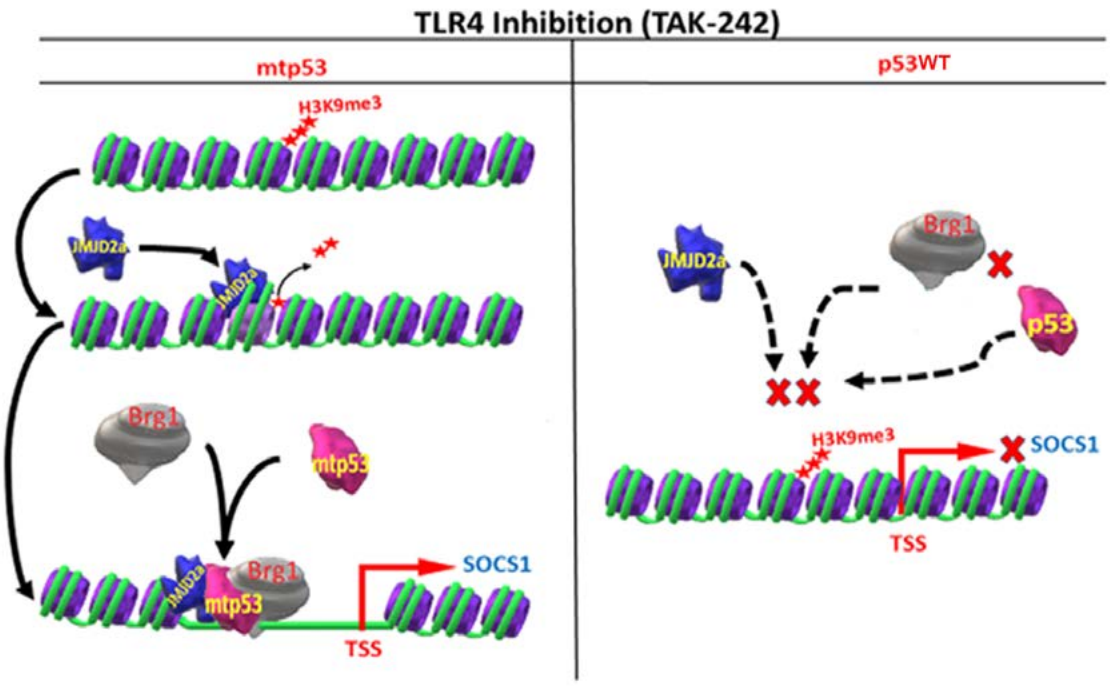

Figure 1. Model demonstrating the p53 affects epigenetic signature on SOCS1 promoter in response to TLR4 inhibition. High MNase accessibility, diminished H3K9 methylation coupled with increased enrichment of p53-Brg1-JMJD2A complex on SOCS1 promoter characterized its enhanced transcription in TAK242 treated p53-mutant glioma. Solid lines indicate increased interaction and dashed lines indicate decreased or no interaction in p53 mutant and wild type cells under TLR4 inhibition, respectively

inflammation, and dysregulated metabolism. Though epidemiological evidence implicates the association of circadian disruption with either dysregulated metabolism or aberrant inflammation in cancer, their inter-relationship in cancer etiology is largely unknown. We are therefore investigating the link between misaligned circadian rhythm, altered metabolic programming and aberrant inflammation in gliomas.

(iii) Somatic mutations in the isocitrate dehydrogenase 1 (IDH1) gene in glioma have been associated with better prognosis than those harboring wild-type IDH1. Importantly, IDH1 is increasingly being recognized as an independent prognostic marker in glioma as occurrence of IDH1 mutations (IDH1-MT) has been associated with improved clinical outcomes. Yes-associated protein 1 (YAP1) - a major mechano-transducer implicated in cancer progression, is at the hub of signaling network that serves as a major determinant of clinical aggressiveness and has strong correlation with patient survival. As TCGA dataset analysis revealed decreased YAP1 levels in IDH1MT patients as compared to those harboring IDH1wild-type, the role of YAP1 in conferring better prognostic value in IDH1 mutants is currently being investigated.

(iv) Abnormal tau phosphorylation is a pathological characteristic of traumatic brain injury (TBI), and inflammatory cytokine directly affects cellular tau level and its phosphorylated form. As altered brain metabolism in TBI is accompanied by dyregulated inflammation, we are investigating the possible link between inflammation mediated changes in metabolic regulators on tau phosphorylation.

Understanding the mechanisms leading to cytokine storms in SARS-COV2 infection

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (COVID-19) characterized by acute respiratory distress syndrome (ARDS) is associated with uncontrolled inflammatory responses. Cytokine storm triggered by SARS-CoV2 induces strong pro-inflammatory response in the lungs and hyper- inflammation is a characteristic feature of severe SARS-COV2 infection. As aberrant hyper-inflammation in severe COVID-19 is indicative of macrophage activation syndrome (MAS), we investigated whether the cytopathic effects of viral protein on lung cells could disrupt macrophage homeostasis leading to the exaggerated inflammatory responses and pulmonary sequelae that characterizes SARS-CoV2 infection. We demonstrate that SARS-CoV-2 viral proteins S-RBD- and Orf3a-induced extracellular release of

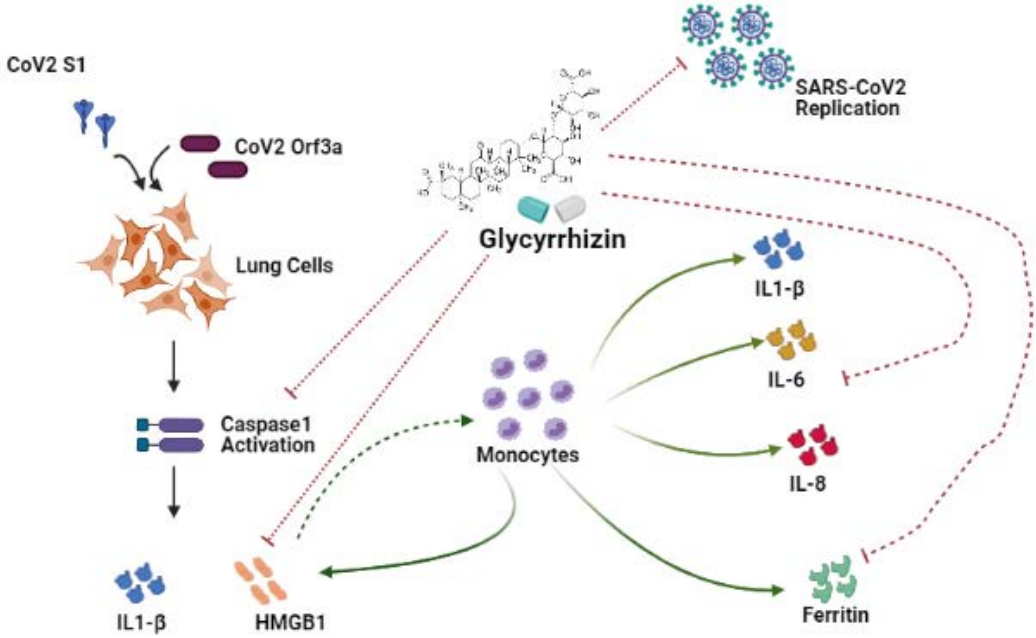

Figure 2. Schematic depiction of the multifaceted potential of Glycyrrhizin to inhibit viral replication and ameliorate host-inflammatory responses. SARS-CoV-2 viral proteins S-RBD and Orf3a-induced extracellular HMGB1 triggers pyroptosis in lung cells. HMGB1 inhibitor glycyrrhizin inhibits SARS-CoV-2 viral protein induced release of HMGB1, pro-inflamamtory cytokines and ferritin from macrophages. Glycyrrhizin also inhibits SARS-CoV-2 replication in Vero E6 cells

pro-inflammatory mediator High mobility group box 1 (HMGB1) triggers pyroptosis in lung cells. Treatment with HMGB1 inhibitor Glycyrrhizin dampened viral proteins triggered inflammation-mediated death in lung cells. The increase in release of pro-inflammatory cytokines as well as ferritin from macrophages cultured with conditioned media from SARS-CoV-2 S-RBD and 3a-transfected lung cells, was also rescued by glycyrrhizin. In addition to preventing the surge of cytokines and thereby protecting the lung epithelial cells, glycyrrhizin also attenuated replication of the virus. By preventing (i) SARS-CoV-2 viral protein induced inflammation mediated death in lung cells (ii) dampening inflammatory responses in macrophages and (iii) replication of SARS-CoV-2 virus; Glycyrrhizin serves both as anti-viral and anti-inflammatory.

Publications:

1. Gowda, P., Patrick, S., Joshi, S. D., Kumawat, R. K., & **Sen, E.** (2021). Repurposing Methotrexate in Dampening SARS-CoV2-S1-Mediated IL6 Expression: Lessons Learnt from Lung Cancer. *Inflammation*, 1-8.

2. Gowda, P., Lathoria, K., Sharma, S., Patrick, S., Umdor, S. B., & **Sen, E.** (2021). Rewiring of lactate-IL-1β auto-regulatory loop with Clock-Bmal1: A feed-forward circuit in glioma. *Molecular and Cellular Biology*, MCB-00449.

3. Gowda, P., Patrick, S., Joshi, S. D., Kumawat, R. K., **Sen, E.** Glycyrrhizin prevents SARS-CoV-2 S1 and Orf3a induced high mobility group box 1 (HMGB1) release and inhibits viral replication (2021). *Cytokine*.; 142: 155496.

4. Sheikh, T., **Sen, E.** p53 affects epigenetic signature on SOCS1 promoter in response to TLR4 inhibition (2021). ***Cytokine***.; 140:155418.

Presentations:

Funding:

1. Inflammation regulated metabolic reprogramming: Implications in tumor progression. Unit of excellence in cancer biology DBT. (#BT/MED/30/SP11016/2015)

2. Early diagnosis of structural and functional decline in brain circuits stemming from traumatic brain injuries in professional athletes playing contact sports. Ministry of Youth Affairs and Sports. (K-15015/42/2018/SP-V, February 2019)

3. NBRC core funds

Cellular and Molecular Mechanisms of HIV-1, Zika and SARS-CoV2 Virus Induced Neuropathogenesis

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Several viral outbreaks reported in the last two decades were associated with increasing incidence of neurological disorders, ultimately impacting human population worldwide. Microcephaly, caused by Zika virus, and neurological complications or ***Brain fog*** following SARS-CoV-2 infection in long-COVID cases have been of special interest to both basic and clinical researchers as the outcomes have affected large population. Furthermore, HIV-1-induced cognitive and motor deficits among AIDS survivors continue to be a challenge for all researchers, as the cure for HIV/AIDS patients remains at large.

One of the biggest challenges for studying neuropathogenesis is to develop physiologically relevant experimental models that represent the disease and offer insights into the cellular–molecular mechanisms of the disease pathogenesis. In this area, our laboratory has made significant breakthrough and developed a well-characterized model of primary cells of human brain derived neural stem cells (hNSCs). We routinely differentiate human astrocytes, neurons and oligodendrocytes from hNSCs. We continue to improve our existing models and develop new ones that can be used to study how viruses and their proteins affect properties and functions of human brain cells. In this direction, we have started using a recently developed model of human blood–brain barrier (BBB) comprising primary cultures of human brain microvascular endothelial cells and human astrocytes. The model is used for studying the effects of Zika virus proteins on BBB. Moreover, we studied the role of hypoxia on differentiating and fully differentiated oligodendrocytes. These studies are supported by extramural grants from the Department of Biotechnology, Government of India. We have extended the use of these models to understand how SARS-CoV-2 viral proteins cause neuronal damage. Detailed experiments are in progress to identify viral proteins that cause neuronal damage and discover the details of cell death pathways. We have made some novel discoveries that are being confirmed with more experiments and will be presented in the subsequent reports from our lab.

Although, there has been a gradual decrease both in new HIV-1 infections and severe cases of neuroAIDS, mild cognitive impairments and memory impairments are still being reported in AIDS patients, owing to HIV-1-induced neurodegeneration. As shown by us and other investigators, most of the neuronal damage is believed to be mediated through glial cells. We continue to study mechanisms of astrocyte-mediated neuronal damage. In the HIV-1 project, our research was focused on how mortalin levels in astrocytes modulate HIV-1 Tat-induced neuronal damage. Although Mortalin belongs to HSP70 family, it is heat uninducible protein encoded by nuclear gene-HSPA9B (GeneID-3313). Mortalin, also known as mitochondrial mortalin (mtHSP70), plays an important role in regulating mitochondrial biogenesis. It is a multifaceted protein that participates in the normal functioning of cell. Clinical samples of neurodegenerative patients have showed reduced expression of mortalin in the brain. Concurrently, our histopathological studies of the autopsied brain samples of HIV-1/AIDS patients revealed reduced expression of mortalin. Given the multifunction role of mortalin in maintaining the normal physiological condition of the cell, we examined the detailed signalling pathway of mortalin in HIV-1 Tat-induced neuropathogenesis.

HIV-1 Tat-transfected astrocytes significantly reduced the expression of mortalin both at mRNA and protein levels. Further, to understand the role

of mortalin in pathological condition, we overexpressed mortalin with HIV-1 Tat in astrocytes. This led us to some very interesting and unexpected observations. The overexpression of mortalin reduced the expression of HIV-1 Tat protein. Immunocytochemistry results revealed no cell death in co-transfected (with mortalin and HIV-1 Tat) astrocytes, whereas most of the astrocytes were HIV-1 Tat negative. Our bioinformatics and coimmunoprecipitation analysis suggest that HIV-1 Tat and mortalin interact with each other. The binding of mortalin with HIV-1 Tat efficiently drives Tat protein towards ubiquitin machinery followed by its degradation.

To understand whether mortalin-associated Tat degradation rescues the cell from Tat-mediated toxicity, mitochondrial morphology and functions were analysed. Our study revealed that co-transfection of mortalin with HIV-1 Tat significantly rescues mitochondrial membrane potential along with reduced mitochondrial fragmentation, as observed in HIV-1 transfected cells. Additionally, the overexpression of mortalin maintained cytochrome c oxidase levels, an essential component of the electron transport chain in cells. Together with this, ATP burst was found to be regulated by mortalin. Ultrastructural evaluation of mitochondria through electron microscope revealed intact mitochondrial cristae with laminar structure and tightly packed mitochondrial bands in co-transfected astrocytes. However, in HIV-1 Tat alone group, mitochondrial extensions were hardly visible and no clear cristae were visible.

In our experiments, overexpression of mortalin along with HIV-1 Tat in astrocytes reduced mitochondrial damage. This rescue explains the degradation of HIV-1 Tat in astrocytes. Astrocyte mitochondria are not under the influence of Tat in co-transfected cells. As glial activation plays an important role in inducing and releasing various neurotoxicants, we extended our study to examine astrogliosis. In co-transfected astrocytes, immunocytochemistry and western blot analysis revealed reduced GFAP expression. This suggests that co-transfected cells with mortalin and HIV-1 Tat do not cause any stress, instead maintain the normal physiological homeostasis in astrocytes.

Collectively, these results strongly advocate that mortalin is very critical for maintaining normal physiological conditions of astrocytes and rescuing astrocyte-mediated neuronal damage induced by HIV-1 Tat. Our study further emphasizes the therapeutic potential of mortalin as a novel target for designing therapies that could prevent astrocyte-mediated neuronal damage and astrogliosis in neurodegenerative diseases.

Publications

1. Priyanka, R. Wadhwa, R. Chaudhuri, TC. Nag, and **P. Seth** (2020)**.** Novel role of mortalin in attenuating HIV-1 Tat mediated astrogliosis. *Journal of Neuroinflammation* Sep 20;17(1):276-296. doi:10.1186/s12974-020-01912-3.

2. CMS Singal, P. Jaiswal and **P. Seth** (2020). SARS-CoV-2 more than a respiratory virus: Its potential role in neuropathogenesis. *ACS Chem Neuroscience* July 1;11(13):1887-1899. doi: 10.1021/acschemneuro.0c00251. Epub 2020 Jun 18.

3. IO. Saliu, R. Bhagat, OB Ojo, AC. Akinmoladun, MT. Olaleye, **P. Seth,** R. Velayudhan (2021). Reduction of anoxia-induced bioenergetic disturbance in astrocytes by methanol fruit extract of *Tetrapleura tetraptera* and *in silico* evaluation of the effects of its antioxidant constituents on excitotoxicity. *Toxicology Reports* January 8:264-276. doi.org/10.1016/j.toxrep.2021.01.015

4. A. Bhattacharya, V. Jha, K. Singhal, M. Fatima, D. Singh, G. Chaturvedi, D. Dholakia, R. Kutum, R. Pandey, TE. Bakken, **P. Seth**, B. Pillai, M. Mukerji (2021). Multiple Alu exonization in 3'UTR of a primate specific isoform of CYP20A1 creates a potential miRNA sponge. *Genome Biology and Evolution* Jan 7;13(1):evaa 233, 2021. doi: 10.1093/gbe/evaa233

5. SS. Raza, P. Seth, MA. Khan (2021). 'Primed' Mesenchymal Stem Cells: A Potential Novel Therapeutic for COVID19 Patients. *Stem Cell Reviews and Reports* 17: 153-162 Feb (2021) doi:10.1007/s12015-020-09999-0

6. R. Rajan, Divya KP, RM. Kandadai, VP. Satagopam, Madhusoodanan UK, P. Agarwal, N. Kumar, T. Ferreira, H. Kumar, S. Prasad Av, K. Shetty, S. Mehta, S. Desai, S. Kumar, Prashanth L K, M. Bhatt, P. Wadia, S. Ramalingam, G M Wali, S. Pandey, F. Bartusch, M. Hannussek, J. Krager, A. Kumar-Sreelatha, S. Grover, M. Sturm, P. Lichtner, J. Roeper, V. Busskamp, GR Chandak, JC. Schwamborn, **P. Seth**, T. Gasser, O. Riess, V. Goyal, PK. Pal, R. Borgohain, R. Krueger, A. Kishore and M. Sharma (2020). (Article type: Study Protocol). Genetic architecture of Parkinson's disease in the Indian population: Harnessing genetic diversity to address critical gaps in Parkinson's disease research. *Frontiers in Neurology (section Neurogenetics)* Jun; 11:524. doi: 10.3389/fneur.2020.00524. eCollection 2020.

7. B. Prajapati, M. Fatima, M. Fatma, P. Maddhesiya, H. Arora, **P. Seth** and S. Sinha (2020). Temporal transcriptome analysis of neuronal commitment reveals the preeminent role of the divergent lncRNA biotype and a critical candidate gene during differentiation. *Cell Death Discovery*. Apr 24; 6: 28.

doi: 10.1038/s41420-020-0263-6. eCollection 2020.

Presentations

1. **Guest Speaker,** *Brain Fog in COVID-19 patients*. During National Science Week, Indira Gandhi University, Meerpur, Harayana, India February 22, 2021.

2. **Invited Speaker,** *SARS-CoV2 is more than a respiratory virus-implications in COVID19 patients*. Jagrukta Abhiyan (General Awareness) on COVID-19, a national level online program organized by National Academy of Sciences (India), January 12, 2021.

3. **Speaker,** *How viruses affect human brain?* NBRC Foundation day talk series, National Brain Research Centre, Manesar (Gurgaon), December 13, 2020.

4. **Invited Speaker,** *SARS CoV2 is more than a respiratory virus, its consequences on Brain,* Motilal Nehru National Institute of Technology, Allahabad, and Indian Young Academy of Sciences (INYAS), New Delhi, India, November 7, 2020.

5. **Invited Speaker,** *COVID19 and Brain connection,* at the 38th Annual meeting of Indian Academy of Neurosciences, organized by University of Hyderabad, Hyderabad, during October 4-7th, 2020.

6. **Invited Speaker,** *How do viruses affect the human brain?* During the 150 years celebrations of the Rawenshaw University, Odhisha, Health and Disease: Contemporary concerns, September 12, 2020.

7. **Invited Speaker,** *SARS-CoV-2 is more than a respiratory virus -its potential in neuropathogenesis in COVID19 patients,* at the Bilateral Indo-US Webinar on COVID Biology jointly organized by IISER-Kolkata, IISC-Bangalore, University of Pennsylvania, USA and University of Colorado, USA, on August 17, 2020.

8. **Symposia Speaker,** *Viruses make Friends Turn Foe: implications in neuropathogenesis following HIV and Zika infections,* Monsoon Brain meeting organized jointly by Indian Institute of Science (IISc), Bangalore and IIT- Kanpur, during June 24-26, 2020.

9. **Invited Speaker,** *Molecular Mechanisms used by viruses to affect human brain,* at Webinar which was part of the Lecture Series on contemporary issues in biosciences organized by School of Life Sciences, Mahatma Gandhi Central University (MGCU), Motihari, Bihar, India June 15, 2020.

10. **Guest Faculty,** *Research methodologies for beginners* as a Webinar for an Online workshop organized by the Department of Biotechnology, Maharishi Dayanand University, Rohtak, Haryana, India May 5, 2020.

Funding

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S. Singh, BHU, Varanasi, India.

S. Sen and P. Chattopadhyay, AIIMS, New Delhi, India.

C. Mukhopadhyay, Jawaharlal Nehru University, New Delhi, India.

M. Sharma, University of Tubingen, Germany.

R. Wadhwa, AIST, Japan.

A. Nath, D. Wang, National Institutes of Health, Bethesda, USA.

Awards/Honors

- **Elected Council Member** - Asia Pacific Society for Neurochemistry (APSN), an international society of countries in Asia Pacific, 2020-2024
- **Elected Fellow** - the National Academy of Medical Sciences (NAMS) (2020)
- **Appointed Senior Editor** – American Society of Neurochemistry – Neuro (2020)

Degrees Awarded (M.Sc.):

Misha Parmar

Degrees Awarded (Ph.D.):

None

Translational and Computational Neurosciences

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Scientist VII/ Senior Professor

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Ms Khushboo Punjabi (Ph.D student)

Ms. Shallu Joon (Neuropsychologist)

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Mr. Saurav Roy (R&D Engineer)

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Ms. Palak Handa

Dr. Yashika Arora

Ms. Komal

Overview

The research at NeuroImaging and NeuroSpectroscopy (NINS) Laboratory is focused on the identification of early diagnostic markers for Alzheimer's disease (AD) using noninvasive imaging modalities, including magnetic resonance imaging (MRI), MR spectroscopy (MRS), functional MRI (fMRI), susceptibility-weighted imaging (SWI), and magnetoencephalography (MEG). For AD-associated therapeutic advancements, causal molecular processes, which transform healthy brain to diseased condition, should be thoroughly understood. The study groups consisting of normal healthy control (HC), those with mild cognitive impairment (MCI), and those with AD are used for the assessment of following features:

- Glutathione (GSH) levels
- Brain iron quantitation
- Working memory performance
- *Visuospatial perception*
- Retinal imaging

Neuropsychological test scores for *cognitive reserve*

The major focus of research at our laboratory is the assessment of these neurochemical levels and functional performance in quantitative terms and its correlation with disease progression. Additionally, NINS lab emphasizes on the development of tools and platforms to aid in the processing of neuroimaging data.

Major Projects

PRATEEK: Integration of multimodal neuroimaging data to facilitate advanced brain research

In vivo neuroimaging modalities, such as MRI, fMRI, MEG, MRS, and quantitative susceptibility mapping (QSM), are useful in understanding brain anatomical structure, functional activity, source localization, neurochemical profiling, and tissue susceptibility, respectively. A multimodal data integration scheme has been developed to integrate the outcomes from multiple neuroimaging data (namely fMRI, MEG, MRS, and QSM) spatially. Furthermore, the entire scheme has been incorporated into a user-friendly toolbox PRATEEK—a unique novel state-of-the-art package to generalize and integrate imaging data received from multiple neuroimaging techniques. Integrating unique and distinct information from these neuroimaging modalities will further help to enhance the understanding of complex neurological disease. The proposed methodology and toolbox have been tested for viability among 14 healthy young participants for bilateral occipital cortices as the region of interest. This scheme can also be extended to other anatomical regions of interest. Overlap percentage from each combination of two modalities (fMRI–MRS, MEG–MRS, fMRI–QSM,

and fMRI–MEG) has been computed and qualitatively assessed for combinations of the three (MEG–MRS–QSM) and four (fMRI–MEG–MRS–QSM) modalities. This user-friendly toolbox minimizes the need of an expertise in handling different neuroimaging tools for processing and analyzing multimodal data. This easy-to-use package is beneficial for hospitals, laboratories, and clinical research.

Development of neuro-tool "SWADESH" by integration of various modules for early diagnosis of AD

This project aims to develop an advanced automated neuro-tool that aids in the early diagnosis of AD for better patient management and improved decision-making outcomes. This disease-predictive system will be accomplished with data quality-check module GANGOTRI, data processing module KALPANA, and statistical analysis module NINS-STAT, and machine learning-based comprehensive package GAURI. Eventually, the system would substantially improve the quality of life of patients and help in curbing the increased burden of mental illness on the nation by early detection and medical intervention.

Clinical trial for AD

Affecting millions of people worldwide, AD is a devastating disorder with its fundamental cause remaining unknown so far. Considering the disease pathophysiology, a normal healthy person initially develops MCI phase, which eventually progresses to AD. However, based on recent discoveries, oxidative stress also seems to be involved in the development of AD due to impaired oxidative defense system. In such patients, the hippocampal area reveals significant depletion of antioxidant GSH. This upcoming double-blinded clinical trial will involve patients from AIIMS, Delhi. GSH supplementation in patients with MCI will be tested, and mental health will be regularly monitored. The proposed trial will be completed in 24 months. With this trial, the research will likely open up avenues of possible therapeutic development for preventing AD. Moreover, a trial for dietary GSH supplementation is being planned. The relevant discoveries and findings would help sustain the quality of life of patients suffering from neurodegenerative disorders, such as AD. NBRC aims to translate these clinical results with the help of supportive in-house advanced technological platforms, namely KALPANA, BRAHMA, and BHARAT into better treatments for mental health patients.

NINS-STAT: An automated statistical package for data analysis

The success of the research depends on the use of appropriate prior study design, execution and statistical test for proper sample size to address the objectives of the study. There are many statistical tests of significance that are used in different conditions. Each test is specific to a particular situation under certain set of assumptions. Most frequent problem encountered by a researcher is to select appropriate statistical test as per the data and study objectives. Further after the appropriate choice of statistical test different software are used for analysis. Each software package has its own data requirement format, frequency of user inputs and requires prior statistical knowledge of the user. To address these difficulties and ease to use, a modality for statistical analysis to facilitate the researcher in deciding which test is appropriate for respective data is developed. We

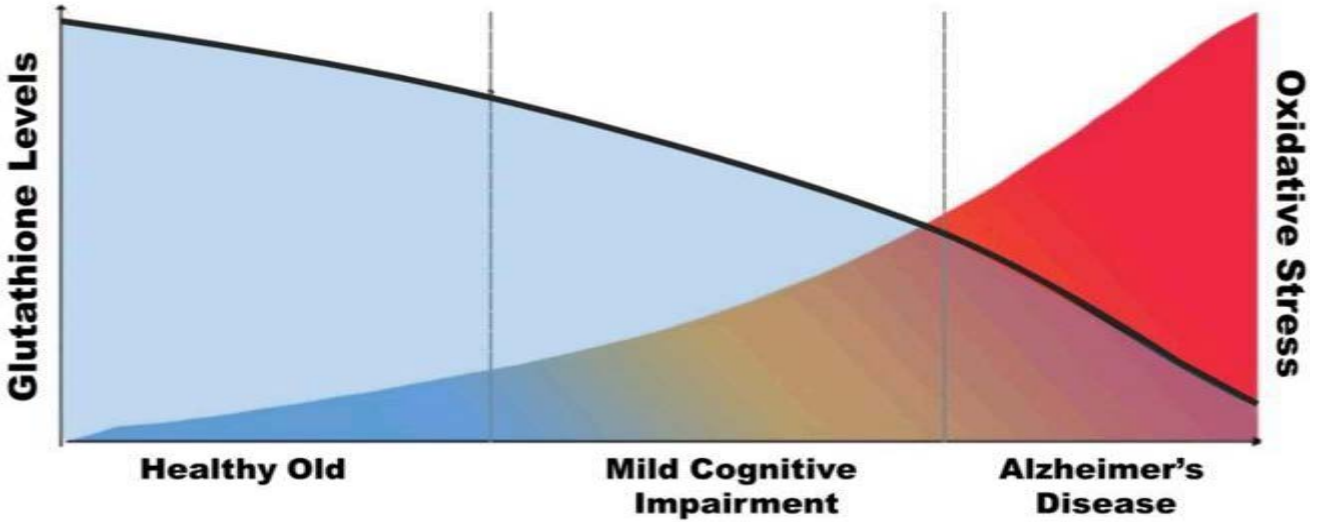

Figure 1: GSH change in AD pathogenesis

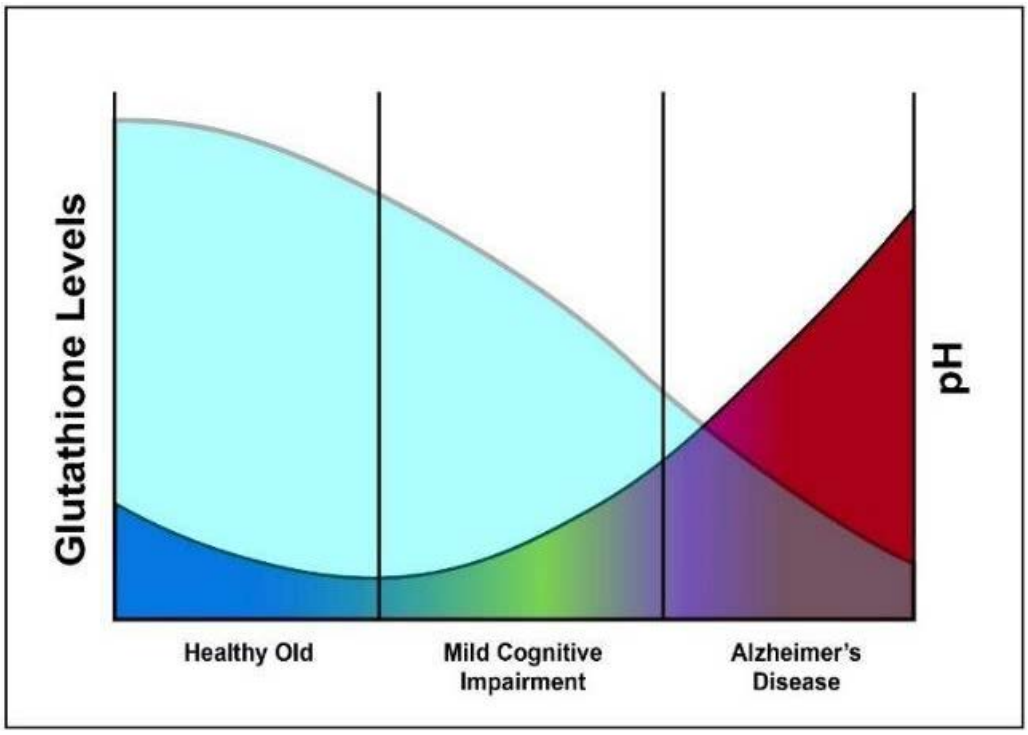

Figure 2: Schematic representation illustrating the relationship between GSH (blue) and pH (maroon) concentration levels in healthy old people, MCI, and AD

developed a Matlab-based automated software package NINS-STAT also helps in identification of the appropriate statistical test for hypothesis testing according to the study design, objectives, and data provided. NINS-STAT with a user-friendly GUI to facilitate the user to execute an automatic statistical analysis based on the study design and study objectives without prior experience of undertaking analysis with ease-of-use approach. The results have been authenticated and verified on wide range of datasets covering major study designs and objectives.

Efforts for COVID-19 Research

Brain Stress Mapping in COVID-19 Survivors Using MR Spectroscopy: New Avenue of Mental Health Status Monitoring

Coronavirus (COVID-19) has emerged as a human catastrophe worldwide, and it has impacted human life more detrimentally than the combined effect of World Wars I and II. Various research studies reported that the disease is not confined to the respiratory system but also leads to neurological and neuropsychiatric disorders suggesting that the virus is potent to affect the central nervous system (CNS). Moreover, the damage to CNS may continue to rise even after the COVID-19 infection subsides which may further induce a long-term impact on the brain, resulting in cognitive impairment. Neuroimaging techniques is the ideal platform to detect and quantify pathological manifestations in the brain of COVID-19 survivors. In this context, a scheme based on structural, spectroscopic, and behavioral studies could be executed to monitor the gradual changes in the brain non-invasively due to COVID-19 which may further help in quantifying the impact of COVID-19 on the mental health of the survivors. Extensive research is required in this direction for identifying the mechanism and implications of COVID-19 in the brain. Cohort studies are urgently required for monitoring the effects of this pandemic on individuals of various subtypes longitudinally.

Brain stress monitoring in COVID-19 survivors: a national program on mental health for early prediction

SARS-CoV-2 has created a global human catastrophe. With adverse impact on human body, coronavirus disease 2019 (COVID-19) has multiple impacts on mental health of patients. COVID-19-associated psychiatric disorders include anxiety, major depressive disorder, post-traumatic stress disorder, and obsessive-compulsive disorder, which have been reported in patients suffering and/or recovered from COVID-19.

NBRC scientists have developed a non-invasive imaging tool for humans using 3T MRI, which measures the levels

of stress (GSH) and neurotransmitters (e.g., GABA). Outcome of this clinical research can be correlated with various neuropsychological testing scores for psychiatric and cognitive functions. All necessary technology and schemes are in place and ready to be deployed.

Brain Imaging in COVID-19

Considering the neurological and neuropsychiatric manifestations of coronavirus disease 2019 (COVID-19), its early diagnosis is crucial. This viewpoint aims to highlight these manifestations through multimodal neuroimaging studies reflecting neurochemical and structural impairment.

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2. Shukla, D., Mandal, P. K., Mishra, R., Punjabi, K., Dwivedi, D., Tripathi, M., Badhautia, V., & **Mandal, P. K.** (2021) Hippocampal Glutathione Depletion and pH Increment in Alzheimer's Disease: An in vivo MRS Study. *Journal of Alzheimer's Disease*, 84(3): 1-14.

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6. Samkaria, A., Punjabi, K., Sharma, S., Joon, S., Sandal, K., Dasgupta, T., ... & **Mandal, P. K**. (2021). Brain Stress Mapping in COVID-19 Survivors Using MR Spectroscopy: New Avenue of Mental Health Status Monitoring. *Journal of Alzheimer's Disease*, (Preprint), 1-8.

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5. Dr. Joseph C. Glorioso, PhD University of Pittsburgh,

Patents

1. **KALPANA**: A comprehensive MATLAB based package for MRS signal processing

International Patent Application no: PCT/IB2016/054978 (submitted for USA, Europe and home country- India), dated 19th August, 2016.

Indian National Patent Application No: 201611001944, dated 19th January, 2016.

Funding: Ministry of Electronics and Information Technology (MEITY)

Role: Principal Inventor

Description: It is an integrative platform for visualization, preprocessing and quantitation of MRS data acquired using single voxel, multi voxel magnetic resonance spectroscopy imaging (MRSI) and MEshcher-GArwood Point-RESolved Spectroscopy (MEGA-PRESS) acquisition methods. The method integrates both time- and frequency-domain signal processing methods on a single platform. The method is optimized for proton (1H) and phosphorous (31P) MRS data. It employs the use of iterative baseline estimation and fitting procedure to provide improved quantitation accuracy. The method can be used in both interactive and automatic mode to cater to the needs of researchers and clinicians.

2. **A method and a system using multi-modal neuroimaging for early diagnosis of Alzheimer's Disease**

National Patent Application No.: 201911019667 dated 17th May, 2019

Funding: Ministry of Electronics and Information Technology (MEITY)

Role: Principal Inventor

Update: International Patent Filling under progress

Description: The present invention relates to a method and a system for early predictive diagnosis of Alzheimer's disease (AD) by integrating multi-modal information derived from non-invasive imaging techniques with the use of statistical and machine learning methods. The present invention aims to assess and classify an unknown case based on the learned disease specific information on the anatomical, functional, neurochemical and behavioral changes. The said information can be obtained from one or more modalities but not restricted to the Magnetic Resonance Imaging (MRI), Magnetic Resonance Spectroscopy (MRS) and Neuropsychological test scores. More particularly, the present invention relates to the inclusion of neuro-metabolites obtained from MRS along with the associative structural and/or functional changes, to ensure improved diagnostic accuracy as the disease specific changes in neurochemicals are reflected at much early stage than they can be observed in the anatomical, functional or other imaging techniques. The present method and system

further include the data integrator for multi-modal imaging and/or behavioral information which are passed to the dedicated statistical and classification analysis platforms and/or for disease specific baseline estimation using multi-modal biomarker(s)/ feature(s) for early diagnosis and prediction of the disease. Present invention comprises of different classifier model(s) and/or deep learning based on neural network to provide appreciable distinction of the selected disease groups which further incorporates statistical significance and confidence interval level on the diagnostic accuracy of the disease. The derived information from the classifier model generated from multi-modal information as input can be used for finding sensitive biomarkers of the respective disease, which further can be used for diagnosing the initial stages of diseases such as mild cognitive impairment (MCI) in case of AD pathology but not restricted to thereof.

Alteration of nucleosomal histone protein acetylation in RML-Scrapie prion infected mouse brain

Prof. Ranjit Kumar Giri

Principal Investigator:

Ranjit Kumar Giri, Ph.D.

Lab. Attendant:

Lalit Bidla

Prion diseases (PD) are a group of irreversible fatal neurodegenerative disorders. It comprises Kuru, Cruetzfeldt-Jakob Disease (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS) and fatal familial insomnia (FFI) in humans; scrapie in sheep and goat, and bovine spongiform encephalopathy (BSE) in cattle. The hallmark neuropathology of prion disease is the post-translational conversion of normal cellular prion protein (PrP^{C}) to pathological, infectious and alternatively folded isoforms (PrP^{Sc}). Infectivity associated with PrP^{Sc} makes this a unique neurodegenerative disease. Astrogliosis, microgliosis, neuronal cell death followed by spongiform degeneration of brain are other neuropathologies of prion disease. However, very little is known about the mechanisms by which PrP^{Sc} mediates PD-associated pathology. Genes which are silenced in healthy and functional neurons but active in cell cycle, are re-activated in various neurodegenerative diseases suggesting the alteration of normal chromatin architecture is a prerequisite condition for neuropathology in prion disease. The gene expression programs governing cellular proliferation, differentiation and cell death involve multiple epigenetic changes beyond the level of transcription factor recruitment. Major epigenetic mechanisms include DNA methylation and nucleosomal histone proteins modification. The acetylation and deacetylation of core histones on chromatin are most important histone modifications and are essential for biological processes, including proliferation, differentiation and gene activation or silencing. Such events are not reported in PD. Therefore, understanding the alteration of histone acetylation and its effect on chromatin might correlate the cellular pathology of various mature brain cells in prion disease. In order to understand this core biological process in prion disease we developed a mouse model of prion disease.

Ten (10) C57BL6/J mice were injected intracerebrally with RML scrapie mouse prions and allowed to manifest the disease completely. All animals were monitored regularly and pathological symptoms were recorded. Animals during advanced stage show plastic tail, circling and ataxic phenotype, typical to prion disease. Brains from terminally sick animals were harvested and frozen immediately in liquid nitrogen. Brain sections or brain homogenates were obtained. Similar samples were also obtained from age matched control mice. Brain histoblots were obtained from brain sections, and western blots were obtained from brain lysates and were immuno-blotted with anti-PrP antibody. Our results clearly demonstrated the presence of Proteinase-K resistant PrP^{Sc} only in RML scrapie prion infected mice brain but not at all in normal mice brain (Figure 1A and B respectively). Brain histoblot results not only demonstrated the presence of PK-resistant prion protein in brain but also indicated differential spatial prion accumulation in brain. Maximum accumulation was seen in thalamus followed by cortex and hippocampus, and cerebellum and other parts of the brain (Figure 1A). Furthermore, mouse which were infected with RML scrapie prion, exhibit increased GFAP expression throughout the brain than normal

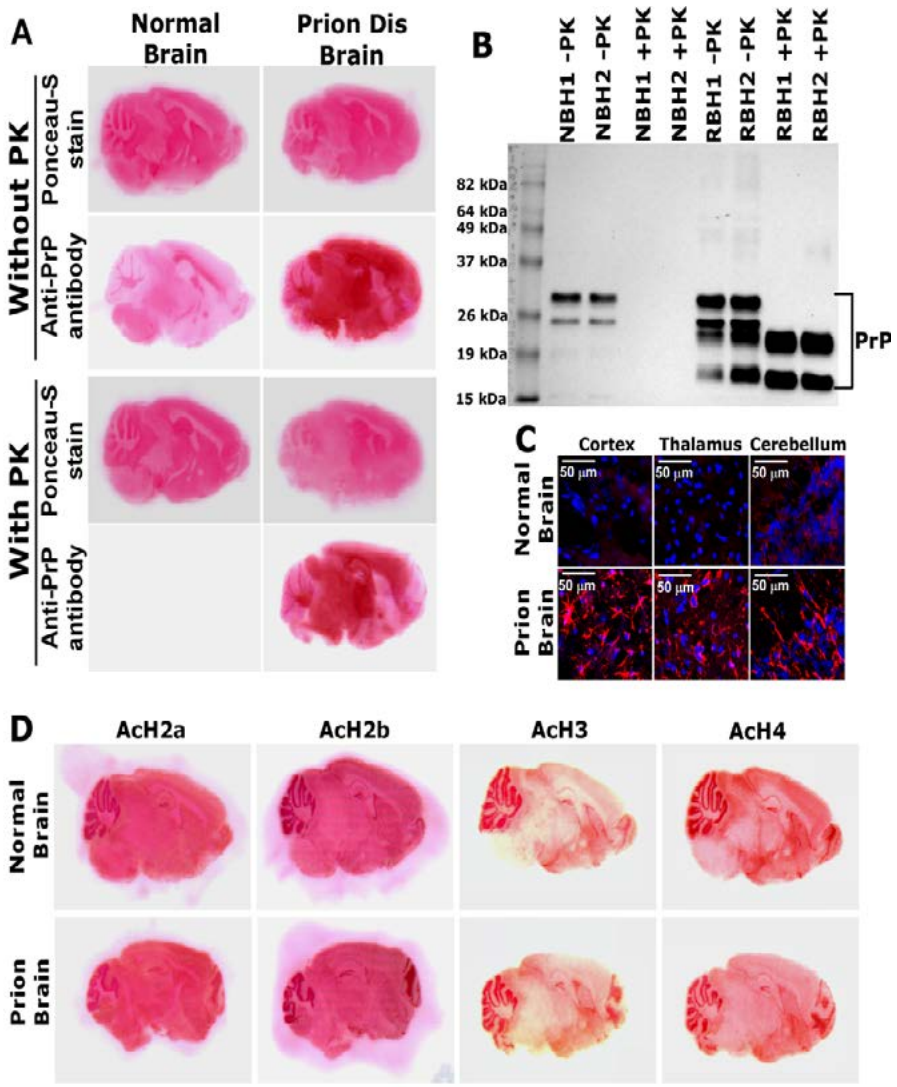

Figure 1: Alteration of nucleosomal histone acetylation in RML-Scrapie infected prion disease in mouse brain. A) Histoblot analysis of proteinase-K (PK) resistant prion accumulation in diseased brain but not in normal mouse brain. Cortex, hippocampus, thalamus, mid brain and granule layer of cerebellum exhibit higher accumulation than other brain region. B) Western blot analysis of characteristic gel electrophoresis pattern of PK-resistant PrPSc protein only in diseased brain than normal brain. C) Immunofluorohistochemistry analysis of GFAP expression exhibit reactive astrogliosis in diseased brain than normal mouse brain. D) Immunohistoblot analysis of nucleosomal histone protein exhibit remarkable decrease in H3 and H4 histone acetylation level in RML Scrapie infected prion disease than normal mouse brain. Mild decrease in H2a and H2b histone protein is also seen in diseased brain than normal brain

mouse brain, suggesting activated astrogliosis, which is an important neuropathology of prion disease.

Once prion disease was established, level of nucleosomal histone acetylation, was studied using immunohistoblot technique. This simple technique not only exhibit the changes in the level of histone acetylation but also its involvement in specific brain area of current mouse model of prion disease. Results from brain histoblots, immunoblotted with anti-acetylated H2a, H2b, H3 and H4 histone antibodies strongly demonstrated remarkable hypoacetylation of H3 and H4 histone proteins in RML scrapie infected C57BL/6J mice brain. In addition, maximum deacetylation was seen in cortex, hippocampus, thalamus and internal granule cell layer of cerebellum. However, H2a and H2b histone acetylation level show mild decrease in prion diseased mouse brain than normal counterpart. Collectively, our results for the first time suggest chromatin remodeling could be one of the main molecular processes behind cellular pathology seen in prion disease.

Funding:

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Effects of a small peptide on spatial memory and amyloid plaque load in an animal model of Alzheimer's disease

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The focus of my laboratory has been to understand how memories are formed in the brain, and to examine the processes involved in the memory impairment condition, the Alzheimer's disease (AD). One of the fundamental features of the brain is to make memories, and retrieve them when needed. We know that memories play critical roles in our proper day-to-day functioning. Thus, researchers have directed significant efforts towards understanding how memories are formed in the brain. In my laboratory, we use a combination of approaches to examine the processes involved in memory formation. This multidisciplinary approach consists of molecular analyses, electrophysiological recording, and behavioral studies. This is driven by the fact that a multidisciplinary approach is better suited to understand mechanisms of memory formation than a single approach. The other aspect of our research involves the most common memory impairment condition, the Alzheimer's disease. This is an age-associated disease that robs the patients of memory and other cognitive abilities. It is devastating not only to the individual who develops this condition, but also to the caregivers and family members and the society. Amyloid beta is considered to be the primary causative agent in AD. This small peptide, among several effects, causes neuronal cell death. Thus, significant research is devoted to understand the mechanisms of amyloid beta-induced neuronal cell death, and how to prevent it. My laboratory has focused on identification of compounds that can prevent neuronal cell death by amyloid beta. In addition, we have tried to understand how amyloid beta affects the signaling molecules that play critical roles in synaptic plasticity and memory.

Previously, I have presented our work on mechanisms of memory formation, and on AD. In continuation with our work on AD, this year, I will discuss the effects of a small peptide on spatial memory and amyloid plaque burden in an animal model of AD. The animal models have been tremendously useful in elucidating the mechanisms involved in various disease conditions. These models have also been useful in evaluating the therapeutic potential of various agents. Amyloid beta is produced by the proteolytic processing of a larger protein, the amyloid precursor protein. This processing is carried out by gamma secretase complex. Presenilin 1 is a part of the gamma secretase. A commonly used model for AD is the double transgenic animal which expresses mutant forms of amyloid beta and presenilin 1. These mutations lead to higher production of amyloid beta. Similar to human patients, these animals develop amyloid plaques, and display impairment in memory. Since these animals show features of AD, they have been used extensively to understand disease mechanisms, and also to examine the effects of compounds on amyloid plaque load and other features.

In this study, we examined the effects of a small peptide on two prominent features in the transgenic AD animals: impairment in memory, and amyloid plaque load. For memory experiments, we

used a spatial memory task, the Morris water maze task. In this task, the tank is filled with opaque water and contains a platform hidden below the water surface. The task for the animals is to find the location of the hidden platform using the cues that are present in the surrounding. After several training sessions, the animals are able to learn the location of the platform to get out of the water. As expected, the transgenic AD animals showed deficit in learning the task. They also showed deficit in long-term memory which was tested 24 h after the end of training. Thus, the animals showed features observed in human patients. Importantly, the animals that were given the small peptide showed improvement in learning the task. They also showed improved long-term memory.

Next, we asked whether the peptide under investigation had any effects on the amyloid plaque load in the transgenic AD animals. The control group of transgenic animals which were not given the peptide showed substantial number of plaques in the hippocampus, a brain region critically involved in several kinds of memories. However, the animals that were treated with the peptide showed significant reduction in the amyloid plaque load in the hippocampus. We assessed the plaque load in the cortex also. Similar to the hippocampus, the transgenic AD animals without peptide treatment showed substantial number of amyloid plaques. But, the animals which were given the peptide showed reduction in plaque number in the cortex.

Collectively, the results show that a small peptide is able to reduce amyloid plaque burden and improve memory in an animal model of AD. The peptide may provide beneficial effects in this condition, an aspect that needs further investigation.

Publications:

1. Pandey, K., & **Sharma, S. K.** (2020). Activity- and memory training-induced acetylation of α-tubulin in the hippocampus. *Neurobiology of learning and memory*, *171*, 107226.

2. **Sharma S. K.** (2020) COVID-19 and Mental Health. *Annals of Pharmacology and Pharmaceutical Sciences* 2020.

Presentations:

1. Delivered a lecture on "Dementia and herbal compounds" as part of India International Science Festival (IISF2020) curtain raiser event organized by Indian National Science Academy, New Delhi.

2. Delivered a lecture on "Mechanisms of memory impairment in Alzheimer's disease", on World Alzheimer's Day, 21st September, 2020 in "Online Webinar and Panel Discussion" organized by Interdisciplinary Brain Research Centre, Aligarh Muslim University, Aligarh.

Collaborators:

Dr. Pankaj Seth, NBRC.

Funding:

This work is supported by NBRC Core funds from Department of Biotechnology, India.

Degrees awarded:

Ph. D. Degree:

1. Kautuk Kamboj

2. Tushar Arora

M. Sc. Degree:

Sharmistha Panda

The Mark Test and Behavioral Responses to Mirrors in Adult Male Zebra Finches and House Crows

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We have been interested in studying the relationship between cognition and brain structure in songbirds including corvids (Indian house crows, *Corvus splendens*) and zebra finches (a species of Australian songbird, *Taenopygia guttata*). One of the most interesting aspects of cognition is self-awareness or to possess a sense of self. Despite the fact that self-awareness involves all senses including proprioception, self-generated vocalizations and odors, visual self-recognition in mirrors using the "mark" test developed by Gordon Gallup (1970) for testing self-recognition in chimpanzees has been most widely used. For this test, an individual is marked with a contrasting dye or paint on a part of the body which can only be seen with the aid of a mirror. If the animal attempts to remove the mark while looking at the mirror, it is said to pass the mark test since it can recognize the image in the mirror as its own reflection.

Earlier evidence suggests that besides humans, some species of mammals and birds demonstrate visual self-recognition, assessed by the "mark" test. Whereas there are high levels of inter-individual differences amongst a single species, some species such as macaques and pigeons which do not spontaneously demonstrate mirror self-recognition (MSR) can be trained to do so. We were surprised to discover that despite being widely used as a model system for avian research, the performance of zebra finches on the mark test had not been studied earlier. Additionally, since we have been studying brain-behavior interactions in house crows, we decided to study their responses to mirrors and the MSR mark test.

In our experiments, birds were placed singly in a cage, facing a mirror after a mark had been placed on a part of the body that they would only be able to view in a reflection (the head and neck; **Fig 1**). Birds were first acclimatized to the mirror (mirror exposure phase). During this phase as well as the test phase after the mark was placed, a number of behaviors were analyzed including (i) time spent in front of the mirror (**Figures 2A and 2B**), (ii) number of vocalizations directed at the mirror, (iii) contingency testing, that is, behaviors performed in front of the mirror which enables the individual to perceive a visual—kinesthetic match between the behavior and its reflection, (iv) mark-directed preening (**Fig. 2C**), (v) generalized preening over the rest of the body, (vi) aggressive/exploratory responses such as pecking on the frame of the mirror and (vii) search responses were analyzed. We also tested the responses of birds to a board of similar dimensions as the mirror (control board) and used a wet brush (without paint) as a control for somatosensory stimuli.

Although a small number of adult male zebra finches appeared to display heightened responses toward the mark while observing their reflections, we could not rule out the possibility that these were a part of general grooming rather than specific to the mark. In the case of house crows, despite the fact that an earlier study had demonstrated that this species of birds recognizes their reflections following the mark test, none of the house crows in our experiments demonstrated mark-directed behavior

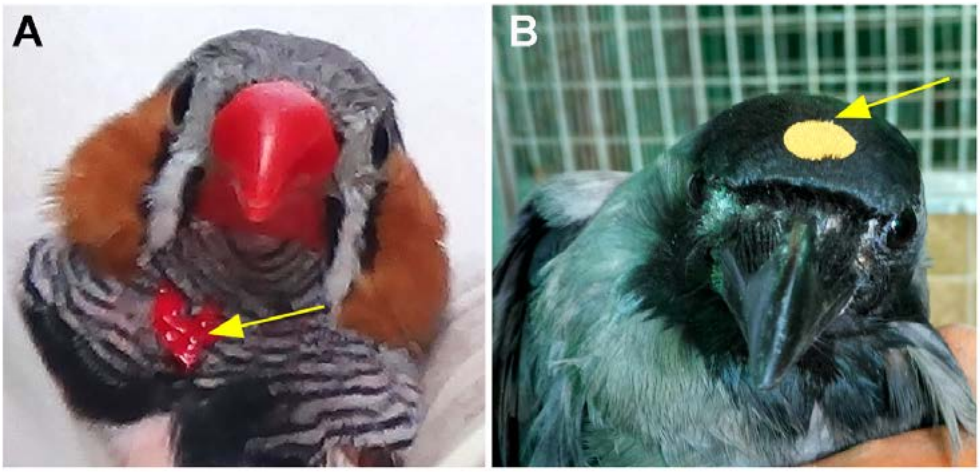

Figure 1: Placement of the "mark" on the (A) neck of an adult male zebra finch and (B) on the head of a house crow. Arrows indicate the position of the mark

or increased self-exploratory behaviors when facing mirrors. However, our results are supported by reports on other species of crows (New Caledonian crows and jungle crows), which do not pass the mark test. Our study suggests that self-directed behaviors need to be tested more rigorously in adult male zebra finches while facing their reflections and these findings need to be replicated in a larger population, given the high degree of variability in mirror-directed behaviors.

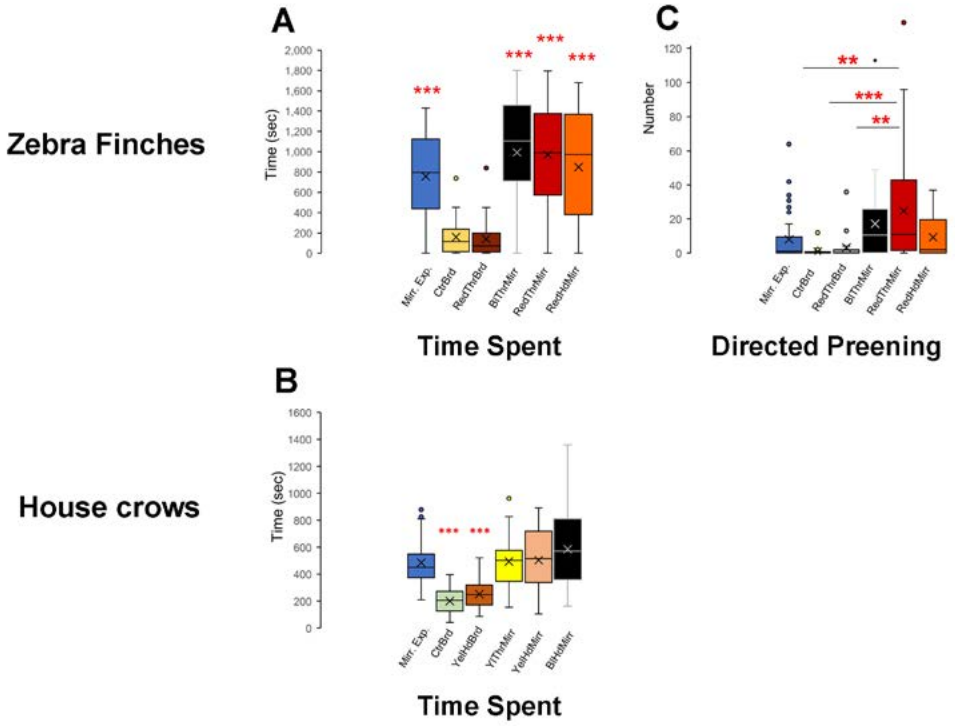

Figure 2: Examples of behaviors demonstrated by zebra finches and house crows while facing their reflections. Both (**A**) Zebra finches and (**B**) house crows spent significantly higher amounts of time facing the mirror during both mirror exposure and mirror test trials compared to facing a board of similar dimensions (control), suggesting that there was an increase in attention paid toward their reflections (post-hoc Tukey test ***, $P < 0.001$). (**C**) For zebra finches, there was a significant increase in directed preening on the neck in the RedThrMirr condition compared to that in the CtrBrd (Control board), RedThrBrd (Red Throat board), and mirror exposure period (post-hoc Tukey test; $P < 0.01$; **; $P < 0.001$***). House crows did not demonstrate mark-directed preening in any of our experimental birds

Abbreviations: Mirr Exp, Mirror Exposure phase; CtrBrd, Control Board;

For Zebra finches: RedThrBrd, Red Throat Board; BlThrMirr, Black Throat Mirror, RedHdMirr, Red Head Mirror. For House crows: YelHdBrd, Yellow Head Board; YlThrMirr, Yellow Throat Mirror; YelHdMirr, Yellow Head Mirror; BlHdMirr, Black Head Mirr.

Publication:

1. Parishar, P., Sehgal, N., & **Iyengar, S.** (2021). The expression of delta opioid receptor mRNA in adult male zebra finches (Taenopygia guttata). *Plos one*, *16*(8), e0256599.

2. Parishar, P., Mohapatra, A. N., & **Iyengar, S.** (2021). Investigating Behavioral Responses to Mirrors and the Mark test in adult male Zebra Finches and House crows. *Frontiers in psychology*, *12*, 1198.

3. Kumar, S., Mohapatra, A. N., Pundir, A. S., Kumari, M., Din, U., Sharma, S., Dutta, A., Arora, V., **Iyengar, S.** (2020) Blocking Opioid Receptors in a Songbird Cortical Region modulates the Acoustic Features and Levels of Female-Directed Singing. *Frontiers in Neuroscience*; September 2020; Volume 14; Article 554094; doi: 10.3389/fnins.2020.554094.

Funding:

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Degree Awarded (Ph.D.):

Sandeep Kumar : Thesis entitled 'Opioid Modulation of Singing in Adult Male Zebra Finches'. Degree awarded: 3rd February, 2021.

Long non-coding RNAs at the synapse: Implications in synaptic plasticity and memory

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Neural circuits engaged in cognitive functions, such as memory formation and storage, are dynamically modulated by persistent structural and functional changes at the synapse. The activity-dependent modifications at the synapses are reversible and occur at various temporal resolutions ranging from minutes to an hour time scale. To meet the demand for input-specific modifications at the synapses during memory formation, dendritic spines acquired some degree of autonomy at the molecular level to make de novo change in the proteome. Synapses are equipped with machineries regulating protein synthesis from transcripts encoding plasticity proteins that are transported to dendritic spines as translationally silenced packets. Therefore, translation from subset of transcripts at specific location and time within neuronal dendrite perceived as an energy efficient process that is pre-requisite to make de novo changes at the synapse.

Advancement of genome-wide sequencing has identified number distinct classes of non-coding RNAs and subset of these transcripts has already been linked with memory formation and storage. Of particular interest, we have focused on long non-coding RNAs (>200 nucleotides) and explored the mechanisms of synaptic plasticity and memory operated at the hippocampus. Long non-coding RNAs (lncRNAs) is expressed in specific cell type within a particular neuroanatomical region, such as hippocampus, cerebral cortex and cerebellum. These non-coding transcripts are originated from intergenic, intronic and imprinted loci and many of these non-coding RNAs are either antisense or overlapping to protein coding transcripts associated with various nervous system function. These observations point towards an intriguing possibility that long non-coding RNAs may localize at the synaptic compartment and regulate gene expression for development of neuronal circuitry and its function.

We have focused on some of the fundamental questions that include: (i) what is the identity of long non-coding RNAs at the synapse? (ii) how these non-coding transcripts are selectively transported to the synapse? (iii) how activity-regulated long non-coding RNAs modulate de novo protein synthesis? and (iv) how synapse-specific control of protein synthesis influence memory?

To explore the function of lncRNAs in synaptic plasticity and memory, we have employed a genome-wide screen of lncRNAs expressed in hippocampal synapses. Synaptic compartments or synaptoneurosome were purified from mouse hippocampus. Transcriptomics analysis was performed using synaptoneurosomal or total RNA. The bioinformatics analysis of the transcriptomics data set identified ~173 lncRNAs (annotated lncRNAs present in both GENCODE and Ensembl data bases) that are significantly enriched at the synaptic compartment (Figure 1).

Among this subset of lncRNAs, we have verified synaptic enrichment of ~10 lncRNAs by *in situ* hybridization.

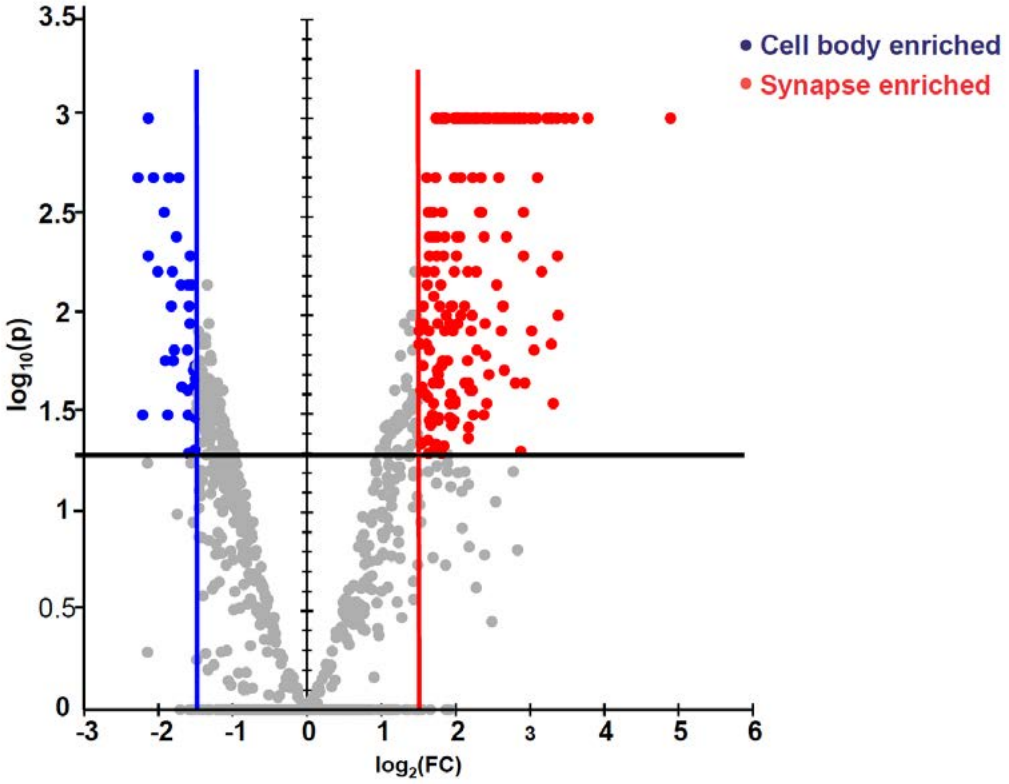

Figure 1: Synapse enriched long non-coding RNAs in hippocampal synapses identified by genome-wide sequencing. Transcriptomics analysis of synaptoneurosomal and total cell transcripts followed by bioinformatics analysis identified subset of lncRNA significantly enriched at the synapses or cell body

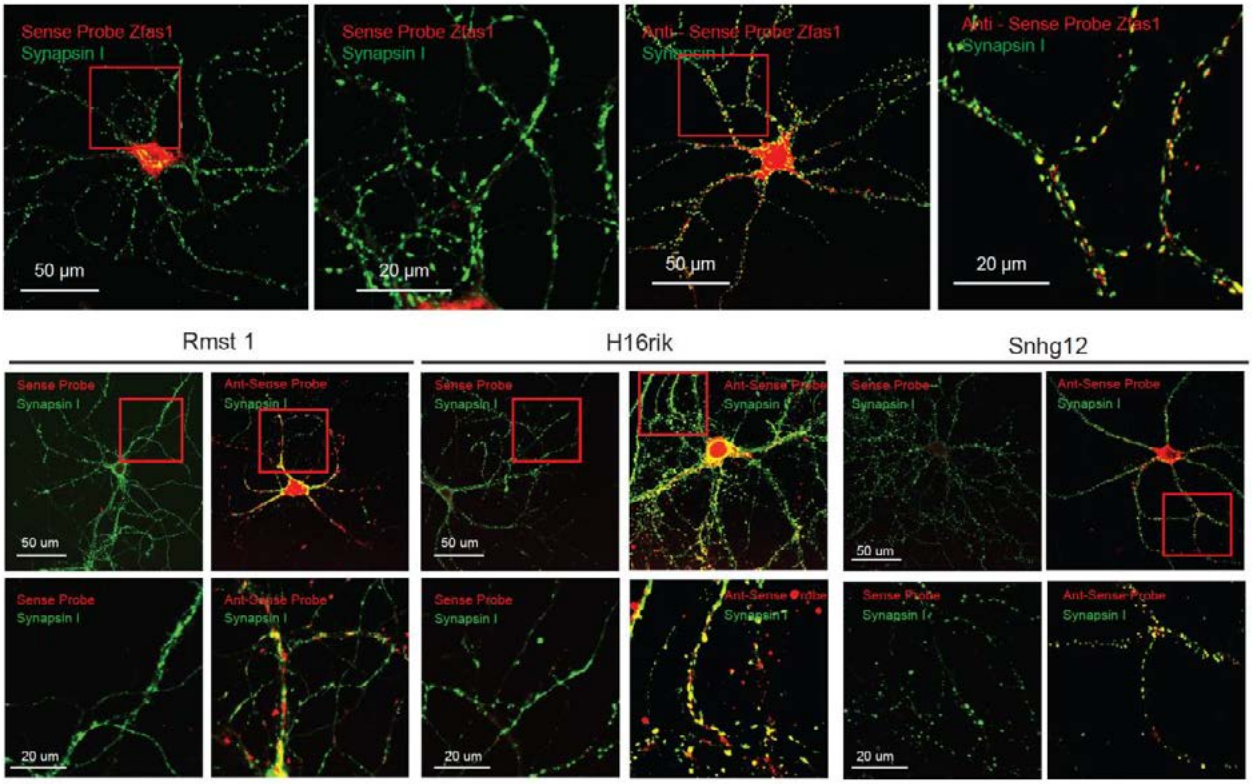

Figure 2: Localization lncRNA at hippocampal synapses visualized by in situ hybridization. Synapses are marked by pre-synaptic marker, Synapsin I

Following visualization of lncRNAs by *in situ* hybridization (Figure 2), we have focused onto few synapse-enriched lncRNAs and investigated their impact on spine development, synapse formation and synaptic transmission. Loss of lncRNA function was achieved by short-hairpin RNA (shRNA) –mediated RNA interference (RNAi) methodology. shRNA against specific lncRNA were cloned into a vector that co-express red fluorescent protein mCherry. To gain insights into synapse development in vivo, shRNA-expressing vector and GFP-expressing vector were co-electroporated into CA1 region of the hippocampus using in utero electroporation. Hippocampus from P28 brain was imaged using confocal microscopy and analyzed for dendritic and spine development (Figure 3).

To analyze the role of lncRNA in functional synapse development, synaptic activity was measured using whole-cell patch clamp recording. Hippocampal neurons were transduced by recombinant lentivirus expressing shRNA against specific lncRNA along with red fluorescent protein – mCherry. After effective knockdown of respective lncRNA by RNAi, amplitude and frequency of miniature Excitatory Post Synaptic currents (mEPSC) were measured from mCherry expressing hippocampal neurons (Figure 4). Our patch clamp recording data showed that loss of Pvt1 significantly reduced amplitude and frequency of mEPSC (Figure 4).

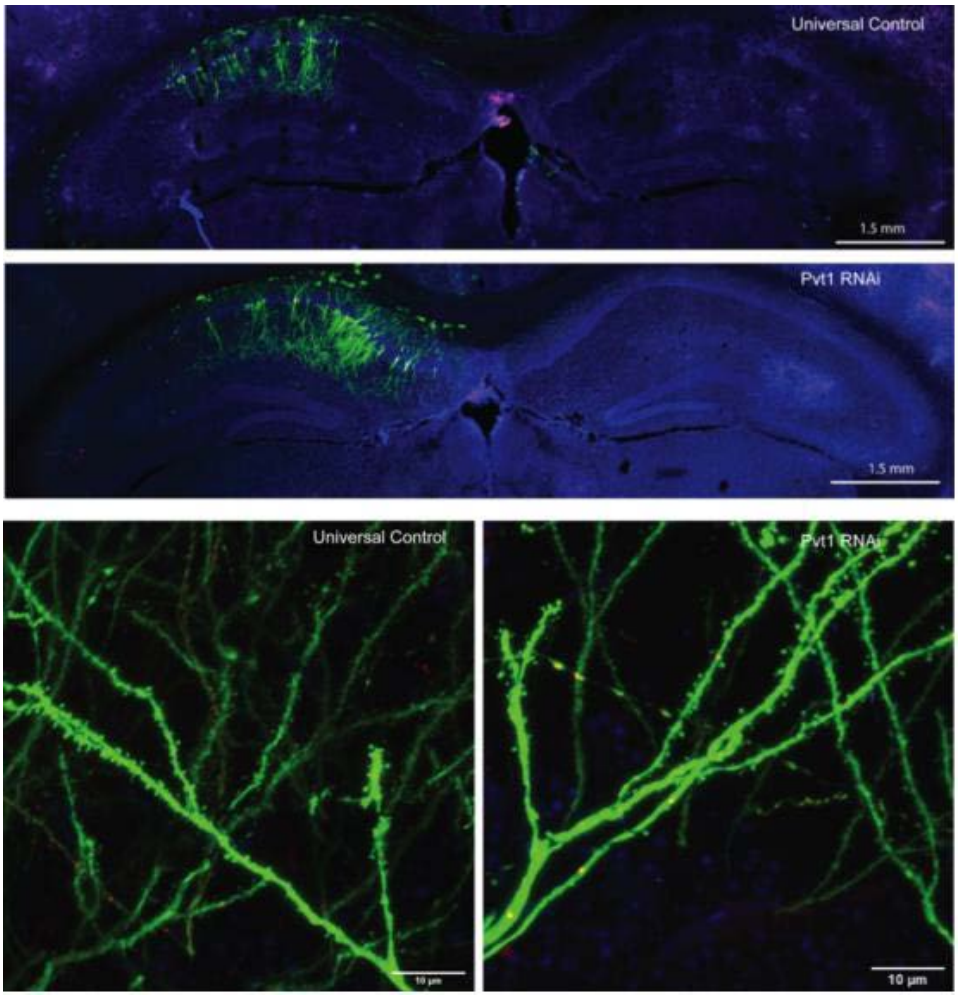

Figure 3: *In utero* electroporated CA1 neurons of the hippocampus showing morphological difference in dendritic spines. Hippocampal neurons expressing respective shRNA for loss of function experiment. The vector expressing specific shRNA along with mCherry co-electroporated with GFP *in utero*. Hippocampal neurons were imaged at postnatal day 28. Scales as indicated

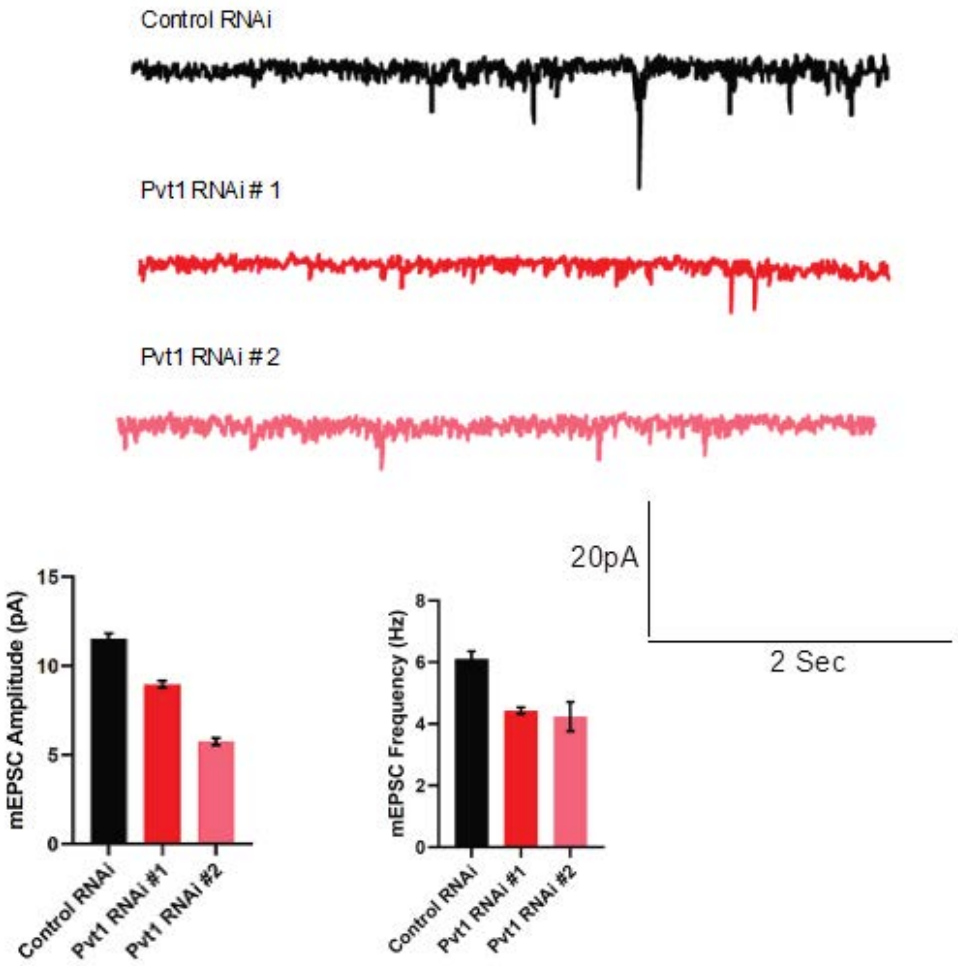

Figure 4: Whole-cell patch clamp recording showing significant reduction in mEPSC amplitude and frequency following loss of Pvt1 lncRNA function

Furthermore, we have analyzed the expression of subset of lncRNAs upon contextual fear conditioning—a memory paradigm that involves the hippocampus. To gain insights into how memory-responsive lncRNAs potentially regulates translation, we analyzed their association with actively translating RNA or polysome. We then assessed how the interaction between specific lncRNA and translating transcript antisense to cognate lncRNA is impacted by deprivation of sleep—a key regulator of memory storage. It was observed that sleep deprivation leads to enhanced association of Rbm3OS lncRNA with actively translating fraction, whereas polysome association of its cognate antisense transcripts encoding RNA binding protein (Rbm3) is significantly reduced. These data demonstrate that sleep deprivation resulted in the reciprocal polysome association of lncRNA and mRNA pair (Figure 5).

Western blot analysis revealed that sleep deprivation significantly reduces the translation of Rbm3. Our ongoing analysis regarding RNA–protein interaction studies will delineate how this lncRNA-mRNA interaction is dynamically regulated by sleep loss and how it impacts protein synthesis-dependent form of memory formation.

We anticipate that our study will provide clues to broaden our understanding of memory deficits—a pathological condition associated with aging or age-related neurodegenerative disorders.

Publications

1. Srinivasan, B.[$], Samaddar, S.[$], Mylavarapu, S. V., Chelliah, J. P., & **Banerjee, S.** (2021). Homeostatic scaling is driven by a translation-dependent degradation axis that recruits miRISC remodelling. Accepted in *PLOS Biology*.[$] Equal Contribution.

2. Samaddar, S., & Banerjee, S. (2021). Far from the nuclear crowd: Cytoplasmic lncRNA and their implications in synaptic plasticity and memory. **Neurobiol Learn Mem**, 107522. doi: 10.1016/j.nlm.2021.107522. Online ahead of print.PMID: 34547434.

3. Liau, W. S., Samaddar, S., **Banerjee, S.**, & Bredy, T. W. (2021). On the functional relevance of spatiotemporally-specific patterns of experience-dependent long noncoding RNA expression in the brain. **RNA biology**, 18(7):1025-1036. doi: 10.1080/15476286.2020.1868165.

4. Dagar, S., Pushpa, K., Pathak, D., Samaddar, S., Saxena, A., **Banerjee, S.**, & Mylavarapu, S. V. (2021). Nucleolin regulates 14-3-3ζ mRNA and promotes cofilin phosphorylation to induce tunneling nanotube formation. *The FASEB Journal*, 35(1), e21199. doi: 10.1096/fj.202001152R.

Presentations

Dr. Sourav Banerjee: Balakumar Srinivasan and Sarbani Samaddar. Balancing act : Mechanism of homeostatic synaptic scaling by Proteastasis. "Synapse and System Plasticity of Learning and Memory" virtual meeting at iCEMS, Kyoto University, Japan, September 2020

Funding

Science and Engineering Research Board

Department of Biotechnology

NBRC Core fund

Collaborators

1. Dr. Dasradhi Palakodeti, in-Stem, Bangalore

2. Dr. Sivaram Mylavarapu, RCB, Faridabad

3. Dr. James Chelliah, JNCASR, Bangalore

4. Prof. Ted Abel, University of Iowa, USA

5. Dr. Timothy Bredy, University of Queensland, Australia

M.Sc. Degree Awards

Ms. Rekha Singh

Vesicular trafficking pathways in neuroendocrine cells and their consequent role in physiology, health and diseases

Dr. Bhavani Shankar Sahu

Principal Investigator:
Bhavani Shankar Sahu

Postdoc Student
Sushma Dagar

Ph.D. Student
Chandramouli Mukherjee

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Aamna Jain

Project assistant
Vinayak Gupta

Project assistant
Souren Sadhukhan

Our lab investigates vesicular trafficking pathways in neuroendocrine cells. Towards understanding the specialised vesicular trafficking pathways, we study a specific type of vesicles called dense-core vesicles (DCVs). DCVs are specialised sub-cellular organelles present in specific neurons/neuroendocrine cells and regulate diverse physio-metabolic functions. They undergo stimulus-dependent secretion, and the phenomenon is called "regulated secretion". Components of regulated secretion include neuropeptides, neurotransmitters that regulate various physiological functions. Although the research on DCVs has been pursued for the past four decades, many aspects related to sub-cellular trafficking/secretion is unknown, and we study this in our lab in two broad themes.

Research Theme I: Investigating DCV Biogenesis

We are specifically interested in understanding the fundamental mechanisms related to post Golgi vesicular trafficking pathways in DCV biogenesis and the proteins associated with regulated exocytosis, a phenomenon of controlled secretion in neuronal/neuroendocrine cells. To understand this, we employ Rat PC-12 and INS-1 neuroendocrine cells as model systems to study. The main goal is to discover novel proteins regulating DCV formation, maturation and secretion. To address this, we use gene manipulation (gene knockdown & CRISPR/Cas9), subcellular fractionation, proteomics, electron and confocal microscopy as tools. In this theme, for a specific project, we are investigating the role of adaptor protein complex-3(AP-3) in DCV formation and function by using inducible shRNA systems. We have established a stable cell line enabling inducible shRNA depletion of Ap-3 complex and are currently carrying out the functional studies. Our studies suggest a significant loss of DCV function, and current research is being pursued to dissect the molecular mechanisms of the same.

Research Theme II: Investigating the contribution of the regulated exocytosis and neuroendocrine proteins in health and disease

Besides fundamental biology, we are also interested in understanding how DCV proteins regulate metabolic and physiological functions at the cell/organism level. Towards this, we are investigating the role of Chromogranins and related peptides co-stored and released by regulated exocytosis from DCVs of neuroendocrine cells in regulating cellular and physiological functions by using cell culture and mice models. In a specific project, we are studying the role of DCV derived peptides, catestatin and TLQP-21 in modulating glial cell functions by using rodent N9 and BV-2 microglial cell lines. We are also interested in investigating the regulated exocytosis in neuronal diseases. We plan to understand the functional status of regulated exocytosis in Huntington's disease by using cell culture and mice models in a specific project. Our futuristic projects include the use of knock out mice of DCV proteins to

characterise the physiological phenotypes and functional characterisation of gene variants of DCV proteins associated with clinical conditions such as mental health disorders and metabolic syndrome (Diabetes, Obesity and Cardiovascular disease).

Publications:

None.

Presentations:

5-9-2020, Webinar on Neuroendocrine regulation of metabolic physiology, GN Ramachandran Science club, Vigyan Prasar, MAC FAST, Kerala.

Funding:

Department of Biotechnology, International Brain Research Organisation, Paris, ICGEB, Trieste & NBRC core funds.

Collaborators:

Internal:

Prof Anirban Basu

External:

Professor Sushil Mahata, University of California, Sandiego, USA.

Dr Alessandro Bartolomucci, University of Minnesota, Twin Cities, USA.

Dr Sanjeev Upadhay, MS University, Baroda, India.

Dr Saleem Mohammad, NISER, BBSR

Dr Yusuf Akther, BR Ambedkar Central University, Lucknow.

Dr Dileep Vasudevan, DBT- Institute of Life sciences, Bhubaneswar, India.

Awards: (if any):

DBT-Ramalingaswami fellowship award, IBRO(International Brain Research Organisation) start-up grant, ICGEB Trieste(Early career research award).

Degrees awarded:

1 MSc

Drosophila models of human neurodevelopmental and neurodegenerative disorders

Dr. Mayanglambam Dhruba Singh

Principal Investigator

Mayanglambam Dhruba Singh

Research Associate/Post-doctoral Fellows: 1

PhD Students: 0

MSc Students: 0

Project Assistants: 2

Technical Assistant: 2

I joined National Brain Research Centre (NBRC) as Scientist III in October 2020. My research at NBRC focuses on neurodevelopmental and neurodegenerative disorders using Drosophila models. Simple model organism such as Drosophila provides several advantages including short life span, availability of large number of genetic toolkit and transgenic lines. Presently, our lab is working on identification of novel genetic modifiers of human neurodegenerative diseases such as, Huntington's disease, Ataxia-3 and Alzheimer's disease. We use UAS-Gal4 system to manipulate the expression of gene in tissue-specific manner. Overexpression of mutant ataxin-3 protein with 78 poly(Q) repeats or huntingtin with 138 poly(Q) repeats induces depigmentation and roughness of eye when compared to control flies (Fig. 1A-C). On the hand, overexpression of mutant tau causes roughness in posterior region of eye (Fig. 1D).

Using rough eye as readout, genetic screens will be performed to identify modifiers of these neurological disorders. In addition to external eye morphology, the number of photoreceptor neurons and level of poly(Q) protein aggregates will also be examined. For example, overexpression of mutant Htt-138-mRFP causes roughness of eye and loss of photoreceptor neurons (Fig. 2A-D). Our modifier screening will examine for the improvement in the structure and number of photoreceptor neurons of the eye. Furthermore, the level of abnormal protein aggregates caused by overexpression of Htt-138Q will be examined (Fig. 2E-G). If the improvements in eye morphology and reduction in the level of protein aggregates are observed, molecular mechanisms for the suppression of the poly(Q) phenotypes will be investigated in detail.

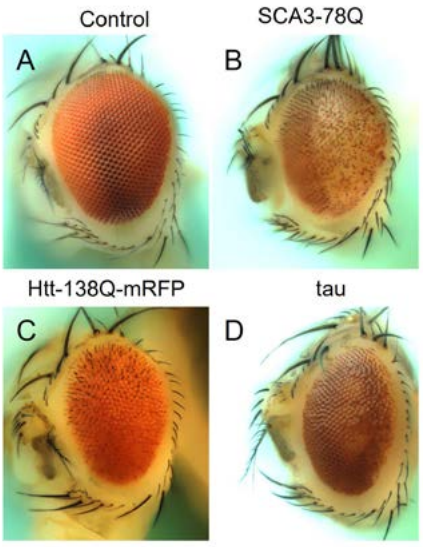


Figure 1: Drosophila models of neurodegenerative disorders. (A) Wild type. (B) Overexpression of ataxin-3 with 78 poly(Q) repeats causes roughness and depigmentation of eye. (C) Overexpression of mutant huntingtin with 138 poly(Q) causes rough eye phenotype. (D) Overexpression of mutant tau causes roughness in posterior region of the eye

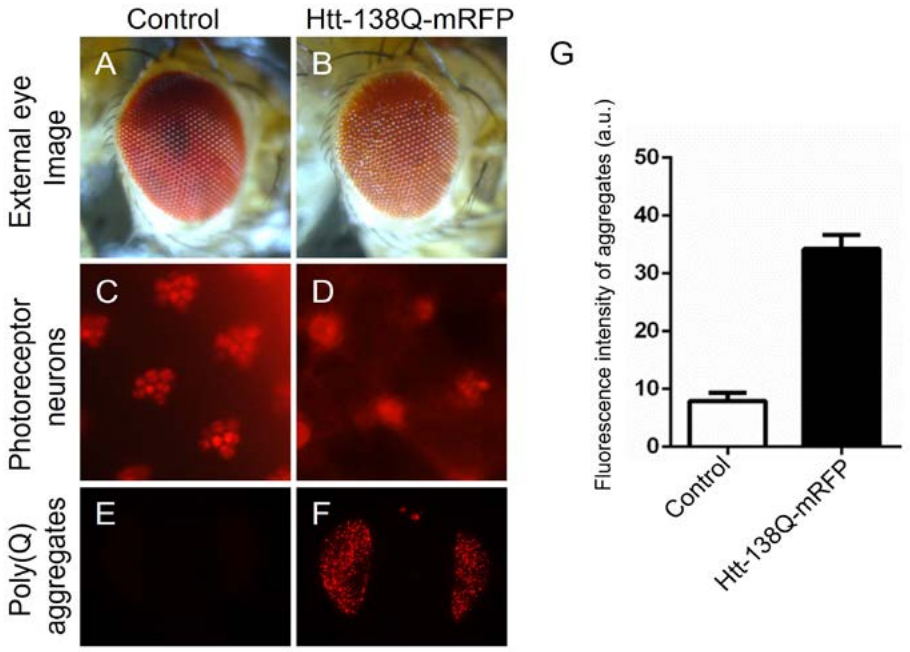


Figure 2: Overexpression of Htt-138Q-mRFP causes degeneration of eye tissue. (A-B) External eye morphology. (C-D) No. of photoreceptor neurons are reduced by overexpression of mutant huntingtin with 138 poly(Q) repeats. (E-F) Observation of poly(Q) aggregates in adult head. (G) Bar graph showing fluorescence intensity of poly(Q) aggregates

Our lab is also interested in understanding the biological functions of neurodevelopmental genes involved in Autism and Intellectual disability. Large scale DNA sequencing of individuals with Autism and Intellectual disability have identified more than 100 genes. However, the functions and role of many of the genes are largely unknown. Drosophila is well-known model system for investigating various neurodevelopmental disorders. So, our study will utilize the Drosophila model system for dissecting the genetic basis of these neurodevelopmental disorders. DRSC integrated ortholog prediction tool (DIOPT) was used to identify the fly homologs of those neurodevelopmental genes. The RNAi lines of these genes will be procured to perform gene knockdown using UAS-Gal4 system. GMR-Gal4, elav-Gal4 and insc-Gal4 will be used to knockdown the expression of the genes in eye, brain and neuroblast cells respectively to investigate the role of the genes during neurodevelopment. Larval brain will be stained with dlg, prospero and deadpan antibodies to investigate any defect in neuronal proliferation and differentiation. Furthermore, behavioral defects caused by gene knockdown will be investigated. For example, Drosophila Grooming assay which mimics repetitive behavior and social distance assay which allows to examine social deficits will be performed. As Autism and Intellectual disability causes sleep defects, circadian activity of flies will be monitored. From these studies, we will identify novel genetic modifiers of neurodegenerative disorders and reveal biological functions of neurodevelopmental genes involved in Autism and Intellectual disability.

Publications:

NA

Presentations:

Mayanglambam Dhruba Singh: Neuroscience with Fruit fly, Indian International Science Festival (IISF), presented virtually, December 2020.

Funding:

This work is supported by NBRC Core funds.

Collaborator:

NA

Awards (if any)

NA

Degrees Awarded (Ph.D.):

0

Meetings/Conferences organized:

NA

Evaluating Interhemispheric Effective Connectivity During Midline Object Recognition using EEG

Dr. Nivethida Thirugnanasambandam

Principal Investigator

Nivethida Thirugnanasambandam

Research Associate/ Post-doctoral Fellows: 0

Ph.D. Students: 1

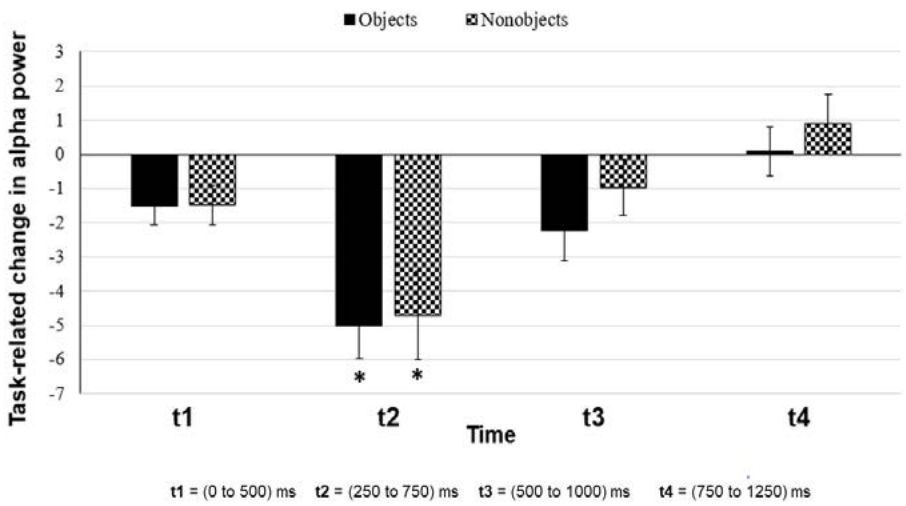
MSc Students: 1

Project Assistants: 1

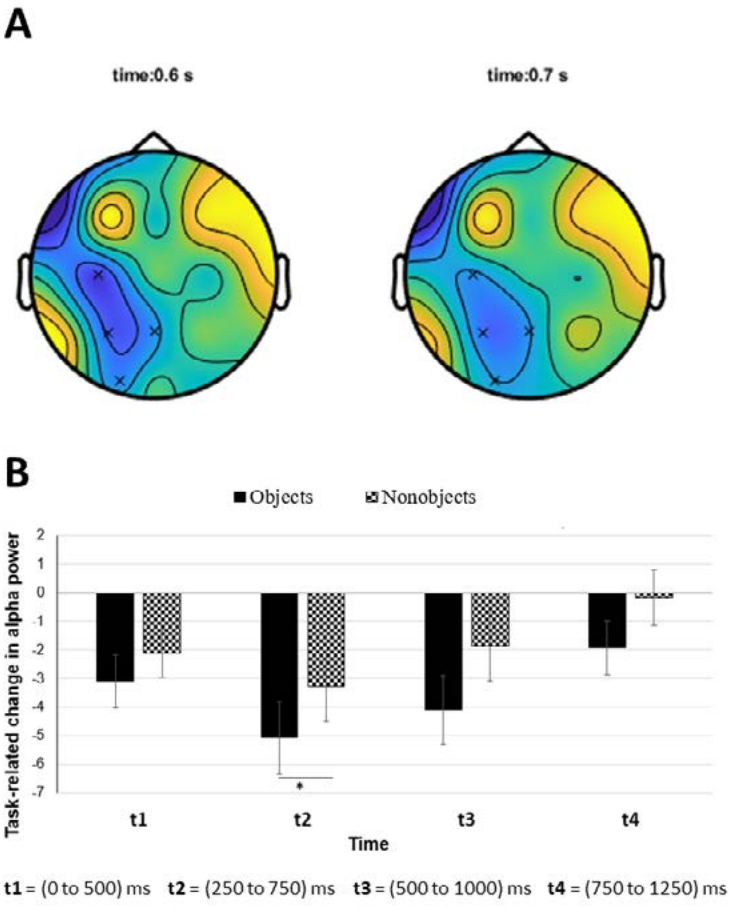
Technical Assistant: 0

When visual stimuli are presented in the centre of our visual field, the images formed in the right and left visual cortices are integrated to form a single image that can then be recognized. This phenomenon is called perceptual binding. For perceptual binding to occur, there has to be some form of communication or functional integration between the two hemispheres. The process of object recognition involves exchange of visual information between the two hemispheres and further successfully retrieving features and names of objects from our memory. One of the widely-accepted views on neuronal communication is the 'communication through coherence' hypothesis. It proposes that distinctly located neuronal groups can communicate with one another by virtue of phase-locked neuronal oscillations known as neuronal coherence. The communication between the two hemispheres during object recognition may also occur via such neuronal synchronisation or coherence. Researchers in the past have studied these neuronal synchronizations using EEG. A study by Mima and colleagues identified transient interhemispheric occipito-temporal synchronization of alpha rhythm during a midline object recognition task using 29-channel EEG. Their study demonstrated a significant increase in interhemispheric coherence in the 117-373 ms time window when healthy human subjects viewed object stimuli compared to when they viewed non-object stimuli. They also observed that in the later time window of 373-639 ms, there was a significant decrease in alpha power for both object and non-object stimuli that were displayed in the midline of the visual field. However, the study was not without limitations. The authors presumed that the power and coherence changes would be best observed over a predetermined set of 4 electrodes that were positioned over the occipito-temporal regions. We know that volume conduction is clearly a significant problem with EEG and also that coherence is a connectivity measure that could be greatly influenced by volume conduction. Furthermore, due to the smaller number of EEG electrodes, they were not able to localize the brain sources which could justify their choice of electrodes of interest. They also did not determine the direction of functional connectivity.

In the current study, we aimed to replicate the results of Mima et al using high density EEG while subjects performed the midline object recognition task and to resolve the limitations of their study. Precisely, we intended to replicate the task-related alpha desynchronization and the transient increase in interhemispheric coherence during midline object recognition. We further sought to localize the sources in the brain that are crucial for midline object recognition and also to determine the direction of this interhemispheric connectivity.



All experiments were carried out at the Human Motor Control Section. The protocol was approved by the Combined Neuro Sciences Institutional Review Board (CNS IRB) of the National Institutes Health (NIH) and conformed to the guidelines of the Declaration of Helsinki. All participants gave written informed consent prior to the study. We recruited 11 healthy adult volunteers and recorded EEG from 64 channels while they performed a midline object recognition task. Task-related power and coherence were estimated in sensor and source spaces. Further, effective connectivity was evaluated using Granger causality.

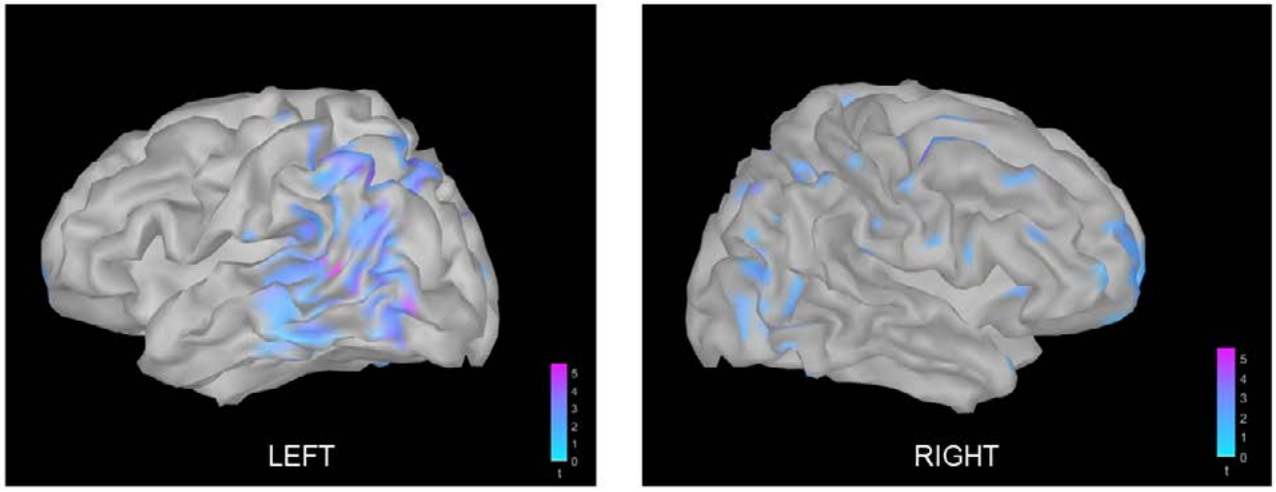


In the current study, we were able to partially replicate the results of Mima et al. We confirmed that the stimulus non-specific alpha desynchronization occurs in the time window around 500 ms. However, we could not replicate the transient increase in interhemispheric alpha coherence associated with object recognition using the same electrode pairs of interest.

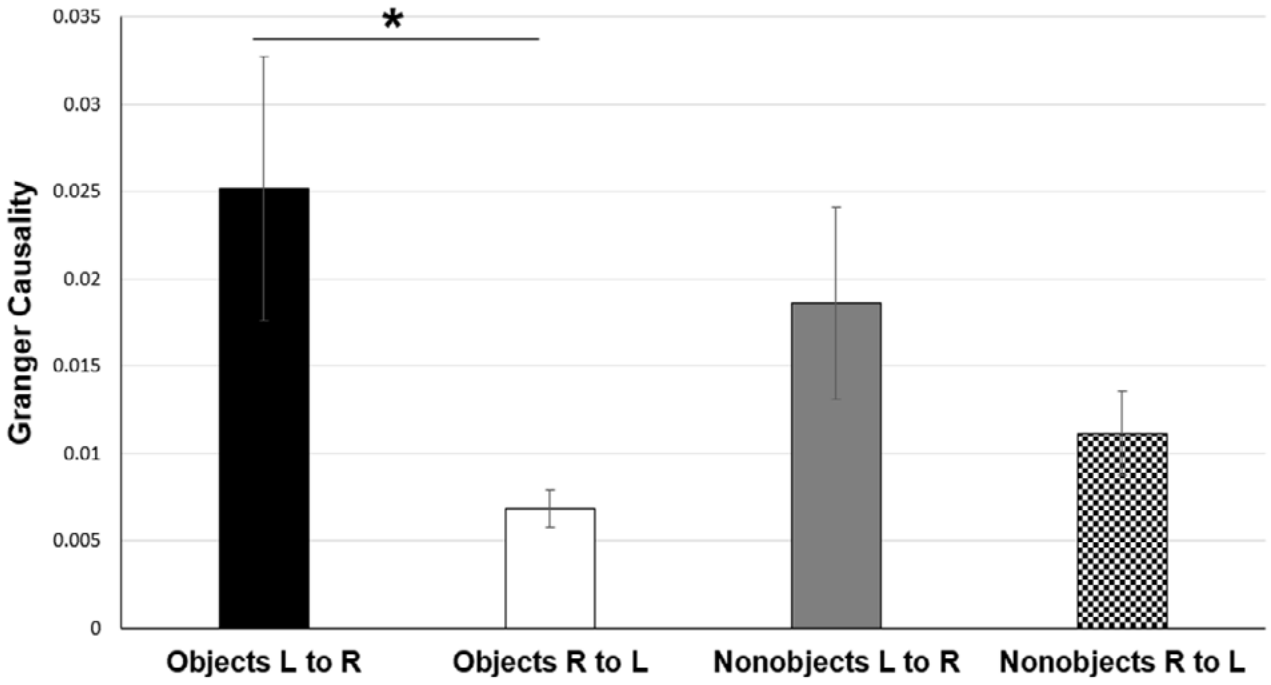
Further, with detailed analysis using a data-driven approach we showed that there is significantly high alpha desynchronization associated with object recognition over C3, P3, Pz and O1 electrode cluster.

We localized the source of this alpha desynchronization to be in the left occipito-temporal region and showed that interhemispheric connectivity (imaginary part of coherency) between the homologous regions increased significantly for object recognition during the 250-500 ms time window.

Using Granger causality, we also showed that the interhemispheric connectivity associated with object recognition is driven by the left occipito-temporal region.



In conclusion, our results confirm that midline object recognition requires integration of information from the two hemispheres. This interhemispheric exchange of information is mediated by transient synchronisation of neuronal activity between the ventral pathways of visual processing. Interhemispheric coherence has relevance not only in the visual sensory system but is also crucial for a broad repertoire of cognitive functions including language, visuospatial attention and manual preference. Consequently, disruption of interhemispheric coherence may impact behavioral and cognitive functioning. Thus, our study may have implications in many neuropsychiatric disorders like schizophrenia, stroke, focal hand dystonia where interhemispheric coherence is known to be impaired.



Publications:

None

Presentations:

Thirugnanasambandam N. Site-specific decrease in cortical reactivity during sensory trick in cervical dystonia patients. 7th Asia-Oceania Congress of Clinical Neurophysiology, Kualalumpur, Malaysia (virtual), January 2021.

Thirugnanasambandam N. Bridging the gap between intracortical mechanisms and behavior. 7th International Conference on Non-invasive Brain Stimulation, Bbaden-Baden, Germany (virtual), November 2020.

Thirugnanasambandam N. Rhythms of the Brain. National Brain Research Centre (NBRC) Outreach event at the India International Science Festival, India (virtual), December 2020.

Thirugnanasambandam N. Evaluating cortical connectivity with non-invasive brain stimulation, Monsoon Brain Meeting, India (virtual), June 2020.

Funding:

DBT/WT India Alliance CPH Fellowship (Intermediate) – IA/CPHI/16/1/502624

Collaborators:

Dr. Arpan Banerjee, NBRC

Dr. Dipanjan Roy, NBRC

Dr. Roopa Rajan, Department of Neurology, AIIMS, New Delhi

Awards (if any):

Har Gobind Khorana Innovative Young Biotechnologist Award 2020

Degrees Awarded (Ph.D.):

None

Meetings/Conferences organized:

(give a brief description of the meeting)

None

Evaluating Interhemispheric Effective Connectivity During Midline Object Recognition using EEG

Dr. Swagata Dey

Principal Investigator
Swagata Dey

Research Associate/ Post-doctoral Fellows: 0

Ph.D. Students: 0

M.Sc. Students: 0

Project Assistants: 0

Technical Assistant: 0

Human behavior is defined by the underlying neural circuits that change during the lifetime of an organism. The polarized structure of the neurons allows them to establish and maintain connections through time for the unidirectional flow of information. The input processes of the neurons are the dendrites that define the sensory area of the neuron and attribute a characteristic morphology and function to the neuron. Dendritic morphology is correlated to certain neurodevelopmental disorders like Autism, Schizophrenia, and Alzheimer's disease. Certain chronic or acute conditions also may lead to disrupted or aberrant dendritic structure as often observed during neuronal excitotoxicity, drug abuse, and traumatic brain injuries. Unlike other compartments of the neuron, dendrites can remodel extensively throughout the lifetime of an organism especially during major interventions like development, consolidation, refinement, or repair of circuitry. Failure in this process can lead to degeneration of the neuronal compartments, loss of the sensory and motor modalities, and a manifestation of neuropathological symptoms. It is therefore imperative to understand the processes and regulators of dendritic remodeling in different physiological conditions.

Remodeling of the dendrites is known to depend on extrinsic factors like adhesion with the substratum and clearance of debris following pruning. Intrinsic factors like calcium transients, transcriptome, cytoskeleton, and polarized transport may influence the dendritic remodeling however, the mechanistic understanding of the process is unclear. The dendritic cytoskeleton is mainly composed of the microtubules and actin with scaffolding proteins like spectrins and septins. The core machinery for microtubule maintenance consists of End binding proteins like EBP and Patronin, depolymerizing motor, Kinesin-13, assembly factors like CRMP, and motors like Kinesin-1 that transports the majority of cargoes including tubulins and MT protofilaments. Similarly, actin is maintained by polymerization factors like Profilin, depolymerization factors Cofilin, and branching factors like Arp-2/3 and WASP/WAVE which have been implicated in the formation of the dendritic arbor. Dendritic arborization also depends on the microtubule and actin nucleators in the form of Golgi outposts, kinetochore proteins, endoplasmic reticulum, actin blobs which enrich at the presumptive dendritic branch points. Due to dendritic complexity and lack of in vivo models, it is not well understood how the neuronal cytoskeleton is organized and regulated for proper dendritic arborization during development or regeneration.

Using the PVD neurons of C. elegans, we are investigating:

1. How dendrites of PVD neuron remodel during development?
2. How is the underlying cytoskeleton reorganized during the dendritic remodeling?

Live imaging paradigm to understand the dynamics of dendrite development

Previous studies have elucidated that the gross characteristics of the dendrite regrowth after injury are different from the dendrite growth during development. However, it is unclear if the cytoskeleton processes regulating the initiation and growth of the dendrite branches are conserved during development and regeneration.

To understand the dynamics of dendrite initiation and development, I established a live imaging paradigm. The worms were synchronized and harvested for live imaging for a duration of 4 hours (Figure 1A). PVD neurons in C. elegans have a well-defined axon and stereotyped dendritic arbor (Figure 1A-B). The branches are formed orthogonally in anatomy and hierarchy with a distinct cytoskeletal constitution. The primary branches run in the anterior-posterior direction are mostly microtubule rich whereas the higher-order branches have an actin-based cytoskeleton (Figure 1C).

Depending on the stage of the harvested worms, the PVD dendrite development is promiscuous at certain hierarchical levels. For example, by 38.5 hours after egg laying (h AEL), the worms reach the L3 stage by which primary branches are laid out and secondary branches are initiated.

During the course of development, the branches show growth and retraction (Figure 2A-B). Simultaneously, the tertiary branches are also assessing the neighboring territory for consolidation or avoidance (Figure 2E) purposes. During this process, they tend to break apart from (Figure 2C) or fuse to a neighboring branch (Figure 2D). The quaternary branches show growth and retraction before stabilization at 48h AEL and culmination of the larval stages.

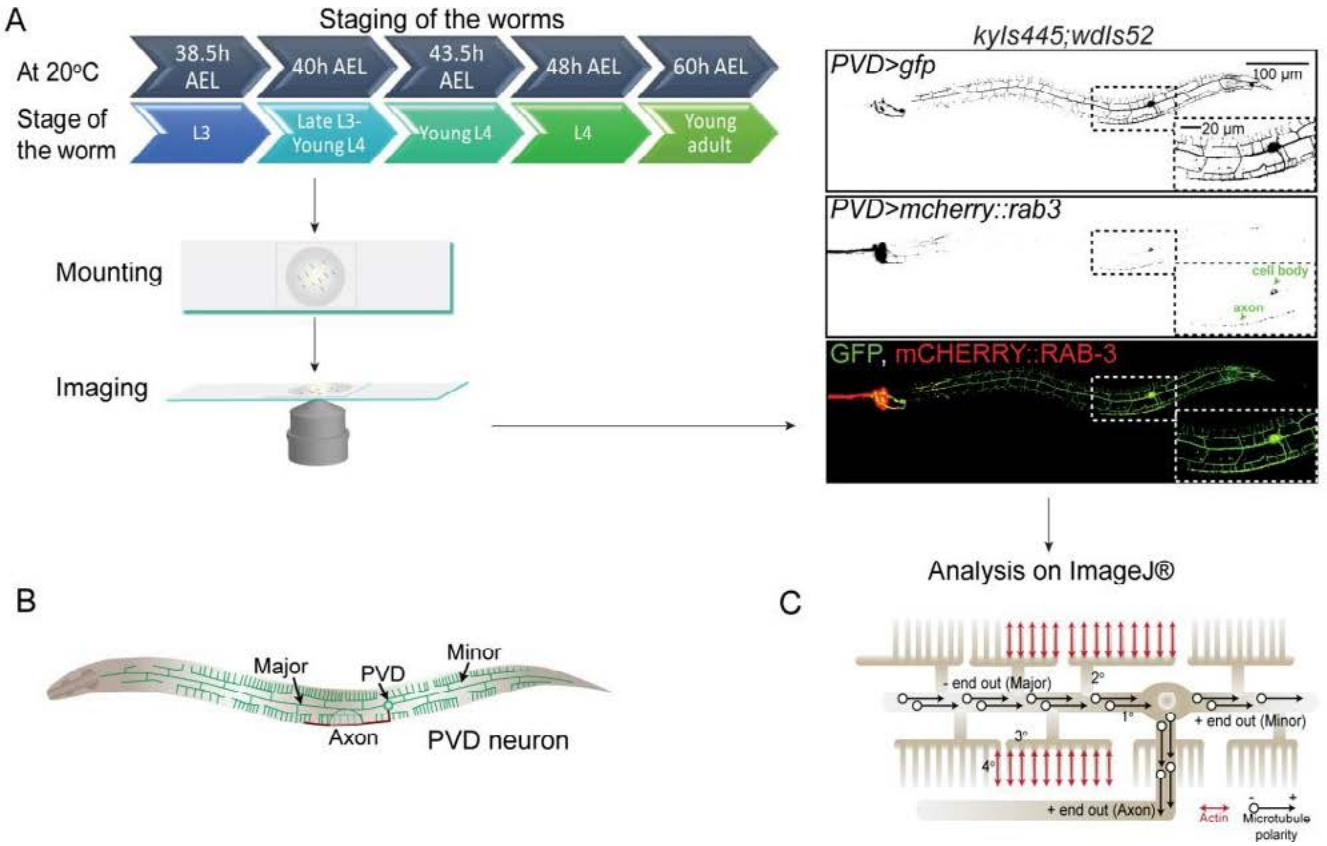


Figure 1: PVD neurons as a model to understand dendrite development.

Schematic depicting synchronized staging and imaging of worms at 20o C with correlated developmental stage and representative images of PVD neuron labeled with GFP (*pF49H12.4*>GFP, green) and mCHERRY::RAB-3 (*pdes-2*>mCherry::RAB-3, red) with the region of interest (white dashed rectangle) magnified in the respective insets. mCHERRY::RAB-3 punctae localized to the cell body and axon (marked in the inset). (B-C) Schematic depicting the neurite arbor of PVD neurons (B) and the cytoskeletal constitution (C)

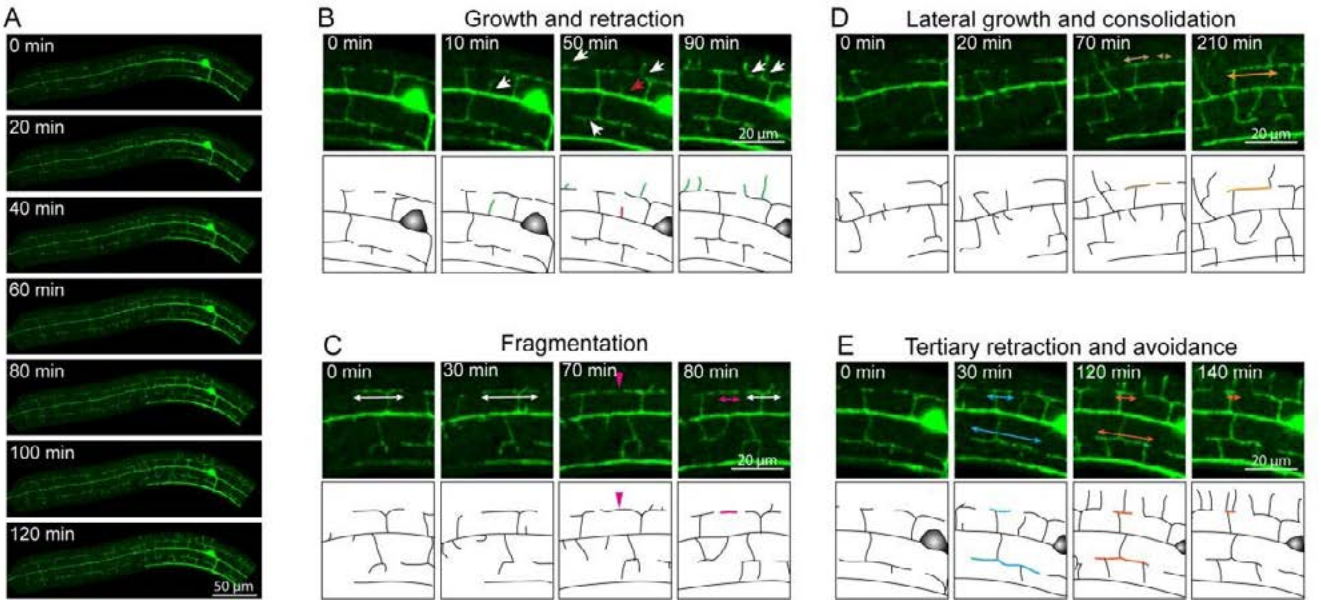


Figure 2: Live imaging of dendrite development in PVD neurons. (A). Timelapse images of the PVD neurons expressing GFP. Dynamics are observed at secondary, tertiary, and quaternary levels. (B-E). Dynamic events observed during the dendrite development include growth (white arrows) and retraction (red arrow) (B), fragmentation (magenta arrowhead) of the tertiary dendrite (double-sided arrows) (C), Lateral growth of the tertiary branches (brown arrows) and consolidation (yellow arrow) (D), and retraction (orange arrows) and avoidance of the tertiary branches (blue arrows) (E). We speculated that this growth and retraction of the branches is facilitated by correlated dynamics of microtubules and actin. We will further investigate this using live imaging of the microtubule and actin reporters, expressible in the PVD neurons.

Kinesin-13 (KLP-7) regulates dendrite arborization of PVD neuron and localizes to its branch points

KLP-7 is a motor of the Kinesin-13 family that depolymerizes the microtubules at both its plus and minus ends. DLK-1 triggered by the axonal injury inhibits the KLP-7 to stabilize the microtubules and promote axon regeneration. Dendrite regeneration is independent of DLK-1 suggesting an alternate pathway. Although KLP-7 is a major microtubule regulator, the role of this protein is not known in the dendrites. I hypothesize that dendrite regeneration in PVD neurons may depend on KLP- 7 mediated microtubule dynamics. However, it is essential to characterize the developmental role of KLP-7 in the remodeling of dendrites.

We investigated the dendritic arbor of the GFP labeled PVD neurons in the wild type and loss of function mutant of *klp-7* in age-matched animals (Figure 3A). We scored the branch density which is the number of branches at a particular hierarchical level normalized to the branches at the previous hierarchical level. For example, secondary branch density is obtained by the number of secondary branches normalized to the length of the primary branch. Similarly, the number of tertiary branches per secondary and the number of quaternary branches per tertiary were measured.

In the loss of *klp-7* mutants, the secondary branch density was drastically high as compared to the age-matched wild type control from 40h AEL onwards (Figure 3B). On the other hand, the density of tertiary and quaternary branches was different in the *klp-7(0)* than that of the wild type animals (Figure 3C-D). The live imaging further revealed that there is a dynamic equilibrium in the branch density during the development which was perturbed in the *klp-7* null mutant.

We further investigated the localization of KLP-7 in the PVD dendrites by using a single copy reporter line of KLP-7::GFP (Figure 4A-B). Endogenously expressed KLP-7::GFP was localized to some of the branch points especially the incipient branch points on the primary dendrite (Figure 4A-B). During the adult stages of the worm, these incipient branches grow more in number. We also observed a correlated increase in the localization of KLP-7::GFP to these ectopic branches (Figure 4A-C).

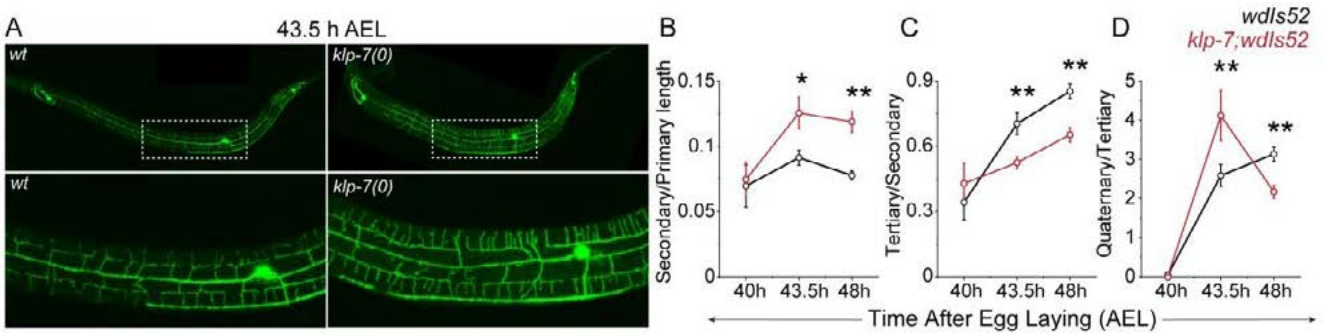


Figure 3: KLP-7 regulates the dynamics of dendrite development. (A) Representative confocal images of the PVD dendritic arbor of wildtype and klp-7 loss of function mutant with magnified views in the lower panel. (B-D) Quantification of the density of secondary branches per unit length of primary (B), the density of the tertiary branches per secondary (C), and density of quaternary branches per tertiary was obtained from the major dendrite of the wild type and klp-7(0) mutant PVD neurons. Comparison of means is done using ANOVA with p<0.05*, 0.01**

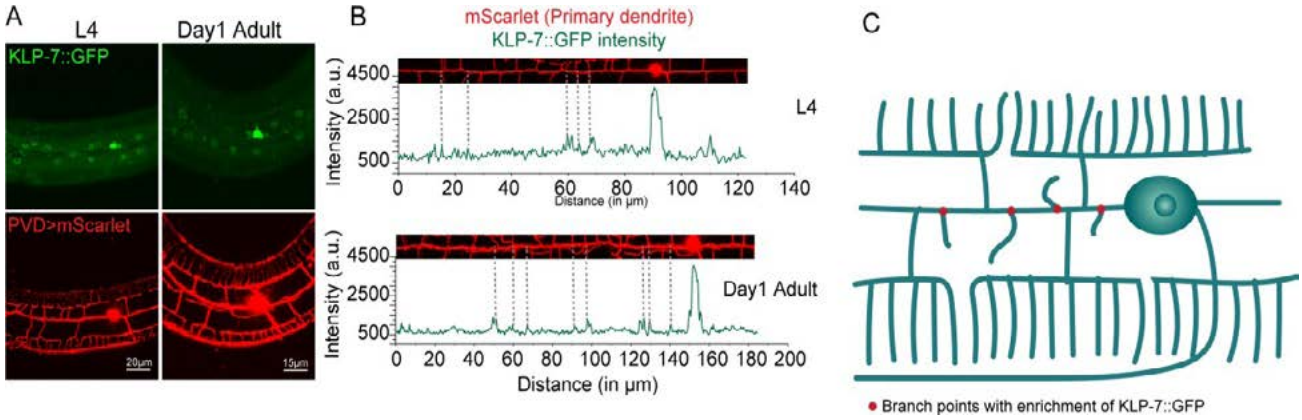


Figure 4: Distribution of KLP-7 in the PVD neurons. (A) Representative images of endogenously expressed KLP-7::GFP with the PVD marker mScarlet at L4 and Day 1 adult stages. (B) Line scan profile of KLP-7::GFP in the primary dendrite of PVD neurons correlated with the branch points as observed by mScarlet. (C) Schematic representing enrichment of KLP-7::GFP at the incipient branch points of PVD neuron

KLP-7 determines the dendritic localization of dynamic microtubules and neuronal polarity

As KLP-7 is known to affect the MT dynamics and orientation, we observed the dynamic microtubules by tracking the plus ends by EBP- 2::GFP. We expressed EBP-2::GFP under the PVD specific promoter where the reporter specifically localized to the primary dendrites in the wild type. The primary dendrite towards the anterior direction i.e., major dendrite had minus-end out comets whereas the posterior primary (minor) dendrite had plus-end out orientation of the comets. The axon of PVD neurons was also having a plus-end out orientation of the comets (Figure 5A-B).

In the *klp-7(0)* mutant, we did not observe any drastic change in the relative orientation of the microtubules in the primary dendrites (Figure 5C). Interestingly, in the *klp-7(0)* mutant, dynamic microtubules were also observed in the secondary and tertiary dendrites (Figure 5A, C). It is possible that stabilization of the microtubules due to loss of *klp-7* function causes ingression of microtubules in the higher-order branches. However, as the microtubule orientation was not changed drastically, KLP-7 is not the sole regulator of microtubule dynamics and orientation. Other microtubule regulators like Patronin and Kinesin-1 are known to regulate the microtubule dynamics and orientation in the PVD dendrites.

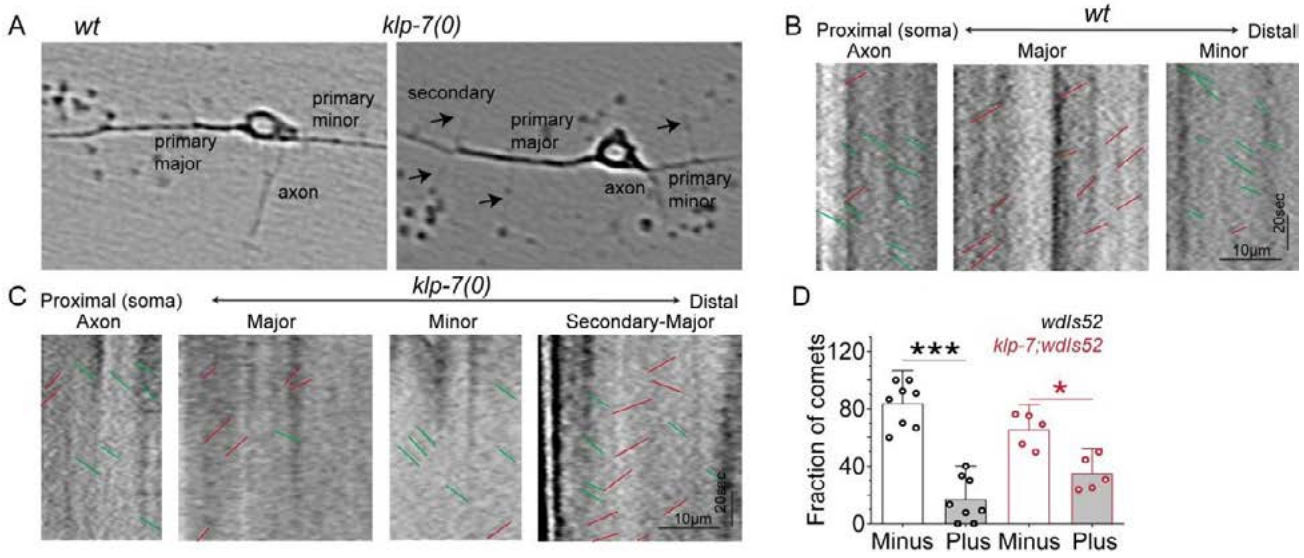


Figure 5: Microtubule dynamics and orientation in the PVD neurons in wildtype and klp-7(0). (A) Representative images of reporter EBP-2::GFP expressed in the PVD neurons of wildtype and klp-7(0) mutant. (B-C) Kymographs extracted from the axons, major, and minor dendrite of wildtype (B) and klp-7(0) mutant (C). In the klp-7(0) mutant, EBP-2::GFP comets were also observed in higher-order branches like secondary (black arrows in A), also represented in the corresponding kymograph (C). (D) Quantification of the relative number of the comets in plus and minus end out orientation in the major dendrite of wildtype and *klp-7(0)* mutant. Comparison of means is done using ANOVA with p<0.05*, 0.001***

Previous studies have documented the conversion of the dendrites to axon-like identity as a consequence of the loss of Kinesin-13. We also made similar observations in the PVD neurons by using an axonal cargo reporter, mCherry::RAB-3 (Figure 6A). This reporter is present only in the axons of wild type neurons whereas, in the loss of *klp-7* function, mCherry::RAB-3 was mislocalized to the secondary, tertiary, and sometimes to the quaternary dendrites (Figure 6A-B). This dendro-axonal conversion is reminiscent of taxol-treated developing neurons. Since the overall dendritic morphology is maintained, it is possible that during the neuritogenesis of PVD in *klp-7(0)* mutant, the stabilized microtubules misdirected the axonal cargoes to the dendrites.

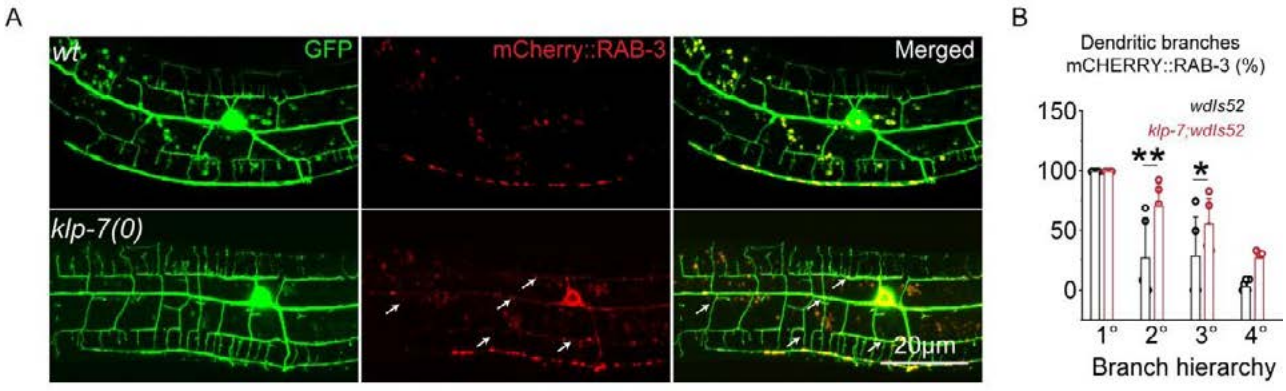


Figure 6: Neuronal polarity in the wildtype and klp-7(0). (A) Representative images of PVD neurons labeled with GFP and mCHERRY::RAB-3 at the L4 stage in the wildtype and loss of klp-7 function mutants. White arrows mark the mCHERRY::RAB-3 punctae in dendrites. (B) Quantification of the percentage of branches containing mCHERRY::RAB-3 punctae in the major dendrite of wildtype and klp-7 loss of function mutant at the L4 stage. Comparison of means is done using ANOVA with p<0.05*, 0.01**, 0.001***

In conclusion, the growth and shrinkage of the branches is an alternative to dendrite pruning. This dynamic equilibrium of the branch formation might be a calibrated way of building the dendritic territory. KLP-7 maintains the dynamic equilibrium of the dendritic branch density. Possible functions of KLP-7 may include generating dynamic microtubules for branch initiation, or excessive microtubule depolymerization for branch retraction. During mitosis, Kinesin-13 localizes with the γ-tubulin and the centrosomes where it regulates the movement of chromosomes by poleward chewing of the microtubules. As γ-tubulin localizes to the branch points of the PVD dendrites, KLP-7 might be employing similar mechanisms to regulate the microtubule dynamics locally and branch density (Figure 7). We will be investigating this further using live imaging of microtubule dynamics, KLP-7, and γ-tubulin in the dynamic branch points.

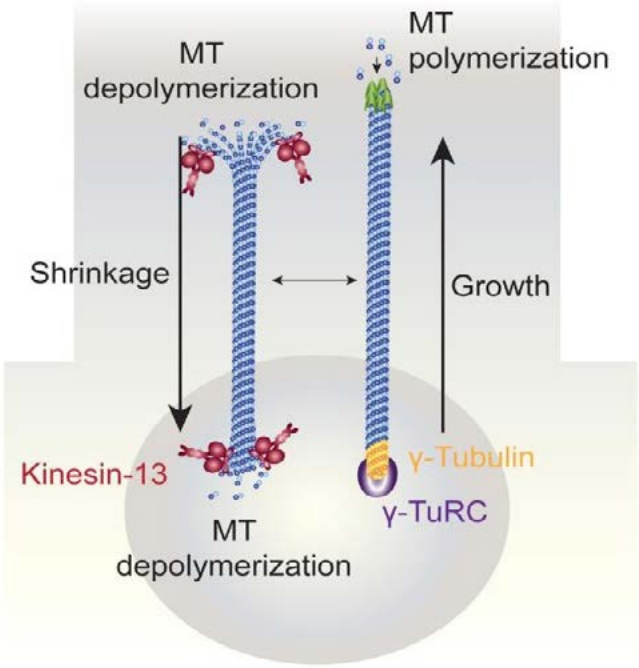


Figure 7: Possible role of KLP-7 in dendrite branching

Publications:

None

Presentations:

Swagata Dey, Harjot Kaur Brar, Shirshendu Dey, and Anindya Ghosh-Roy: Dendrite regeneration is independent of DLK/MLK pathways of axon regeneration in PVD neurons of *Caenorhabditis elegans* (Virtual) organized by Cold Spring Harbor Laboratory. September 2020

Funding:

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Collaborator:

0

Awards

0

Degrees Awarded (Ph.D.):

0

Meetings/Conferences organized:

0

Major Research Programs

Dementia Science Programme

A national level research programme

Funded by

Department of Biotechnology

Coordinated by

National Brain Research Centre

Dementia is a devastating condition. It is a progressive disorder that is characterized by impairment in memory and other cognitive abilities. Alzheimer's disease accounts for majority of the dementia cases. Other conditions include vascular dementia, fronto-temporal dementia and Lewy body dementia. With increase in the number of people with advanced age due to increase in life expectancy combined with other risk factors, the prevalence of dementia is predicted to increase substantially in the coming years. This will lead to tremendous increase in the burden on families, the care givers, the healthcare system and the society at large. The increase in dementia cases is predicted to be more in the developing countries including India than the developed countries. For these reasons, it is important to understand the processes involved in the development of dementia, and to identify potential window of therapeutic interventions for this devastating condition.

This chronic disorder is relatively less explored in our country. Given that it is extremely important to understand different aspects of Dementia, Department of Biotechnology provided funding for Dementia Science Programme aimed at comprehensive investigation on different facets of this disorder. The Programme aims to collect reliable data regarding prevalence, incidence, biomarkers and risk and protective factors for this disorder. This multi-centric Programme involves researchers and clinicians from across the country. As part of the Programme, long-term population-based and hospital-based cohorts of dementia patients will be established, and followed up. A unique feature of the Programme is that for diagnosis and classification of dementia, all the participating centres will use robust and uniform criteria that have been internationally accepted and validated in the Indian context. The Programme also involves imaging, basic biology and genetic studies. Another important aspect of the Programme is to set up a Bio-repository of samples from normal individuals and dementia patients, and to establish a long-term data storage facility at NBRC. The samples stored in the bio-repository and the data stored in the data storage facility will serve as very valuable resources for further studies.

The Dementia Science Programme is coordinated by Director, NBRC with Dr. Shiv Sharma, NBRC and Dr. NK Arora, INCLEN Trust International, as co-coordinators. The participating sites, in alphabetical order, are given below. All India Institute of Medical Sciences, New Delhi.

- Bangur Institute of Neurosciences, Kolkata
- National Brain Research Centre, Manesar
- National Institute of Mental Health and Neurosciences, Bengaluru
- North Eastern Indira Gandhi Regional Institute of Health and Medical Sciences, Shillong
- Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram
- The INCLEN Trust International, New Delhi

University of Calcutta, KolkataTo formulate robust and uniform criteria for diagnosis and classification of Dementia, several discussion meetings with participating investigators and other experts were held. These meetings helped shape the final Protocol, which is now ready. Standard operating procedures have almost been finalized for different aspects of the study. Before starting large scale recruitment of participants, it was necessary to conduct pilot studies in using the uniform Protocol. Thus, pilot studies were undertaken in Bengaluru, an urban community site, and in Palwal, a rural community site. These pilot studies identified certain limitations. Discussion meetings were then held to tweak the Protocol in the light of the lessons learnt during the pilot studies. After these important activities, the participating sites are almost ready to start recruiting the participants.

NBRC Flagship program

PIs (Brain Imaging)

Dr Arpan Banerjee

Dr Dipanjan Roy

PIs (Bio-banking and genetic analysis)

Dr Shiv Kumar Sharma

Dr Anindya Ghosh Roy

Tracking mental health over lifespan The science of well being

NBRC has secured funds from Department of Biotechnology in support of its flagship program for brain mapping of common mental disorders (CMD) of India. In Phase I the project involves collection of brain imaging and biological sample by building cohorts and identifying imaging phenotypes from anxiety, depression, bipolar and post-traumatic stress disorder (PTSD) – together defined as CMD. In Phase 2, the project will involve linking of brain imaging and molecular phenotypes by Artificial intelligence / machine learning tools. Goals of the flagship program (Phase 1) are following

- To build a new big-data repository comprising of brain imaging (structural and functional) data of normal and patients with common mental disorders (CMD) comprising of anxiety, depression, OCD and PTSD, where AI-based techniques can be implemented over the long-term.
- Quantification of changes in brain's structural and functional networks during resting state and with naturalistic stimuli in a cross-sectional adult life span data from general population (in the age range between 18-80 years).
- Identification of parameters to differentiate between different etiologies of cognitive impairment in CMD subtypes comprising of anxiety, depression, OCD and PTSD.
- Predictive regression-based model to extract relationship among mental health measures in different phases of CMD in the age range between 18-80 years and overlapping brain connectivity patterns • Collection of blood samples to build a biobank for genetic analysis in Phase II

The flagship project is of immense clinical and research importance to the nation and will be impactful in treatment and drug discovery. The project will provide normative data in populations that has not been accessed by previous studies and be an important resource hub for a variety of researchers from eminent technological institutions in the country. The expected outcomes from this project are as follows:

- Creation and dissemination of a public database that will include normative data from 200 healthy volunteers and 200 mental health affected patients.
- Several imaging derived phenotypes (IDP) that include information about specific brain structures and their connections by the end of Phase 1.
- Creation of a bio-bank in Phase I that will store blood samples from healthy and patient participants. In phase 2 genomic DNA will be prepared from the blood samples and subsequently whole-genome sequencing and analysis will be done

Publications, Patents & Presentations

Publications

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Presentations

Anindya Ghosh Roy: IBRO school organized by IGIB, "Regulatory RNAs and the Brain: Development to Disease".

Anindya Ghosh Roy: Keynote speaker in the Brain Awareness Week on 17th March 2021 organized by Centre for Cognitive and Brain Sciences at the Indian Institute of Technology Gandhinagar, Title of seminar: "C. elegans as a model for nerve regeneration study"

Anirban Basu: (2021) Host MicroRNA: An important modulator of antiviral immunity in Japanese Encephalitis virus infection. IBRO-APRC Associate school of Neuroscience, CSIR-IGIB, Work shop theme: Regulatory RNAs and the Brain: Development to Disease" Session theme: Neurovirology: Regulation by RNA, 20-26th January, 2021.

Anirban Basu: (2020) Delivered a lecture on "Brain and it's health", curtain raiser by INSA for IISF-2020. 4th of December, 2020

Anirban Basu: (2020) Modulation of Neural Stem/Progenitor Cell response following Japanese Encephalitis Virus infection, Annual meeting of Society for Neurochemistry (SNCI):11-13th December, 2020

Anirban Basu: (2020) Innate Immunity in the central nervous system: Redefining the relationship between "Immune system" and "Nervous system". KT Shetty Memorial Oration of Indian Academy of Neurosciences (IAN) for the year 2019. Oration delivered in the occasion of XXXVIII Annual Conference of Indian Academy of Neurosciences, October 4 – 7, 2020.

Anirban Basu: (2020) Modulation of Neural Stem/ Progenitor Cell response following Japanese Encephalitis Virus infection: Webinar entitled: Host-Microbe Interactions: Present and Future Perspectives; School of Biotechnology, Department of Life Sciences, Presidency University, Kolkata, 7th August, 2020

Anirban Basu: (2020) "ACS Science Talks." "Drug repositioning/repurposing: Promising strategy to develop therapy against viral infections." 31st July 2020.

Arpan Banerjee: Invited Talk: March 2021: Invited speaker at Royal Society Yusuf Hamied Workshop for India and the UK organised by the Royal Society (UK) and the Indian National Science Academy (INSA)

Arpan Banerjee: Invited Talk: Jun 2020: Git Commit Show (International Online developer conference) Can data science lead us to the grand unified theory of brain function?

Dipanjan Roy: Invited Talk: Tracking cognitive brain dynamics through lifespan" Fifth Brain Mapping & AI workshop IIT Delhi 11-20th March 2021.

Dipanjan Roy: Invited Talk: Tracking Structure-Function relationship and causal dynamics associated with lifespan" Global Neuroscience Partnership meeting NBRC, NIMHANS, and University of Iowa USA Organized by Sourav Banerjee, Ted Abel and Marco Hefti November 13, 2020.

Dipanjan Roy: Invited Talk: Characterizing Variability of Audiovisual Speech Perception Based on Prestimulus Oscillatory Features of Electrophysiological Brain Signals Vinsea A V Singh,Vinodh G. Kumar , Arpan Banerjee, Dipanjan Roy Bernstien Conference 2020, Annual Meeting of the Bernstein Network in Computational Neuroscience, Germany. September 29- October 2, 2020; Virtual Mode.

Dipanjan Roy: Invited Talk: Dynamic Repertoire of Brain During rest and task Nisha Shastry, Dipanjan Roy, Arpan Banerjee, Bernstien Conference 2020, Annual Meeting of the Bernstein Network in Computational Neuroscience, Germany. September 29- October 2, 2020; Virtual Mode.

Pankaj Seth: Guest Speaker: *Brain Fog in COVID-19 patients*. During National Science Week, Indira Gandhi University, Meerpur, Harayana, India February 22, 2021.

Pankaj Seth: Invited Speaker: *SARS-CoV2 is more than a respiratory virus-implications in COVID19 patients*. Jagrukta Abhiyan (General Awareness) on COVID-19, a national level online program organized by National Academy of Sciences (India), January 12, 2021.

Pankaj Seth: Invited Speaker: *How viruses affect human brain?* NBRC Foundation-day talk series, National Brain Research Centre, Manesar (Gurgaon), December 13, 2020.

Pankaj Seth: Invited Speaker: *SARS CoV2 is more than a respiratory virus, its consequences on Brain,* Motilal Nehru National Institute of Technology, Allahabad, and Indian Young Academy of Sciences (INYAS), New Delhi, India, November 7, 2020.

Pankaj Seth: Invited Speaker: *COVID19 and Brain connection,* at the 38th Annual meeting of Indian

Academy of Neurosciences, organized by University of Hyderabad, Hyderabad, during October 4-7th, 2020.

Pankaj Seth: Invited Speaker: *How do viruses affect the human brain?* During the 150 years celebrations of the Rawenshaw University, Odhisha, Health and Disease: Contemporary concerns, September 12, 2020.

Pankaj Seth: Invited Speaker: *SARS-CoV-2 is more than a respiratory virus -its potential in neuropathogenesis in COVID19 patients,* at the Bilateral Indo-US Webinar on COVID Biology jointly organized by IISER-Kolkata, IISC-Bangalore, University of Pennsylvania, USA and University of Colorado, USA, on August 17, 2020.

Pankaj Seth: Invited Speaker: *Molecular Mechanisms used by viruses to affect human brain,* at Webinar which was part of the Lecture Series on contemporary issues in biosciences organized by School of Life Sciences, Mahatma Gandhi Central University (MGCU), Motihari, Bihar, India June 15, 2020.

Pankaj Seth: Symposia Speaker: *Viruses make Friends Turn Foe: implications in neuropathogenesis following HIV and Zika infections,* Monsoon Brain meeting organized jointly by Indian Institute of Science (IISc), Bangalore and IIT- Kanpur, during June 24-26, 2020.

Pankaj Seth: Guest Faculty: *Research methodologies for beginners* as a Webinar for an Online workshop organized by the Department of Biotechnology, Maharishi Dayanand University, Rohtak, Haryana, India May 5, 2020.

Pravat Mandal and Sourav B sir's talks pending

Shiv Kumar Sharma: Delivered a lecture on "Dementia and herbal compounds" as part of India International Science Festival (IISF2020) curtain raiser event organized by Indian National Science Academy, New Delhi.

Shiv Kumar Sharma: Delivered a lecture on "Mechanisms of memory impairment in Alzheimer's disease", on World Alzheimer's Day, 21st September, 2020 in "Online Webinar and Panel Discussion" organized by Interdisciplinary Brain Research Centre, Aligarh Muslim University, Aligarh.

Bhavani Shankar Sahu: 5-9-2020, Webinar on Neuroendocrine regulation of metabolic physiology, GN Ramachandran Science club, Vigyan Prasar, MAC FAST, Kerala.

Mayanglambam Dhruba Singh: Neuroscience with Fruit fly, Indian International Science Festival (IISF), presented virtually, December 2020.

Thirugnanasambandam N: Site-specific decrease in cortical reactivity during sensory trick in cervical dystonia patients. 7th Asia-Oceania Congress of Clinical Neurophysiology, Kualalumpur, Malaysia (virtual), January 2021.

Thirugnanasambandam N: Bridging the gap between intracortical mechanisms and behavior. 7th International Conference on Non-invasive Brain Stimulation, Bbaden-Baden, Germany (virtual), November 2020.

Thirugnanasambandam N: Rhythms of the Brain. National Brain Research Centre (NBRC) Outreach event at the India International Science Festival, India (virtual), December 2020.

Thirugnanasambandam N: Evaluating cortical connectivity with non-invasive brain stimulation, Monsoon Brain Meeting, India (virtual), June 2020.

Swagata Dey, Harjot Kaur Brar, Shirshendu Dey, and Anindya Ghosh-Roy: Dendrite regeneration is independent of DLK/MLK pathways of axon regeneration in PVD neurons of *Caenorhabditis elegans* (Virtual) organized by Cold Spring Harbor Laboratory. September 2020

Externally Funded Research Projects

List of Extra Mural Projects as on date 31.03.21 (of the Financial Year 2020-21)

S. No.	Name of P.I.	Project S.No.	Name of Project	Name of the Implementing Agency	Date of Sanction of Project	Original Sanctioned Cost (Rs. In Lakh)	Actual Sanctioned/ Rele ase Amount for F.Y. 2020-21	Date of Complition	Sanction No.
1	Prof. Anirban Basu	1	Deciphering ANTIVIRAL Properties of Statins against Japanese Encephalitis Virus Infections	D.B.T.	26.12.2018	30.00	4.35	25.12.2021	BT/PR27796/ MED/29/1301 /2018
		2	MicroRNA mediated regulation of neural stem/ progenitor cell fate in neurotropic flaviviral infection	D.B.T.	29.12.2017	77.07	0.00	28.06.2021	BT/PR22341/ MED/122/ 55/2016
		3	Understanding the therapeutic role of adult stem cell derived exosome in combating virus induced neurodegenerative disease	D.B.T.	20.03.2018	25.50	8.10	25.12.2021	BT/PR15984/ MED/31/ 325/2015
		4	MicroRNAs as a potential therapeutic target in Neuro tropic viral infection (Tata Innovation fellowship)	D.B.T.	01.05.2015	45.00	0.00	30.04.2020	BT/HRD/35/01 /02/2014
		5	Elucidating the role of long non coding RNAs (lncRNAs) in neuronal cell death during Japanese Encephalitis (JE)	D.B.T.	05.03.2019	19.00	2.16	04.03.2022	BT/PR26590/ MED/122/ 133/2017
2	Dr. Ellora Sen	6	Inflammation regulated metabolic reprogramming Implications in tumor progression (UOE)	D.B.T.	30.03.2015	172.90	0.00	29.03.2021	BT/MED/30/ SP11016/2015
	Dr. Soumya Iyengar	7	Exploring Auditory Perception in House Crows using Functional Magnetic Resonance	D.S.T.(SERB)	20.05.2020	21.86	7.87	19.05.2023	CRG/2019/ 002672
		8	Autism spectrum disorders, genes and the gut microbiome: Utilizing song birds (Zebra Finches) as a model system	D.S.T.	24.02.2021	41.45	13.51	23.02.2024	DST/ CSRI/2017/69
		9	The sensitive period of the human auditory cortex a neuroanatomical study	ICMR	25.09.2019	42.10	0.00	24.09.2022	51/4/2019- ANA/BMS
4	Prof. Pankaj Seth	10	Differentiation of fetal neural stem cells to oligodendrocytes- a disease model to decipher the pathogenesis and devise therapeutic strategies for cerebral plasy	D.B.T.	19.03.2018	20.80	0.00	18.03.2021	BT/PR17581/ MED/31/ 333/2016
		11	Insights into role of a dyslexia linked long non-coding RNA(lncRNA) in human neural stem cell	D.S.T.	18.07.2017	76.97	0.00	17.07.2020	SR/CSRI/210 /2016(G)
		12	Hypoxia Induced Changes in Blood Brain Barrier	D.B.T.	12.09.2018	34.66	6.50	11.09.2021	BT/PR23625/ MED/122 /77/2017

S. No.	Name of P.I.	Project S.No.	Name of Project	Name of the Implementing Agency	Date of Sanction of Project	Original Sanctioned Cost (Rs. In Lakh)	Actual Sanctioned/ Rele ase Amount for F.Y. 2020-21	Date of Complition	Sanction No.
		13	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	66.96	19.85	15.10.2021	BT/PR21413/ MED/122 /40/2016
		14	Role of Ephrins/Eph receptors in HIV mediated neuropathogensis	D.B.T.	27.06.2019	73.16	15.56	26.06.2022	BT/PR27512 /122/146/2018
5	Dr. Dipanjan Roy	15	Oscillatory network dynamics in perceptual learning	D.S.T.	23.08.2017	50.55	0.00	22.08.2020	SR/CSRI/21 / 2016(G)
		16	Role of default mode brain network is normal cognitive function	D.B.T.	26.05.2016	88.00	0.00	26.05.2021	BT/RLF/ Re-entry/07/2014
		17	Dementia Science Program- Tissue MRI Studies	D.B.T.	18.12.2017	35.41	0.00	17.12.2021	BT/HRD/ Dementia/2017
		18	Unravelling the causes of stroke and cognitive decline in general population A cross-Cultural perspective (DBT Netherland Grant)	D.B.T.	21.04.2016	73.66	0.00	20.04.2022	BT/IN/ Netherlands/ 03/ KP/2012
		19	Novel Imaging Diagnostics for Alzheimer's Disease	D.B.T.	24.01.2018	151.26	28.14	23.01.2021	BT/Indo-Aus/ 10/31/2016
		20	Dementia Science Program- Imaging Studies	D.B.T.	18.12.2017	35.41	0.00	17.12.2021	BT/HRD/ Dementia/2017
		21	Construction of an India population specific brain templete	D.S.T.	11.05.2016	26.12	0.00	09.11.2020	SR/CSRI/ 229/2014(G)
		22	Artificial intelligence for early predictive diagnosis of alzheimer's disease using multi model imaging data	Meity	17.09.2019	59.96	0.00	16.09.2022	4(5)/ 2019-ITEA
	Dr. Sourav Banerjee	23	CRISPR-Cas13- mediated engineering of endogenous long non-coding RNAs for fluorenscent tagging to study RNA dynamics	D.B.T.	29.02.2020	72.00	0.00	28.02.2023	BT/RLF/ PR31811/ GET/119/ 285/2019
		24	Regulation of Fear memory formation by long non-coding RNAs and RNA biniding protiens: Mechanism of combinational control	D.S.T.(SERB)	25.03.2019	52.29	11.00	24.03.2022	SERB/F/ 12655/2018-19
8	Director NBRC	24	Magentoncephalography (MEG) Resource Facility	D.B.T.	27.03.2018	1498.86	361.31	26.03.2022	BT/MED/122/ SP24580/2018
		25	Dementia Programme	D.B.T.	14.09.2007	37.50	0.00	31.03.2021	BT/PR-NBRC/2008
		26	Delcon (E- Laibrary Consortia) Project	D.B.T.	18.03.2009	37123.67	2384.35	31.3.2022	BT/ BI/12/053/2012
		27	Comparative Mapping of common mental disorders (CMD) over lifespan	D.B.T.	29.09.2019	477.05	0.00	28.09.2022	BT/MED/-III/ NBRC/Flagship/ program/2019
		28	Demetia Science Programme- Coordination Administration & Management setup at DBT	D.B.T.	18.12.2017	530.42	0.00	17.12.2021	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	Original Sanctioned Cost (Rs. In Lakh)	Actual Sanctioned/ Rele ase Amount for F.Y. 2020-21	Date of Complition	Sanction No.
9	Dr. Shiv Kumar Sharma	29	Demetia Science Programme- Basic Biology Studies (i) Genetic Studies at lab	D.B.T.	18.12.2017	106.64	0.00	17.12.2021	BT/HRD/ Dementia/2017
10	Dr. Arpan Banerjee	30	Early Diagnosis of Structural and Functional Decline in Brain Circuits Stemming from Traumatic Injuries in Professional Athletes Playing Centact Sports	MYAS	14.02.2019	40.00	0.00	13.02.2022	K-15015/ 42/2018/SP-V
11	Dr. Anindya Ghosh Roy	31	Wellcom Trust/DBT Indian Alliance	D.B.T.	01.12.2013	321.93	0.00	25.02.2021	IA/I/13/1 /5000874
		32	Study of Neuronal Regeneration after Injury using Caenorhabditis Elegans	SERB	20.05.2020	50.72	21.35	19.05.2023	CRG/2019/ 002194
12	Dr. Bhavani Shankar Sahu	33	Understanding the regulated secretory pathway and its role regulating physio-metabolic fuctions	D.B.T.	16.12.2019	_	17.00	15.12.2021	BT/RLF/Re-entry/38/2016
		34	IBRO Returned Home Start-up Grant	IBRO	19.06.2020	_	8.12	18.06.2022	IBRO Return Home Program 2018
		35	ICGEB Research Grant	ICGEB	01.01.2021	$4500	$1500	31.12.2024	BSS/ICGEB/ 0121/122
13	Dr. Sandeep Kumar	36	Post Doctoral Fellowship	SERB	06.04.2018	19.20	0.00	05.04.2020	PDF/2017 /001610
15	Dr. Swagata Dey	38	Wellcom Trust/DBT Indian Alliance	D.B.T.	01.01.2020	167.73	36.75	31.12.2025	IA/E/18/ 1/504331
17	Dr. Ashraful Hassan	40	DBT-TWAS Fellowship	D.B.T.	11.10.2019	5.67	5.67	10.10.2020	DO/CCSTDS/ 144/2019
18	Dr. Nivethida	41	Wellcom Trust/DBT Indian Alliance	D.B.T.	01.07.2017	378.7	1.58	30.06.2022	IA/CPHI/16/ 1/502624

Distinctions, Honors & Awards

Course-Work

M.Sc. 2019

Ms. Debapriya Roy

This M.Sc. student has been awarded first rank upon completion of course-work during the year 2019–20, and a certificate was given to her.

Ms. Aamna Jain

This M.Sc. student has been awarded first rank upon completion of course-work during the year 2019–20, and a certificate was given to her.

Mr. Ankit Kumar Shah

This M.Sc. student has been awarded second rank upon completion of course-work during the year 2019–20, and a certificate was given to him.

Ph.D. 2019

Mr. Chandramouli Mukherjee

This Ph.D. student has been awarded first rank upon completion of course-work during the year 2019–20, and a certificate was given to him.

Ms. Sakshi Shukla

Academic Programmes

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 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.

1. Ph.D. 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 ₹ 31,000/- per month for Junior Research Fellows and ₹ 35,000/- per month for Senior Research Fellows.

2. M.Sc. 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 a fellowship of ₹12,000/- per month. NBRC inducts students for its M.Sc. (Neuroscience) and Ph.D. programmes from diverse backgrounds having Bachelors or Master's degree in any branch related to Neurosciences, Psychology or M.B.B.S., B.E., or B.Tech. NBRC recognizes that understanding brain functions requires a fusion of knowledge from multiple disciplines.

3. 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.

NBRC
Facilities

Information Technology Communications Cell (ITCC)

The Information Technology and Communications Cell (ITCC) of NBRC manages the overall Information and Communications infrastructure of the Institute apart from aiding in R&D activities. Previously, ITCC functioned as Distributed Informatics Centre (DIC) under the BTISNET initiative of Department of Biotechnology. ITCC manages the campus converged network (data and voice traffic), communications links (Network and PSTN), Institute's Data centre, cloud resources running from NIC cloud, application servers, software development, ICT Modernization, e-Governance initiatives, technical support to users, common computing facility etc. Some of them are summarized as under:

1. Campus Converged Network: (NBRC-IntraNet)

The NBRC campus network consists of campus wide Local Area Network running on 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 is built in the network architecture. The network is supplemented with 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 a IPv6 compliant and IPv6 services are functional in 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 1 Gbps optical fibre link provided by BSNL that is further supplemented with a 50Mbps 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 NBRC-AIIMS data pipeline for MEG as part of 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.

2. IP-PBX Facility

The tele-communication 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 user endpoints are IP Phones connected to LAN. The facility is running on automatic failover mode on virtualized servers from institute's datacenter. 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 end-points.

3. Institute Core and Application Servers

The computing facility manages and maintains the server infrastructure of the institute which are housed and maintained in the data centre facility. In essence, the institute currently has five fully utilized 42U server racks in the datacenter facility. The various services running on these servers can be classified as follows:

- Web-servers for the institute and various webservers related to ongoing computational projects and applications of various scientific groups . The primary webserver for official website - running from VM's installed on NIC cloud.
- Acting as liaison with NIC for maintaining emails of core employees on NIC mail services (gov. in/nic.in). Management of in-house email on list server (nbrc.ac.in) for temporary staff, students, project personnel for broadcast and academic purpose.

- DNS servers for the official and hosted domains which are running from NBRC datacenter as well as VM's hosted on NIC cloud.
- Virtualization servers for providing virtualized hardware to run various applications and service in a more systematic manner and to consolidate and utilize the existing physical server infrastructure.
- New Central Storage servers of 400TB has 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 g. 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.
- Antivirus and security servers for providing protection to user end-points across the campus.

4. Other Facilities & Services

- **NIC Cloud and Email Services:** The DIC unit 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 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's etc. An online support ticketing system with automated workflow management is functional for support activities.
- **CCTV Monitoring and Management:** The DIC has also installed IP-Cameras connected to the core network which has 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 requirements, several scientific and e-Governance applications have been developed in-house.
- **Infrastructure Improvement:** The computing facility also undertakes planning and implementation of new computational infrastructure facilities and services, software/ hardware/network upgradations of Institute computers/peripherals etc.
- **Video-conferencing:** Management of videoconferencing for official meetings, interviews and academic activities.

Animal Facility

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.

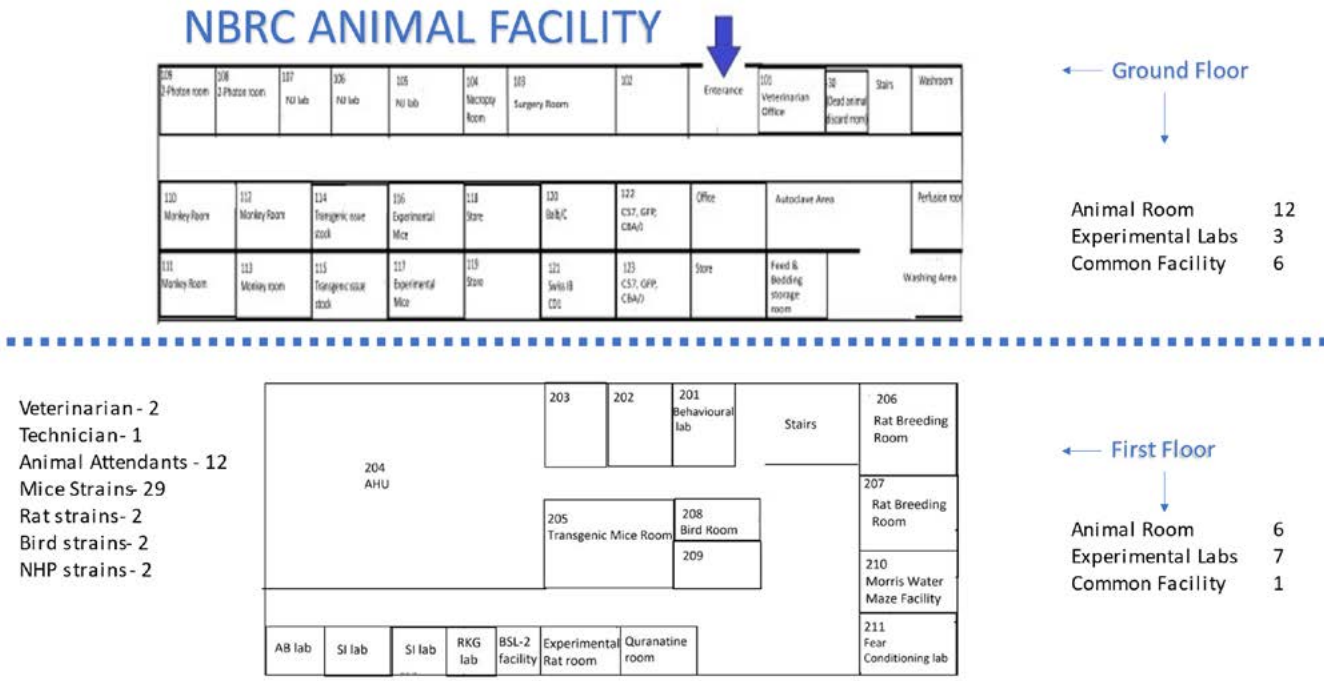
The Animal Facility is registered with the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Ministry of Environment and Forests, Government of India, New Delhi. (Registration number: 464/GO/ReBi-S/Re-L/01/CPCSEA; initially registered on 24/08/2001. All activities 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 CPCSEA.

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.

All the animal species are housed in species appropriate cages, which are designed as per the CPCSEA 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 the moved to fresh cages in cage-changing station under hepa-filtered air.



The animals are maintained under controlled environmental conditions as specified in CPCSEA guidelines, with temperature maintained between 22 ± 2° 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 CPCSEA 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.

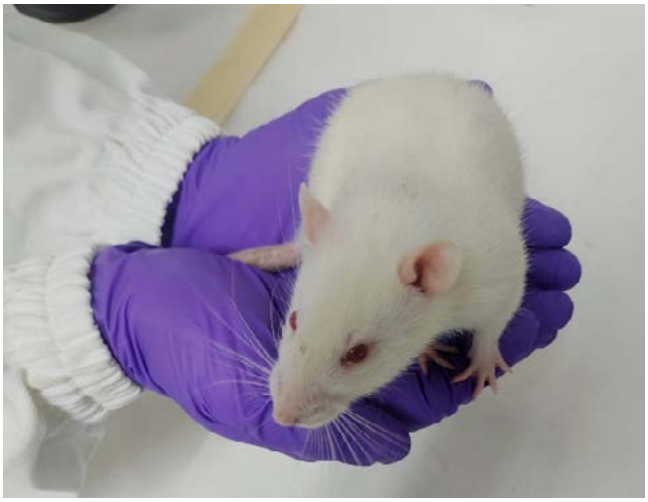
The animal facility has a state-of-art surgical suite equipped with intensity controlled surgical lights, advanced surgical microscopes, gas anesthesia machines, equipment for monitoring the physiological state of the animals, including heart rate monitor, pulse oximeter and rectal thermometer. For cleaning 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 is 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)
- UBC-GFP (Green fluorescent protein)
- B6CBA-Tg (Hdexon1) 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
- C57BL/6J-Tg(Thy1-GCaMP6s)GP4.3Dkim/J

Knock Out Mice

- UBE3A null mice (Angelman syndrome model)

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.

Digital 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 994 online journals through the DBT e-Library Consortium (DeLCON), 5 specialized journals, and 119 freely accessible online journals. It also maintains digital archives and news clips about the Centre and subscribes to 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 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 entertained an average of approximately 94+ requests for articles during this financial year (2020-21) 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 994 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
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

1. Dibrugarh University, Assam
2. Assam University, Silchar
3. North Eastern Regional Institute of Science & Technology, Arunachal Pradesh

4. North East Institute of Science & Technology, Assam
5. Mizoram University, Mizoram
6. D. M. College of Science (DMC), Manipur*
7. Sikkim University, Gangtok
8. College of Veterinary Science, Assam Agricultural University, Guwahati
9. Guwahati University, Assam
10. Manipur University, Imphal
11. College of Veterinary Science & Animal Husbandry Central Agricultural University, Mizoram
12. Rajiv Gandhi University, Arunachal Pradesh
13. Nagaland University, Nagaland
14. North-Eastern Hill University (NEHU), Shillong
15. St. Anthony's College (SAC), Meghalaya*
16. Indian Institute of Technology Guwahati, Assam
17. Tezpur University, Tezpur, Sonitpur, Assam
18. 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 → 8 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 → 40 Journals
- Nature Publications → http:// www.nature.com → 34 Journals
- Oxford University Press (OUP) → http://www. oxfordjournals. org → 22 Journals
- Springer India → http://www. springerlink.com → 343 Journals
- Microbiology Society (MBS) → http:// mic. sgmjournals.org → 3 Journals
- Wiley-Blackwell → http:// www3.interscience. wiley.com/cgi-bin/home → 86 Journals
- Elsevier Science (ScienceDirect) → http:// www. sciencedirect.com → 434 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 Journal (Only Archives from 2009-2018)
- American Society of Plant Biologists (ASPB) → http://www.aspb.org/→ 2 Journals (Only Archives from 2009-2018)
- American Association for Advancement of Science (AAAS)→ http://www.sciencemag.org → 3 Journals (Only Archives from 2009-2019)

- American Society for Hematology (ASH)→ http://bloodjournals.hematologylibrary.org →1 Journals (Only Archives from 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 minimum cap.
- 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 headquarters 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

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 equipment:

1. 3 Tesla Magnetic Resonance Imaging (MRI): Philips Achieva 3.0 T scanner
2. Electroencephalography (EEG): 64-channel Synamps 2 EEG system, Compumedics Neuroscan, Inc
3. Transcranial magnetic stimulation (TMS): Magventure MagPro

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 studies. 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 as follows:

1. MR Spectroscopy (MRS) which provides noninvasive 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 (see Fig 1) 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)

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 in 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 magnetic field. These 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.

Translational Research: Clinical Unit

The National Brain Research Centre (NBRC) has always been on the forefront when it comes to helping the nation to reduce the burden of neurological disorders in our country. Realizing the social responsibility and the need for serious efforts to address the growing cases of neurological diseases in our country, the National Brain Research Centre extended its expertise and support to the Government General Hospital (GGH), Gurgaon more than a decade ago. As the Government General Hospital, lacked the expertise of a neurologist and did not have a neurology department, the needy patients coming from Gurgaon neighboring districts and villages were turned away or refereed to other hospitals. We established the Translational and Clinical Neuroscience Unit and provided a neurology outpatient department to the government hospital to help the citizens and also assess the occurrence of neurological cases in this region.

The Translational and Clinical Neuroscience Unit initially started from room number 7 of the Government General Hospital, Gurgaon near that city bus stand in heart of the city. In 2019, as the GGH was shifted to Sector 10A in Gurgaon, and the unit was also transferred. We continue to cater the service to the needy citizens of state of Haryana.

Investigation facilities

As the unit is established at the hospital, patients visit the unit and get access to several facilities listed below through the hospital or its associated clinics:

1. MRI system: Siemens Magnetom 1.5 Tesla scanner with various study protocols
2. CT (computed tomography) system
3. Ultrasonography
4. X-ray and Contrast imaging
5. *Laboratory facilities*: Biochemistry, Microbiology, Hematology, Pathology & Immunology

The NBRC unit has highly qualified consultants and a support team of following personnel:

1. Consultant Clinical Professor, Neurology: Dr Rajnish Kumar
2. Consultant Clinical Professor, Pediatric Neurology, Dr. Rakesh Jain
3. Clinical Psychologist: Priyanka Kaushik
4. Clinic Assistant: Hanuman Singh

Understanding basic biology of a disease can only be achieved with a close coordination and crosstalk between clinicians and basic researchers with a common aim to reduce the suffering of patients Translational & Clinical Neuroscience Unit and minimizing their hospital stay and visits. The translational and clinical neuroscience unit of NBRC was established at Gurgaon Government Hospital with this aim and has succeeded to some extent. Previously, due to lack of neurological OPD, most of the patients were unattended and suffering. The Clinical Research Unit of NBRC along with the Hospital provides a bouquet of much needed services such as - Neurology, Neuropsychology, Neuropsychiatry, Behavioral therapy, Psychology, and Psychometry. The unit is supported by qualified consultants, including a DM in Neurology, at the outpatient department of the centrally located Government General Hospital. The consultant clinical faculty offer their services on one of the designated days of the week. As we had several pediatric patients visiting the NBRC unit, we added a Pedaitric Neurologist, Dr. Rakesh Jain, to the list of consultants. This has been much appreciated by the visiting patients and the hospital. The NBRC Unit has registered an increase in number of referrals of such cases by the pediatric department since last year.

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consultants. This has been much appreciated by the visiting patients and the hospital. The NBRC Unit has registered an increase in number of referrals of such cases by the pediatric department since last year.

Patients attending the OPD at Civil Hospital come from old Gurgaon township and the villages and towns in the surrounding districts of Haryana, while some come from neighbouring states as Rajasthan, Delhi, Uttaranchal, Himachal Pradesh, Punjab and Uttar Pradesh. Patients requiring advanced specialist neurology in-patient care are referred to All-India Institute of Medical Sciences (AIIMS), Institute of Postgraduate Medical Education & Research – Rohtak, Institute of Human Behaviour & Allied Sciences (IHBAS), New Delhi or to other tertiary hospital as per the choice of the patient, if he/she so desires.

We are in process of creating a database of all the patients that visit the neurology OPD, and wish to create a computer database with relevant patient data along with any planned imaging/molecular/neurophysiological studies at the NBRC laboratories. This would help in creating a thoroughly documented clinical window for NBRC and the neuroscience research community. In this effort to narrow the gap between Basic Neuroscience and Applied Neuroscience, an ethics committee has been formulated jointly with the Government General Hospital/Government of Haryana.

The NBRC Unit acknowledges the cooperation from the Ministry of Health - Government of Haryana, and the Deputy Commissioner - Gurgaon, and also from the Chief Medical Officer & Civil Surgeon and Principal Medical Officer of the Hospital. The translational and clinical research unit of NBRC provides the much-needed neurological OPD services for the patients from Gurugram and adjoining districts.

Magnetoencephalography Resource Facility

Under DBT-SAHAJ Scheme, Funded by 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:

Prof. Pravat K Mandal,

Director-in-charge, NBRC

Investigator from ACBR:

Dr. Aparna Dixit

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 as well as 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. Magnetoencephalography (MEG) resource facility is one of its kind in Northern India, proved very much fruitful in managing these patients. Till date, 1994 patients were evaluated using this facility from all over India (state-wise distribution of patients were shown in figure1. In fact, international patients have been also benefitted, especially patients from SAARC countries. 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 outcome following surgery in patients with drug refractory epilepsy. It is also important to know the extent of overlap of 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. SEEG implantation is usually done in MRI negative cases, when there is discordance between electro clinical and MRI, when there is overlap with eloquent cortex and dual pathology cases. Placement of SEEG electrodes is safe and effective technique to localize epileptogenic zone.

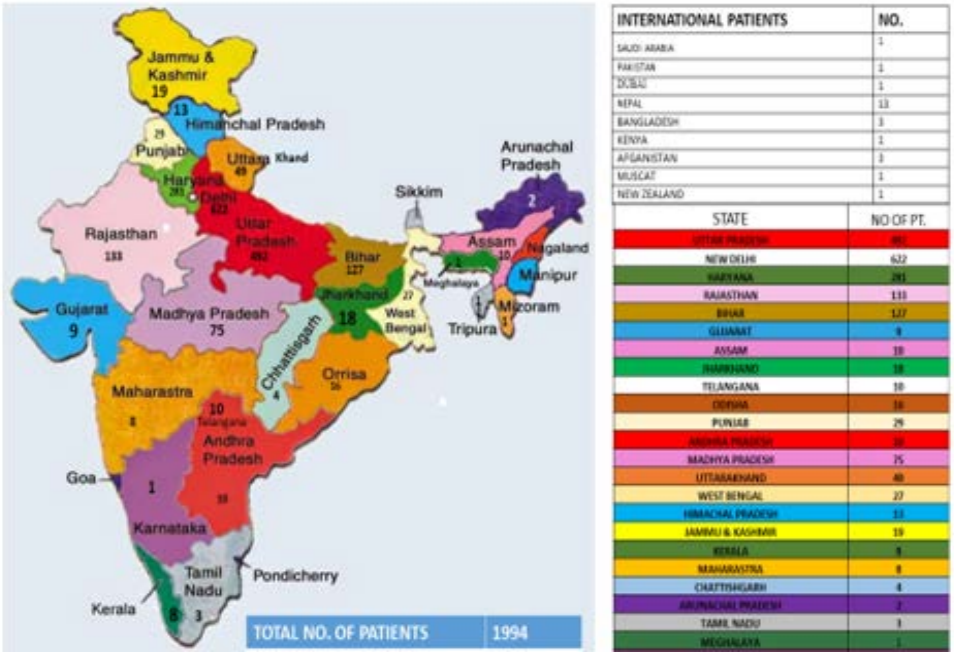
Magnetoencephalography (MEG) Resource Facility has established in NBRC it is collaborative between National Brain Research Centre (NBRC) and All India Institute of Medical Sciences (AIIMS) under the aegis of Department of Biotechnology (Government of India). MEG facility has entered in the prestigious **DBT-SAHAJ** structure and primary goal of DBT-SAHAJ infrastructure is to create "National service Facility" to provide access to resource.

This one of the few facilities in the world which brings together a premier medical science institute and a dedicated neuroscience research centre to study difficult-to-treat epilepsy. The main aim of the centre is to develop a cure for drug-resistant epilepsy by bridging the gap between

Figure 1: *MEG unit in MSR -NBRC-AIIMS*

clinical and basic research which is mediated by the close coordination between NBRC and 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. The NBRC component of the centre is using non-invasive protocol of magnetoencephalography (MEG) for the localization of epileptogenic focus

Total Number of patients scanned till Now =1994



INTERNATIONAL PATIENTS	NO.
SAUDI ARABIA	1
PAKISTAN	1
DUBAI	1
NEPAL	13
BANGLADESH	1
KENYA	1
AFGANISTAN	1
MUSCAT	1
NEW ZEALAND	1

STATE	NO OF PT.
UTTAR PRADESH	482
NEW DELHI	622
HARYANA	283
RAJASTHAN	133
BIHAR	127
GUJARAT	9
ASSAM	10
JHARKHAND	18
TELANGANA	10
ODISHA	16
PUNJAB	29
ANDHRA PRADESH	18
MADHYA PRADESH	75
UTTARAKHAND	49
WEST BENGAL	27
HIMACHAL PRADESH	13
JAMMU & KASHMIR	19
KERALA	8
MAHARASTRA	8
CHATTISHGARH	4
ARUNACHAL PRADESH	2
TAMIL NADU	3
MEGHALAYA	1

Figure 2: Showing the national and international distribution of total patients being evaluated at MEG, Resource facility

New Methods for MEG Analysis:

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

Patients have been analysed by various methods given below (01April 2020-till date):

No. of patients Analysed in DANA	No. of patients Analysed in CURRY	No. of patients Analysed in sLORETA	No. of patients Analysed in PSSL	No. of patients Analysed in High Frequency Interictal Ripples	No. of patients Analysed in High Frequency Ictal Ripples	Total No. of Patients have been Analysed
169	280	290	48	79	10	876

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.

Procedure of Electrode Implantation: Initial evaluation of patients with drug resistant epilepsy (DRE) usually done in three steps. First, detailed history of seizure semiology help in diagnosing the epilepsy syndromes and severity. Secondly, long term Video electroencephalography recording will help in identification of type and location of seizure onset. Third, high resolution Magnetic resonance imaging under epilepsy protocol to identify any structural lesion and developmental abnormalities. Additional physiological studies such as positron emission topography (PET) and Single-photon emission computed tomography (SPECT) and magnetoencephalography (MEG) may be required to strengthen the anatomo-electro-clinical (AEC) hypothesis. Invasive investigations are required when non -invasive studies not conclusive or discordant or dual pathologies are present. Invasive EEG includes placement of grid, strip and depth electrodes. These investigations can be used to map the eloquent areas of brain along with localization of epileptogenic focus.

The current research methodology deals with the localization of the epileptogenic networks using intra operative robotic driven electrodes to be placed in different areas of the brain (as decided pre operatively). This is followed by long term monitoring (5-7 days) till the patient develops habitual seizures. This may be further augmented by electrical stimulation of the different contact points of the electrodes using a 256 channel EEG system with a grid matrix cortical stimulation system. Such a strategy allows the physician to localize the epileptogenic networks.

Following this, the patient is subjected to a resective surgery which allows the surgeon to remove the abnormal epileptogenic network thus providing the best possible chance for the patient to become seizure free. Following surgery, the tissues will be provided various grading of abnormality and accordingly will be subjected to cellular electrophysiological and molecular characterization.

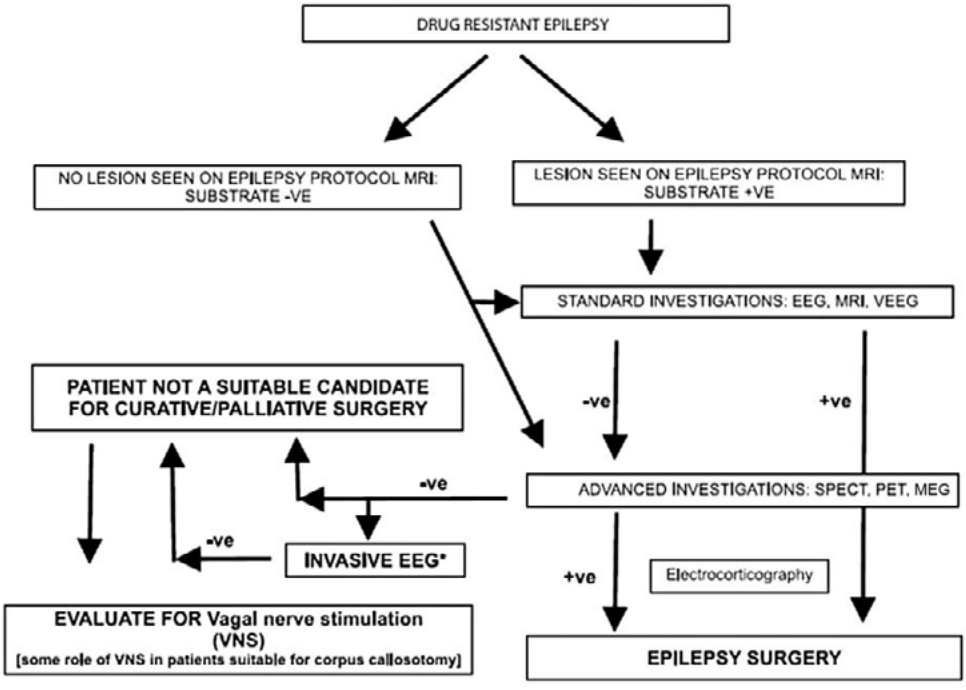


Figure 3: Showing various diagnostic algorithm used for epilepsy surgery protocol

The flowchart depicted in Figure 3 shows the various diagnostic algorithm used for patients with drug refractory epilepsy. As we can see, imaging forms the first modality of investigation in patients with DRE following standard investigations for localization and lateralization are carried out. Patients, where even advanced non-invasive modalities are not sufficient to pinpoint the seizure focus, undergo invasive stereotactic EEG and based on those

findings and patient suitability, are finally taken up for surgery or undergo other procedures.

We analysed data from our centre, of patients undergoing stereotactic electrode implantation from 2015 – 2019. 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. 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 anesthesia 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 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 and to ensure no hemorrhage. 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 anesthesia and sedation.

Results

Twenty-one patients (Table 1) underwent SEEG implantation during the study period at our center (AIIMS). Out of them 17 were males (80.9%). Mean age of patients is 21.7 years (Range- 1.5years to 44 years). Five patients were less than 18 years of age (35.7%). Reasons for SEEG implantation are tabulated in Table 1. Two patients (Patient 5 and 17) were not operated in view low frequency of seizures and 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 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 moths (range-5months 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 intresting reorganisation patters to the other hemisphere/ ant and superior to pars triangularis. Motor function also shifted to more anterion in 1 and posterior in another/ A.

Case No.	Age/ Sex	MEG Localisation	Language and motor functions by TMS navigated done/ clinical	Reason for SEEG implantation	No. of Electrodes	No. of Contacts	Region of Implantation	Surgery	Postop Complications (Post definitive surgery)	Follow Up (Months)	Seizure Outcome (ILAE Class)
1	15/M	Bilateral temporal	Usual/Average IQ, Impaired visual memory	Seizure localized, not lateralized	4	40	B/L hippocampi	Right ATL + AH	None	36	Class I
2	22/M	Left temporal and basitemporal	Aberrant/Average IQ, impaired verbal memory	Seizure localization discordant with other data	3	30	Left basifrontal, Posterior insula and left cingulate gyrus	ECOG guided Left Basifrontal and Insular Resection	Transient right hemiparesis	36	Class I
3	18/M	B/L temporal	Aberrant/Impaired verbal memory and attention	Seizure localisation is discordant with other data	4	40	B/L Hippocampi	Left ATL + AH	2 episodes of seizures in immediate post op period	28	Class I
4	10/M	Left posterior temporal, temporo-occipital and occipital	Aberrent/Average intelligence, Intact verbal and visual memory	Dual pathology	3	30	B/L occipitotemporal and left anterior temporal	ECOG guided left ATL and left basitemporal resection	None	23	Class I
5	22/F	Left lateral and basitemporal, temporooccipital, right posterior temporal	Aberrent/Extremely low IQ, impaired visual memory	Seizures were neither localized nor lateralized	6	64	B/l amygdala, hippocampus, lingula	No surgery (Infrequent seizures from eloquent cortex-lingula)			No surgery done
6	1.5/F	Right basitemporal and temporo-occipital	Not possible/Delayed milestones	Multiple heterotropias	3	30	B/l frontal and left atrial periventricular	RFA PVNH- Right frontal horn, left ventricular body and left atrial	Operated twice 3 months apart	22	Class V
7	30/M	Bilateral frontoparietal and bilateral temporal	Aberrant/Average IQ	Seizures were neither localized nor lateralized	6	64	Right amygdala, hippocampus, para hippocampus, Peri Rolandic, insula, Left insula	Corpus callosotomy with Right ATL + AH	None	19	Class IV
8	35/M	Bilateral posterior temporal, left angular gyrus and parieto-occipital	Aberrant/Average IQ	Seizures were neither localized nor lateralized	10	100	B/l superior temporal gyri, B/l amygdala, B/l hippocampi, B/l posterior temporal, B/l Temporo occipital	ECOG guided Left Superior Temporal Gyrus Excision	None	12	Class IV
9	21/M	Left basifrontal	Aberrant/Average IQ, Broca's area is posterior to the lesion	Seizures lateralized but not localized	6	60	B/l temporal, Right basifrontal, Left medial and lateral basifrontal, Left fronto insular	ECOG Guided Resection of Left Basifrontal FCD	None	10	Class I
10	13/M	Left parito-occipital, Wernicke's area, right parieto-occipital	Usual/Above average IQ, intact verbal and visual memory	Right insular FCD operated, with recurrent seizures with ?residual FCD. Seizure localization was discordant with other data	5	50	Right oblique insular, Right basifrontal, Amygdala & hippocampus, Residual FCD-posterior insula, Right temporo occipital	ECOG Guided Resection of Residual FCD and Basifrontal region	Left hemiplegia which improved to 3/5	11	Class I

Case No.	Age/ Sex	MEG Localisation	Language and motor functions by TMS navigated done/ clinical	Reason for SEEG implantation	No. of Electrodes	No. of Contacts	Region of Implantation	Surgery	Postop Complications (Post definitive surgery)	Follow Up (Months)	Seizure Outcome (ILAE Class)
11	11/M	Left lateral temporal neocortex, right parietal and mesial temporal	Aberrant /Intact memory with impaired abstract ability	Dual Pathology	4	40	Right premotor, motor and sensory, left thalamic	Excision of NCC on right motor cortex with MST	None	14	Class I
12	31/M	Right basifrontal, frontoparietal, left supramarginal gyrus	U/Average IQ, intact verbal and visual memory	Seizure localization discordant with other data	5	48	Left Broca's area, left superior frontal, middle frontal and lateral orbitofrontal cortex and medial orbitofrontal cortex	RFA of PVNH	None	14	Class I
13	21/M	Right posterior temporal	U/Average IQ, impaired visual memory	Dual Pathology	4	40	1 electrode anterior to right middle temporal DNET, right frontobasal, 2 electrodes anterior and posterior to right frontal gliosis	Right ATL + AH	None	8	Class I
14	28/F	Bilateral posterior tempral and left anterior temporal	Aberrant /High average IQ, impaired visual memry and immediate recall	Seizure localization discordant with other data	12	144	B/l anterior, middle and posterior temporal, left basi temporal, b/l cingulate, b/l anterior insular and right posterior insular	Left ATL +AH	None	10	Class V
15	22/M	Left pars triangularis	Usual /Intact verbal and visual memory, Motor speech area-bilateral represenation	Broca,s area mapping in relation to FCD	7	76	left pars orbitalis, triangularis, opercularis, anterior insula, anterior and posterior to FCD, anteroinferior to FCD	ECOG guided resection of FCD	None	10	Class I
16	27/M	Right frontal and temporal	High average IQ, Impaired visuospatial memory	Bilateral MTS	3	34	left amygdala,left hippocampus and right hippocampus	Left ATL +AH	None	10	Class I
17	24/M	Left temporal and occipital	Aberrant /Low IQ, delayed recall	Dual pathology	4	52	Left amygdala, hippocampus and bilateral occipital	Not operated			Surgery was not done in view of low frequency of seizures and involvement of eloquent cortex

Case No.	Age/ Sex	MEG Localisation	Language and motor functions by TMS navigated done/ clinical	Reason for SEEG implantation	No. of Electrodes	No. of Contacts	Region of Implantation	Surgery	Postop Complications (Post definitive surgery)	Follow Up (Months)	Seizure Outcome (ILAE Class)
18	44/M	Right posterior temporal, basitemporal, temporo-occipital, parietal operculum,	U/Borderline IQ, impaired visual memory	MRI negative DRE	13	156	B/L hippocampus, basitemporal, occipital and right superior temporal mid and posterior, left Superior temporal , Right Amygdala, right PHG, right temperooccipital, right Cingulate	Not yet operated			Not yet operated
19	21/M	Bilateral posterior temporal	U/Low average IQ, impaired verbal memory and attention	Seizure localization discordant with other data	12	145	B/L hippocampii, amygdala, Broca's area, right ant and post superior temporal, right anterior and posterior inferior temporal, right anterior insula, right lingula, right basitemporal and right frontal operculum	ECOG Guided resection of right basitemporal area	None	5	Class I
20	20/M	Right occipital, posterior and superior temporal, left temporal	U/Average IQ, Verbal and visual memory are impaired	Dual pathology	11	139	B/L hippocampii, right amygdala, right superior temporal, cingulate, supra and infracalcarine, 2 basifrontal and inferior temporal	Not yet operated			Not yet operated
21	21/F	Right frontoparietal, frontotemporal opercular, right frontal and posterior temporal, left angular gyrus	U/Low average IQ	Seizure localization discordant with other data	9	126	B/L hippocampii, right motor cortex, cuneus, precuneus, angular and ligular gyrus, superior and inferior temporal gyrus	ECOG guided resection of right precuneus	None	07	Class I

The resected tissues of all these cases have been provided for cellular electrophysiological and molecular characterization and the preliminary results of the same have been presented in other R&D projects.

Next to do:

1. More number of epilepsy patients will be evaluated at MEG Resource facility for better therapeutic and surgical outcome

2. We are currently in process of acquiring a language mapping system which will also allow us to characterize the epileptogenic networks and its relationship to the eloquent cortex.

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Lectures, Meetings & Workshops

Lectures, Meetings & Workshops

Sr. No.	Title and Speaker of the Talk	Date	Host Faculty
1	Royal Society Dr. Yusuf Hamied Workshop for India and the UK, 3-4th March 2021 (virtual platform). Co-Organizer (from India) for the Neuroscience session: Understanding brain structure and function: from molecules to mind	3rd–4th Mar 2021	Prof. Anirban Basu
2	"Brain dynamics of interactions between cognition, emotion, and motivation" by Prof. Luiz Pessoa, Professor, University of Maryland, USA	25th Nov 2020	Dr. Dipanjan Roy
3	Brain dynamics and flexible behaviours: Insights from network neuroscience " by Dr. Lucina Q. Uddin, University of Miami, Department of Psychology, USA	27th Oct 2020	Dr. Dipanjan Roy
4	"Mapping the spatiotemporal dynamics of hippocampal-cortical dynamics in health and Alzheimer's disease"- by Dr. Majid H. Mohajerani, the Canadian Centre for Behavioural Neuroscience, the University of Lethbridge	22nd Dec 2020	Dr. Dipanjan Roy

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 Anti-terrorism day, Sadbhavana Diwas, Independence Day, Vigilance Awareness week, International Yoga Day etc. The Administration achieved excellence in execution of the following activities at NBRC:

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

- The 17th Foundation Day of NBRC was held on 16th day of December, 2020. On this occasion, due to COVID-19 pandemic virtual program was performed.

On this august occasion, Prof. Josef P. Rauschecker, Department of Neuroscience & Cognition, Georgetown University, USA delivered the lecture virtually to the students and scientific community.

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 use of Official Language in all the administrative jobs such as internal official meetings, interviews, debates, general applications etc.

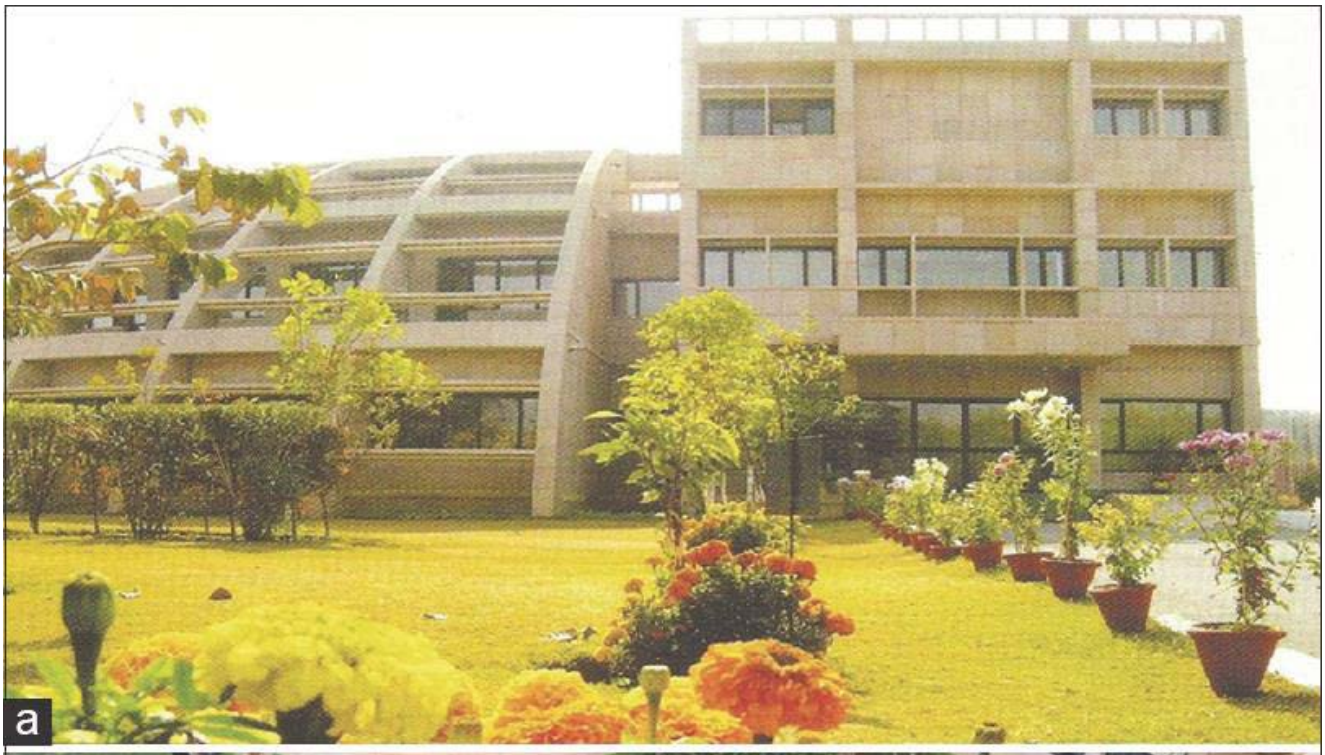
RTI Act : The provisions of RTI Act are being followed at NBRC in letter and spirit. All RTI applications received during 2020-21 seeking information on various matters concerning NBRC were provided the requisite information within the prescribed time limit. The quarterly reports containing number of 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 can 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, and 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 guidelines of DBT, one of the scientists of NBRC has been nominated as Part time Chief Vigilance Officer of the Centre.

Institutional Governance
Structure & People at NBRC


a


b

Members of The General Body of NBRC Society (As on 31-03-2021)

Prof. P.N. Tandon (President)	Chairman
Dr. Renu Swarup Secretary, Department of Biotechnology, New Delhi	Member **(Ex-Officio)**
Prof. Ashutosh Sharma Secretary, Department of Science & Technology, New Delhi	Member **(Ex-Officio)**
Prof. Balram Bhargava Director-General Indian Council of Medical Research, New Delhi	Member **(Ex-Officio)**
Dr. Sandip K. Basu JC Bose Chair Professor, National Institute of Science Commination & Information Resources (NISCAIR), New Delhi	Member
Financial Advisor, Department of Biotechnology, New Delhi	Member **(Ex-Officio)**
Director General CSIR, Institute of Genomics & Integrative Biology, New Delhi	Member
Dr. Sundeep Sarin Advisor, Department of Biotechnology, New Delhi	Member **(Ex-Officio)**
Dr. M. Gourie Devi Director (Retd.), NIMHANS, Bangalore	Member
Dr. L. M. Patnaik CSA Department Indian Institute of Science Bangalore	Member
Dr. Kalluri Subba Rao (INSA Hon. Scientist & Professor) School of Medical Sciences University of Hyderabad Hyderabad	Member
Prof. Gomathy Gopinath Flat # 001, Kanchanjunga Apartments, 122/2, Nagavarapalaya, Varthur Road, Bangalore – 560093	Member
Director National Centre for Biological Sciences, GKVK Campus, GKVK P.O., Bangalore – 560 065	Member
Prof. Neeraj Jain Director, National Brain Research Centre Nainwal Road, Manesar – 122052 Haryana	Member **(Ex-Officio) Till 31.12.2020**
Prof. Pravat K. Mandal Director (I/C) National Brain Research Centre, Manesar	Member **(Ex-Officio) w.e.f. 01.01.2021**

Members of The Governing Council (As on 31-03-2021)

Prof. Renu Swarup (Chairperson) (Ex-officio), Secretary Department of Biotechnology, New Delhi	Chairperson **(Ex-Officio)**
Prof. P.N. Tandon, New Delhi (Chairperson, SAC)	Member **(Ex-Officio)**
Prof. Upinder S. Bhalla Scientist, National Centre for Biological Sciences (NCBS), Bangalore	Member
Prof. Dinakar M. Salunke Director, International Centre for Genetic Engineering and Biotechnology, New Delhi	Member
Dr. A.K. Agarwal, Dean, Director, Professor & HOD (Retd.), New Delhi	Member
Professor G. Mehta, FNA, FRS, Bhartia Chair School of Chemistry University of Hyderabad	Member

Dr. Chitra Sarkar, Department of Pathology, All India Institute of Medical Sciences (AIIMS), New Delhi	Member
Prof. Seyed E. Hasnain, Vice Chancellor Jamia Hamdard University, New Delhi	Member
(Financial Advisor), Department of Biotechnology, New Delhi	Member **(Ex-Officio)**
Prof. Balram Bhargava Director General, Indian Council for Medical Research, New Delhi	Member **(Ex-Officio)**
Prof. Ashutosh Sharma Secretary, Department of Science & Technology (DST), New Delhi	Member **(Ex-Officio)**

UGC nominee	Member **(Ex-Officio)**
Dr. Sundeep Sarin Advisor, Department of Biotechnology, New Delhi	Member
Mr. C. P. Goyal JS (Admin), Department of Biotechnology, New Delhi	Member **(Ex-Officio)**
Dr. Neeraj Jain Director, National Brain Research Centre, Manesar	Member **(Ex-Officio) Till 31.12.2020**
Prof. Pravat K. Mandal Director (I/C), National Brain Research Centre, Manesar	Member **(Ex-Officio) w.e.f. 01.01.2021**

Members of The Finance Committee (As on 31-03-2021)

Chairman
Additional Secretary & Financial Advisor
Department of Biotechnology, Lodhi Road,
CGO Complex, NEW DELHI – 110 003

Prof. Dinakar M. Salunke Member
Director
International Centre for Genetic Engineering & Biotechnology, Aruna Asaf Ali Marg,
New Delhi

Prof. Seyed E. Hasnain, Member
IIT, Delhi
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Shri Sundeep Sarin,
Member
Advisor, DBT,
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Prof. Neeraj Jain, Member
Director
National Brain Research Centre,
Nainwal Mode, Manesar-122051
Haryana
Till 31.12.2020

Prof. Pravat K. Mandal
Director (I/C)
National Brain Research Centre,
Nainwal Mode, Manesar-122051
Haryana
From 01.01.2021

F&AO Non-Member Secretary
National Brain Research Centre
Nainwal Mode
Manesar-122051, Haryana

Members of The Building Committee (As on 31-03-2021)

Prof. Dinakar M. Salunke (Chairperson)
Director International Centre for Genetic Engineering and Biotechnology Aruna Asaf Ali Marg, New Delhi

Dr. Amulya K. Panda
Ex-Director National Institute of Immunology (NII), New Delhi

Dr. S. K. Gupta
Deputy Director (Retired) & Emeritus Scientist National Institute of Immunology (NII), New Delhi

Sh. V. H. Rao
Senior Consultant Engineering, Mehrauli - Badarpur Road, Near Batra Hospital, Hamdard Nagar, New Delhi

Mr. M. K. Gupta
Engineer-In-Charge (Civil), IUAC, New Delhi

Prof. Sidhartha Satpathy
HOD Hospital Administration, AIIMS, New Delhi

Prof. Pravat Kumar Mandal
Director (I/C), Member (Ex-Officio), NBRC, Manesar

Members of The Scientific Advisory Committee (As on 31-03-2021)

Prof. P. N. Tandon (Chairperson)
President, NBRC Society
Email: tandon@nbrc.ac.in

Prof. Upinder S. Bhalla (Co-Chairperson)
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International Members

Prof. ARIEL RUIZ i ALTABA
Professor,
Faculty of Medicine, University of Geneva,
Department of Medicinal Genetics, 8242 CMU,
1 rule Michel Servet, CH-1211, Geneva 4,
Switzerland

Prof. Baroness Susan Greenfield
Professor,
Department of Pharmacology, Lincoln College,
Oxford University, UK

Prof. Thomas D. Albright
Professor,
The Salk Institute for Biological Studies,
La Jolla, California, USA 92037

Michael W. Weiner
MD, Director of the Center for Imaging of
Neurodegenerative Diseases,
SFVAMC, Professor of Radiology,
Medicine, Psychiatry and Neurology, UCSF

Members of Board of Studies (As on 31-03-2021)

Prof. Pravat K Mandal, Chairperson
Director I/C
National Brain Research Centre
Manesar, Haryana

Prof. Ranjit K. Giri, Member
National Brain Research Centre
Manesar, Haryana

Prof. Kunzang Chosdol, External Member
Department of Biochemistry,
All India Institute of Medical Sciences, New Delhi

Dr. Anindya Ghosh Roy, Member
National Brain Research Centre
Manesar, Haryana

Dr. Sushil Kumar Jha, External Member
Associate Professor,
Jawaharlal Nehru University,
New Delhi

Dr. Arpan Banerjee, Member
National Brain Research Centre
Manesar, Haryana

Prof. Soumya Iyengar, Member
National Brain Research Centre
Manesar, Haryana

Dr. Dipanjan Roy, Member
National Brain Research Centre
Manesar, Haryana

Prof. Ellora Sen, Member
National Brain Research Centre
Manesar, Haryana

Offg. Registrar, Member
National Brain Research Centre
Manesar, Haryana

DST-INSPIRE Faculty (As on 31-03-2021)

Dr. Yogita Kapil Adlakha (Till 04/05/2020 (A.N.))

Wellcome Trust India Alliance / DBT India Alliance Early Career Fellow

Dr. Swagata Dey

Ph.D. Degrees Awarded

1. Shashi Shekhar Kumar
2. Vikas Pareek
3. Touseef Ahmad Sheikh
4. Kautuk Kamboj
5. Vipendra Kumar
6. Pruthvi SG
7. Tushar Arora
8. Sandeep Kumar

M.Sc. Degrees Awarded

1. Akanksha Goyal
2. Akanksha Gupta
3. Ankit Dhoundiyal
4. Thakar Darshit Mahesh
5. Mantosh Patnaik
6. Mishaben Parmar
7. Nitish Kumar
8. Pratibha Ahirwal
9. Rekha Singh
10. Rishika Tiwari
11. S Indira Priya
12. Sharmistha Panda
13. Shashwati Tripathi
14. Surbhi
15. Vinsea AV Singh

Ph.D. Students

1. Mr. Apoorv Sharma
2. Mr. Sandeep Kumar (Till 18.01.2021)
3. Mr. John Thomas (Till 08.01.2021)
4. Mr. Shashi Shekhar Kumar (Till 15.06.2020)
5. Mr. Kautuk Kamboj (Till 29.10.2020)
6. Mr. Vipendra Kumar (Till 04.12.2020)
7. Mr. Biswaranjan Sahoo
8. Mr. Indrajith R. Nair
9. Mr. Touseef Ahmad Sheikh (Till 30.09.2020)
10. Mr. Tushar Arora (Till 18.01.2021)
11. Mr. S Balakumar
12. Ms. Arti Kumari
13. Mr. Dharmendra Puri
14. Ms. Mukta Kumari
15. Mr. Raghav Shankar
16. Md. Tipu Khan
17. Ms. Priyanka Ghosh
18. Ms. Sarbani Samaddar
19. Ms. Shruti Patrick
20. Mr. Surajit Chakraborty
21. Ms. Bindu
22. Mr. Shiladitya Laskar (Till 27.11.2020)
23. Mr. Sibaram Behera
24. Ms. Tripti Joshi
25. Mr. Abhishek Singh Narvaria
26. Ms. Deepti Dama
27. Mr. Karthick R
28. Ms. Nisha Chetana Sastry
29. Ms. Shivangi Sharma
30. Ms. Sunanda Sharma
31. Ms. Vanshika Singh (Till 31.12.2020)
32. Ms. Himali Arora
33. Ms. Meenakshi Bhaskar
34. Mr. Neeraj Kumar
35. Ms. Dipanjana Banerjee
36. Ms. Gargi Majumdar

37. Ms. Kamakshi Garg
38. Ms. Khushboo Vinod Punjabi
39. Ms. Partika
40. Ms. Shalini Sharma
41. Ms. Stuti Mohapatra
42. Mr. Anagh Pathak
43. Ms. Kirti
44. Ms. Ritu Moni Borah
45. Ms. Anjali
46. Mr. Ankit Yadav
47. Ms. Archana Mehta
48. Mr. Chandramouli Mukherjee
49. Ms. Sakshi Shukla
50. Ms. Sonia Balahun Umdor
51. Mr. Arkaprovo Sarkar
52. Mr. Azman Akhter
53. Ms. Guneet Kaur
54. Ms. Kirti Saluja
55. Ms. Pallavi Singh
56. Ms. Guncha Bhasin
57. Ms. Uzma Din
58. Ms. Chitra Mohinder Singh Singal
59. Ms. Pooja Parishar
60. Mr. Apurva Agrawal
61. Mr. Atanu Datta
62. Mr. Hriday Shanker Pandey
63. Ms. Atrayee Basu
64. Ms. Priyanka
65. Mr. Gourav Sharma
66. Ms. Harjot Kaur Brar
67. Mr. Shubham Krishna
68. Ms. S Indira Priya
69. Ms. Shashwati Tripathi
70. Ms. Vinsea AV Singh

M.Sc. Students

1. Ms. Akanksha Goyal (Till 31.07.2020)
2. Ms. Akanksha Gupta (Till 31.07.2020)
3. Mr. Ankit Dhoundiyal (Till 31.07.2020)
4. Mr. Thakar Darshit Mahesh (Till 31.07.2020)
5. Mr. Mantosh Patnaik (Till 31.07.2020)
6. Ms. Mishaben Parmar (Till 31.07.2020)
7. Mr. Nitish Kumar (Till 31.07.2020)
8. Ms. Pratibha Ahirwal (Till 31.07.2020)
9. Ms. Rekha Singh (Till 31.07.2020)
10. Ms. Rishika Tiwari (Till 25.03.2021)
11. Ms. S Indira Priya (Till 31.07.2020)
12. Ms. Sharmistha Panda (Till 31.07.2020)
13. Ms. Shashwati Tripathi (Till 31.07.2020)
14. Ms. Surbhi (Till 31.07.2020)
15. Ms. Vinsea A V Singh (Till 31.07.2020)
16. Ms. Aamna Jain
17. Mr. Ankit Kumar Shah
18. Ms. Anwesha Das
19. Ms. Bhanumita Agrawal
20. Ms. Debapriya Roy
21. Ms. Bapat Ojasee Ajinkya
22. Ms. Pooja Kri Gupta
23. Mr. Rudradeep Mukherjee
24. Mr. Akshay Kumar Tiwari
25. Ms. Dyutika Banerjee
26. Ms. Janhvi Mahesh Dhongdi
27. Ms. Mohima Mukherjee

28. Ms. Mrittika Dey
29. Ms. Muskaan Verma
30. Ms. R. Madhumita
31. Mr. Rajat Joshi
32. Mr. Shuvrangshu Guha
33. Ms. Yamini Gupta (Till 29.10.2020)

Project Assistants

1. Ms. Keerthana. P (Till 19/06/2020 (A.N.))
2. Dr. Fahd M Yasin
3. Mr. Vivek Sharma (05/03/2021 (A.N.))
4. Ms. Smriti Bhardwaj (26/02/2021 (A.N.))
5. Mr. Devashish Arvind Pande (11/11/2020 (A.N.))
6. Mr. Paritosh Jaiswal (31/03/2021 (A.N.))
7. Mr. Rishabh Kapoor (17/12/2020 (A.N.))
8. Ms. Sigar Priyanka Jaipal
9. Ms. Reshma Raj (06/10/2020 (A.N.))
10. Mr. Varun Madan Mohan
11. Ms. Bhavya Gohil (Till 31/07/2020 (A.N.))
12. Mr. Shubham Singhal
13. Ms. Kulshrestha Shruti (29/05/2020 (A.N.))
14. Mr. Mainak Ghosh (30/09/2020 (A.N.))
15. Ms. Dimpi
16. Ms. Richa Sharma (Till 27/11/2020 (A.N.))
17. Mr. Vinayak Ghosh
18. Mr. Santhosh Kumar S
19. Ms. Rimil Guha Roy
20. Ms. Kavinila S
21. Mr. Souren Sadhukhan
22. Ms. Bhavya Gohil

Research Associates

1. Ms. Deepali Singh, Research Associate-2
   (Till 09/06/2020 (A.N.))
2. Dr. Kanu Megha, Research Associate-1
   (Till 03/04/2020 (A.N.))
3. Dr. Sonika, Research Associate-2
4. Dr. Sandeep Kumar, Research Associate-3
   (Till 23/09/2020 (A.N.))
5. Dr. Soibam Shyamchand Singh,
   Research Associate-3
6. Mr. Anuj Mishra (Till 08/01/2021)
7. Ms. Nidhi Sharma (Till 05/03/2021)
8. Dr. Karthick C (Till 10/03/2021 (A.N.))
9. Dr. Rituparna Chaudhuri
10. Mr. Pruthvi S. G
11. Dr. Atreye Majumdar
12. Dr. Shubhi Kansal
13. Dr. Priyanka Chakraborty
14. Dr. Sushma Dagar
15. Ms. Km Nisha
16. Dr. Atreye Majumdar
17. Dr. Shubhi Kansal

Research Associate (Project)

1. Dr. Amit Naskar (Till 31/03/2021 (A.N.))
2. Dr. Rituparna Chaudhuri (Till 14/01/2021 (A.N.))
3. Dr. Jasleen Gund (Till 01/12/2020 (A.N.))
4. Dr. Md. Ashraful Hasan, DBT-TWAS Postdoctoral Fellowship (Till 20/07/2020 (A.N.))

Research Fellows

1. Mr. Vikas Pareek
   (From 24/01/2020 till 23/07/2020)
2. Mr. Touseef Ahmad Sheikh
   (From 13/03/2020 till 12/09/2020)

Project Employees

1. Ms. Radhika Shivhare, Senior R&D Engineer (Project) (Till 11/03/2020 (A.N.))
2. Ms. Ruchika Mittal, Programmer (Project)
   (Till 12/02/2020 (A.N.))

3. Dr. Rini Dhawan, Scientist "B" (Project) (Till 03/04/2020 (A.N.))
4. Ms. Apoorva Misra, Research Manager (Project) (Till 31/12/2020)
5. Dr. P. Prarthana Chandra, CMO
6. Mr. Gaurav Singh, Technologist
7. Mr. Prem Chand, Manager
8. Ms. Srimathi P., Technician
9. Ms. Meenu Yadav, Technician
10. Mr. Om Prakash Jakhar, Nurse
11. Ms. Priya Shrivastav, Nurse
12. Ms. Divyasree V.M., Nurse
13. Mr. Amit Kumar Srivastva, Nursing Orderly
14. Mr. Deepak Kumar, Nursing Orderly
15. Mr. Manjit, Lab Attendant
16. Mr. Sachin Kumar, Lab Attendant
17. Mr. Sukhvir Singh Pundir, Tech. Associate (Computer /IT)
18. Ms. Shalini, Technical Assistant
19. Mr. Saurav Roy, R&D Engineer
20. Mr. Kuldeep Singh, R&D Engineer
21. Ms. Shallu Sharma, Scientist
22. Ms. Divya Dwivedi, Clinical Coordinator
23. Ms. Shallu, Neuropsychologist
24. Ms. Komal Jindal, Senior R&D Engineer
25. Ms. Anshika Goel, Research Scientist
26. Dr. Rini Dhawan, Project Coordinator –I
27. Dr. Jasleen Gund, Project Scientist I
28. Dr. Shah Zinkal Atul, Project Scientist –III
29. Mr. Anupam Das, JRF
30. Ms. Ruby Goel, Scientist B (Project)
31. Mr. Krishn Kant, Technical Assistant
32. Ms. Pallavi Pant, JRF
33. Ms. Sruthy Raviverma, Project Assistant
34. Mr. Mantosh Patnaik, Research Assistant
35. Dr. Deepali Singh, ICMR-RA
36. Dr. Swagta Dey, The Welcome Trust / DBT India Alliance
37. Dr. Nivethida T, The Welcome Trust / DBT India Alliance

Scientific Staff

Name	Designation
Prof. Neeraj Jain	Director (Retired on 31.12.2020)
Prof. Pravat Kumar Mandal	Scientist – VII & Director (I/C) (w.e.f. 01.01.2021 as Director (I/C))
Prof. Pankaj Seth	Scientist – VII
Prof. Shiv Kumar Sharma	Scientist – VII
Prof. Nandini C. Singh	Scientist-VI (Working at UNESCO, New Delhi on deputation basis)
Dr. Ellora Sen	Scientist – VI
Prof. Soumya Iyengar	Scientist – VI
Prof. Anirban Basu	Scientist – VII
Dr. Ranjit Kumar Giri	Scientist – VI
Dr. Sourav Banerjee	Scientist – V
Dr. Arpan Banerjee	Scientist – V
Dr. Anindya Roy Ghosh	Scientist – V
Dr. Dipanjan Roy	Scientist - IV
Dr. Mayanglambam Dhruba Singh	Scientist-III
Dr. Bhavani Shankar Sahu	Scientist-III
Mr. Mahender Kumar Singh	Information Scientist

Administrative Staff

Name	Designation
Mr. Tanmoy Bhattacharyya	Chief Administrative Officer
Mr. Santosh Kumar Choudhary	Deputy Finance Officer
Mrs. Pooja Gosain	Administrative Officer
Ms. Shiwani Tanwar	Administrative Officer (Acad.)

Name	Designation
Mr. Ravinder Pal	Stores & Purchase Officer
Sanjay Kumar Gupta	Office Assistant
Mr. Suraj Bhan	Office Assistant
Mr. Rakesh Kumar Yadav	Office Assistant
Mr. Himanshu Mal	Office Assistant (Working at ICSSR, New Delhi on deputation basis
Mr. Ajay Kumar Dehariya	Office Assistant
Mr. Parmander Singh Rawat	Office Assistant
Mr. Jitendra Kumar Meena	Office Assistant
Mr. Bhupender Pal Sharma	Driver Grade-I
Mr. Satish Kumar	Driver Grade-II

Technical Staff

Name	Designation
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Dr. D.D. Lal	Technical Officer
Mr. Jitender Ahlawat	Technical Officer – B
Mr. Arvind Singh Pundir	Technical Officer – B
Dr. Inderjeet Yadav	Veterinarian
Mr. Kedar Singh Bajetha	Computer Operator
Ms. Seepika	Computer Operator
Mr. Sachin Kumar	Computer Operator
Ms. Tarnnum Mansoori	Computer Operator
Mr. Sanjeev Bhardwaj	Computer Operator
Mr. Kanhaiya Lal Kumawat	Technician-C
Mr. Shankar Datt Joshi	Technician-C
Mr. Sumit Kumar Sinha Mahapatra	Technician-C
Mr. D. Narender	Technician-C
Mr. Sanjay Kumar	Technician-B
Mr. Mithlesh Kumar Singh	Technician-B
Mr. Ankit Sharma	Technician-B
Mr. Yunis Khan	Technician-B
Mr. Durga Lal Meena	Technician-B
Md. Irshad Alam	Technician-B
Mr. Manish Kumar	Technician-B
Mr. P. Manish	Technician-B
Mr. Dil Bahadur Karki	Technician-A
Mr. Rammehar	Technician-A
Mr. Hari Shankar	Technician-A
Mr. Mahendra Singh	Technician-A
Mr. Sanjay Kumar Singh	Technician-A

Contractual Staff

Name	Designation
Dr. P. Raghunathan	Consultant
Dr. Rema Velayudhan	Sr. Consultant (Relieved on18.12.2020)
Mr. Om Prakash Nagar	Consultant (Admin.)
Mr. M.V.S. Visweswara Sai	Consultant (Acad.) (Relieved on 16.11.2020)

DIC Project Staff

Name	Designation
Ms. Reema Saxena	Computer Operator (Relieved on 30.06.2020)
Ms. Sunita	Computer Operator (Relieved on 30.06.2020)
Mr. R. Ganesh Gurumoorthy	Computer Operator (Relieved on 30.06.2020)
Mr. Amit Kumar	Computer Operator (Relieved on 30.06.2020)

Annual Financial Statements

MAHESHWARI PA & ASSOCIATES
Chartered Accountants
1st Floor, Saraswati Plaza, E.K. Road, Meerut
Ph: 0121-4059052, 4059065, +91-7217000890, 9997179121,
Email : maheshwarica9@gmail.com

INDEPENDENT AUDITOR'S REPORT

Report on the Financial Statements

1. We have audited the accompanying financial statements of **M/S NATIONAL BRAIN RESEARCH CENTRE** ("the Institute"), which comprise the Balance Sheet as at March 31, 2021, the Statement of Income & Expenditure A/c for the year then ended, and a summary of the significant accounting policies and other explanatory information, which we have signed under reference to this report.

Management's Responsibility for the Financial Statements

2. The Institute's Management is responsible for the matters with respect to the preparation of these financial statements that give a true and fair view of the financial position and financial performance of the Institute in accordance with the accounting principles generally accepted in India, including the Accounting Standards specified. This responsibility also includes the maintenance of adequate accounting records for safeguarding of the assets of the Institute 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 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.

Auditor's Responsibility

3. Our responsibility is to express an opinion on these financial statements based on our audit. We conducted our audit in accordance with the Standards on Auditing issued by the Institute of Chartered Accountants of India and in accordance with the Standards on Auditing specified. Those Standards require that we comply with ethical requirements and plan and perform the audit to obtain reasonable assurance about whether the financial statements are free from material misstatement.

4. An audit involves performing procedures to obtain audit evidence about the amounts and disclosures in the financial statements. The procedures selected depend on the auditor's judgment, including the assessment of the risks of material misstatement of the financial statements, whether due to fraud or error. In making those risk assessments, the auditor considers internal control relevant to the Institute's preparation and fair presentation of the financial statements 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 Institute's internal control. An audit also includes evaluating the

206, 1st Floor, Vikas Complex, Opp. Metro Pilar No. 57,37,
Veer Savarkar Block, Nirman Vihar, New Delhi-92

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

appropriateness of accounting policies used and the reasonableness of the accounting estimates made by management, as well as evaluating the overall presentation of the financial statements.

5. We believe that the Audit evidence we have obtained is sufficient and appropriate to provide a basis for our audit opinion.

Opinion

6. In our opinion and to the best of our information and according to the explanations given to us, the aforesaid financial statements give the information, in the manner so required and give a true and fair view in conformity with the accounting principles generally accepted in India:

   a) In the case of the Balance Sheet, of the state of affairs of the Institute as at March 31, 2021;

   b) In the case of the Statement of Income & Expenditure A/c of the Institute for the year ended on that date.

7. *Report on Other Legal and Regulatory Requirements*

   1) As required, we report that:

   a) We have obtained all the information and explanations which to the best of our knowledge and belief were necessary for the purpose of our audit;

   b) In our opinion, proper books of account as required by law have been kept by the Institute so far as it appears from our examination of those books;

   c) The Balance Sheet and Statement of Income & Expenditure A/c dealt with by this Report are in agreement with the books of account;

   d) In our opinion, the Balance Sheet, Statement of Income & Expenditure A/c, Receipt & Payment A/c comply with the Accounting Standards;

   e) In our opinion and to the best of our information and according to the explanations given to us, we report as under with respect to other matters to be included in the Auditor's Report:

      i) The Institute does not have any pending litigations which would impact its financial position, except one case which is pending.

      ii) There are some payables pending since last 3 years.

      iii) Donation Received should be transferred to indirect income.

      iv)TDS should be deducted on due basis.

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

v) The Institute did not have any long term contracts including derivative contracts; as such the question of commenting on any material foreseeable losses thereon does not arise.

FOR MAHESHWARI P A AND ASSOCIATES
(Chartered Accountants)

CA ANKUR AGARWAL
PARTNER
M. NO. 409197

Date: 08th October, 2021
Place: Meerut
UDIN: 21409197AAAAED8279

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM, HARYANA
BALANCE SHEET AS AT MARCH 31,2021

(Amount-Rs.)

CORPUS / CAPITAL FUND AND LIABILITIES	Schedule	Current Year	Previous Year
Corpus/Capital Fund	1	1,40,35,02,000.00	1,37,35,02,000.00
Reserve and Surplus	2	(23,80,89,863.34)	(11,18,40,570.41)
Earmarked/Endowment Funds	3	63,86,15,715.38	83,68,24,080.29
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	4,22,39,582.51	4,85,50,775.64
Total (Liabilities)		**1,84,62,67,434.55**	**2,14,70,36,285.52**
ASSETS			
Fixed Assets	8	98,34,66,108.13	1,09,21,58,034.51
Investments - From Earmarked/Endowment Funds	9	0.00	0.00
Investments-Others	10	1,35,63,572.19	2,51,08,109.99
Current Assets, Loans, Advances etc.	11	84,92,37,754.23	1,02,97,70,141.02
Miscellaneous Expenditure (to the extent not written off or adjusted)		0.00	0.00
Total (Assets)		**1,84,62,67,434.55**	**2,14,70,36,285.52**
Significant Accounting Policies	24		
Contingent Liabilites and Notes on Accounts	24		

SHIWANI TANWAR
FINANCE & ACCOUNTS OFFICER (I/C)
NBRC

वित्त एवं लेखा अधिकारी
Finance & Account Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
मानेसर / Manesar-122050
हरियाणा / Haryana

PROF. PRAVAT KUMAR MANDAL
DIRECTOR (I/C)
NBRC

Prof. Pravat K. Mandal
प्रो. प्रवात कुमार मंडल
प्रभारी निदेशक / Director-in-Charge
राष्ट्रीय मस्तिष्क अनुसंधान केन्द्र
National Brain Research Centre
मानेसर- 122 052 / Manesar-122 052
हरियाणा / Haryana
फोन नः 0124-2338929/ Tel. No. 0124-2338929
फोन नः 0124-2845207/ Tel. No. 0124-2845207

As per our separate report of even date attached

For Maheshwari PA & Associates
Chartered Accountants
(FRN-012023C)

MAHESHWARI P.A. & ASSOCIATES
CHARTERED ACCOUNTANTS

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
Meerut

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM, HARYANA
INCOME AND EXPENDITURE ACCOUNT FOR THE YEAR ENDED March 31, 2021

(Amount-Rs.)

	Schedule	Current Year	Previous Year
INCOME			
Income from Sales/Services	12	0.00	0.00
Grants/ Subsidies (Revenue) from DBT	13	25,45,00,000.00	29,13,00,000.00
Fees/Subscriptions	14	10,64,770.67	11,60,404.00
Income from Investments (Income on Invest. From earmarked/endow. Funds transferred to funds)	15	12,67,054.00	60,29,338.92
Income from Royality, Publication etc.	16	0.00	0.00
Interest Earned	17	2,89,24,668.99	3,89,84,253.22
Other Income	18	19,37,443.00	20,41,033.00
Increase/(decrease) in stock of Finished goods and work-in-progress	19	0.00	0.00
Total Income (A)		**28,76,93,936.66**	**33,95,15,029.14**
EXPENDITURE			
Establishment Expenses	20	9,52,70,171.00	8,95,29,003.00
Other Administrative etc.	21	17,63,33,987.56	16,93,76,010.40
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 8)		9,15,93,133.03	9,92,57,469.55
Total Expenditure (B)		**36,31,97,291.59**	**35,81,62,482.95**
Balance being excess of Income over Expenditure (A-B)		(7,55,03,354.93)	(1,86,47,453.81)
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		**(7,55,03,354.93)**	**(1,86,47,453.81)**
Significant Accounting Policies	24		
Contingent Liabilites and Notes on Accounts	24		

SHIWANI TANWAR
FINANCE & ACCOUNTS OFFICEER (I/C)
NBRC

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फोन नं: 0124-2338929/ Tel. No. 0124-2338929
फोन नं: 0124-2845207/ Tel. No. 0[illegible]

As per our separate report of even date attached

For Maheshwari PA & Associates
Chartered Accountants
(FRN-012023C)

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
Meerut

NATIONAL BRAIN RESEARCH CENTRE, MANESAR
NH-8, NAINWAL MORE, MANESAR, GURGAON, HARYANA
RECEIPTS AND PAYMENTS FOR THE YEAR ENDED March 31, 2021

RECEIPTS	CURRENT YEAR	PREVIOUS YEAR	PAYMENTS	CURRENT YEAR	PREVIOUS YEAR
	Amount in (Rs.)			Amount in (Rs.)	
I. Opening Balances			I. Expenses		
a) Cash in Hand	2,62,526.00	1,48,195.00	i) Establishment Expenses	84,72,715.00	1,00,47,485.00
b) Bank Balances			ii) Administrative Expenses	29,49,240.14	27,31,821.47
i) In Deposit Accounts	-	-	II. Payment Made Against Funds For Various Projects		
ii) Saving Accounts	19,80,80,018.04	55,72,26,483.43	i) Recurring /Capital expenditure	2,35,92,665.03	1,35,76,622.58
iii) CPF Investments	2,31,321.99	91,12,172.79	ii) Capital Grant Refunded to DBT	-	-
II. Grants Received			iii) Refund to RCGB	-	-
a) From Government of India			iv) Refund of Interest	5,07,45,938.00	1,40,21,000.00
Plan			v) Bank Deposits	21,00,00,000.00	1,01,00,00,000.00
i) Recurring Income	25,45,00,000.00	29,13,00,000.00	III. Maintenance Cost		
ii) Non-Recurring Income	3,00,00,000.00	-	i) Lab Maintenance Expenses	16,86,923.24	31,38,234.93
Plan (Recurring)			ii) Office Maintenance	3,45,13,748.00	3,50,92,734.00
b) Fellowship Grant	4,66,170.00	2,70,501.00	iii) Vehicle Running & Maintenance	5,60,581.00	7,19,199.00
c) Delcon Projects (Including Interest)	24,17,79,366.00	58,71,93,205.00	IV. Investment and Deposit Made		
III. Receipt made against funds for various projects			i) Out of Earkmarked/Endowment funds	32,44,000.00	2,69,18,669.00
i) Recurring Receipt/ Capital Grant	6,38,82,011.50	8,93,93,279.14	V. Expenditure of Fixed Assets & Capital Work-in-progress		
(Including Interest)			i) Purchase of Fixed Assets	46,93,298.11	46,77,725.69
ii) Bank Deposits	69,00,00,777.00	68,81,18,520.00	VI. Training Expenses	16,20,957.00	32,24,477.82
IV. Interest Received			VII. Other Payments(Specify)		
i) On Bank Deposits			i) Advances to Supplier	1,24,53,768.53	2,84,72,772.96
ii) Savings Account	91,15,353.99	86,01,651.06	ii) Advances to Staff	13,86,771.00	31,86,169.00
iii) On CPF Fund	1,15,18,624.00	2,07,85,226.00	iii) Leave Encashment/ LTC/ Bonus	7,33,167.00	3,79,046.00
iv) Other Interest	-	2,26,185.00	iv) Security Deposit Paid	9,46,232.00	22,31,336.00
V. Any Other Receipt			v) EMD Refunded	8,12,500.00	26,56,439.00
Indirect Income			vi) TDS Paid	4,22,96,949.00	6,71,93,163.56
i) Advance to Supplier Received	1,13,076.00	7,00,228.00	vii) Imprest	1,63,578.00	1,95,578.00
ii) Advance to Staff Received	8,46,472.89	16,98,360.00	viii) Payment of Current Liabilities	61,91,56,292.48	85,25,62,364.38
iii) Sale of Tender Documents	4,99,620.00	43,100.00	ix) Prepaid Insurance	-	-
iv) Misc. Receipts.	3,93,693.67	4,97,581.00	VIII. Closing balances		
v) Earnest Money Deposit Received	10,31,300.00	33,85,139.00	a) Cash in Hand	39,098.00	2,62,526.00
vi) Sale of Scrap	-	1,12,731.00	b) Bank Balance		
vii) Guest House Charges	65,369.00	2,24,150.00	i) In Deposit Accounts	-	-
viii) Hostel Deposit	3,00,000.00	3,39,000.00	ii) Saving Accounts	49,71,09,024.36	19,80,80,018.04
ix) CPF Fund Received	2,15,82,528.00	1,64,10,480.00	iii) CPF Investments	87,78,018.19	2,31,321.99
x) Library Deposit	1,08,000.00	1,16,000.00			
xi) Current Liabilities Rec.	9,95,298.00	36,34,115.00			
xii) Other Receipts	1,83,938.00	62,402.00			
TOTAL	1,52,59,55,464.08	2,27,95,98,704.42	TOTAL	1,52,59,55,464.08	2,27,95,98,704.42

SHIWANI TANWAR
FINANCE & ACCOUNTS OFFICEER (I/C)
NBRC

वित्त एवं लेखा अधिकारी
Finance & Account Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
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PROF. PRAVAT KUMAR MANDAL
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फोन नं: 0124-2338929/ Tel. No. 0124-2338929
फोन नं: 0124-2845207/ Tel. No. 0124-2845207

As per our separate report of even date attached

For Maheshwari PA & Associates
Chartered Accountants
(FRN-012023C)

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
Meerut

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021 (Amount-Rs.)

SCHEDULE 1-CORPUS/CAPITAL FUND:

	Current Year		Previous Year	
1 Grant-in-Aid - Balance as at the beginning of the year		1,37,35,02,000.00		1,37,35,02,000.00
Add: Contribution towards Corpus/Capital Fund	3,00,00,000.00		0.00	
Add/(Deduct): Balance of net income/(expenditure) transferred from the Income and Expenditure Account		3,00,00,000.00		0.00
Balance as at the year end		1,40,35,02,000.00		1,37,35,02,000.00

SHIWANI TANWAR
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For Maheshwari PA & Associates
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(FRN-012023C)

Ankur Agarwal
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Meerut

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021 (Amount-Rs.)

SCHEDULE 2 - RESERVES AND SURPLUS:

	Current Year		Previous Year	
1 **Capital Reserve:**				
As per last Account	0.00		0.00	
Addition during the Year	0.00		0.00	
Less : Deductions during the year (deficit)	0.00	0.00	0.00	0.00
2 **Revaluation Reserve:**				
As per last Account	0.00		0.00	
Addition during the Year	0.00		0.00	
Less : Deductions during the year (deficit)	0.00	0.00	0.00	0.00
3 **Special Researve:**				
As per last Account	0.00		0.00	
Addition during the Year	0.00		0.00	
Less : Deductions during the year (deficit)	0.00	0.00	0.00	0.00
4 **General Reserve**				
As per last Account	(11,18,40,570.41)		(7,91,72,116.60)	
Addition during the Year	-			
Surplus during the yar (as per I&E A/c)	(7,55,03,354.93)		(1,86,47,453.81)	
Less : Deductions during the year (deficit)	5,07,45,938.00	(23,80,89,863.34)	1,40,21,000.00	(11,18,40,570.41)
Balance as at the year end		**(23,80,89,863.34)**		**(11,18,40,570.41)**

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NBRC
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As per our separate report of even date attached

For Maheshwari PA & Associates
Chartered Accountants
(FRN-012023C)

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
Meerut

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021 — Amount in (Rs.)

FUND-WISE BREAK UP SCHEDULE 3 - EARMARKED/ENDOWMENT FUNDS				TOTALS			
Endowment Fund for Building		Donation		Current Year		Previous Year	
	0.00		26,31,788.00		83,68,24,080.29		91,17,44,376.60
0.00		0.00		31,95,78,988.34		69,42,33,036.38	
0.00		0.00		0.00		0.00	
0.00	0.00	0.00	0.00	72,67,311.65	32,68,46,299.99	1,05,09,839.94	70,47,42,876.32
	0.00		26,31,788.00		1,16,36,70,380.28		1,61,64,87,252.92
0.00		0.00		1,21,10,217.04		2,46,63,489.18	
0.00		0.00		0.00		0.00	
	0.00		0.00		1,21,10,217.04		2,46,63,489.18
0.00		0.00		1,79,04,677.00		1,75,15,018.00	
0.00		0.00		0.00		0.00	
0.00		0.00		43,39,43,140.86		68,74,20,514.45	
0.00		0.00		4,25,36,309.00		4,84,40,074.00	
	0.00		0.00		49,43,84,126.86		75,33,75,606.45
	0.00		0.00		50,64,94,343.90		77,80,39,095.63
				1,85,60,321.00		16,24,077.00	
					1,85,60,321.00		16,24,077.00
	0.00		26,31,788.00		63,86,15,715.38		83,68,24,080.29

SHIWANI TANWAR
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Prof. Pravat K. Mandal
प्रो. प्रवात कुमार मंडल
प्रभारी निदेशक / Director-in-Charge
राष्ट्रीय मस्तिष्क अनुसंधान केन्द्र
National Brain Research Centre
मानेसर- 122 052 / Manesar-122 052
हरियाणा / Haryana
फोन नः 0124-2338929/ Tel. No. 0124-2338929
फोन नः 0124-2845207/ Tel. No. 0124-2845207

As per our separate report of even date attached

For Maheshwari PA & Associates
Chartered Accountants
(FRN-012023C)

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
Meerut

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021

Amount in (Rs.)

SCHEDULE 3 - EARMARKED/ENDOWMENT FUNDS	FUND-WISE BREAK UP							
	Project Fund		Fixed Assets Fund (Project)		Contributory Provident Fund		DeLcon E-library Consortium	
a) Opening Balance of Project Fund		14,98,40,752.98		27,60,06,559.32		87,68,736.00		39,95,76,243.99
b) Additions to the Funds:								
i. Donations/grants/Additions to Fund	6,78,27,511.30		1,21,10,217.04		12,05,740.00		23,84,35,520.00	
ii. Income from investments made on account of funds	0.00		0.00		0.00		0.00	
iii. Other additions (Interest Earned)	39,23,465.65	7,17,50,976.95	0.00	1,21,10,217.04	0.00	12,05,740.00	33,43,846.00	24,17,79,366.00
Total (a+b)		22,15,91,729.93		28,81,16,776.36		99,74,476.00		64,13,55,609.99
c) Utilisation/Expenditure towards objectives of funds								
i. Capital Expenditure								
Fixed Assets (net)	1,21,10,217.04		0.00		0.00		0.00	
Others	0.00		0.00		0.00		0.00	
Total		1,21,10,217.04		0.00		0.00		0.00
ii. Revenue Expenditure								
-Salaries, Wages and allowances etc	1,66,02,572.00		0.00		0.00		13,02,105.00	
-Rent	0.00		0.00		0.00		0.00	
-Others	2,94,97,380.71		0.00		32,44,000.00		40,12,01,760.15	
-Depreciation	0.00		4,25,36,309.00		0.00		0.00	
Total		4,60,99,952.71		4,25,36,309.00		32,44,000.00		40,25,03,865.15
Total (C)		5,82,10,169.75		4,25,36,309.00		32,44,000.00		40,25,03,865.15
d) Refund of unspent fund during the year	1,85,60,321.00							
		1,85,60,321.00						
NET BALANCE AS AT THE YEAR-END (a+b-c-d)		14,48,21,239.18		24,55,80,467.36		67,30,476.00		23,88,51,744.84

Notes
1) Disclosures shall be made under relevant heads based on conditions attaching to the grants
2) Plan funds received from the Central/State Governments are to be shown as separate Funds and not to be mixed up with any other Funds.
3) Net additions during the year represents additions net of deductions during the year.

SCHEDULE 4 - SECURED LOANS AND BORROWINGS: NIL

SCHEDULE 5 - UNSECURED LOANS AND BORROWINGS: NIL

SCHEDULE 6 - DEFERRED CREDIT LIABILITIES: NIL

SHIWANI TANWAR
FINANCE & ACCOUNTS OFFICER (I/C)
NBRC

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Finance & Account Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
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PROF. PRAVAT KUMAR MANDAL
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As per our separate report of even date attached

For Maheshwari PA & Associates
Chartered Accountants
(FRN-012023C)

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
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MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
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SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021 — Amount in (Rs.)

SCHEDULE-7 CURRENT LIABILITIES AND PROVISIONS

	Current Year		Previous Year	
A. Current Liabilities				
1. Acceptances		0.00		0.00
2. Sundry Creditors				
-For Goods	0.00		0.00	
- Others	11,52,789.00	11,52,789.00	11,83,854.00	11,83,854.00
3. Advances Received		41,94,866.42		44,05,236.42
4.Interest accured but not due on:				
-Secured Loans/borrowings	0.00		0.00	
-Unsecured loans/borrowings	0.00		0.00	
		0.00		0.00
5. Satutory Liabilities:				
-Overdue	0.00		0.00	
-Others (TDS payable)	3,94,316.50		3,92,532.50	
		3,94,316.50		3,92,532.50
6. Others current Liabilities		2,84,40,059.79		3,45,11,601.92
Total (a)		**3,41,82,031.71**		**4,04,93,224.84**
B. Provisions				
1. For Taxation		0.00		0.00
2. Gratuity		61,37,980.00		61,37,980.00
3. Superannuation/Pension		0.00		0.00
4. Accumulated Leave Encashment		19,19,570.80		19,19,570.80
5. Trade Warranties/Claims		0.00		0.00
6. Others (Specify)		0.00		0.00
Total (b)		**80,57,550.80**		**80,57,550.80**
Balance as at the year end (a+b)		**4,22,39,582.51**		**4,85,50,775.64**

SHIWANI TANWAR
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फोन नं: 0124-2338929/ Tel. No. 0124 2338
फोन नं: 0124-2845207/ Tel. No. 0124-284520

As per our separate report of even date attached

For Maheshwari PA & Associates
Chartered Accountants
(FRN-012023C)

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
Meerut

MAHESHWARI P.A. & ASSOCIATES
CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURUGRAM
NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGAON
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021

SCHEDULE 8 - FIXED ASSETS/ DEPRECIATON

DESCRIPTION	Rate of Dep.	GROSS BLOCK					DEPRECIATION				NET BLOCK	
		Cost /valuation As at beginning of the Year	Additions during the Year		Deductions during the Year	Cost /valuation As at end of the Year	As at the beginning of the Year	Depreciation for current year	On Deductions during the year	Total Depn. Upto 31.03.21	As at Current year-end	As at Previous year-end
			More than 6 Months	Less than 6 Months								
A. FIXED ASSETS:												
1 LAND												
a) Freehold		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
b) Leasehold		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2 BUILDINGS:												
a) On Freehold Land		0.00			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
b) On Leasehold Land	10%	75,39,65,741.73	0.00	0.00	0.00	75,39,65,741.73	10,77,60,466.63	6,46,20,527.51	0.00	17,23,80,994.14	58,15,84,747.59	64,62,05,275.10
c) Ownership Flats/Premises		0.00			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
d) superstructures on land not belonging to the entity		0.00			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		0.00			0.00	0.00	0.00	0.00		0.00	0.00	0.00
3 PLANT MACHINERY & EQUIPMENT	15%	35,87,23,302.58	15,10,898.86	1,04,37,468.75	0.00	37,06,71,670.19	22,93,79,696.53	2,04,10,985.89	0.00	24,97,90,682.42	12,08,80,987.77	12,93,43,606.05
4 VEHICLES	15%	41,45,034.00	0.00	0.00	0.00	41,45,034.00	23,04,103.83	2,76,139.53	0.00	25,80,243.36	15,64,790.64	18,40,930.17
5 FURNITURE, FIXTURES	10%	4,09,88,269.00	87,110.00	48,372.00	0.00	4,11,23,751.00	2,55,51,579.66	15,54,798.54	0.00	2,71,06,378.20	1,40,17,372.80	1,54,36,689.34
6 OFFICE EQUIPMENT	15%	4,78,68,753.95	20,900.00	6,12,417.00	0.00	4,85,02,070.95	2,89,40,436.95	28,88,313.83	0.00	3,18,28,750.78	1,66,73,320.18	1,89,28,317.00
7 COMPUTER/PERIPHERALS	40%	1,23,77,214.81	4,67,670.00	1,35,222.00	0.00	1,29,80,106.81	91,93,990.68	14,87,402.05	0.00	1,06,81,392.73	22,98,714.08	31,83,224.13
8 ELECTRIC INSTALATIONS		0.00			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9 LIBRARY BOOKS	40%	5,54,544.00	4,240.00	3,000.00	0.00	5,61,784.00	2,25,801.80	1,33,792.88	0.00	3,59,594.68	2,02,189.32	3,28,742.20
10 TUBEWELLS & W. SUPPLY		0.00			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11 OTHER FIXED ASSETS (Patents & Copyrights)	25%	53,55,643.00	0.00	0.00	0.00	53,55,643.00	44,70,951.81	2,21,172.80	0.00	46,92,124.61	6,63,518.39	8,84,691.19
TOTAL OF THE CURRENT YEAR		1,22,39,78,503.07	20,90,818.86	1,12,36,479.75	0.00	1,23,73,05,801.68	40,78,27,027.89	9,15,93,133.03	0.00	49,94,20,160.92	73,78,85,640.78	81,61,51,475.18
CAPITAL WORK IN PROGRESS		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
B (Building)												
C PROJECT EQUIPMENTS	15%	80,74,59,759.16	30,27,454.86	90,82,762.18	0.00	81,95,69,976.20	53,14,53,199.85	4,25,36,309.00	0.00	57,39,89,508.85	24,55,80,467.35	27,60,06,559.31
TOTAL (A+B+C)		2,03,14,38,262.23	51,18,273.72	2,03,19,241.93	0.00	2,05,68,75,777.88	93,92,80,227.74	13,41,29,442.03	0.00	1,07,34,09,669.77	98,34,66,108.13	1,09,21,58,034.49

(Note to be given as to cost of assets on hire purchase basis included above)

SCHEDULE 9 - INVESTMENT FROM EARMARKED/ENDOWMENT FUNDS: NIL

As per our separate report of even date attached

SHIWANI TANWAR
FINANCE & ACCOUNTS OFFICEER (I/C)
NBRC

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Finance & Account Officer
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National Brain Research Centre
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हरियाणा / Haryana
फोन नं: 0124-2338929/ Tel. No. 0124-2338929
फोन नं: 0124-2845207/ Tel. No. 0124-2845207

For Maheshwari PA & Associates
Chartered Accountants
(FRN 012023C)

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
Meerut

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021 Amount in (Rs.)

SCHEDULE 10 - INVESTMENTS-OTHERS

	Current Year	Previous Year
1 In Government Securities	0.00	0.00
2 Other approved Securities	0.00	0.00
3 Shares	0.00	0.00
4 Debentures and Bonds	0.00	0.00
5 Subsidiaries and Joint Ventures	0.00	0.00
6 Others (CPF Fund)	1,35,63,572.19	2,51,08,109.99
Total	**1,35,63,572.19**	**2,51,08,109.99**

SHIWANI TANWAR
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फोन नं: 0124-2338929/ Tel. No. [illegible]
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For Maheshwari PA & Associates
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(FRN-012023C)

Ankur Agarwal
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MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021

Amount in (Rs.)

SCHEDULE 11 - CURRENT ASSETS, LOANS, ADVANCES ETC.	Current Year		Previous Year	
A. Current Assets				
1 Inventories:				
a) Stores and Spares	0.00		0.00	
b) Loose Tools	0.00		0.00	
c) Stock-In-Trade				
Finished Goods	0.00		0.00	
Wrok-in-progress	0.00		0.00	
Raw Materials	0.00	0.00	0.00	0.00
2 Sundry Debotrs:				
a)Debts Outstanding for a period exceeding six months	0.00		0.00	
b) Others	0.00	0.00	0.00	0.00
3 Cash balances in hand (including cheque/drafts and imprest)		39,098.00		2,62,526.00
4 Bank Balances:				
a) With Scheduled Banks:				
-On Current Accounts	0.00		0.00	
-On Deposit Accounts (includes margin money)	32,82,09,084.00		80,06,36,949.00	
-On Savings Accounts	49,71,09,024.36		19,80,80,018.04	
		82,53,18,108.36		**99,87,16,967.04**
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	0.00	0.00
5 Post Office-Savings Accounts		0.00		
Total (A)		**82,53,57,206.36**		**99,89,79,493.04**

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Amount in (Rs.)

SCHEDULE 11 - CURRENT ASSETS, LOANS, ADVANCES ETC. (Contd.)	Current Year		Previous Year	
B. LOANS, ADVANCES AND OTHER ASSESTS				
1 Loans:				
a) Staff	52,14,241.87		60,14,946.87	
b) Other Entities engaded in activities/objectives similar to that of the entity	0.00		0.00	
c) Other (Imprest)	48,775.00		1,12,922.00	
		52,63,016.87		61,27,868.87
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)	11,01,173.00		12,08,124.00	
C) Other - Advance to Parties	9,92,024.75		65,56,871.86	
- Other Advances	57,62,502.45		57,62,502.45	
		78,55,700.20		1,35,27,498.31
3 Income Accured:				
a) On Investments from Earmarked/Endowment Funds	0.00		0.00	
b) On Investments-Others	0.00		4,89,840.00	
c) On Loans and Advances	0.00		0.00	
d) Others (SB A/C)	0.00	0.00	17,740.00	5,07,580.00
b) (includes income due unrealised-Rs.....)				
4 Claims Receivable (TDS Receivable) & Income Tax		1,07,61,830.80		1,06,27,700.80
Total (B)		2,38,80,547.87		3,07,90,647.98
Total (A+B)		84,92,37,754.23		1,02,97,70,141.02

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SCHEDULE 12 - INCOME FROM SALES/SERVICES	Current Year	Previous Year
1 Income from Sales	0.00	0.00
2) Income from Services	0.00	0.00

SCHEDULE 13 - GRANTS/SUBSIDIES		
(Irrevocable Grants & Subsidies Receivecd)	Current Year	Previous Year
1 Central Government	0.00	0.00
2 State Government(s)	0.00	0.00
3 Government Agencies	0.00	0.00
4 Institutions/Welfare Bodies	25,45,00,000.00	29,13,00,000.00
5 International Organisations	0.00	0.00
6 Others (Specify)	0.00	0.00
Total	25,45,00,000.00	29,13,00,000.00

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National Brain Research Centre
मानेसर / Manesar-122050
हरियाणा / Haryana

PROF. PRAVAT KUMAR MANDAL
DIRECTOR (I/C)
NBRC

Prof. Pravat K. Mandal
प्रो. प्रवात कुमार मंडल
प्रभारी निदेशक / Director-in-Charge
राष्ट्रीय मस्तिष्क अनुसंधान केन्द्र
National Brain Research Centre
मानेसर- 122 052 / Manesar-122 052
हरियाणा / Haryana
फोन नः 0124-2338929/ Tel. No. 0124-2338
फोन नः 0124-2845207/ Tel. No. 0124-

As per our separate report of even date attached

For Maheshwari PA & Associates
Chartered Accountants
(FRN-012023C)

Ankur Agarwal
PARTNER
Membership No. 409197
Date: 08.10.2021
Meerut

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED March 31, 2021

SCHEDULE 14 - FEES/ SUBSCRIPTIONS — Amount in (Rs.)

		Current Year	Previous Year
1	Entrance Fees	3,69,480.67	4,12,404.00
2	Annual Fees/Subscriptions	6,95,290.00	7,48,000.00
3	Seminar/Program Fees	0.00	0.00
4	Consultancy Fees	0.00	0.00
5	Others (Fellowship Grants)	0.00	0.00
	Total	**10,64,770.67**	**11,60,404.00**

SHIWANI TANWAR
FINANCE & ACCOUNTS OFFICEER (I/C)
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NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF BALANCE SHEET AS AT March 31, 2021

Amount in (Rs.)

SCHEDULE 15 - INCOME FROM INVESTMENTS	Investment from Earmarked Fund		Investment-Others	
(Income on invest. From Earmarked/Endowment Funds transferd to Funds)	Current Year	Previous Year	Current Year	Previous Year
1 Interest				
a) On Govt. Securities	0.00	0.00	0.00	0.00
b) Other Bonds/Debentures	0.00	0.00	0.00	0.00
2 Dividents:				
a) On Shares	0.00	0.00	0.00	0.00
b) On Mutual Fund Securities	0.00	0.00	0.00	0.00
3 Rents	0.00	0.00	1,20,539.00	2,83,250.00
4 Others (Project Receipts)	0.00	0.00	11,46,515.00	57,46,088.92
Total (B)	0.00	0.00	12,67,054.00	60,29,338.92
TRANSFERRED TO EARMARKED/ENDOWMENT FUNDS				

SCHEDULE 16 -INCOME FROM ROYALITY, PUBLICATION ETC. NIL

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NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED March 31, 2021

SCHEDULE 17 -INTEREST EARNED — Amount in (Rs.)

		Current Year	Previous Year
1	On Term Deposits:		
	a) With Scheduled Banks	1,99,22,990.00	2,99,53,243.00
	b) With Non-Scheduled Banks	0.00	0.00
	c) With Institutions	0.00	0.00
	d) Others	0.00	0.00
2	On Savings Accounts:		
	a) With Scheduled Banks	89,85,042.99	87,68,825.22
	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	16,636.00	2,62,185.00
4	Interest on Debtors and Others Receivables	0.00	0.00
	Total	**2,89,24,668.99**	**3,89,84,253.22**

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NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED March 31, 2021

SCHEDULE 18 -OTHER INCOME — Amount in (Rs.)

	Current Year	Previous Year
1 Profit on Sale/disposal of Assets:		
a) Owned assets	0.00	0.00
b) Assets acquired out of grants, or received free of cost	0.00	0.00
2 Export Incentives realized		
3 Fees of Miscellaneous Services		
4 Miscellaneous Income	19,37,443.00	20,41,033.00
5 Prior Period Income	0.00	0.00
Total	**19,37,443.00**	**20,41,033.00**

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

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MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

NATIONAL BRAIN RESEARCH CENTRE
NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED March 31, 2021

SCHEDULE 20 -ESTABLISHMENT EXPENSES — Amount in (Rs.)

	Current Year	Previous Year
a) Salaries and Wages	6,21,16,648.00	5,95,76,344.00
b) Allowances and Bonous	0.00	0.00
c) Contribution to Provident Fund	0.00	6,46,110.00
d) Contribution to Pension Scheme	6,02,870.00	0.00
e) Staff Welfare Expenses	41,963.00	0.00
f) Expenses on Employees Retirement and Terminal Benefits	0.00	0.00
g) Others - Children education reimbursement	15,66,000.00	14,85,000.00
- Leave encashment	2,94,790.00	3,68,391.00
- LTC expenses	7,88,571.00	8,52,917.00
- Medical reimbursement	13,43,800.00	12,74,250.00
- NPS(employer subscription)	46,61,052.00	50,76,188.00
- overtime allowance	3,077.00	0.00
- Skilled manpower	2,17,47,667.00	1,86,44,639.00
- Medical insurance (Staff)	17,21,182.00	13,75,902.00
- Office expenses	3,82,551.00	2,29,262.00
Total	**9,52,70,171.00**	**8,95,29,003.00**

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NH-8, NAINWAL MORE, MANESAR, GURGRAM
SCHEDULE FORMING PART OF INCOME AND EXPENDITURE FOR THE YEAR ENDED March 31, 2021

SCHEDULE 21 -OTHER ADMIISTRATIVE EXPENSES

Amount in (Rs.)

		Current Year	Previous Year
1	Purchases	0.00	0.00
2	Labour and processing expenses	0.00	0.00
3	Cartage and Carriage inwards	0.00	0.00
4	Electricity and Power	3,64,07,260.00	3,63,03,226.00
5	Water Charges	0.00	0.00
6	Insurance	4,94,310.00	3,77,530.00
7	Repairs and maintenance	4,26,32,884.55	4,68,95,323.03
8	Excise Duty	0.00	0.00
9	Rent (Lease Rent), Rates and Taxes	14,05,777.00	14,05,777.00
10	Vechicles Running and Maintenance	52,719.00	1,37,090.00
11	Postage, Telephone and Communication Charges	5,38,013.00	4,88,506.00
12	Printing and Stationary	7,34,727.00	13,79,395.00
13	Travelling and Conveyance Expenses	20,10,200.00	63,25,034.47
14	Expenses on Seminar/Workshops	1,50,114.00	10,99,275.00
15	Subscription Expenses	18,05,151.71	28,25,204.40
16	Expenses on Fees	0.00	0.00
17	Auditor Remuneraion	24,000.00	24,000.00
18	Hospitality Expenses	1,95,930.00	2,02,323.00
19	Professional Charges	6,67,067.00	11,63,000.00
20	Provision for bad and Doubtful Debts/Advances	0.00	0.00
21	Irrecoverable Balances Written-off	0.00	0.00
22	Packing Charges	0.00	0.00
23	Freight and Forwarding Expenses	0.00	0.00
24	Distribution Expenses	0.00	0.00
25	Advertisement and Publicity	39,80,176.98	9,23,791.00
26	Foreign Exchange Fluctuation Loss/Gain	0.00	0.00
27	Prior Period Expenses	11,11,292.00	0.00
28	Others - Bank charges	9,366.66	7,612.00
	- Misc. expenses	3,07,772.00	4,26,790.00
	- Books and Periodicals	90,912.00	1,26,858.00
	- Honorarium (others)	3,86,500.00	4,06,500.00
	- Petrol, Diesel & CNG etc.	6,23,480.00	7,05,879.00
	- Manpower	2,02,75,107.00	81,77,283.00
	- Horticulture	24,76,425.00	34,91,410.00
	- Training and networking expense	3,88,70,367.00	3,72,43,448.00
	- Laboratory & Animal Consumables	2,08,33,661.66	1,92,40,755.50
29	Accreditation Fee	2,50,774.00	0.00
	Total	17,63,33,987.56	16,93,76,010.40

SCHEDULE 22 -EXPENDITURE ON GRANTS, SUBSIDIES ETC. NIL

SCHEDULE 23 -INTEREST PAID NIL

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MAHESHWARI P.A. & ASSOCIATES
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NATIONAL BRAIN RESEARCH CENTRE, MANESAR, GURGAON

• SIGNIFICANT ACCOUNTING POLICIES & NOTES ON ACCOUNTS FORMING PART OF THE BALANCE SHEET AS AT 31ST MARCH, 2021 AND INCOME & EXPENDITURE ACCOUNT FOR THE YEAR ENDED 31STMARCH,2021.

• 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, unless otherwise stated.

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.

3. Fixed Assets:

3.1 Fixed 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 Fixed 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 Fixed Assets have been created mainly out of grants received from the Department of Biotechnology, Ministry of Science and Technology, Government of India.

4. Depreciation:

4.1 Depreciation provided for current year on the fixed assets of Project for Rs. 4,25,36,309.00 (previous year Rs. 4,84,40,074.00) 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 for other than project assets amounting to Rs. 9,15,93,133.03 for current financial year (Rs. 9,92,57,469.55 for previous year) had been debited to Income & Expenditure Account. Depreciation is being charged as per Income Tax Act 1961 on W.D.V basis.

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

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 only a few employees are under CPF (as they had joined NBRC before 01st January, 2004). The employees joined New Pension Scheme (NPS).

9.2 The NBRC has not made any provision for gratuity and leave encashment during financial year 2020-2021 as against the requirement of AS-15 issued by ICAI. However, the amount of gratuity and leave encashment to the extent of Rs. 61,37,980.00 and Rs. 1919570.80 respectively already exists on 31st March, 2021, (Rs. 61,37,980.00 and Rs. 1919570.80 respectively as on 31st March, 2020) against provision made earlier.

10. Taxation:

In view of the tax exemption status of the National Brain Research Centre, has been registered society registration Act-1860 at Autonomous bodies.

11. Current Assets, Loans & Advances:

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. However, advances appearing under the head Current assets, Loans & Advances under Schedule-11 are subject to confirmation from respective parties. Further an amount is showing under head **Advance to Parties** amounting to **Rs. 9,92,024.75** against which no bills is received till date.

12. Bank Balance:

All Banks accounts have been reconciled till 31st March, 2021.

14. Fraud/Manipulation of funds encountered by NBRC:

No Fraud was detected during the year.

15. Outstanding Balances of Closed Projects:

As on 31st March, 2021, 40 Projects have already been closed which amounting of Rs. 32.17 Lakh transferred to DBT & Rs.15.88 to DST & reaming other project of Rs.19.68 (Debit balance) is still pending.

16. Contingent Liabilities

1.1 Claims against the Entity not acknowledged as debt. Rs. NIL (Previous year Rs. NIL).
1.2 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).

1.3 Disputed demands in respects of Income tax (TDS) Rs. 56,77,370.00 (Previous year Rs. 1,06,27,700.80) which is under representation before the concerned authorities. Further, TDS deducted and to be received as refund amounts to Rs. 47,66,561.80 out of which Rs. 40,30,217.52 is pending since 2008-09. The appeal has already been made to Income Tax Authority (CIT - Chandigarh).

17. 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). However, reference is drawn to para 3.5 above.

18. Lease Obligations

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

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

19. Foreign Currency Transactions

20.1 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.2,05,45,268.66

20.2 Expenditure in foreign currency:

a) Travelling charges Rs. 25,929/-
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 .

20.3 Earnings:

Value of Exports of FOB basis Rs. NIL .

20. Remuneration to auditors:

- As Auditors Rs. 24,000.00 (Previous year Rs. 24,000.00).

21. Others

22.1 The Balance in the name of various parties under the head Current Liabilities are subject to confirmation/ reconciliation by respective parties. The total amount payable to Sundry Creditors is Rs. 11,52,789 (previous year Rs. 11,83,854.00).

22.2 Schedules 1 to 23 along with Annexures 1 to 112 are annexed to and form an integral part of the Balance Sheet as at 31st March, 2021 and the Income and Expenditure Account for the year ended on that date.

22.3 Corresponding figures for the previous year have been regrouped/ rearranged, wherever necessary.

22.4 Accounting polices not referred to otherwise be consistent with Generally Accepted Accounting Principles (GAAP).

22.5 There is outstanding balance of Expenses Payable of Rs. 1,91,57,345.33 out of which most of the expenses is related to next financial year but the same are booked in current financial year. Further no voucher has been received for the verification of the same.

22.6 There are outstanding Income Tax Demands for A.Y. 2017-18 amounting to Rs. 2,99,76,350.00 and A.Y. 2018-19 amounting to Rs. 136,03,15,740.00 respectively against which appeals to CIT- Chandigarh has already been filed.

22.7 There is an expense of Rs. 2,09,682.00 relating to insurance of building some part of which relate to FY 2019-20 (i.e. prior period) and This bill was received in current period and it was booked in FY 2020-21. Further Accrued interest of Rs. 9,01,610.00 reversed in current period as it is already assessed in the FY 2019-20.

SHIWANI TANWAR

(FINANCE & ACCOUNTS OFFICER (I/C)

वित्त एवं लेखा अधिकारी
Finance & Account Officer
राष्ट्रीय मस्तिष्क अनुसंधान केंद्र
National Brain Research Centre
मानेसर / Manesar-122050
हरियाणा / Haryana

PRAVAT KUMAR MANDAL

(DIRECTOR I/C)

Prof. Pravat K. Mandal
प्रो. प्रवात कुमार मंडल
प्रभारी निदेशक / Director-in-Charge
राष्ट्रीय मस्तिष्क अनुसंधान केन्द्र
National Brain Research Centre
मानेसर- 122 052 / Manesar-122 052
हरियाणा / Haryana
फोन नः 0124-2338929/ Tel. No. 0124-2338929
फोन नः 0124-2845207/ Tel. No. 0124-2845207

As per our separate report of even date attached

For Maheshwari P A &Associates
(Chartered Accountants)
(FRN-012023C)

MAHESHWARI P.A. & ASSOCIATES CHARTERED ACCOUNTANTS

(CA ANKUR AGARWAL)
PARTNER
M.NO. 409197

Place: Meerut
Date: 8th October, 2021

NATIONAL BRAIN RESEARCH CENTRE, NH-8, NAINWAL MORE, MANESAR GURGAON
ANNEXURE OF PROJECT GRANTS AND EXPENDITURE FOR THE YEAR ENDED 31.03.2021

S. No./ Annx. No.	NAME OF PROJECT	Opening Balance as on 01.04.2020	Grants received during the year 2020-21	Interest earned during the year 2020-21	Capital Exp. during the year 2020-21	Revenue Expenditure during the year 2020-21			Refund of Unspent Balance	Closing Balance as on 31.03.2021
						Manpower	Others	Total Expenditure		
2	Wellcom Trust/DBT Indian Alliance -Dr. Anindya Ghosh Roy	23,41,435.48	0.00	70,885.65	20,12,811.54	0.00	3,05,913.59	3,05,913.59	93,596.00	0.00
3	CSIR Japanese Enccephalities - Dr. Anirban Basu	(86,110.00)	4,34,943.00	0.00	0.00	0.00	2,41,966.00	2,41,966.00	0.00	1,06,867.00
4	Elucidating the role of long non coding RNAs (lnCRNAs) (Dr. Anirban	7,98,006.00	2,15,578.00	0.00	0.00	0.00	9,62,882.00	9,62,882.00	0.00	50,702.00
5	MicroRNA mediated Reg. of Neural stem(Dr. Anirban Basu)	15,82,816.62	0.00	28,317.00	0.00	0.00	4,70,523.00	4,70,523.00	0.00	11,40,610.62
6	Tata innovation fellowship award- Dr. Anirban Basu	2,83,482.47	0.00	0.00	0.00	0.00	2,89,258.00	2,89,258.00	0.00	-5,775.53
7	Therapeutic Role DBT(Dr. Anirban Basu)	17,148.96	8,10,005.00	10,979.00	0.00			0.00	0.00	8,38,132.96
8	Multifaceted Kinase CDK5 - Dr. Aparna Dixit	-1,223.93	1,223.93	0.00	0.00			0.00	0.00	0.00
9	Mapping of Common Mental Disorders Over Lifespan- Dr Arpan Banerjee	2,61,56,450.00	0.00	5,65,128.00	7,63,875.00	8,91,734.00	3,13,288.00	12,05,022.00	0.00	2,47,50,681.00
10	Early Dignostics of structural and functional- Dr Arpan Banerjee	31,16,539.00	0.00	37,337.00	0.00	6,48,000.00	10,63,153.00	17,11,153.00	0.00	14,42,723.00
11	Dementia Tissue MRI studies(Dr. Dipanjan Roy)	17,89,897.00	0.00	17,831.00	0.00			0.00	80,897.00	17,26,831.00
12	Oscillatory network dynamics DST(Dr. Dipanjan Roy)	6,91,210.00	0.00	3,012.00	0.00	4,92,439.00	1,20,000.00	6,12,439.00	0.00	81,783.00
13	Ramalinga Swamy - Dr. Dipanjan Roy	8,91,252.26	0.00	8,805.00	0.00	1,20,900.00	5,33,697.79	6,54,597.79	0.00	2,45,459.47
14	Implications in tumor progression- Dr. Ellora Sen	14,54,224.49	0.00	12,334.00	0.00	0.00	4,21,213.00	4,21,213.00	0.00	10,45,345.49
15	National Bioscience Award- Dr. Ellora Sen	26,403.29	0.00	0.00	0.00			0.00	0.00	26,403.29
16	Neurobiology of Dyslexia Brain & Behavior-Dr.Nandini C.Singh	6,14,968.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6,14,968.00
17	Fetal neural stem cells to oligodendrocytes (Dr. Pankaj seth)	1,94,376.00	0.00	0.00	0.00	0.00	0.00	0.00	1,94,376.00	0.00
18	Dyslexia Linked RNA(Dr. Pankaj Seth)	11,22,525.05	0.00	0.00	0.00	0.00	3,24,714.00	3,24,714.00	0.00	7,97,811.05
19	Effect of Hypoxia on different Neural-Dr Pankaj Seth	6,75,182.30	19,85,256.00	16,922.00	6,96,000.00	2,37,097.00	9,54,182.00	11,91,279.00	53,501.00	7,36,580.30
20	Hypoxia Ind. Change in Blood Brain- Dr Pankaj Seth	10,79,010.00	6,50,000.00	15,358.00	0.00	67,355.00	6,23,230.08	6,90,585.08	19,256.00	10,34,526.92
21	A CROSS-CULTURE PERSPECTIVE(DBT-NETHERLANDS) -Dr. PRAVAT MANDAL	4,85,631.00	0.00	0.00	0.00	9,04,264.00	0.00	9,04,264.00	30,394.00	-4,49,027.00
22	Characterizing biomarkers of Alzheimer's disease Dr. Pravat Mandal	1,828.50	0.00	0.00	0.00	0.00	11,840.00	11,840.00	0.00	-10,011.50
23	Dementia Imaging studies(Dr. Pravat Kumar Mandal)	17,89,897.00	0.00	16,966.00	6,79,784.00	67,742.00	1,55,000.00	2,22,742.00	80,897.00	8,23,440.00
24	Novel Imaging Dignostics Indo-Aus grant(Dr. Pravat Kumar Mandal)	40,04,060.00	28,13,925.00	0.00	33,68,000.00	21,30,689.00	46,016.00	21,76,705.00	94,195.00	11,79,085.00
25	SPECIFIC BRAIN TEMPLATE DST Dr. PRAVAT K MANDAL	-1,87,110.50	0.00	0.00	0.00	1,80,000.00		1,80,000.00	0.00	-3,67,110.50
26	Tata innovation fellowship Award - Dr. Pravat Mandal	23,038.00	0.00	0.00	0.00	25,000.00		25,000.00	3,250.00	-5,212.00
27	Crspri System - Dr. Sourav Banarjee	-7,25,783.32	0.00		0.00	0.00	0.00	0.00	0.00	-7,25,783.32
28	DBT Mirna Meditate Control - Dr. Sourav Banarjee	1,89,009.97	0.00	0.00	0.00			0.00	0.00	1,89,009.97
29	Mechanism of Combinational Control(SERB)- Dr Sourav	19,83,103.00	11,00,000.00	0.00	9,77,000.00	4,00,065.00	12,35,417.00	16,35,482.00	0.00	4,70,621.00
30	Innovation In Science Pursuit For Inspired Research(INSPIRE)- Dr. Yogita	3,56,492.18	0.00	0.00	0.00	1,34,483.00		1,34,483.00	0.00	2,22,009.18
31	Influence of social cues omapatial cognition - Dr. Chetan Yadav	0.00	0.00	0.00	0.00			0.00	0.00	0.00
32	DST-CSRI (Dr. Prem Chand)	28,916.16	0.00	0.00	0.00			0.00	0.00	28,916.16
33	Centre for Excellence for Epilepsy(Phase-II)	4,96,51,421.00	3,61,30,695.00	7,46,223.00	0.00	65,08,650.00	20,01,042.00	85,09,692.00	52,66,192.00	7,27,52,455.00
34	Dementia Meeting	25,67,077.00	0.00	0.00	0.00	0.00		0.00	25,67,077.00	0.00
35	Dementia Science Programme	1,21,49,021.32	1,12,611.00	0.00	11,88,952.00	8,41,990.00	74,909.00	9,16,899.00	11,99,515.00	89,56,266.32
36	Dementia Basic Biology(Dr. Shiv Kumar Sharma)	44,18,140.50	0.00	42,706.00	1,99,395.00	0.00	30,739.00	30,739.00	2,22,779.00	40,07,933.50
37	Distributed Information Centre	8,42,613.33	1,28,300.67	0.00	0.00	8,86,367.00	3,32,347.00	12,18,714.00	0.00	-2,47,800.00
38	Epilepsy Project of NBRC	53,82,067.30	0.00	0.00	0.00			0.00	0.00	53,82,067.30
39	PDF-SERB-Dr Sandeep Kumar	2,04,015.00	0.00	0.00	0.00	0.00	64,000.00	64,000.00	0.00	1,40,015.00
40	PDF-SERB(Soibam Shyamchand)	-58,472.00	0.00	0.00	0.00			0.00	0.00	-58,472.00
41	Mechanisms Of Adult Brain Reorganisation- Dr. Neeraj Jain	0.00	0.00	0.00	0.00			0.00	0.00	0.00
42	DST- Inspired Fellow (Sriparna Mukherjee)	4,288.00	0.00	0.00	0.00			0.00	0.00	4,288.00
43	TWAS-DBT (Saliu Ibrahim)	11,045.00	0.00	0.00	0.00			0.00	0.00	11,045.00
44	Workshop & Conference(NBRC)	-12,96,823.58	8,22,794.27		0.00		46,911.00	46,911.00	0.00	-5,20,940.31
45	IBRO RETURNED HOME START-UP GRANT-DR BHAVANI SHANKAR SAHU	7,73,173.00	8,12,260.00	27,173.00	0.00		2,73,544.00	2,73,544.00	0.00	13,39,062.00
46	ROLE REGUATING PHYSIO-METABOLIC FUNCTION- Dr BHAVANI	16,29,893.00	17,15,703.00	25,967.00	0.00	0.00	21,39,051.53	21,39,051.53	0.00	12,32,511.47
47	ROLE OF EPHRINS/EPH RECEPTORS IN HIV MEDIATED NEUROPATHOGENSIS- DR PANKAJ SETH	21,35,943.90	15,56,576.00	41,150.00	14,13,458.31	76,000.00	16,45,173.02	17,21,173.02	43,424.00	5,55,614.57
48	ARTIFICIAL INTELLIGENCE - DR PRAVAT K. MANDAL	25,59,180.00	0.00	19,420.00	3,11,330.00	14,21,987.00	93,945.00	15,15,932.00	19,420.00	7,31,918.00
49	NEUROANATOMICAL STUDY- DR SOUMYA IYENGAR	11,47,395.00	0.00	20,257.00	0.00	0.00	3,33,011.37	3,33,011.37	0.00	8,34,640.63
50	CRISPR-CAS13- DR SOURAV BANERJEE	35,81,520.00	0.00	58,046.00	99,880.00	6,643.00	12,85,319.00	12,91,962.00	0.00	22,47,724.00
51	EMOTIONAL PRIMING- DR SHUBHAM KUMAR	6,58,747.00	0.00	0.00	0.00			0.00	0.00	6,58,747.00
52	WELLCOME TRUST/DBT INDIA ALLIANCE FELLOW- DR SWAGATA DEY	10,48,581.00	36,75,469.00	20,025.00	2,44,969.00	0.00	13,57,867.00	13,57,867.00	0.00	31,41,239.00
53	WELLCOME TRUST/DBT INDIA ALLIANCE FELLOW- DR NIVETHIDA T.	21,58,769.00	1,58,172.00	48,026.00	0.00	83,000.00	16,69,984.00	17,52,984.00	0.00	6,11,983.00
54	DBT-TWAS FELLOWSHIP- DR ASHRAFUL HASSAN	3,88,242.00	0.00	0.00	0.00		3,88,242.00	3,88,242.00	0.00	0.00
55	BBNSC - Dr. Rema	18,09,628.00	0.00	0.00	0.00			0.00	18,09,628.00	0.00
56	BBNSC - Dr. Shyamala	-3,92,947.00	3,92,947.00	0.00	0.00			0.00	0.00	0.00
57	COGNITIVE NEUROSCIENCE DBT-PROJECT- (Dr. Aditya)	-4,37,464.00	4,37,464.00	0.00	0.00			0.00	0.00	0.00
58	C V Raman Interest Income Fellow(Dr. Rolland Kipre)	26,543.00	0.00	0.00	0.00			0.00	0.00	26,543.00
59	Chandipura Virus Infection - Dr. Anirban Basu	-22,580.53	22,580.53	0.00	0.00			0.00	0.00	0.00
60	CSIR -II Study the Role of Neural Immune Respnce - Dr. Anirban Basu	-1,68,365.93	1,68,365.93	0.00	0.00			0.00	0.00	0.00
61	DBT National Bioscience Award 2010 - Dr. Anirban Basu	585.65	0.00	0.00	0.00		585.65	585.65	0.00	0.00
62	Study of Mole. Mechanism - Dr. Anirban Basu	-68,830.00	68,830.00	0.00	0.00			0.00	0.00	0.00
63	Neuro -Cognitive Networks Underlying Dr. Arpan Banerjee	18,438.00	0.00	0.00	0.00			0.00	0.00	18,438.00
64	Vision Guide Speech Percention- Dr. Arpan Banerjee	5,85,272.00	0.00	0.00	0.00			0.00	0.00	5,85,272.00
65	DST Inspired Faculty Award- Dr. Deepashri	19,00,000.00	0.00	0.00	0.00		19,00,000.00	19,00,000.00	0.00	0.00
66	BBNSC - Dr. Ellora	-4,03,419.00	4,03,419.00	0.00	0.00			0.00	0.00	0.00
67	Understanding the Signaling Circuities - Dr. Ellora Sen	-5,75,915.39	5,75,915.39	0.00	0.00			0.00	0.00	0.00
68	DST Austism Spectrum Disorder - Dr. Nandini C. Singh	82,849.00	0.00	0.00	0.00		82,849.00	82,849.00	0.00	0.00

S. No./ Annx. No.	NAME OF PROJECT	Opening Balance as on 01.04.2020	Grants received during the year 2020-21	Interest earned during the year 2020-21	Capital Exp. during the year 2020-21	Revenue Expenditure during the year 2020-21			Refund of Unspent Balance	Closing Balance as on 31.03.2021
						Manpower	Others	Total Expenditure		
69	Autism Behavior and Diffusion Tensor Imaging - Dr. Nandini C. Singh	60,474.00	0.00	0.00	0.00			0.00	0.00	60,474.00
70	Comp. Analysis of Speech Imp. - Dr. Nandini C. Singh	-5,47,567.00	5,47,567.00	0.00	0.00			0.00	0.00	0.00
71	CSI Development and Validation of Screening Tools -Dr. Nandini C Singh	-3,03,509.00	3,03,509.00	0.00	0.00			0.00	0.00	0.00
72	DBT ITPAR Grant-Dr.Nandini C. Singh	5,56,234.89	0.00	0.00	0.00			0.00	0.00	5,56,234.89
73	Multi Displinary System of Parkinson Disease - Dr. Nandini C. Singh	11,89,000.00	0.00	4,32,883.00	0.00			0.00	16,21,883.00	0.00
74	CSIR -Project Dr. Nihar Ranjan Jana	73,089.50	0.00	0.00	0.00			0.00	0.00	73,089.50
75	animal models of Huntington's disease Dr. Nihar Ranjan Jana	-2,999.59	2,999.59	0.00	0.00			0.00	0.00	0.00
76	Tata Innovation Fellowship- Dr. Nihar Ranjan Jana	3,09,758.91	0.00	0.00	0.00			0.00	0.00	3,09,758.91
77	Cellular & Mole. Basis - Dr. Pankaj Seth	-34,974.00	34,974.00	0.00	0.00			0.00	0.00	0.00
78	INDO-US & NIH R01 - Dr. Pankaj Seth	-6,31,828.42	0.00	0.00	0.00			0.00	0.00	-6,31,828.42
79	National Initiative On Glia Cell Research Project - Dr. Pankaj Seth	92,588.71	0.00	0.00	0.00			0.00	0.00	92,588.71
80	DBT BIRAC Under CRS Scheme Project Grant - Dr. Ranjit Kr. Giri	56,991.50	0.00	0.00	0.00		56,991.50	56,991.50	0.00	0.00
81	Ramalinga Swamy - Dr. Ranjit Kr. Giri	-68,440.70	68,440.70	0.00	0.00			0.00	0.00	0.00
82	Women Scientist Scheme DST -Dr. Sayali Ranade	-1,31,556.00	1,31,556.00	0.00	0.00			0.00	0.00	0.00
83	National Institute Glial Cell Research - Shiv Kumar Sharma	-84,803.61	84,803.61	0.00	0.00			0.00	0.00	0.00
84	Motivated Behaviour in Male Zebra Finches - Dr. Soumya Iyengar	73,194.65	0.00	0.00	0.00		73,194.65	73,194.65	0.00	0.00
85	DST Inspire Faculty Award -Dr. Supriya Bhavani	-12,189.65	12,189.65	0.00	0.00			0.00	0.00	0.00
86	IYBA DBT 2013- Dr. Supriya Bhavanani	49,737.51	0.00	0.00	0.00		49,737.51	49,737.51	0.00	0.00
87	Neural Network Mechanism - Dr. Yoganarashimha	-10,70,838.61	10,70,838.61	0.00	0.00			0.00	0.00	0.00
88	A critical assessment of the dual stream models of visual information processing- DST - Dr. Dipanjan Ray	220.00	0.00	0.00	0.00		220.00	220.00	0.00	0.00
89	EBM Including Alzheimer Disease - Dr. Vijaylaxmi Ravindranath	-2,30,717.00	2,30,717.00	0.00	0.00			0.00	0.00	0.00
90	DST Cognitive Science Research Initiative (CSI) - Dr. Chaitra Rao	-3,24,000.00	3,24,000.00	0.00	0.00			0.00	0.00	0.00
91	Spinal Cord Plasticity ILTP - Dr. Neeraj Jain	-31,869.00	31,869.00	0.00	0.00			0.00	0.00	0.00
92	Multifactorial Risk Factor - Prof. V. Ravindranath	-29,346.00	29,346.00	0.00	0.00			0.00	0.00	0.00
93	Est. of Translational Res. Unit - Prof. P.K. Roy	43,07,442.00	0.00	0.00	0.00		43,07,442.00	43,07,442.00	0.00	0.00
94	Func. Magnetic Resonance Imaging - Prof. V. Ravindranath	-3,55,435.00	3,55,435.00	0.00	0.00			0.00	0.00	0.00
95	DBT INCRE & INCNE Grant (NBRC)	17,99,153.00	0.00	0.00	0.00		17,99,153.00	17,99,153.00	0.00	0.00
96	Programme of Co-Operation Between India and Syria Project	35,58,649.00	0.00	16,01,392.00	0.00			0.00	51,60,041.00	0.00
97	Material Malnutrition - Dr. Shyamala	-5,79,048.00	5,79,048.00	0.00	0.00			0.00	0.00	0.00
98	Mole. Role of Transc. Factors - Dr. Prabodha Kumar Swain	-6,44,021.00	6,44,021.00	0.00	0.00			0.00	0.00	0.00
99	PDF-SERB(AMIT NASKAR)	-1,28,935.00	0.00	0.00	0.00			0.00	0.00	-1,28,935.00
100	PDF-SERB(Ashok Datusalia)	45,920.00	0.00	0.00	0.00			0.00	0.00	45,920.00
101	PDF-SERB(POONAM MEENA)	-1,49,358.00	1,49,358.00	0.00	0.00			0.00	0.00	0.00
102	BBNSC - Dr. Neeraj	2,96,937.00	0.00	0.00	0.00		2,96,937.00	2,96,937.00	0.00	0.00
103	Collaboration for Trans. & Clin. Res. (GLUE) - Prof. P.K. Roy	-3,44,006.00	3,44,006.00	0.00	0.00			0.00	0.00	0.00
104	DBT Tata Innovation Fellowship -Dr P.K.Roy	6,67,424.60	0.00	0.00	0.00			0.00	0.00	6,67,424.60
105	DIT McGILL Linkage (NKN) - Prof. Prasun Kumar Roy	-5,96,926.84	5,96,926.84	0.00	0.00			0.00	0.00	0.00
106	J.C.Bose Fellowship(PROF. SUBRTA SINHA	0.58	0.00	0.00	0.00		0.58	0.58	0.00	0.00
107	Study of Neuronal Regeneration after Injury using Caenorhabditis Elegans- Dr. Anindya Ghosh Roy	0.00	21,35,000.00	26,110.00	1,54,762.19	1,63,267.00	6,96,889.00	8,60,156.00	0.00	11,46,191.81
108	International Centre for Gentic Engineering and Biotechnology(ICGEB)- Dr BHAVANI SHANKAR SAHU	0.00	12,95,352.58	0.00	0.00	0.00	1,15,982.00	1,15,982.00	0.00	11,79,370.58
109	Austin Spectrum Disorders, Genes and the Gut Microblome: Utilizing Song Birds(Zebra Finches) as a Model System- DR SOUMYA IYENGAR	0.00	13,51,520.00	0.00	0.00	0.00	1,01,400.00	1,01,400.00	0.00	12,50,120.00
110	Exploring Auditory Perception in House Crows using functional Magnetic Resonance Imaging and Neuroanatomical Techniques- DR SOUMYA	0.00	7,87,000.00	12,213.00	0.00	0.00	2,07,620.44	2,07,620.44	0.00	5,91,592.56
111	ICMR-Deepali Singh	0.00	0.00	0.00	0.00	3,14,900.00	0.00	3,14,900.00	0.00	-3,14,900.00
112	DST INSPIRE FACULTY AWARD -NBRC(2020-21)	0.00	15,24,000.00	0.00	0.00	0.00	0.00	0.00	0.00	15,24,000.00
	Total (A)	14,98,40,752.98	6,78,27,511.30	39,23,465.65	1,21,10,217.04	1,66,02,572.00	2,94,97,380.71	4,60,99,952.71	1,85,60,321.00	14,40,21,239.18
1	DELCON E-LIBRARY CONSORTIUM (B)*	39,95,76,243.99	23,84,35,520.00	33,43,846.00	0.00	13,02,105.00	40,12,01,760.15	40,25,03,865.15	0.00	23,88,51,744.84
	Grand Total (A+B)	54,94,16,996.97	30,62,63,031.30	72,67,311.65	1,21,10,217.04	1,79,04,677.00	43,06,99,140.86	44,86,03,817.86	1,85,60,321.00	38,36,72,984.02

SHIWANI TANWAR
FINANCE & ACCOUNTS OFFICER (I/C)

वित्त एवं लेखा अधिकारी
Finance & Account Officer
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PRAVAT KUMAR MANDAL
DIRECTOR (I/C)

Prof. Pravat Kumar Mandal
प्रो. प्रवात कुमार मंडल
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फोन नः 0124-2338929/ Tel. No. 0124-2338929
फोन नः 0124-2845207/ Tel. No. 0124-2845207

As per our separate report
of even date attached

For MAHESHWARI PA & ASSOCIATES
Chartered Accountants
(FRN-000237N)

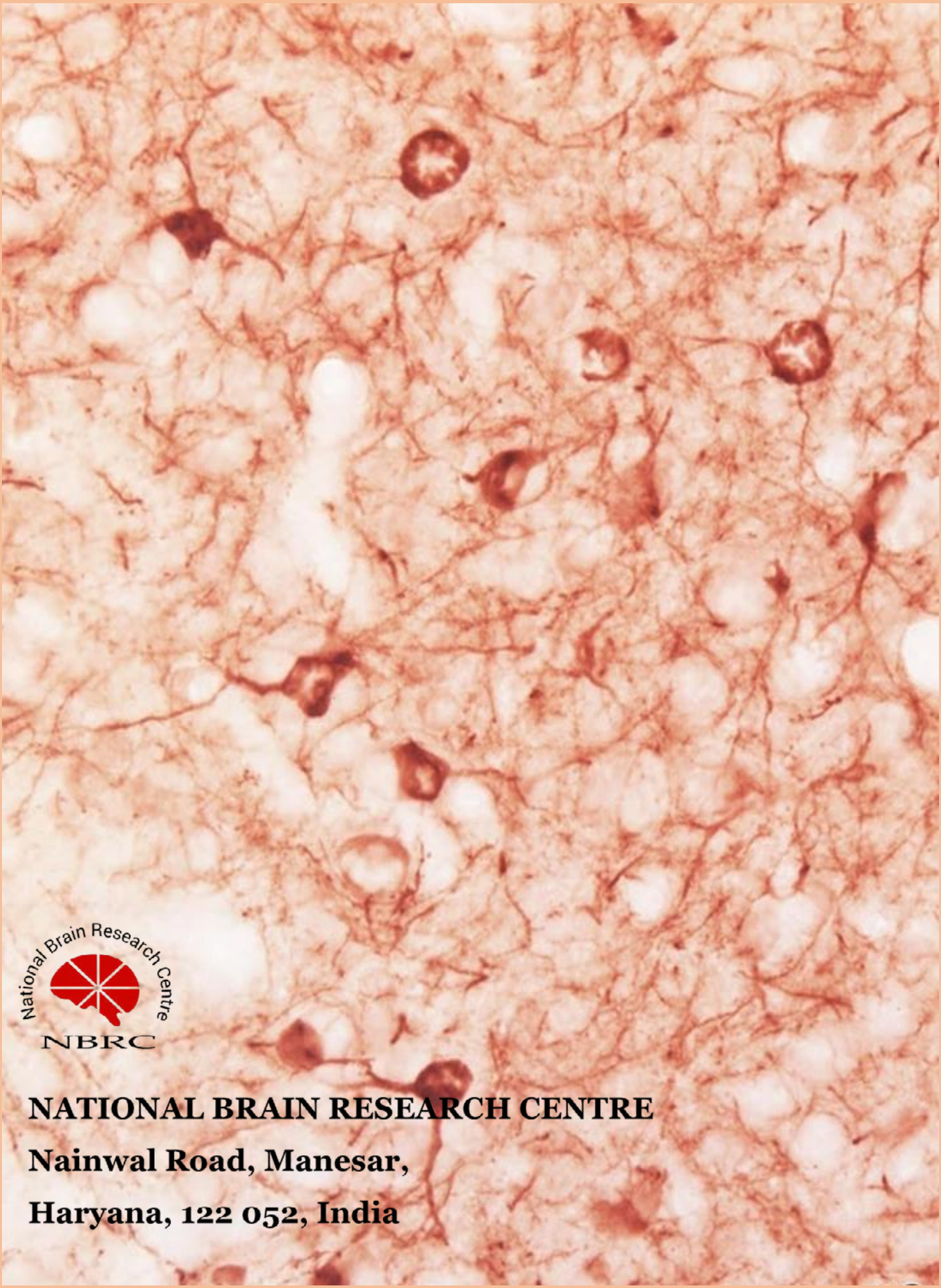
ANKUR AGARWAL
PARTNER
Membership No. 409197
Date:08.10.2021
MEERUT

MAHESHWARI PA & ASSOCIATES
CHARTERED ACCOUNTANTS

Front Cover: A DARPP-32-labelled neuron with elaborate dendritic arbours and well-labelled spines in Field L2b

Back Cover: Secondary auditory areas L1 and L3 are marked by the presence of strongly immunopositive neuronal somata with well labelled proximal dendrites

Both images have been obtained from the publication of Prof. Soumya Iyengar entitled "Singh, U. A., & Iyengar, S. (2019). The expression of DARPP-32 in adult male zebra finches (Taenopygia guttata). Brain Structure and Function, 224(8), 2939-2972."



National Brain Research Centre

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