



Annual Report 2018-19

**National Brain
Research Centre**
Manesar, Haryana



National Brain Research Centre

**Annual Report
2018-19**

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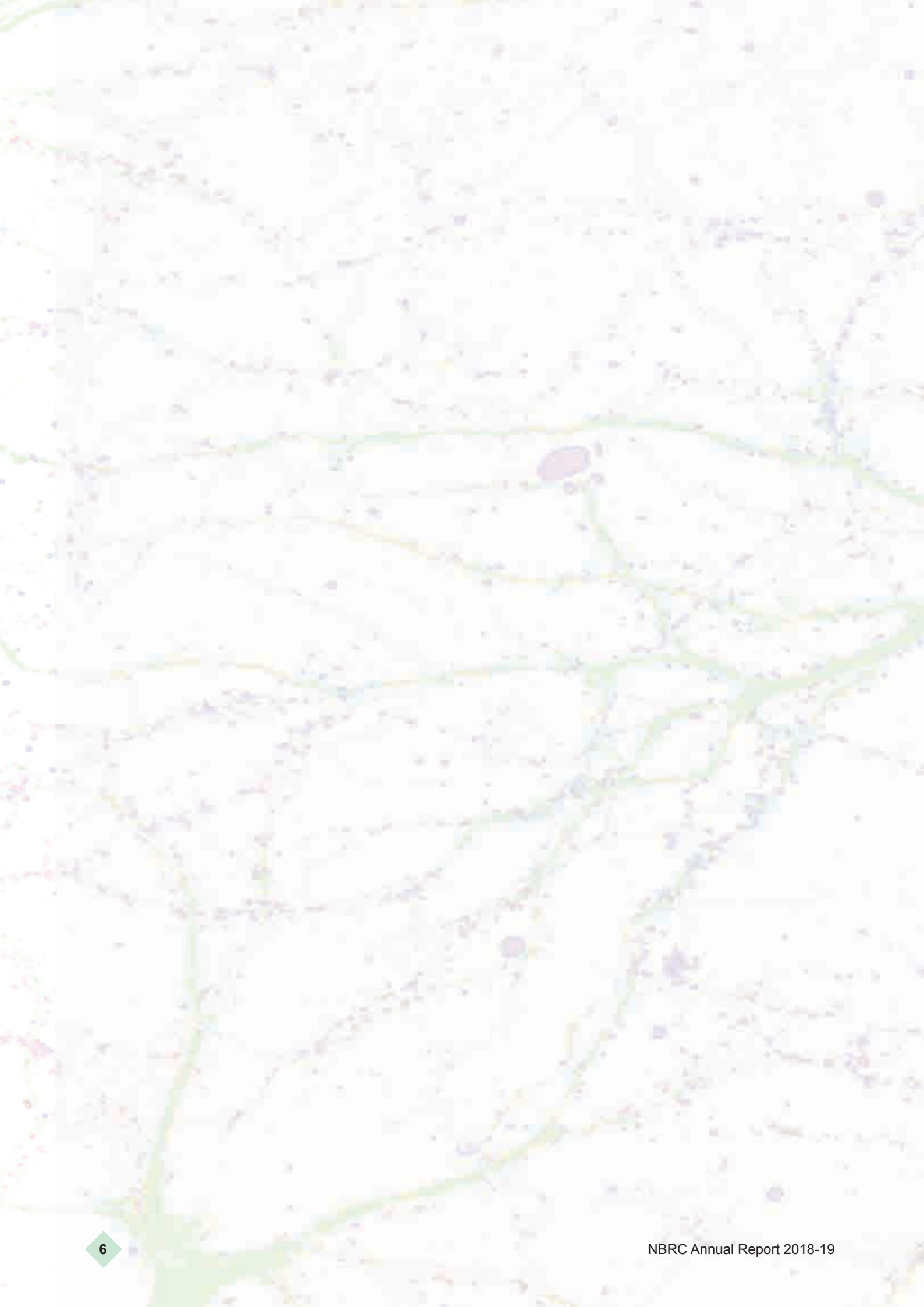
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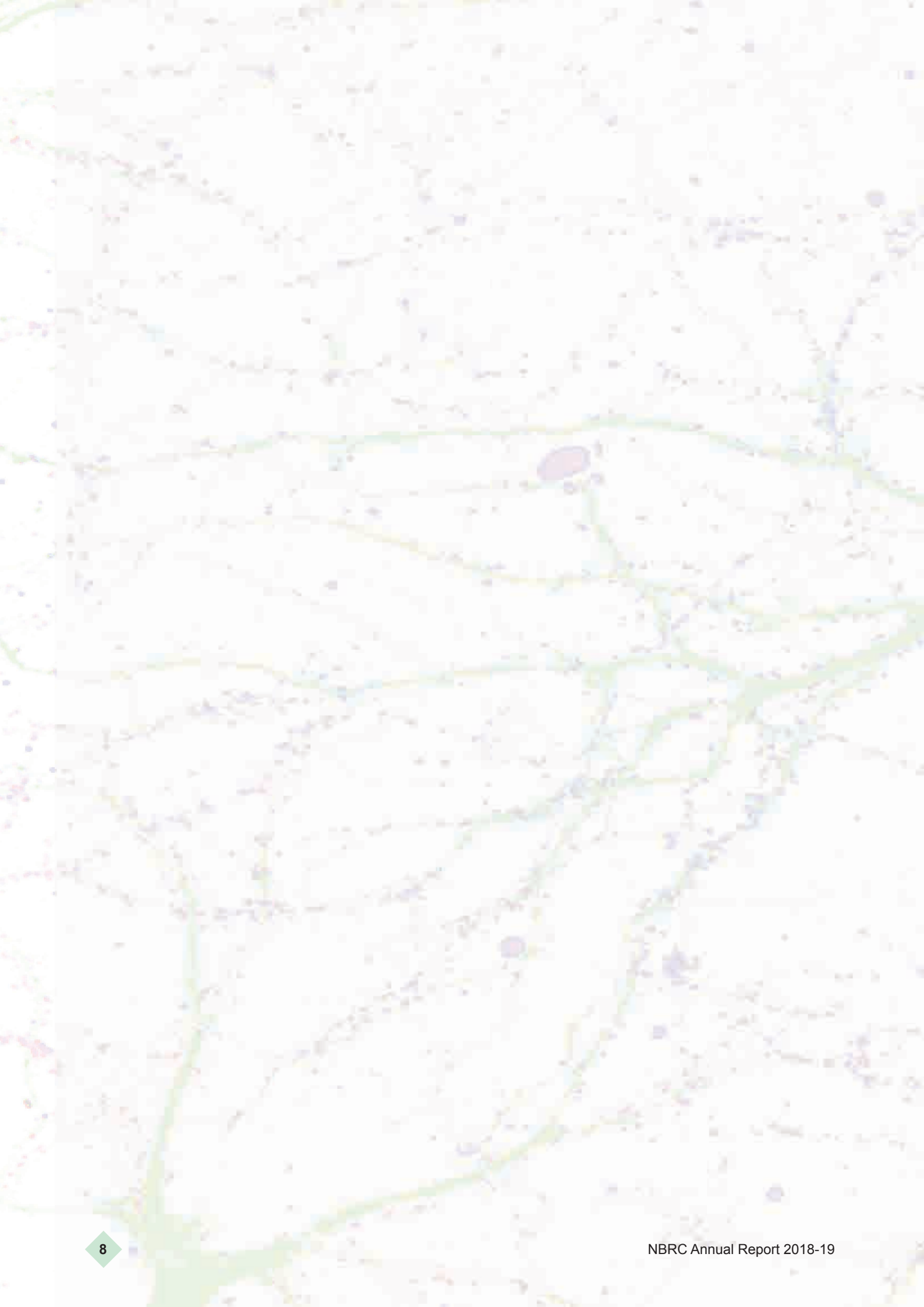
Mandate & Objectives

MANDATE

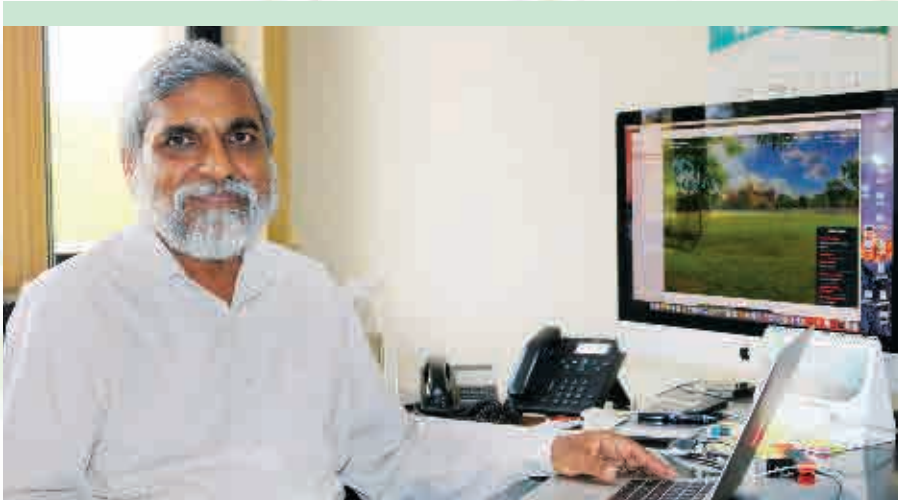
- Pursue basic research to understand brain function in health and disease.
- Generate trained human resources with the capability to carry out inter-disciplinary research in neuroscience.
- Promote neuroscience in India through networking among institutions across the country

OBJECTIVES

- To undertake, aid, promote, guide and coordinate research of high caliber in basic and clinical neuroscience related to diseases and disorders of the nervous system.
- To develop NBRC as the national apex centre for neuroscience research and promote neuroscience research at different centres in the country and to provide consulting services to other institutions, agencies and industries.
- To promote, encourage and augment effective linkages, alliances and affiliations between the Centre and National and International Scientific and Research Institutions, bodies, agencies/laboratories and other organizations working in the field of brain and neurosciences research.
- To establish one or more satellite centers to serve different regions of the country for efficient achievement of the objectives of the Center.
- To collect, assimilate, publish and disseminate data and information on relevant aspects of neuroscience to the scientific community.
- To establish, operate and maintain state-of-the-art facilities as well as databases for carrying research and development activities and make such facilities and databases available to scientists and researchers from all over the country and abroad.
- To provide for instructions and training in such other branches of learning as the Centre may deem fit.
- To provide facilities for the advancement of research and development to facilitate learning and dissemination of knowledge.
- To undertake extramural studies, extension programmes and field outreach activities to contribute to the development of society.
- To promote, develop, collaborate or otherwise assist in providing services of research, training, consulting or guidance related to neurosciences activities comprising biological, psychological, sociological and clinical aspects; and
- To do all such other acts and things as may be necessary or desirable to further the objectives of the Centre.



From the Director's Desk



Prof. Neeraj Jain

National Brain Research Center (NBRC) is a premier Institute dedicated to conducting advanced research in Neuroscience, and excel in providing education and training. The mandate of NBRC is to understand brain function in health and disease. NBRC, as a Deemed to be University, also runs M.Sc. and Ph.D. programs besides other post-graduate and post-doctoral programs to generate trained human resources with the knowledge and skills required to conduct interdisciplinary research in neurosciences. The broader vision is to discover underlying causes, and develop diagnostic markers and cures for various diseases affecting the brain through innovative approaches in basic and translational research.

With immense pleasure, I present this annual report for the year 2018-19, which clearly showcases the commitment of our scientists towards achieving the objectives of NBRC. In order to better reflect the strength of NBRC in multiple subdisciplines of Neuroscience, we now organize NBRC into five divisions – Cellular and Molecular, Systems, Cognitive, Computational, and Translation Research. Using multidisciplinary approaches and various model systems, and supported by intra-institutional and inter-institutional collaborations, NBRC has made significant strides as evidenced by publications, awards and honors. Some of the major achievements during the year are highlighted below.

Encephalitis, caused by Japanese Encephalitis Virus (JEV), and other viruses remains a major public health challenge. The research program developed by Dr Anirban Basu has successfully combined basic and translational research. Their relentless efforts to find a means to tackle this disease has led to identification and classification of hubs in miRNA target gene networks in human neural stem/progenitor cells (NSPCs) following JEV infection. His group has also utilized computational biology approach to analyse networks generated from protein interaction modules and hub proteins, which might be the key host factors for viral infection in NSPCs. On the translational front, his group in association with sister NCR cluster institutes RCB and THSTI has demonstrated that the platelet factor 4 (PF4) promotes rapid replication and propagation of Dengue Virus (DV) and JEV. These findings suggest that the PF4-CXCR3-IFN axis is a potential target for developing methods to treat viral infections including DV and JEV. Moreover, CXCR3 antagonists including

AMG487 can be advantageous for treatment of JEV and DV infections, and possibly for other viruses as well.

Zika virus infection in pregnant mothers has serious neurological consequences for the unborn babies. Dr Pankaj Seth's group at NBRC has identified that the Envelop (E) Protein of the Zika virus is mainly responsible for causing microcephaly, a condition in which size of the head of babies becomes small due to underdevelopment of the brain. They have delineated the cellular and molecular mechanisms by which E-protein of the virus slows down proliferation and differentiation of fetal neural stem cells during brain development, even before the birth. They have identified specific microRNAs that regulate the functions of human neural stem cells thereby affecting their stemness, and overall development of the human brain.

Dr Shiv Kumar Sharma's group is examining molecular changes in the Down syndrome condition. Using hippocampal cell lines derived from trisomic animals, an animal model of Down syndrome, they have found aberrant activation of signaling molecules in the trisomy condition. Dr Ranjit Giri and his group are continuing studies on Prion infection and have identified several factors related to cell death in an animal model of prion disease. They are now identifying which cell types are the main targets of signaling by these death receptors. Dr Ellora Sen continues her efforts to investigate the influence of dysregulated metabolism and aberrant inflammation on the regulation of genes associated with immune evasive responses in Glioblastoma multiforme, a biologically aggressive brain tumor.

Members of the genus Corvidae which include crows, rooks, starlings and jays are increasing being used to study advanced cognitive abilities such as attention, learning and memory, tool use and theory of mind in birds. Dr Soumya Iyengar's group has shown that the expression of tyrosine hydroxylase, the rate-limiting enzyme in the synthesis of catecholamines and DARPP-32 (dopamine

and cAMP-regulated phosphoprotein), which is expressed in dopaminergic neurons can be used to delineate different subdivisions of the caudal midpallidum (NCL, analogous to the prefrontal cortex) in house crows, indigenous to India. These findings lay down a foundation for future studies linking brain structure to complex behaviors and the problem-solving abilities demonstrated by these birds. Dr Anindya Ghosh Roy's group, working on *C. elegans*, another powerful model system for neuroscience research, showed that insulin signaling (IIS) contributes to the age-related decline in axon regeneration. Loss of insulin receptor DAF-2 or any other downstream kinases like AGE-1, PDK-1, AKT-1/2 in the IIS pathway promotes regeneration and functional restoration despite the animal's age. This paves the way to further investigate if similar pathways play a role in regeneration and repair in mammalian systems.

During the year, NBRC took up a challenging program 'Comparative mapping of common mental disorders (CMD) over the lifespan' as its Flagship Programme which aims to understand how information processing networks in the brain are affected in common mental disorders such as anxiety, depression, obsessive-compulsive disorder and post-traumatic stress disorder. The goal is to understand underlying brain mechanisms that differentiate between these disorders, and if these networks are affected in the same manner in different age groups. The data will help establish age-specific diagnostic markers for these disorders. We will expand the programme into a multicentric nationwide programme and carry out genetic investigations in the next phase.

Besides delivering productive outcomes through research, NBRC understands its societal responsibility and stands responsive to the larger needs of society. With an aim to reduce the burden of neurological disorders amongst the general population, NBRC offers neurological outpatient department services at Civil Hospital, Gurgaon to patients from the city as well as neighboring

districts. Patients with epilepsy continue to come to NBRC for sophisticated investigations using the Magnetoencephalography (MEG) facility set up in collaboration with AIIMS, New Delhi. 'DALI' a tool in Indian languages for dyslexia assessment in children developed by Dr Nandini Singh remains in high demand, and is being increasingly adopted countrywide.

NBRC organizes open days for children from various schools, and other citizens to visit the campus and interact with our students and faculty. The visitors are given a tour of the laboratories to introduce them to the excitement of neuroscience research. Given their enthusiastic response, the number of open days was increased to three. This is in addition to the visits of students from schools and colleges organized throughout the year upon request. NBRC also conducts symposia and workshops to exchange knowledge and to facilitate interactions between students and researchers within India and other countries. One of the major events this year was the India-EMBO (European Molecular Biology Organization) symposium

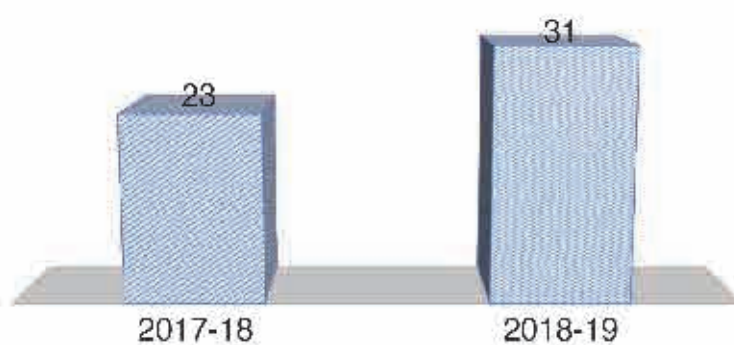
'From synapse to memory: RNA-based regulatory mechanisms'. The thrust of the meeting was memory formation and dysfunction, with a focus on RNA-based mechanisms operating at synapses.

I must express my deepest appreciation for the generous and continuous support of Department of Biotechnology throughout the year that enabled us to succeed in our endeavors. A special thanks to the members of all statutory committees and members of the Indian scientific community for their valuable inputs that continue to contribute immensely to the growth of NBRC.

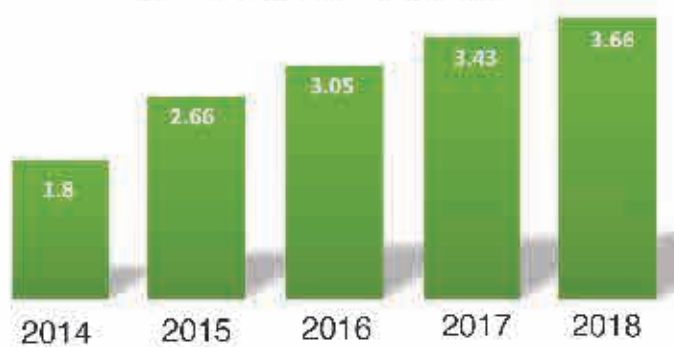
Prof Neeraj Jain
Director

.....at a glance

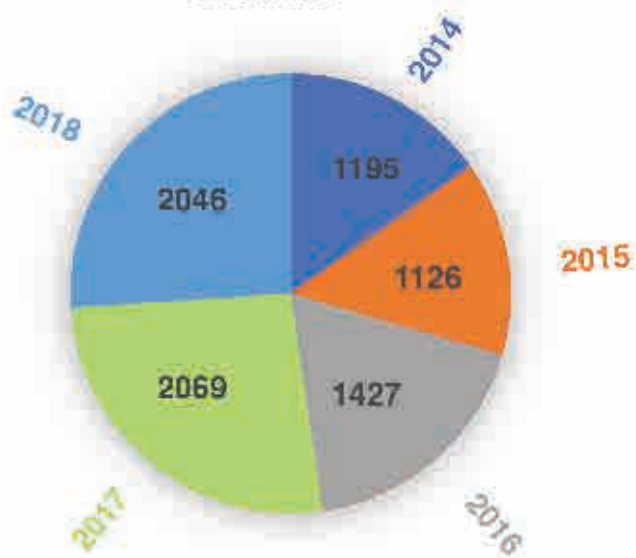
Active Extramural Grants



Publications / Faculty



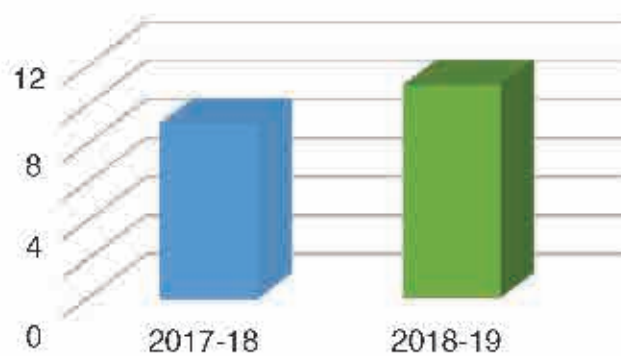
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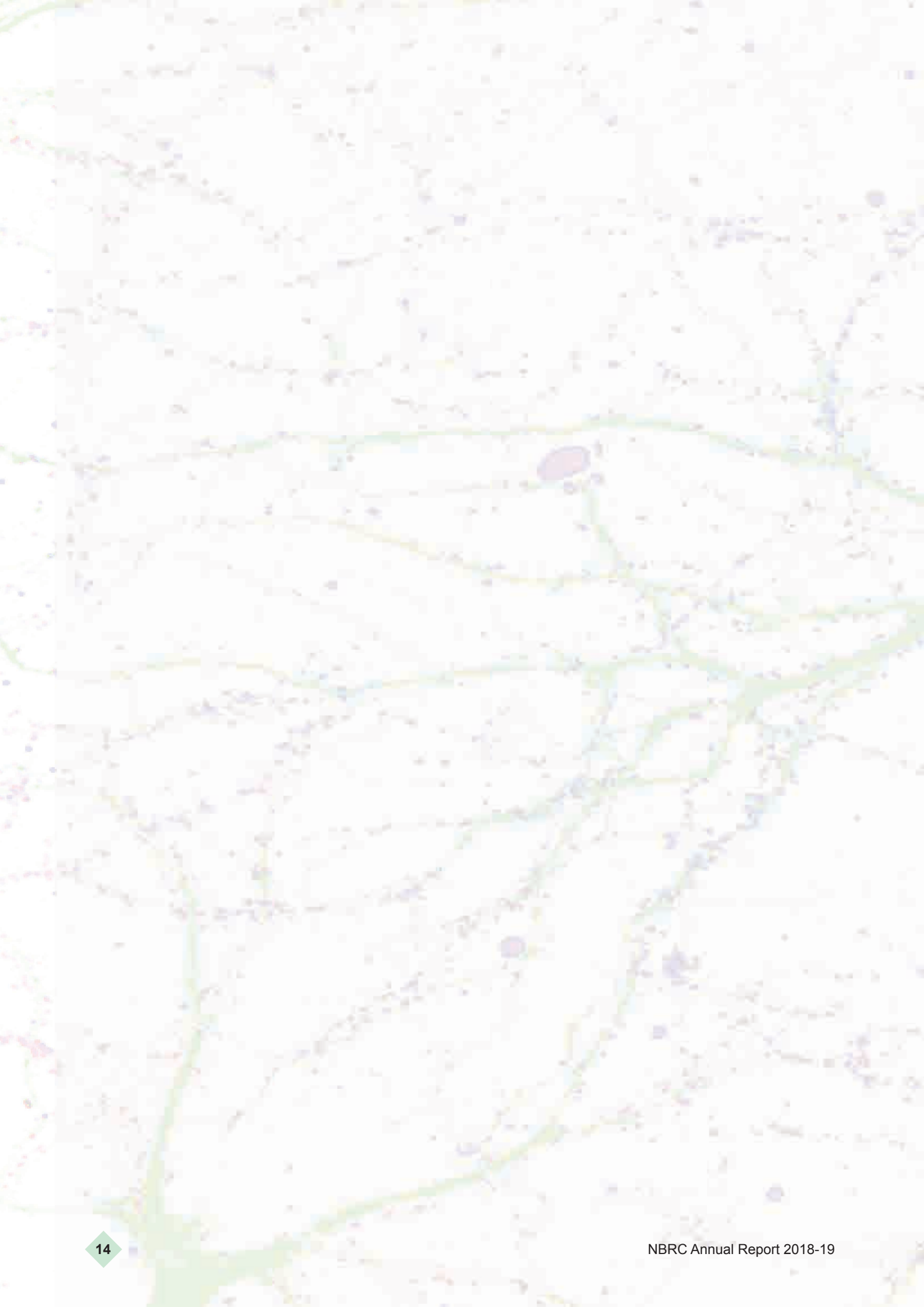


Number of Students Admitted



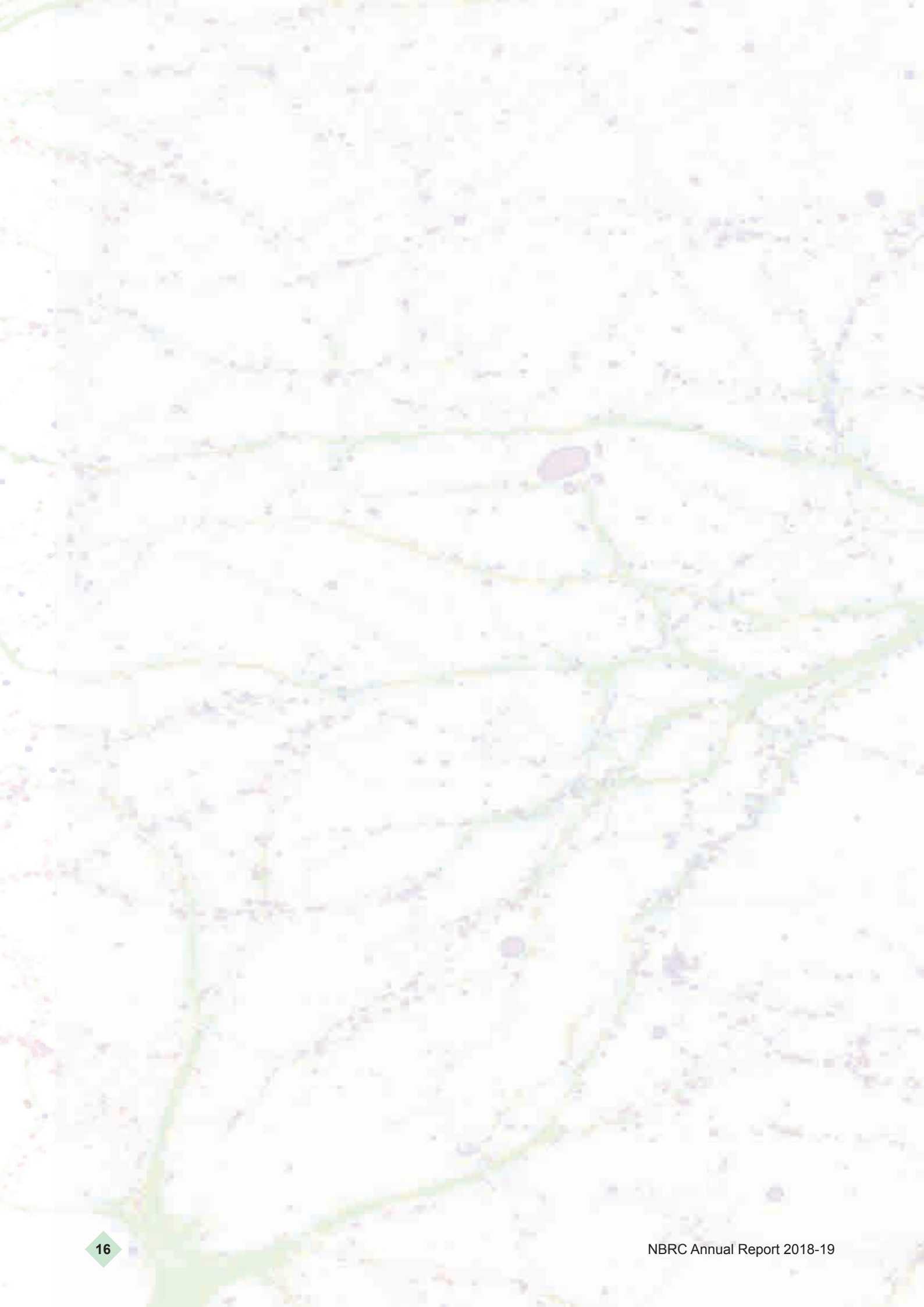
Number of Schools that Visited NBRC







Scientific Reports



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



Anindya Ghosh Roy

Department of Cellular and Molecular Neuroscience,
Systems Neuroscience

Post Doctoral Fellows

Sandeep Kumar
Swagata Dey

PhD Students

Dharmendra Puri
Atrayee Basu
Harjot Kaur
Sibaram Behera
Sunanda Sharma

MSc. Student

Pallavi Singh Rajput

Project Assistants

Keerthana P
Smriti Bharadwaj
Devashish Pande

Technical Assistants

Sumit Mahapatra
Yunis Khan

Background and overall summary

Our lab is interested in understanding how nervous system develops and after injury how it repairs. Towards this goal we use a combination of genetics, molecular biology and imaging in *Caenorhabditis elegans*.

1) Regulation of neuronal polarity:

Microtubule (MT) cytoskeleton is the basis of the polarized structure of neuron. We found that loss of the kinesin-13 family depolymerizing factor KLP-7 stabilizes microtubules and causes multi-polar neuron formation.

We have been able to set up the live imaging system to image microtubule dynamics in vivo. We are using plus end binding protein EBP-2 tagged with GFP to image plus ends of microtubules in touch neurons. We found that microtubules are arranged in a plus-end-out manner in the long anterior process of PLM, whereas MTs are of mixed polarity in the short posterior process. Loss of klp-7 stabilizes the MTs and their polarity becomes plus-end-out in the posterior process. We further found that WNT signalling regulates KLP-7 to establish microtubule polarity in touch neuron (Figure 2).

2) Nerve regeneration study:

To study how nervous system is repaired after neuronal injury, we have established the neurosurgery protocol using femto-second lasers of 2-photon microscope. We showed that axotomy of posterior touch neurons on both sides of a worm leads to a dramatic loss of posterior touch sensation. Using this paradigm, we have shown that initial response to injury is a local raise in axonal Ca^{2+} /cAMP level which activates the a signalling cascade involving dual leucine zipper MAPKKK DLK-1. Several screening using candidate approaches identified the essential, permissive and the inhibitory components of axon regeneration. Rather less is explored whether the axon regeneration of PLM neuron would give rise to functional recovery. We have recently addressed this question and showed that the regrowing axon which could fused to its distal fragment leads to speedy functional recovery [Basu et al 2017]. We also showed that functional recovery declines with age and loss of let-7 miRNA overcomes the age-related decline in functional recovery [Basu et al 2017]. We have seen that the injured axons which

regrows without fusing to its distal end also leads to functional recovery in later time points. Particularly, the axons which reaches the original target area in the ventral nerve cord are likely to give functional recovery. Loss of insulin signaling promotes the functional recovery in these events as well (Figure 4).

We have initiated a project to understand how dendrite responds to mechanical injury. We have found that the DLK-1 MAP kinase pathway that is important for axon regeneration is not required for dendrite regeneration of PVD neuron (Figure 5).

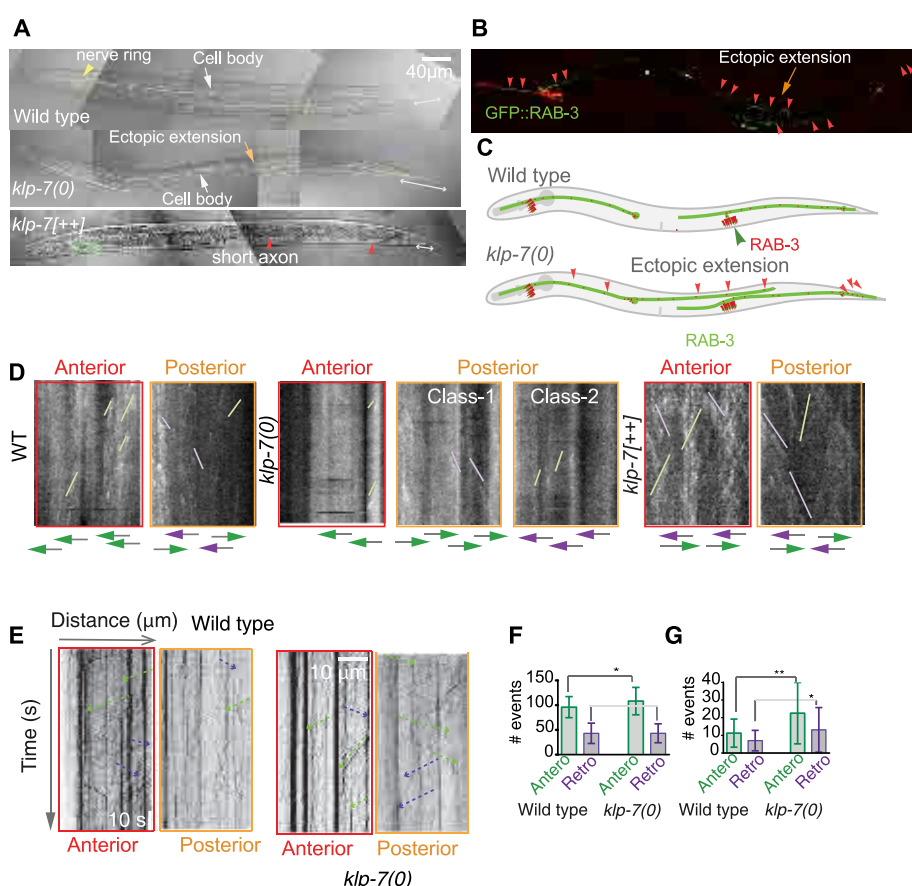
Overall our research on neuronal regeneration has been stimulated by many leads in past few years. We are actively pursuing these discoveries for submission to peer reviewed journals.

Objective 1: WNT signaling establishes microtubule polarity in touch neuron through the regulation of microtubule depolymerizing kinesin KLP-7 in *C. elegans*

Neuronal polarization - a key step in the development of neuronal circuits - is driven by the formation of plus-end-out parallel arrays of

microtubules (MTs) in the axon. In contrast, the orientation of microtubules in dendrites is non-uniform. We used *C. elegans* mechanosensory neuron to address how this differential organization of MTs is achieved. The posterior touch receptor neuron (PLM) has a long anterior process, which connects to its post-synaptic cell, and a short posterior process. In vivo imaging of microtubules and axonal transport with EBP-2::GFP and GFP::RAB-3 reporters respectively, reveals that the MTs in the short process has mixed polarity with mostly static RAB-3 particles. Instead, the long anterior process has parallel bundle of plus-end-out MTs showing consistent bidirectional transport. Loss of Kinesin-13 family microtubule depolymerizing enzyme KLP-7 makes the microtubules unipolar in the posterior process and promotes bidirectional transport; which leads to its overextension (Figure 1A-E). Conversely, overexpression of KLP-7 makes the microtubules oriented randomly even in the anterior process leading to its shortening (Figure 1D). We found that loss of either WNT receptor LIN-17 or its ligand LIN-44 reverses the polarity of MTs in the PLM neuron. In *lin-17* mutant, the MTs in the posterior process are polarized uniformly with plus-end-out organization and that in anterior process

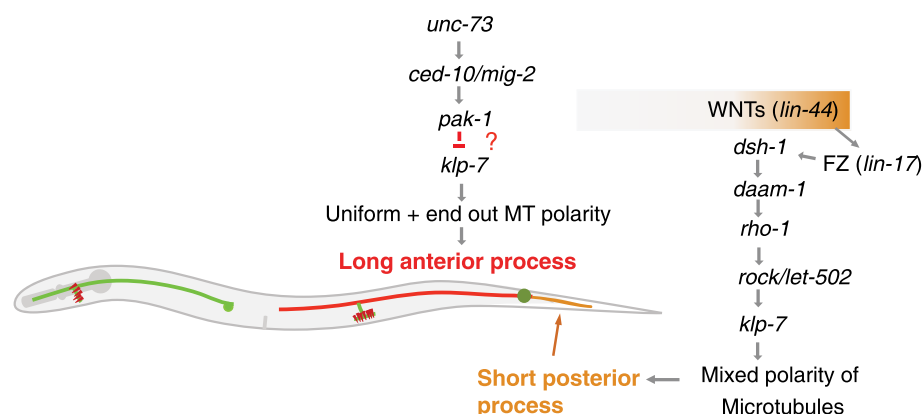
Figure 1: (A-C) Shows GFP labeled PLM and ALM touch neuron. These neurons are polarized during early embryonic development. In *klp-7(0)* mutant very frequently more than one process is extended. (D) In vivo imaging of growing microtubules and determination of their polarity using EBP-2::GFP as reporter in PLM neuron. In *klp-7(0)*, the ectopic processes have unipolar plus end out microtubules. KLP-7 is sufficient to induce mixed polarity in the microtubules in the anterior process of PLM. (E) Representative kymographs showing the movement of GFP::RAB-3 marked vesicles in the anterior and posterior processes of PLM. (F-G) Histogram depict the number of movement events of GFP::RAB-3 vesicles in anterograde (Antero) and retrograde (Retro) directions.



becomes mixed. Removal of *klp-7* in *lin-17* mutant can reset the microtubule polarity and directionality of axonal transport in PLM neuron. We found that in the posterior process, *klp-7* acts downstream of WNT *pcp* components involving *daam-1*/*rho-1*/*rock* to restrain its length. Whereas, in the anterior process *unc-73*/*ced-10*/*pak-1* cascade negatively regulates *klp-7* to promote its growth (Figure 2).

touch neuron in early adult stages. It is not clear among these which are critical for promoting functional regeneration in late adult stages. A comprehensive understanding of how these pathways would be related to the pathways that control organism level aging is also lacking till date.

Figure 2: Model outlining the mechanism by which microtubule depolymerizing kinesin KLP-7 is regulated by WNT signaling for proper organization of microtubules in PLM neuron.



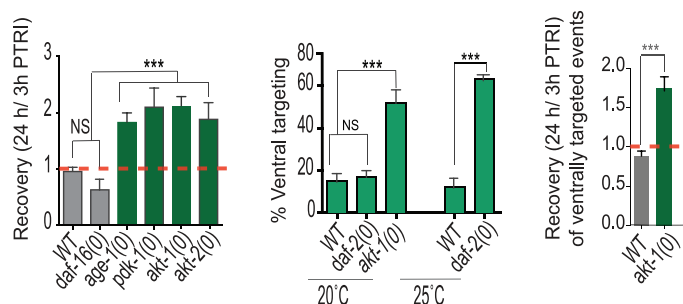
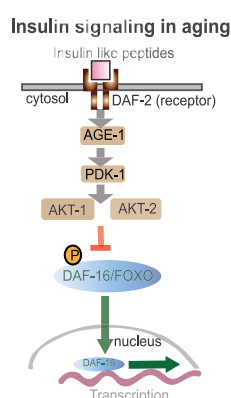
2) Neuronal Regeneration

Objective 2a. Age dependent regulation of functional restoration in touch neuron

Adult nervous system has limited capacity to regenerate after accidental damages. Post injury functional restoration requires proper targeting of the regenerated axon to the postsynaptic partner. In *C. elegans* both mechanosensory and motor neurons show functional recovery following neuronal injury in early stages of life. Functional restoration declines with age in touch neuron. Identification of pathways that inhibit

Towards this goal, we axotomized the PLM (Posterior Touch Receptor Neuron) axons in various mutants and compared the functional regeneration at L4 and Day3 stages using the posterior touch response index (PTRI). We found that although some of the mutants known for enhancing anatomical regrowth rate showed improved functional recovery at L4 stage, only few of them showed functional restoration in A3 stage. Other than the previously known *let-7(0)*, mutants lacking the components of insulin signaling (IIS) and S6 kinase mutant *rsk-1(0)* showed an enhanced recovery at A3 stages.

Figure 3: Functional recovery and guidance parameters in mutants lacking the components of insulin (IIS) signaling. ANOVA, Student's t-Test * $p < 0.01$, ** $p < 0.001$, *** $p < 0.0001$. $n = 33$ & $N = 3$.



regrowth is relevant for informing therapeutic strategies for functional recovery. Several mutants are identified that inhibit regeneration of

Loss of IGF-1 receptor DAF-2 or any other downstream kinases like AGE-1, PDK-1, AKT-1/2 in IIS pathway promotes functional restoration despite

of the animal's age. Upon activation, DAF-2 inhibits translocation of the forkhead transcription factor FOXO/ DAF-16 from cytoplasm to nucleus through the downstream kinases. Enhanced recovery in *daf-2(0)* is completely dependent on *daf-16*. Moreover, we found that in absence of *daf-16*, functional recovery is affected in both L4 and A3 stages. This indicated that DAF-16 is important for regeneration (Figure 4). Other than IIS pathway, we found significant recovery in *rsk-1(0)*. S6 kinase inhibits the downstream kinase AMPK/ AAK-2 and affects DAF-16 activity. We found functional restoration in *rsk-1(0)* depends on *aak-2* but independent of *daf-16*.

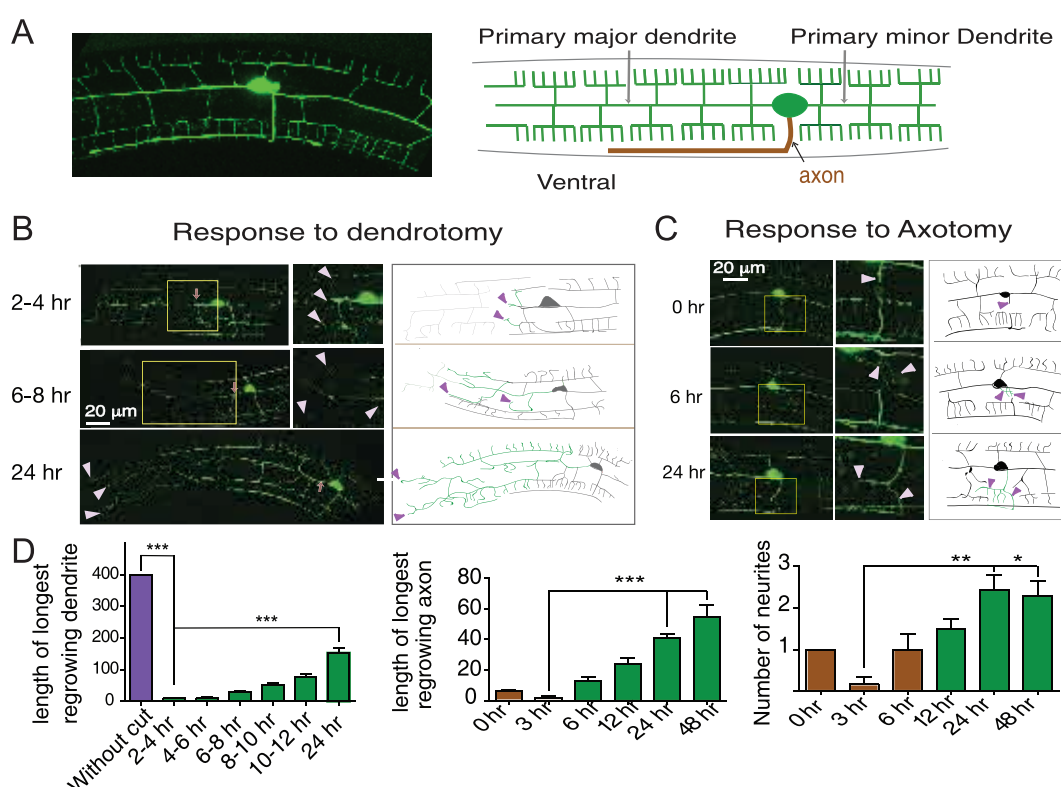
Since both *let-7* miRNA and IIS pathway inhibit axon regeneration, it is important to understand how these two cascades converge and/or diverge. We found that simultaneous loss of *let-7* and *akt-1* increases functional recovery in synergistic manner. Also, loss of *let-7(0)* can completely bypass the requirement of *daf-16* in regeneration. Moreover, functional restoration gets significantly improved in worms lacking both *let-7* and *rsk-1* in comparison to single mutants. Our data reveals that there are independent negative control of axon regeneration and their simultaneous removal might be beneficial in overcoming regeneration barrier after neuronal injury.

Aim2b. Study of Dendrite Regeneration using PVD neuron as Model

Both dendrites and axons are vulnerable to physical insults during the life span of an individual. Several studies recently have focused on understanding the regenerative capacity of an injured axon. The p38 MAP kinase signaling cascade involving Dual Leucine zipper kinase DLK-1 is essential for axon regeneration. The cyclic AMP and mTOR signaling are limiting factors in axon regeneration. But less is known about dendrite regeneration.

To understand the mechanisms of dendrite regeneration, We used PVD neuron in *C. elegans*, which has branched dendrites (Figure 5A). The PVD neurons are responsible for harsh touch sensation. Using femtosecond laser, we severed the dendrites and axon initial segment (AIS) of this neuron. After the primary dendrite was severed near the cell body, we noticed sprouting of new branches from the cut site at 3-hour (Figure 5B). By 24-hours the primary dendrite regrew, following similar trajectory and formed more complex branching patterns unlike the original menorah observed in uninjured PVD (Figure 5A). These branches often lacked self-avoidance phenomenon. We quantified the regeneration pattern in two aspects - length of primary dendrite and number of branches. The primary dendrite

Figure 4:
(A-B) Confocal image and schematics of PVD neuron with branched dendrites. (B) Primary dendrite regrows new processes from the cut dendrite tip. (C) After axotomy, new neurites grow towards ventral nerve cord. (D) Quantification and dendrite and axon regeneration.



regrew in length by $83.5 \pm 64.7 \mu\text{m}$ and $201.1 \pm 56.1 \mu\text{m}$ at 24-hour and 48-hour respectively (Figure 5D). Severing the AIS led to complete retraction of the proximal axon after 3 hours. This was followed by the formation of a new process either from cell body or from sites of primary dendrites adjacent to the cell body. Eventually, these processes were guided to the ventral nerve cord (Figure 5C). This response is reminiscent to the repolarization phenomenon observed in fly and vertebrates after cutting AIS.

The extent of regrowth of the primary dendrite and the number of branches were not affected by the loss of *dlk-1*. This indicated that dendrite regeneration is independent of *dlk-1* as seen in fly. Our future goal is to identify signalling mechanism that is important for the dendrite regrowth. We are prioritizing the kinases specific to dendrite for this direction.

Presentations

1. Anindya Ghosh Roy: "Regulation of functional restoration after neuronal injury by miRNA pathway" in 'India | EMBO Symposium- From synapses to memory: RNA based regulatory mechanisms' 2018 between Oct 15-18, 2018 at NBRC
2. Anindya Ghosh Roy: "Femtosecond laser and live imaging meet the power of genetics in transparent nematode: Intrinsic control of axon and dendrite regeneration" 2nd RCB Bioimaging School, RCB Faridabad, 2019
3. Anindya Ghosh Roy: "C. elegans model and nerve regeneration study" at Dr. B.R. Ambedkar Center for Biomedical Research (ACBR) at University of Delhi during Summer Undergraduate Research Program (SURP) on 8th July 2019

Funding

Wellcome Trust-DBT
NBRC Core

Collaborator

Sandhya Koushika, TIFR, Mumbai, India
Sourav Banerjee, NBRC, India
Smarajit Polley, Bose Institute, Kolkata
Kavita Babu, IISER-Mohali

Student's and Fellows Award

1. Atrayee Basu: TNQ Inspiring Science Award 2019 Finalist for the best published scientific paper in the Life Sciences from India
2. Swagata Dey: Inspire Faculty award 2019
3. Swagata Dey: Early career fellowship of Wellcome Trust-DBT India alliance 2019

Degree Awarded

Pallavi Singh Rajput was awarded MSc degree.

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



Anirban Basu

Department of Cellular and Molecular Neuroscience,
Translational Neuroscience

SERB-National Post-Doctoral Fellowship

Dr Suvodip Mallick

PhD Students

Shalini Swaroop
Abhishek Kumar Verma
Surojit Chakrabarty
Meenakshi Bhaskar
Shivangi Sharma

MSc. Student

Masood Ahmed Wani

DST Inspire Fellow

Sriparna Mukherjee

Project Assistants

Irshad Akbar
Sujata Dev

Technician C

Kanhaiya Lal Kumawat

Technician B

Manish Dogra

The neuro-immune response is generally modulated through multi directional cross talks among immune, endocrine and nervous systems. These cross talks often happened at the physiological level as well as at molecular and cellular level. Spatial and temporal information about inflammatory processes is conveyed to the central nervous system (CNS) where neuro-immune modulation control the extent and intensity of the inflammation. Cytokines and chemokines are molecules that were initially discovered in the immune system as mediators which can communicate between different types of immune cells and occasionally with other cell types. However, it soon became evident that cytokines/chemokines exert profound effects on key functions of the central nervous system, such as food intake, fever, neuroendocrine regulation, long-term potentiation, and behavior. Over the past two decades, tremendous amount of research has revealed various routes by which the nervous system and the immune system communicate with each other. The CNS regulates the immune system via hormonal and neuronal pathways, including the sympathetic and parasympathetic nerves. The immune system signals the CNS through cytokines that act both centrally and peripherally.

The research questions in our laboratory hovers around the concept of neuro-immune interaction and consider its potential clinical application, in an attempt to broaden the awareness of this rapidly evolving as well as emerging area. We are also exploring new avenues that may be helpful in treating brain disorders and the diseases of the nervous system, in particular, the diseases, which has conspicuous inflammatory compartment. We use a model of viral infection to understand this area of research. We also use pro-inflammatory cytokine like IL-1 β to better understand the signaling cascades in an inflammatory milieu or microenvironment.

Micro RNA dysregulation is observed in many viral diseases. RNA viruses modulate host miRNA machinery for their own benefit. Japanese Encephalitis virus (JEV), a neurotropic RNA virus has been reported to manipulate several miRNAs in neuron or microglia. A recent work from our lab has reveals miRNA target gene landscape in human neural stem/progenitor cells following Japanese encephalitis virus infection. We performed a miRNA array of 84 miRNAs in human neuronal

progenitor cell line hNS1 and primary neural precursor cells isolated from aborted fetus. Several fold down-regulation of hsa-miR-9-5p, hsa-miR-22-3p, hsa-miR-124-3p and hsa-miR-132-3p were found in both of the cells. We did a software based search for the target genes of these miRNAs and looked for the biological pathways the genes significantly regulate. Then we sorted out the target genes which are involved in two or more than two pathways. A gene interaction network is built up thereafter. We formed a binary matrix for each gene network on the basis of their interconnection. Different modules or communities were identified in each gene network using MATLAB algorithms. Again, through graph theory approach, we identified the hub genes by analyzing their participation co-efficient in the network. The hub genes were classified either as provincial ($P < 0.4$) or connector hubs ($P > 0.4$). Furthermore, we validated the expression of hub genes in both cell line and primary cells through qRT-PCR post JEV infection and respective miR-mimic transfection. Taken together, our findings integrate the target gene networks of four miRNAs that are affected by JEV infection in NSPCs.

In this study, we address the down-regulation of four miRNAs and we prepared a protein-protein interaction network of miRNA target genes. We have also utilized the computational biology approach to analyze the networks generated from couple of protein interaction modules and hub proteins, which might be the key host factors for viral infection in NSPCs.

In another recent work from our laboratory, we have showed miR-301a regulates inflammatory response to Japanese Encephalitis Virus infection via suppression of NKRF activity. Microglia being the resident macrophage of brain provides neuroprotection following diverse microbial infection. Japanese encephalitis virus (JEV) invades the central nervous system (CNS) resulting in neuro-inflammation, which turns the neuroprotective role of microglia detrimental as characterized by increased microglial activation and neuronal death. Several host factors, including microRNAs (miRNAs), play vital roles in regulating virus-induced inflammation. In the present study, we demonstrate that the expression of miR-301a is increased in JEV-infected microglial cells and human brain. Overexpression of miR-301a augments the JEV-induced inflammatory response, whereas inhibition of miR-301a completely reverses the effects. Mechanistically, NF-

κ B repressing factor (NKRF), functioning as inhibitor of NF- κ B activation is identified as a potential target of miR-301a in JEV infection. Consequently, miR-301a mediated inhibition of NKRF enhances nuclear translocation of NF- κ B, which in turn resulted in amplified inflammatory response. Conversely, NKRF overexpression in miR-301a inhibited condition restores nuclear accumulation of NF- κ B to a basal level. We also observed that JEV infection induces classical activation (M1) of microglia that drive the production of proinflammatory cytokines, while suppressing alternative activation (M2) that could serve to dampen the inflammatory response. Furthermore, in vivo neutralization of miR-301a in mouse brain restores NKRF expression, thereby reducing inflammatory response, microglial activation and neuronal apoptosis. Thus, our study suggests that the JEV-induced expression of miR-301a positively regulates inflammatory response by suppressing NKRF production, which might be targeted to manage viral-induced neuro-inflammation.

Our lab is always interested to translate basic research findings to more applied one, which has clinical relevance. Recently we have found that hypolipidemic drug Atorvastatin exhibits significant anti-viral activity via Heterogeneous nuclear ribonucleoproteins dependent manner. We have used two important neurotropic virus for this study, Japanese Encephalitis Virus (JEV), and Chandipura virus (CHPV). Increased incidences of Central nervous system invasion by the viruses have been reported in case of children and elderly thus culminating into severe neurological damage and long term consequences. Since earlier studies underscore the pleiotropic role of atorvastatin (AT) in neuroprotection against flaviviruses like Hepatitis C and dengue, it was hypothesized that AT might also act as a neuroprotective agent against RNA viruses like CHPV and JEV. AT-mediated antiviral activity was evaluated by assessing survivability of virus-infected mouse (BALB/c) pups treated with the drug. Neuro2A cell line was used as the model for in vitro experiments. Cells subjected to AT treatment were infected by CHPV and JEV followed by evaluation of ER stress-related and apoptosis-related proteins by immunoblotting technique and immunofluorescence microscopy. Interaction of host protein with viral genome was assessed by RNA-Co-IP. AT treatment exhibited significant anti-viral activity against CHPV and JEV infections via hnRNPC-dependent manner. Viral genome-hnRNPC interaction was found to be abrogated upon AT action. AT was also observed to

reduce secretion of proinflammatory cytokines by the neurons in response to viral infection. Moreover, AT treatment was also demonstrated to reduce neuronal death by abrogating virus-induced miR-21

upregulation in hnRNPC-dependent fashion. This study thus suggests probable candidature of AT as antiviral against CHPV and JEV infections.

Figure 1: Network analysis showing miR-9-5p target gene networks, modularity/ community detection in gene networks and the identification of hub genes based on participation coefficients

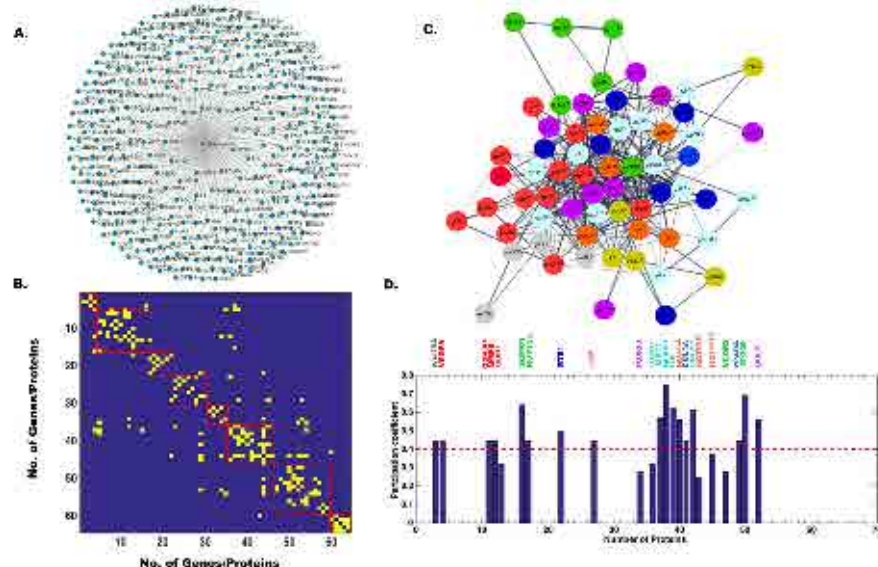
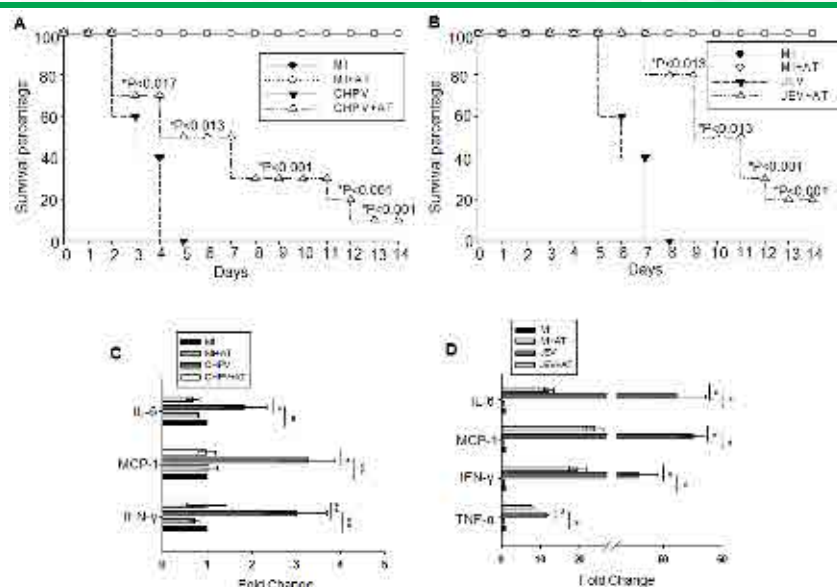


Figure 2: Atorvastatin-treatment increased mice survival and resulted in amelioration of virus-induced neuroinflammation.



Publications

1. A Ojha, A Bhasym^s, S Mukherjee^s, G K Annarapu, T Bhakuni, I Akbar, T Seth, N K. Vikram, S Vrat, Basu A[#], S Bhattacharyya, P Guchhait (2019) Platelet factor 4 promotes rapid replication and propagation of Dengue and

Japanese encephalitis viruses. EBioMedicine 39:332-347. [AB^s, and SM^s equal contribution; all the JE virus related animal study reported in this communication has been done by SM, and IA at NBRC, under the supervision of AB[#]]

2. S Mukherjee, N Sengupta, A Chaudhuri, I Akbar, N Singh, S Chakraborty, A R Suryawanshi, A

- Bhattacharyya, and Basu A (2018) PLVAP and GKN3 Are Two Critical Host Cell Receptors Which Facilitate Japanese Encephalitis Virus Entry Into Neurons. *Scientific Reports* 8(1):11784
 3. A K Verma, T S Waghmare, G R Jachak, S C Philkhana, D S Reddy, and A Basu (2018) Nitrosporeusine ameliorates Chandipura virus Induced inflammatory response in CNS via NF κ B inactivation in microglia. *PLoS Neglected Tropical Diseases* 12(7):e0006648
 4. AK Verma, S Ghosh, and Basu A (2018) Chandipura Virus Induced Neuronal Apoptosis via Calcium Signaling Mediated Oxidative Stress. *Front Microbiol* 6;9:1489. doi: 10.3389/fmicb.2018.01489.
 5. S Swaroop, A Mahadevan, S K Shankar, Y K Adlakha, and Basu A (2018) HSP60 critically regulates endogenous IL-1 β production in activated microglia by stimulating NLRP3 inflammasome pathway. *J Neuroinflammation* 15(1):177. doi: 10.1186/s12974-018-1214-5.
- ### Presentations
1. A Basu (2019) Emerging and Re-emerging Viral Diseases: Host Immunity and Therapeutic Interventions. Sonarpur Mahavidyalaya, Rajpur, South 24 Paraganas, 26th March.
 2. A Basu (2019) Emerging and Re-emerging Viral Diseases: Host Immunity and Therapeutic Interventions. Lady Brabourne College, Kolkata, 25th March. (Special lecture under DBT star college program)
 3. A Basu (2019) Modulation of Interleukin-1 β -HSP60 axis: A potential therapeutic target to ameliorate neuro-inflammatory conditions. 32nd Annual Meeting of SNCI, Dept. of Pharmacology, JSS College of Pharmacy, Mysuru, 14th-16th March.
 4. A Basu (2019) Drug repositioning/repurposing: Promising strategy to develop therapy against viral infections. National Conference on Frontiers in Health Sciences, Institute of Medical Sciences, BHU, Varanasi, 11th-12th March.
 5. A Basu (2019) Neuro-inflammation as a mechanism and therapeutic target. IBRO-APRC associate school, IGNTU, Amarkantak, 3-9th March.
 6. A Basu (2019) Drug repositioning/repurposing: Future drug developmental strategy against viral infections. CCMB, Hyderabad, 27th February.
 7. A Basu (2018) Drug repositioning/repurposing: Promising strategy to develop therapy against viral infections. Centre for Liver Research, School of Digestive and Liver Diseases, IPGME&R, Kolkata, 24th December.
 8. A Basu (2018) Drug repositioning/repurposing: Future drug development strategy against viral infections. C-CAMP, Bangalore Life Science Cluster, 14th December.
 9. A Basu (2018) Modulation of Neural Stem/ Progenitor Cell response following Japanese Encephalitis Virus infection. CIDR-IISC, 13th December.
 10. A Basu (2018) Host MicroRNA: An important modulator of antiviral immunity in Japanese Encephalitis virus infection. International symposium on Infectious Diseases, RCB & Jamia Hamdard, 12-14th, November.
 11. A Basu (2018) Host pathogen interaction in Japanese Encephalitis Virus infection: from bench to bedside. Frontiers in Biotechnology, Dept. of Biotechnology, St Xavier's college, Kolkata, 12th October.
 12. A Basu (2018) Neurotropic Viral Infection Perturbs brain endogenous repairing mechanism. Modern Trends in Microbiology, Dept. of Microbiology, St Xavier's College, Kolkata, 11th October.
 13. A Basu (2018) Interleukin -1 β , a cytokine at the crossroads between infection and inflammation. International conference on Immunology, Dept. of Biotechnology, and School of Bioengineering, SRM University, Chennai, 26th -28th September.
 14. A Basu (2018) Host pathogen interaction in Japanese Encephalitis Virus infection: from bench to bedside, International Society for Neuroimmunology (ISNI)-2018, Workshop 6, CNS infection, Brisbane, Australia, 27th-31st August.
 15. A Basu (2018) Neural stem/progenitor cell response to Japanese encephalitis virus infection. NCR-Biotech cluster meeting, RCB, Faridabad, 29th June.

Scientific Reports

16. A Basu (2018) Host pathogen interaction in Japanese Encephalitis Virus infection: from pathogenesis to therapy. Summer Undergraduate Research Programme- 2018, ACBR, Delhi University, 1st June.

Funding

- 1) microRNAs as a potential therapeutic target in Neuro-tropic Viral infection [Tata Innovation Fellowship from the Department of Biotechnology (BT/HRD/35/01/02/2014)] starting from 01/04/2018, for two years.
- 2) 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.
- 3) 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.
- 4) 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.
- 5) 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.

Collaborators

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Awards

Basanti Devi Amir Chand Prize (ICMR)-2017 (announced in October 2018).

Elected to Fellowship in the American Academy of Microbiology (AAM)-2019

Dr. Y.S. Narayana Rao Oration Award (ICMR)-2018

Student's Award

Surojit Chakrabarty received the best poster award in International symposium on Infectious Diseases, RCB & Jamia Hamdard, 12-14th, November.

Degree Awarded

Shalini Swaroop, (Ph.D.)

Abhishek Verma (M.Sc. & Ph.D.)

Neuro-cognitive network mechanisms using multimodal neuroimaging



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Shrey Dutta
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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 cognitive impairments in epilepsy 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, 2018 - March, 2019. 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: Quantitative evaluation in estimating sources underlying brain oscillations using current source density methods and beamformer approaches

Brain oscillations from electro-encephalogram (EEG) and magneto-encephalogram (MEG) shed light on neurophysiological mechanisms of human behavior. However, to extract information on cortical processing, researchers have to rely on source localization methods that can be very broadly classified into current density estimates such as exact low resolution electromagnetic tomography (eLORETA), minimum norm estimates (MNE) and beamformers such as Dynamic Imaging of Coherent Sources (DICS) and Linearly Constrained Minimum Variance (LCMV). These algorithms produce a distributed map of brain activity underlying sustained and transient responses during neuroimaging studies of behavior. On the other hand, there are very few comparative analyses that evaluates the "ground truth detection" capabilities of these methods. This project evaluated the reliability in estimation of sources of spectral event generators in the cortex using a two-pronged approach. First, simulated EEG data with point dipoles and distributed dipoles are used to validate the accuracy and sensitivity of each one of these methods of source localization

(Fig 1). The abilities of the techniques were tested by comparing the localization error, focal width, false positive ratios while detecting already known location of neural activity generators under varying signal to noise ratios (**Fig 2**). Second, empirical EEG data during auditory steady state responses (ASSR) in human participants were used to compare the distributed nature of source localization (**Fig 3**). All methods were successful in recovery of point

sources in favorable signal to noise scenarios and could achieve high hit rates if false positives are ignored. Interestingly, focal activation map is generated by LCMV and DICS when compared to eLORETA while control of false positives is much superior in eLORETA. Subsequently drawbacks and strengths of each method are highlighted with a detailed discussion on how to choose a technique based on empirical requirements.

Figure 1: **a)** eLORETA (red) vs DICS source localization (green) using frequency-lock analyses on distributed dipolar source generated signals. Overlapping regions are depicted in yellow and locations used for simulated dipole placements are depicted in blue. eLORETA was applied to the simulated sinusoidal signal embedded in resting state EEG, where the simulated signals generated from each hemisphere had 0 phase lag. For DICS, the simulated signal from the right hemisphere had a phase lag of μ with respect to the signal from the left hemisphere. The ratio of power spectrum at 40 Hz between the sinusoidal embedded in EEG and resting state EEG was chosen similar to the ratio found in our empirical results. **b)** eLORETA (red), LCMV (green) and MNE (purple) generated sources using time-lock analysis. No overlapping regions were found. The results indicate 0 time lag scenario between the Gaussian pulses from each hemisphere. Physiologically realistic SNR 25 dB for simulated dipolar sources was chosen for this illustration.

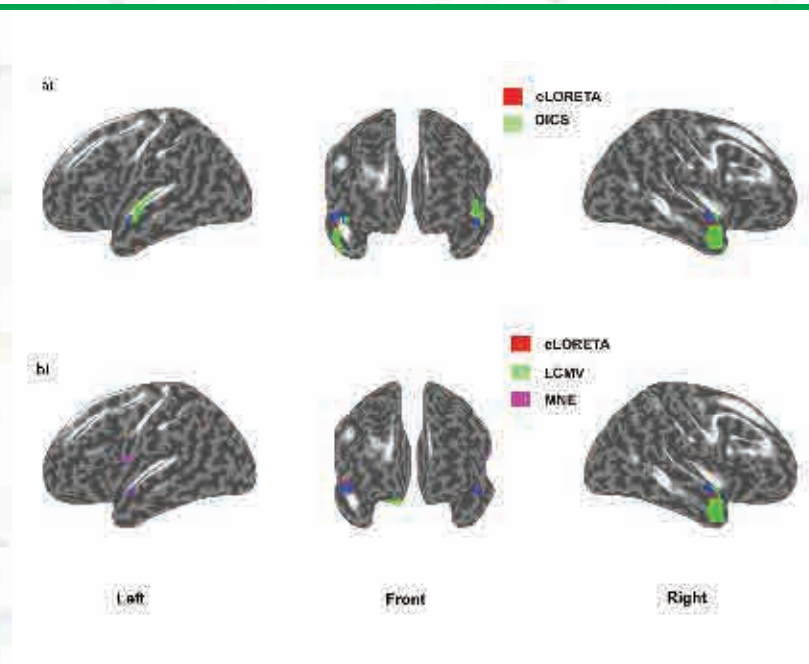


Figure 2: Localization error, Focal width, False positive and Hit rate percentage for (a) Single dipole (b) Two-point dipole and (c) Distributed dipole conditions measured for eLORETA (red) and DICS (blue). Source localization was done for all power ratios (x axis) across different simulated phase lags of 0, $\mu/2$, μ and $3\mu/2$.

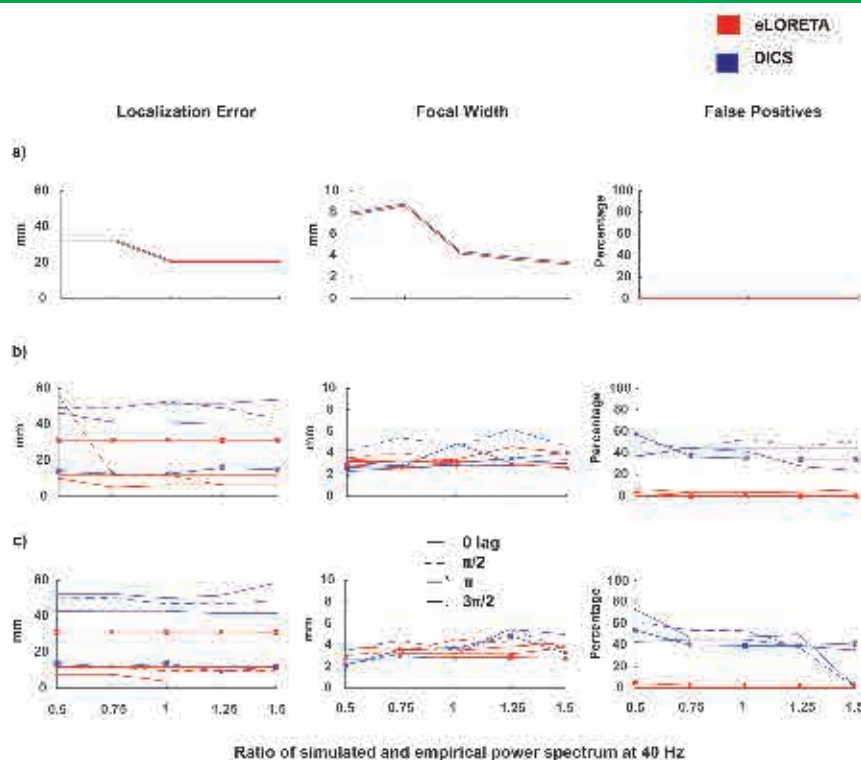
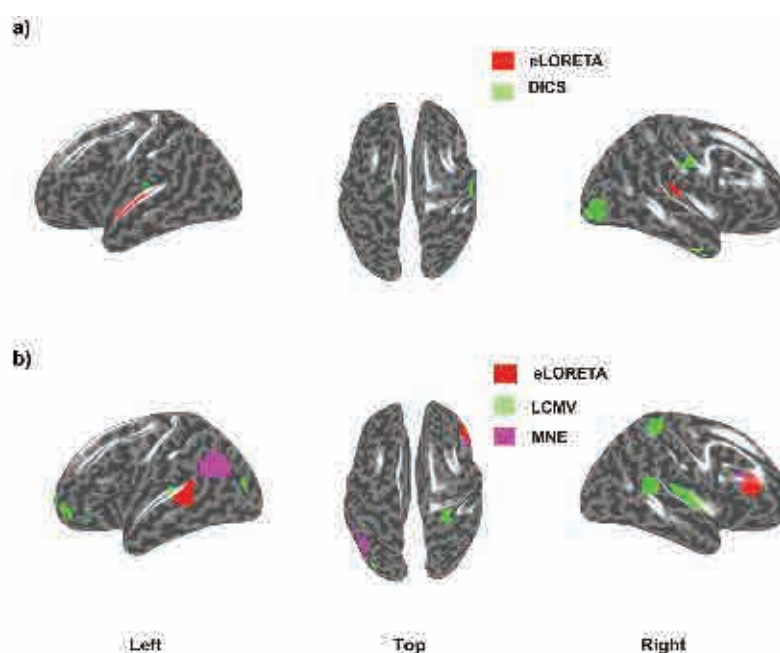


Figure 3: Left, top and right view of significantly active cortical sources underlying for a) 40 Hz auditory steady state response (ASSR) b) N100 component of the event related potential (ERP). The red colored regions show estimated sources from eLORETA while green regions show estimated sources from a) DICS and b) LCMV analysis. The pink regions in b) show MNE source localization results.

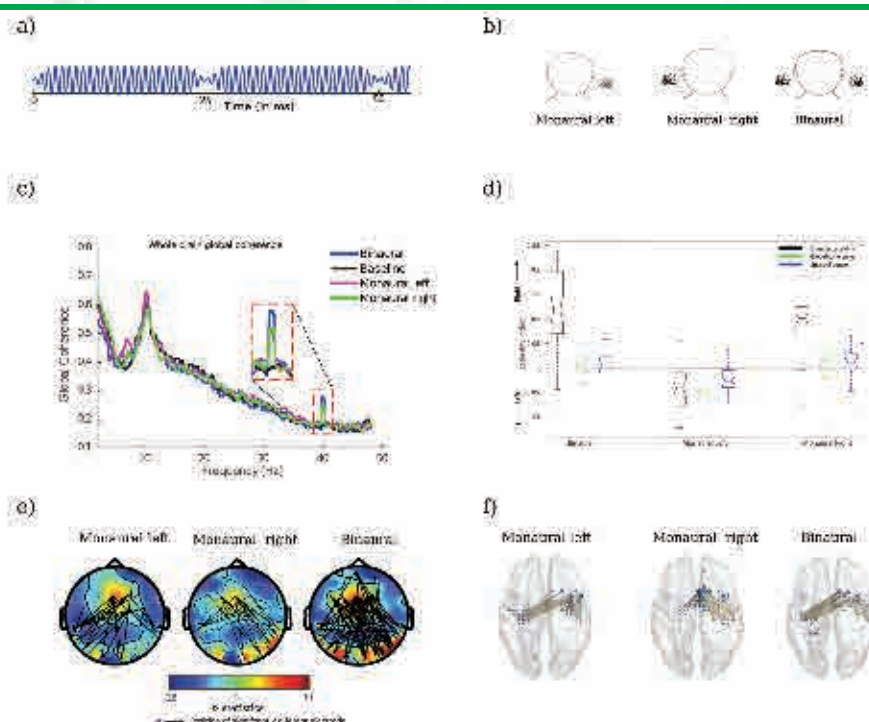


Project 2: Asymmetric organization of functional brain networks underlying auditory steady state responses (ASSR) to rhythmic tonal stimulation.

Several electroencephalogram (EEG) studies have shown that stimulation by periodic auditory stimuli evokes a periodic brain response having the corresponding frequency (**Fig 4a**), with repetition at 40 Hz eliciting maximum response, popularly known as 40 Hz auditory steady-state response or

40 Hz entrainment. ASSRs has been proven as a potential marker of auditory processing. Emerging evidence on the human auditory system suggests that auditory functions are lateralized. Numerous evidence substantiates the fact that neural functions involved in the processing of human speech are biased towards the left hemisphere, however, processing of tonal stimuli are more pronounced in the right auditory cortex. In the present study, we investigate the lateralization of the large-scale functional network that supports information

Figure 4: (a) Auditory stimuli and (b) different stimulation condition. (c) group level global coherence spectra (d) asymmetry in the hemispheric specialization with various network measures (e) sub-network analysis at sensor-level using pairwise imaginary part of coherence and (f) source level coherence during monaural left, monaural right and binaural condition.



processing of periodic auditory stimuli. We collect EEG data from 21 healthy volunteers during rest, binaural and both monaural auditory stimulation (**Fig 4b**). Pure tones at 1 kHz frequency were presented at 40 cycles a second during the periodic stimulation period (**Fig 4a**). We performed multi-taper power spectral analysis to capture 40 Hz spectral content in different stimulation conditions. We observed the enhancement of spectral power at 40 Hz in distributed scalp sensor locations. Subsequently, using global coherence to identify the presence of a large-scale brain network we find task-specific enhancement of global coherence specifically at 40 Hz indicating the entrainment of a large-scale neuronal network in monaural and binaural conditions (**Fig 4c-e**). Subsequently, measurement of pairwise imaginary coherence to detect sub-networks were carried out (**Fig 4e**). Bilateral long-range interactions involving centro-frontal and temporal sensors and parietooccipital sensors were significant. To identify the causal influence in all significantly interacting channel pair we employed Granger causality (GC) analysis. In both monaural and binaural conditions, GC revealed the influence of right temporal region to the frontal areas. Hemispheric analysis revealed the ipsilateral dominance in the processing of monaural stimuli (**Fig 4d**). We showed that rather than a local response, integration at large scale level contributed most to observed asymmetry.

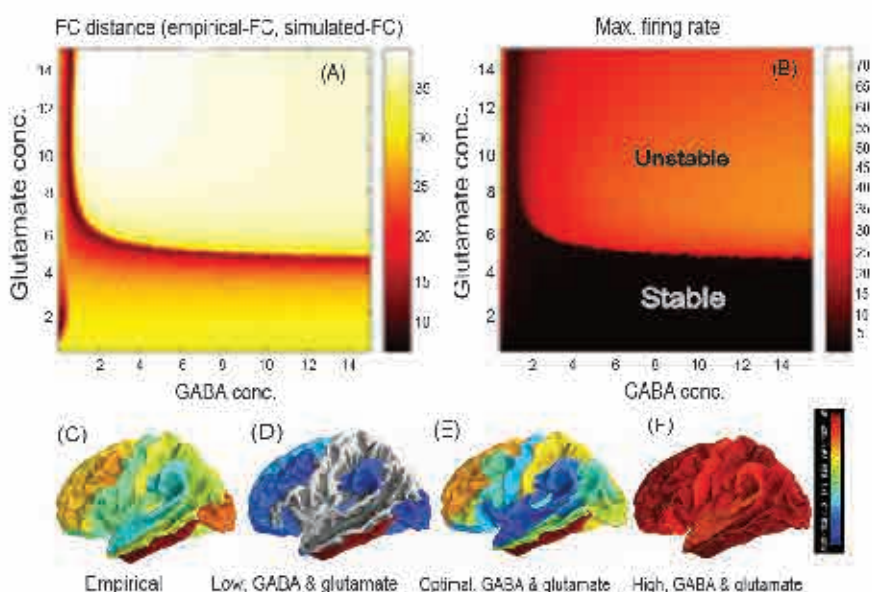
Finally, to identify stimuli specific network present at the source level we performed whole-brain connectivity analysis. From the source network

analysis, we found that irrespective of the stimulation condition number of nodes in the right hemisphere was always higher than that in the left hemisphere, however, the number of nodes in the left hemisphere was greater during monaural left conditions than binaural and monaural right condition. We found during every type of stimulation condition, prominent nodes are temporal gyri and frontal gyri, which was in line with the sensor level results (**Fig 4f**). Moreover, a different set of interactions are present during different auditory conditions. In conclusion, connectivity analysis at sensor and source level revealed right hemispheric dominance during 40 Hz ASSRs that may be the result of processing temporal regularities associated with pitch processing of incoming sound.

Project 3: Multiscale dynamic mean field (MDF) framework to relate resting state brain dynamics with local cortical excitatory-inhibitory neurotransmitter homeostasis in health and disease.

The spontaneous neuronal activity of human brain shows highly structured spatiotemporal pattern, designated as resting state network. Previous neuro-computational studies have established the connection of spontaneous resting state activity with neuronal ensembles (using dynamic mean field approach) and showed the impact of local inhibitory-excitatory balance on this patterned spontaneous resting state activity of brain. But, the role of neurotransmitter dynamics on whole-brain network organization remains unexplored. Empirically,

Figure 5: FC distance and neuronal firing as a function of GABA and glutamate concentrations. (A) FC Distance: Black color region represents highly similar (low FC distance) and yellowish region depicts distant similarity (high FC distance) between empirical rs-FC and model-generated FC. GABA and glutamate concentration ranging from 0.1 to 15 mmol are which captures the biologically realistic parameter regime is used for simulations. (B) Neuronal firing rate: We consider high neuronal firing leads to instability for various neurotransmitter concentration scenarios. Stable region (black color) denotes low rate of neuronal firing (<8 Hz), and unstable region (red-yellow) region represents firing rate more than 8 Hz. Seed-based correlation maps with respect to precuneus plotted for different scenarios (C) empirical FC, and model-generated FCs in (D) low, (E) optimal, or (F) high GABA and glutamate concentrations.



this is beyond the scope of current neuroimaging techniques to monitor the neurotransmitter changes and capture BOLD dynamics simultaneously. We argue that whole-brain computational models offer an attractive platform to test out the possible neural mechanisms that operate at multiple scales of physiological organization namely, brain metabolism and changes in synaptic concentrations of GABA (gamma-aminobutyric acid)/ glutamate. We introduce the multiscale dynamic mean field (MDMF) framework that captures the synaptic gating dynamics as a function of neurotransmitter concentrations using which it is possible to demonstrate how local homeostasis of excitatory-inhibitory neurotransmitters namely GABA/ glutamate ratio could modulate the whole-brain resting state network dynamics of brain. Simulations were carried out across glutamate (0.1 to 15 mmol) and GABA (0.1 to 15 mmol) concentration regimes that span the parameter space reported

from healthy and diseased brains. We have found for optimal configurations of glutamate concentration ranging from 5.6 to 15 mmol and GABA concentration from 0.9 to 15 mmol MDMF predicted functional connectivity matrix show closest match with empirical functional connectivity matrix from resting brain recordings over a cohort of 49 volunteers (Fig 6). Graph theoretical measures of segregation (modularity, clustering coefficient and local efficiency) and integration (global efficiency and characteristic path length) in network information processing reported earlier in healthy and diseased brains were related to optimal GABA and glutamate concentrations and pathological values reported in epilepsy and schizophrenia respectively. In conclusion, the MDMF framework could relate the various scales of observations from neurotransmitter concentrations to dynamics of synaptic gating to whole-brain resting state network topology in healthy and in disorder state.

Publications

Banerjee, A., Kikuchi, Y., Mishkin, M., Rauschecker, J. P., Horwitz, B. (2018) Chronometry on Spike-LFP Responses Reveals the Functional Neural Circuitry of Early Auditory Cortex Underlying Sound Processing and Discrimination, *eNeuro*, 5(3): ENEURO.0420-17.2018.

Presentations

1. Arpan Banerjee: Oct 2018: at Montreal Neurological Institute, Montreal Canada in Auditory processing and plasticity meeting organized by IDRC project from Israel-Canada research consortium (Invited talk)
2. Arpan Banerjee: Oct 2018: at Penn State Medical Centre, Hershey Park, PA, USA, Departmental seminar in radiology (Invited talk)
3. Arpan Banerjee: July 2018: at Indian Institute of Science, Bangalore, International Conference on Signal processing and communications (IEEE). (Invited tutorial)

Funding

- NBRC Core
- Ramalingaswami Fellowship, Department of Biotechnology
- National Postdoctoral fellowship, Department of Science and Technology
- 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.

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Large-scale brain Network dynamics in health and disease using multimodal Neuroimaging



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Our Lab is interested in understanding multi scale brain network dynamics using various imaging (fMRI, EEG, MEG) modalities to understand what gives rise to perception, different forms attention, learning and higher order cognition and their alteration with atypical development and healthy, pathological aging. One of the experimental paradigm that we are interested in known as the resting state - when the wakeful brain neither accomplishes explicit tasks nor is exposed to external stimuli – the brain is organized in distinct networks of correlated activity. Fluctuations of these resting-state networks (RSN) influence human behavior and cognition as has recently been demonstrated. The RSNs, including the default mode network (DMN), Saliency Network (SN), and Central Executive Network (CEN) are highly reproducible across different healthy individuals and are considered to co-ordinate endogenous large-scale interactions such as self-referential cognitive processes, maintenance of memory and cognitive control during attention, perception. Moreover, across wide range of experimental works related to working memory task, visual learning task, tactile stimulation task has found that the activity in this set of brain regions are high when the mind is not engaged in specific behavioral tasks and low during focused attention on the external sensory processing engaging goal driven CEN. SN appear to work as mediator between DMN and CEN by switching their role during on-and-off task. Hence in this project, we aim to investigate interaction between triad of networks and their functional role in health and disease, whose direct impact would span several disciplines, including cognitive neuroscience, pharmacological neuroimaging, clinical neuroscience, and theoretical neuroscience. We investigate three outstanding open problems in cognitive neuroscience (a) Mechanisms that underlies default mode activation, and suppression, (b) Interactions between default mode activations and suppressions with SN, CEN brain networks observed during resting state, (c) Information processing capacity or complexity in the default mode and cognitive functional brain networks. To this end, we use high throughput diffusion tensor imaging, functional MRI, and MEG data to understand spatiotemporal brain dynamics between neurocognitive networks and their relationship with underlying anatomy.

Causality and switching between Salience, Default and Executive brain networks with aging in the presence of Thalamic drive

Introduction

The human brain undergoes both structural and functional changes across the lifespan. It is important to know the dynamics of these changes. Furthermore, functional connections within resting-state networks weaken while connections between resting-state networks strengthen with aging. A recent study by Tsvetanov et al. (2016) shows that effective connectivity within and between large scale functional networks changes over the healthy lifespan. However, the contribution of thalamus in these age-related changes is not adequately explored. In particular, very few studies have investigated how thalamocortical structural and functional connectivity changes with age and how such changes are associated with changes in cognitive functions (Goldstone et al. 2017). Using effective connectivity measures on resting state fMRI data, we examine the age-related changes in cortico-cortical and thalamocortical causal interactions within and between resting state networks.

Methods

We use resting state fMRI, structural MRI and diffusion tensor imaging (DTI) to scan participants from young to elderly in the age group of 18–82 years and identify their Salience Network (SN), Central Executive Network (CEN) and Default Mode Network (DMN). The couplings between the nodes of these spatially extended networks are derived from empirical DTI (Diffusion tensor imaging) data collected from 25 young and 25 old subjects. Thereafter, we calculate the Functional Connectivity (FC) matrix based on BOLD signals from identified Regions of Interests (ROIs) based on Desikan-Killiany (DK) parcellation atlas which are labelled as 68 cortical regions and 2 subcortical regions (left and Right Thalamic regions). Subsequently, various centrality measures are applied on average FC and SC matrix for finding out the most central nodes (hubs). Community structure of these matrices are identified using the algorithm by Blondel et al. (2008). The three of core cognitive networks DMN, SN, CEN networks are identified by spatially matching of hub regions with the important RSNs in the literature. Multivariate GCA is performed to

test for causality index between ROIs with and without the thalamus. Pairwise granger causality indexes are calculated for within network and between network causality analysis. We have also calculated the distribution of weighted net granger causal outflow with the hundred bootstrap samples of Granger Causality matrix. We have performed nonparametric Mann-Whitney U test to test whether there is any significance difference between net causal outflow for two different age groups with and without the thalamus.

Results

In the context of effective connectivity, our analysis revealed causal connections and switching between DMN, SN, CEN alters with age followed a specific pattern. Within network causal connections become weaker, but between network causal connections are getting stronger with aging. Between-network causal influences were stronger for the old individuals compared to younger. Salience network acts as a causal outflow hub for both the age groups. Strongest causal connection was found from SN to DMN for both the young and old groups. Causal connections were found for every pair of nodes in both directions for both the age groups, but connections from SN to CEN, CEN to DMN, DMN to CEN were significantly greater in elderly compared to younger participants (FDR corrected, $p < 0.05$). Between network integrations are stronger for the elderly. Thalamic influence on the SN was the least, whereas, the influence altered causality and net causal outflow significantly in CEN, DMN. For the young cohorts, among the nodes in DMN, RMFC had a significantly greater causal flow than other nodes, while in the elderly age groups right Inferior Parietal Cortex (rIPC) acts as a causal hub. Causal outflows were significantly different ($p < 0.01$) in every nodes for both age groups. Youngs were exhibiting higher causal flow in right Posterior Cingulate Cortex (rPCC), Left Middle Frontal Cortex (IMFC), Right MFC. In other three nodes, namely IPCC, IIPC, rIPC causal flows were significantly higher for elderly individuals. For both groups among the nodes in SN, left insula acted as a causal hub mediating flexible switching between DMN, CEN brain networks. The relative strength of efferent versus afferent connections places the SN at the apex of hierarchy between three large scale networks, suggesting that the SN modulates anticorrelated networks with descending hierarchical connections.

Discussion

The underlying causes and mechanisms for anticorrelated/suppression in DMN in younger individuals and higher activity in DMN networks are not fully explainable by current neurocognitive theories of the aging brain and remain a fundamental open question. To the best of our knowledge, no previous studies have addressed systematically the role of thalamus and other subcortical regions in shaping between network causal connectivity resulting in increased integration between intrinsic resting brain networks with aging. Hence, what emerge from our causality analysis that thalamus indeed has an important causal role as a common driver to influence and shape up within and between large scale network connectivity between three prominent hub networks DMN, SN, and CEN. Furthermore, this influence also significantly alters along descending cortical hierarchy among hub networks with aging. Our findings suggest that effective connectivity strengthens the hypothesis that balancing between within and between network connectivity is an important neural marker to maintain the functionality of brain with aging.

Spatiotemporal reorganization of brain rhythms on slow and fast time scales with healthy aging

Introduction

Cognitive processes are mediated by communication among multiple brain areas using frequency specific interactions. These frequency specific interactions are fundamental for attention, perception, learning and memory as highlighted by extant literature. Individually, brain areas exhibit oscillatory neuro-electromagnetic signals emerging from a cascade of complex physiological processes. Efficient cognition demands seamless interaction between these different oscillatory modules of the brain. Recent models of large-scale spontaneous brain activity have underscored the importance of brain-wide coherence in bringing about facilitative patterns of activity. Additionally, the allied but independent concept of global metastability casts the understanding of the transient nature of stability of brain connectivity patterns within a dynamic framework. Since healthy ageing is accompanied by changes in large-scale spontaneous electromagnetic oscillations, we asked if the variability and complexity of communication between brain areas with aging could be quantified with global

coherence and metastability. Finding such patterns in resting state MEG would naturally pave the way for tracking task relevant changes in metastability and coherence dynamics that could crucially determine cognitive flexibility and performance in elderly individuals. While previously reported observations of a reduction in peak alpha frequency and increased beta power in older adults are reflective of changes at individual sensors (during rest and task), global coherence and metastability truly pinpoint the underlying coordination dynamic across multiple brain areas across the entire lifespan. This study was not only able to replicate the previous observations in a substantially larger lifespan cohort than what was previously reported, we also demonstrate, for the first time to the best of our knowledge, age related changes in coherence and metastability. Furthermore, we observed a marked frequency dependence in changes in global coordination dynamics, which, coupled with the long-held view of specific frequency bands subserving different aspects of cognition, hints at different functional processing roles for slower and faster brain dynamics.

Methods

We analyzed resting-state magnetoencephalogram data of 650 healthy individuals (age 18-88, 322 female) collected as a part of a cross-sectional adult life-span study by Cambridge Centre for Ageing and Neuroscience (Cam-CAN). Spectral power of neuronal oscillations was estimated using Welch's method. Frequency in the alpha band having maximum power was taken to be the representative peak alpha frequency (PAF). We estimated global coherence as a measure of global covariation among the sensors. Since variation in global coherence in delta and theta band with age was similar which was also the case for alpha and beta oscillation we broadly divided the oscillations into two bands i.e. slow oscillations (1-5 Hz) and fast oscillations (10-20 Hz). Upon visual inspection, we found that the overlap in sensor topographies corresponding to alpha and beta oscillations decreased with aging. We established the separation of alpha and beta oscillations sensor level representation with aging by estimating the angle between these topographies and correlating it with age.

Results

We found that global beta power i.e. power in the beta band averaged over all the sensors

was higher in elderly population compared to the younger population. PAF was significantly negatively correlated with age. Interestingly we found a set of sensors in the anterior part of the brain where PAF stayed invariant across life-span. The global coherence of slow oscillations increased with age whereas global coherence of fast oscillations decreased with age. PAF derived from global coherence analysis also significantly decreased with increase in age. The angle between vector representations of alpha and beta sensor topographies was positively correlated with age.

Discussion

Increase in beta power in older population has been well documented and is considered to be the cause of greater motor inhibition in aged individuals. Invariance in PAF in the prefrontal cortex may be reflective of healthy aging. Differential relationship of global coherence of slow and fast oscillations, combined with separation of alpha and beta topographies support the integration and segregation theory of aging. Source level analysis of this data may give insights into the specific brain regions responsible for this spatiotemporal reorganization.

Resting state dynamics meets anatomical structure: Temporal multiple kernel learning (tMKL) model

Introduction

Over the last decade there has been growing interest in understanding the brain activity, in the absence of any task or stimulus, captured by the resting-state functional magnetic resonance imaging (rsfMRI). The resting state patterns have been observed to be exhibiting complex spatio-temporal dynamics and substantial effort has been made to characterize the dynamic functional connectivity (dFC) configurations. However, the dynamics governing the state transitions that the brain undergoes and their relationship to

stationary functional connectivity still remains an open problem. One class of approaches attempts to characterize the dynamics in terms of finite number of latent brain states, however, such attempts are yet to amalgamate the underlying anatomical structural connectivity (SC) with the dynamics. Another class of methods links individual dynamic FCs with the underlying SC but does not characterize the temporal evolution of FC. Further, the latent states discovered by previous approaches could not be directly linked to the SC, thereby motivating us to discover the underlying lower-dimensional manifold that represents the temporal structure. In the proposed approach, the discovered manifold is further parameterized as a set of local density distributions, or latent transient states. We propose an innovative method that learns parameters specific to the latent states using a graph-theoretic model (temporal Multiple Kernel Learning, tMKL) that inherently links dynamics to the structure and finally predicts the grand average FC of the test subjects by leveraging a state transition Markov model. The proposed solution does not make strong assumptions about the underlying data and is generally applicable to resting or task data for learning subject-specific state transitions and for successfully characterizing SC-dFC-FC relationship through a unifying framework. Training and testing were done using the rs-fMRI data of 46 healthy participants. tMKL model performs significantly better than the existing models for predicting resting state functional connectivity based on whole-brain dynamic mean-field model (DMF), single diffusion kernel (SDK) model and multiple kernel learning (MKL) model. Further, the learned model was tested on an independent cohort of 100 young, healthy participants from the Human Connectome Project (HCP) and the results establish the generalizability of the proposed solution. More importantly, the model retains sensitivity toward subject specific anatomy, a unique contribution towards a holistic approach for SC-FC characterization.

Figure 1: Relation between global spectral activity and age. A. Plots of mean power spectral density for 4 non-overlapping age groups i.e. 18-35, 36-50, 51-65 and 66-88. Shaded region denotes standard error of mean. B. Boxplot of distribution of band limited power in the beta band (16-25 Hz). Blue line indicates the median of each distribution. Notch denotes 95% confidence interval of the median. C. Variation of alpha activity with aging has been plotted as an age-spectrogram. Center frequency in the alpha band has been plotted as solid circles and solid black line is the linear fit of these points (labels indicate effect sizes, significance and correlation function) D. Sensor topography of correlation between peak alpha frequency and age. Colorbar represents Spearman's rank correlation value.

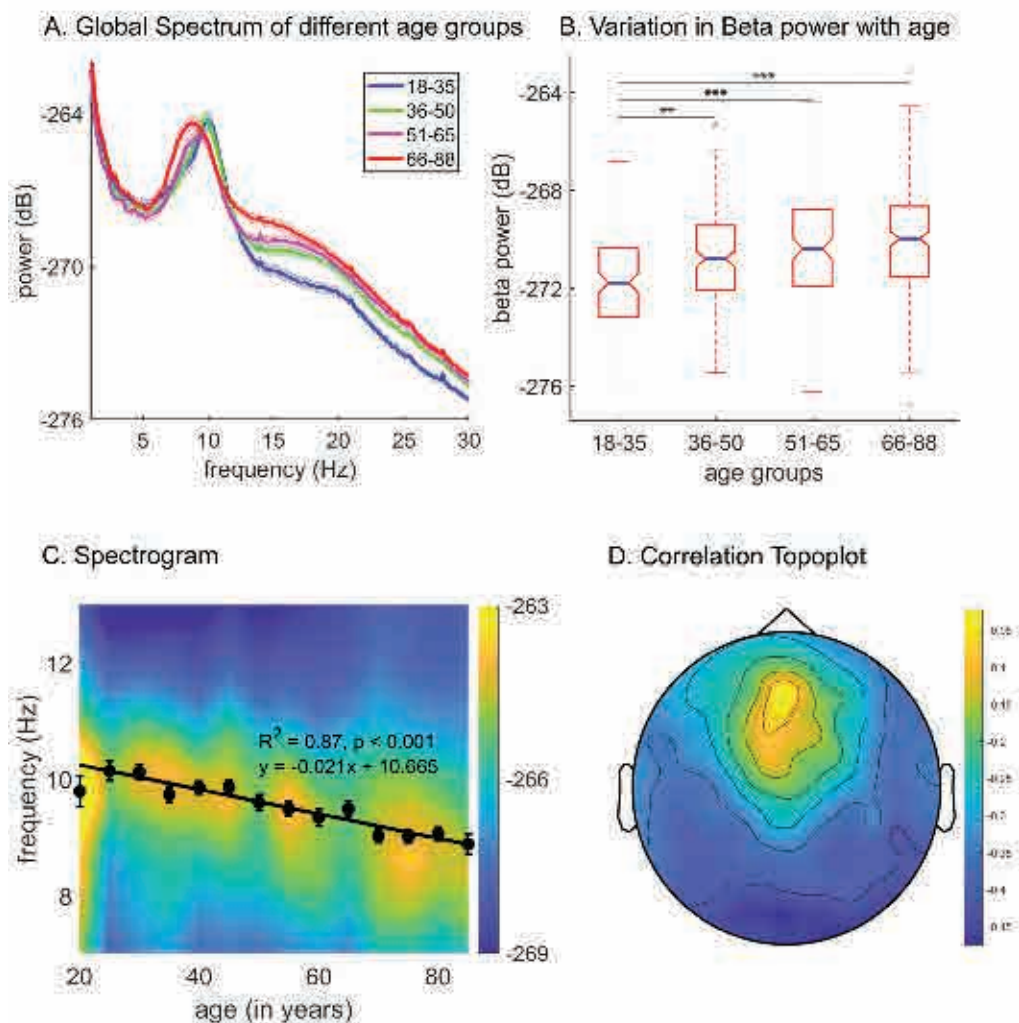


Figure 2:

Differential changes in global coherence with aging. A. Plots of mean global coherence for the four age groups. Shaded region denotes s.e.m. B. Differential variation of global coherence for slow (1-5 Hz) and fast oscillations (10-20 Hz). C. Variation of global coherence with aging. Solid circle denote the average global coherence value for a) Delta oscillations (1-3 Hz) b) Theta oscillations (3-7 Hz) c) Alpha oscillations (8-12 Hz) d) Beta oscillations (16-25 Hz) in each age bin of 5 years. Errorbar denotes s.e.m

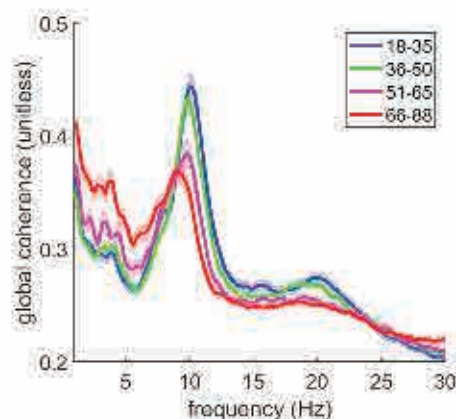
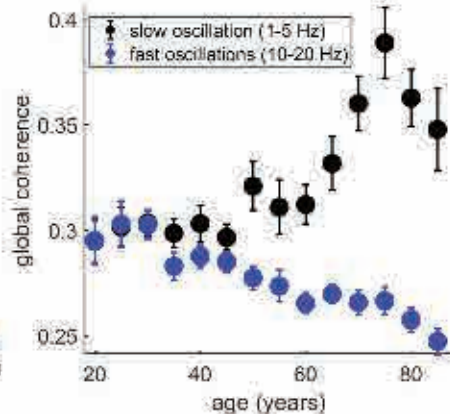
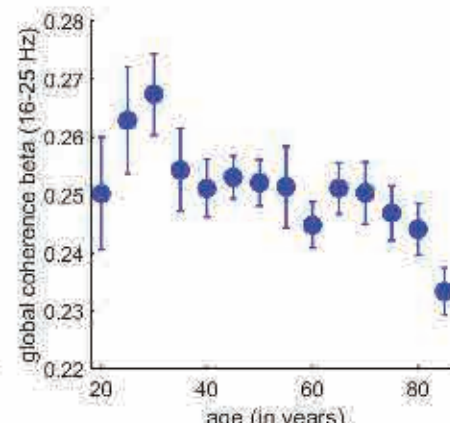
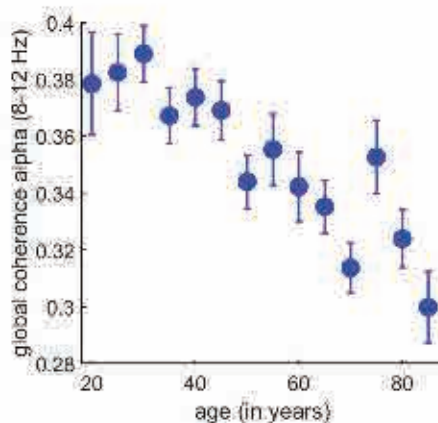
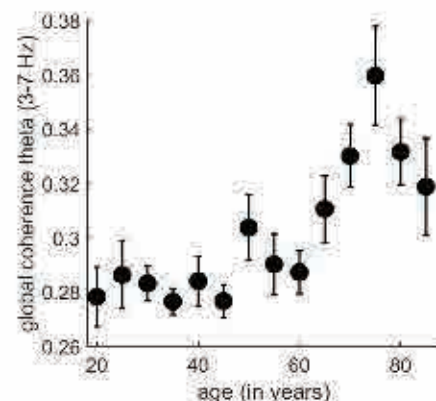
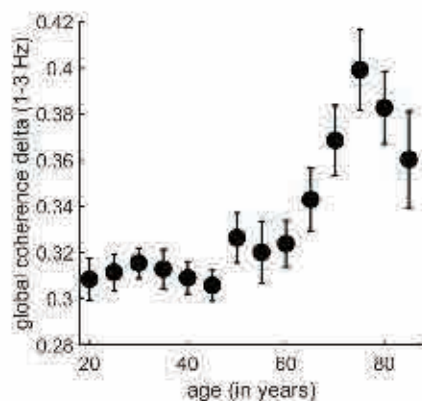
A. Global Coherence of Different Age Groups

B. Global Coherence of Oscillations at slow and fast time scales.

C. Global Coherence in different frequency bands


Figure 3:

Segregation of sensor level topographies with aging. A. Sensor topographies of alpha power at center frequency and average beta power for the two extreme age groups. B. Clusters of sensors with significant differences in power between the oldest and youngest age group for alpha band (8–12 Hz) and beta band (16–25 Hz). White dots represent sensors with a negative difference and black dots represent sensors with a positive difference. C. Boxplot for the distribution of angles between the sensor topographies of center alpha power and average beta power for the four age groups. Blue line denotes the median of the distribution and the notch indicates 95% confidence interval of the median.

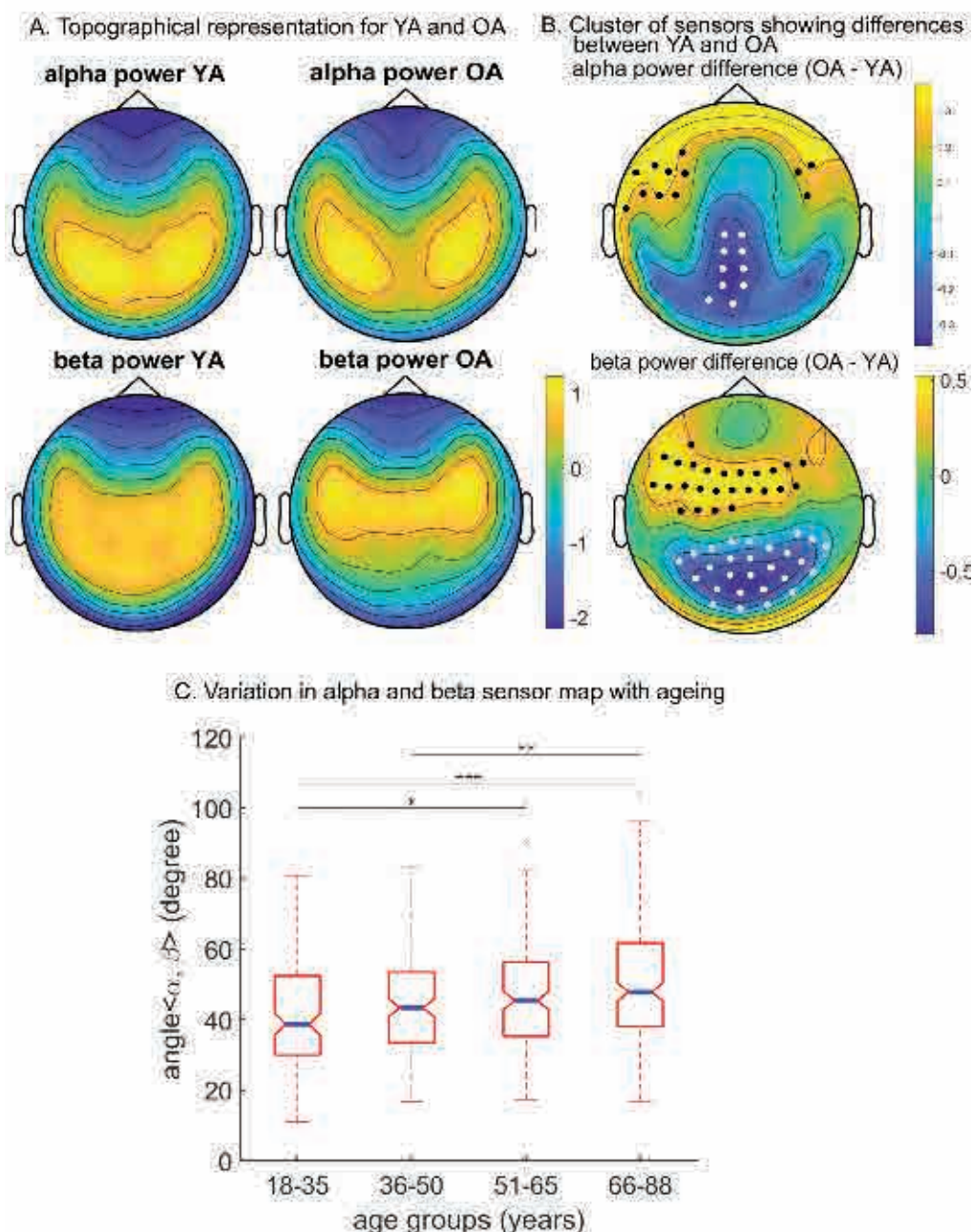
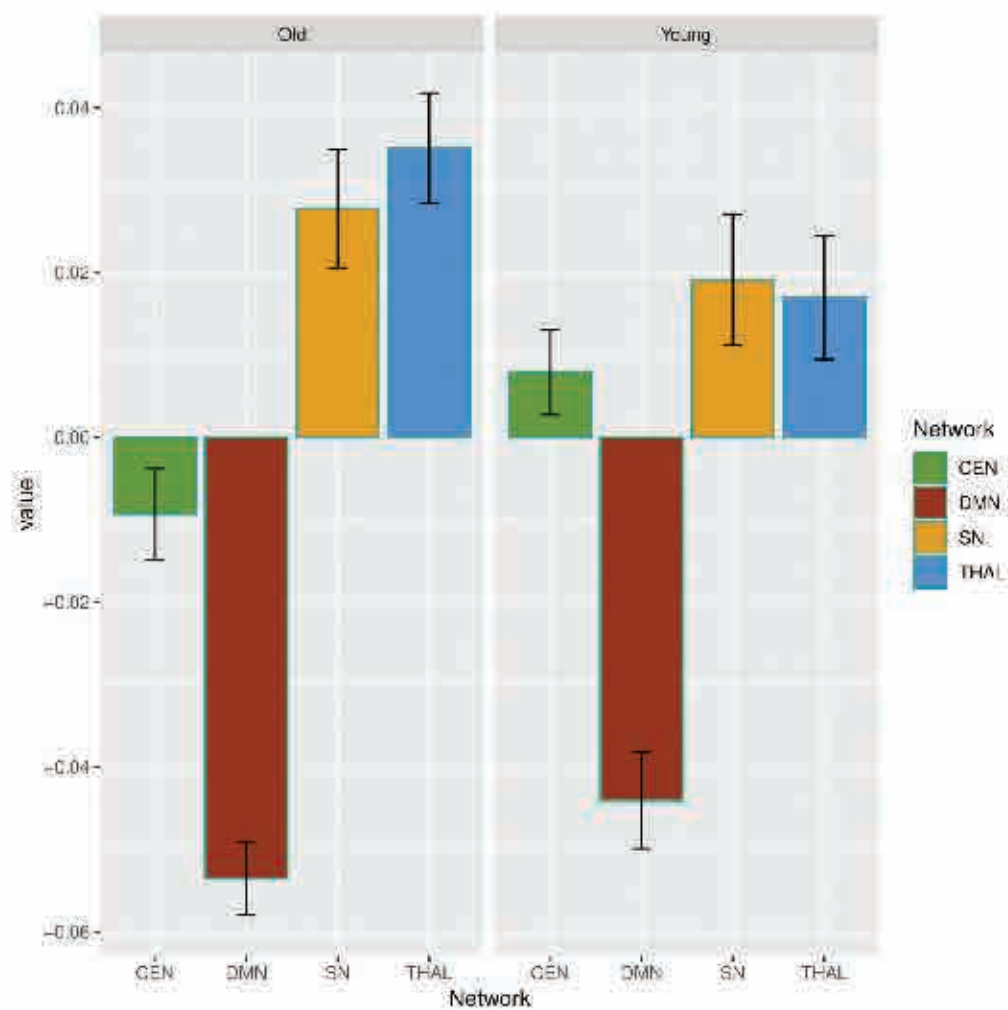
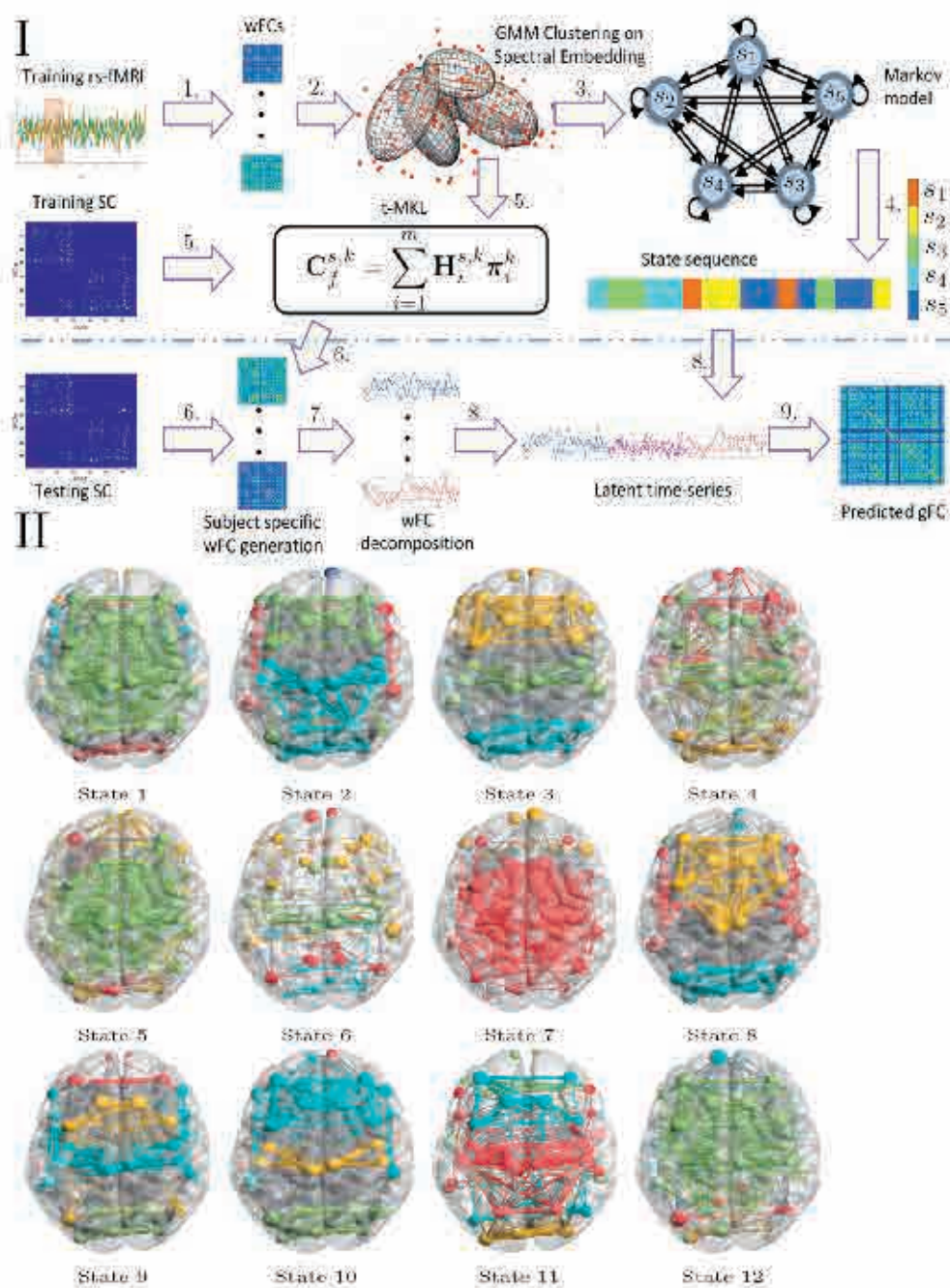


Figure 4: Between Network Causal outflow and Connectivity in the presence of right and left Thalamus for both young and old. Three networks interactions are shown here comprise of DMN, SN and CEN.



Between Network Connectivity in Presence of Thalamus for both Young and Old individuals.

Figure 5: Outline for temporal Multiple Kernel Learning (tMKL) model. Figure shows the entire pipeline for predicting grand FC for a testing subject. The model incorporates subject specificity along with temporal variation characterization. Part (I) of the model, training phase, consists of three blocks. The first one, learns temporal variations in terms of distinct states via GMM clustering over the underlying manifold of wFCs (steps 1. and 2). The second block utilizes the empirical transitions between these distinct states and captures dynamics in the first order Markov chain (steps 3. and 4). The third block learns subject-specificity by modeling each state by its MKL model (Surampudi et al., 2018) (step 5.). Part (II) of the model validates its generalizability on unseen subjects. Importantly, only SC of a testing subject is required (step 6). Each state for the testing subject is characterized in step 7. Each state-specific predicted FC is decomposed into a latent time series which are then concatenated using the steady state distribution of the Markov chain (steps 4. and 8). Finally, grand average FC, static FC, is predicted for that subject (step 9).



Publications

1. Govinda Surampudi, Joyneel Mishra, Bapi Raju Surampudi, Gustavo Deco, Avinash Sharma, and Roy, D (2019) Resting State Dynamics Meets Anatomical Structure: Temporal Multiple Kernel Learning (tMKL) Model. *Neuroimage* Volume 184, 1 January 2019, Pages 609-620
2. Harlalka, V., Bapi, R. S., Vinod, P. K., & Roy, D (2019) Atypical flexibility in dynamic functional connectivity quantifies the severity in autism spectrum disorder January 2019 *Front. Hum. Neurosci.* doi: 10.3389/fnhum.2019.00006
3. Harlalka V, Bapi RS, Vinod PK, Roy, D (2018). Age, Disease, and Their Interaction Effects on Intrinsic Connectivity of Children and Adolescents in Autism Spectrum Disorder Using Functional Connectomics. *Brain Connectivity* 2018 Aug 29. doi:10.1089/brain.2018.0616.
4. Lanke, V., Moolamalla, S. T. R., Roy, D. & Vinod, P. K. (2018). Integrative Analysis of Hippocampus Gene Expression Profiles Identifies Network Alterations in Aging and Alzheimer's Disease. *Frontiers in aging neuroscience*, 10. doi:10.3389/fnagi.2018.00153.

Presentations

1. Dipanjan Roy: Brain Awareness week DBT STAR college program DDU Delhi "Brain Science foundations and fundamentals" March 29, 2019
2. Dipanjan Roy: Invited talk at AIIMS New Delhi 10th of March one day workshop on mathematics in biomedicine.
3. Dipanjan Roy: Invited plenary speaker "Aging related resting and sensory motor brain signals and their reorganization" The Department of Biomedical Engineering, SSNCE Chennai Fifth International conference on bio signals, Images and Instrumentation (ICBSII 2019)
4. Dipanjan Roy: Invited speaker Brain Modes 2018

Computational modelling of Neurodegeneration and aging brain Dec 2-4, 2018 Havana, Cuba.

5. Dipanjan Roy: 5th IISC Bangalore Cognition workshop Center for Neuroscience module Attention at multiple scales June 17-29, 2018.

Funding

- 1) Role of Default Mode Network in Cognitive functions BT/RLF/Re-entry/07/2014 Department of Biotechnology (DBT) Ramalingaswami fellowship (Initiated in 2016 for five years)
- 2) Oscillatory Network Dynamics in Perceptual Learning SR/CSRI/21/2016 Department of Science and Technology (DST) Initiated in 2017 and for three years
- 3) Dementia Science Program: Incidence/ Prevalence/Risk analysis of dementia and basic research thereof. Ageing process in normality and dementia Department of Biotechnology starting from 2018, for three years

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Metabolic reprogramming in Glioblastoma: Implication in immune evasive responses



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Background and significance

Glioblastoma multiforme (GBM)- the most malignant of brain cancers is characterized by dysregulated metabolism. In addition to metabolic reprogramming; aberrant inflammation and genetic alterations promote glioma progression. As metabolic reprogramming deregulates a number of cellular functions and since targeting metabolic remodelers is regarded as a potential anti-cancer strategy; the focus of our study is to dissect molecular circuitries that regulate expression of metabolic modelers to subsequently affect genes associated with cellular bioenergetics and immune evasive responses in GBM.

(i) Silencing of Suppressor of cytokine signaling 1 (SOCS1) is associated with increased inflammation in GBM. SOCS1 is known to regulate Toll like receptor (TLR4) signaling and elevated TLR4 levels are observed in GBMs. On investigating the molecular and functional association between the two, TLR4 was found to negatively regulate SOCS1. However, TLR4 inhibition mediated increase in SOCS1 expression was observed only in p53 mutant glioma cells. Increase in SOCS1 promoter activity was concomitant with diminished nucleosomal occupancy around p53 sites on SOCS1 promoter in glioma bearing p53-mutant. Altered nucleosomal occupancy at this p53 site was accompanied by decreased nuclear H3K9me3, and increased JMJD2a and Brg1 levels. TLR4 inhibition enhanced the recruitment of JMJD2A- Brg1-p53 complex and decreased H3K9me3 levels at the p53 binding site. Interestingly, the ability of JMJD2a and Brg1 to serve as positive regulator of SOCS1 upon TLR4 inhibition was dependent on p53 status. Our findings implicate the importance of p53 mutational status in affecting TLR4-SOCS1 cross-talk through chromatin remodelers.

(ii) Mounting evidences link disruption of circadian rhythms with metabolic reprogramming in cancers. We investigated the role of metabolism in orchestrating circadian rhythm and the involvement of the later in regulating pro-inflammatory mediators crucial for mounting effective immune responses. Pharmacological inhibition of lactate diminished expression of pro-inflammatory cytokine IL-1 β and levels of core-clock genes CLOCK-BMAL1 in glioma. The reverse was observed upon treatment with IL-1 β . By highlighting the interplay between inflammation, metabolism and molecular clock; our study suggest the importance of this network as potential target for anti-cancer therapy.

Publication

Gowda, P., Patrick, S., Singh, A., Sheikh, T., and Sen, E. (2018). Mutant Idh1 Disrupts Pkm2-B-Catenin-Brg1 Transcriptional Network Driven Cd47 Expression. *Molecular and Cellular Biology*. 38(9):1-18.

Presentations

1. Ellora Sen. Immune surveillance in cancer: Therapeutic implications. Gurudas College, Kolkata, Oct 2018.
2. Ellora Sen. TLR4 affects p53-JMJD2A-BRG1 transcriptional network to drive SOCS1 expression. Genome Architecture and Cell Fate Regulation, University of Hyderabad, Hyderabad, December 2018
3. Knowledge in Science: A philosophical Quest. DST Sponsored Faculty Development Program on Entrepreneurship Science, Technology & Management for Society, Deshbandhu College, Delhi, December, 2018
4. Ellora Sen. Bridging metabolism and inflammation in glioma: Role of genetic and epigenetic landscape. IBRO, Associate School of Neuroscience on Advances in Molecular

Neurobiology Research, IGNTU, Amarkantak, March 2019.

5. Ellora Sen Metabolic modulation of immune regulatory responses in glioma. Society for Neurochemistry (India) SNCI & International Conference on Neurochemistry and Neuropharmacology: From bench to bedside Mysuru, March 2019.
6. Ellora Sen. Re-inventing oneself: Science as a Career. Department of Biochemistry, Daulatram College, Delhi, March 2019

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)

Award

Novartis Oration Award (ICMR)-2018

Autistic traits modulate interpersonal vocal communication



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Background

Humans routinely and volitionally modulate their voice in social contexts like striking up a new friendship, arguing or making an emotionally-charged speech. A well-modulated voice carries considerable information about the message, speaker, language, and even on the emotions of the speaker (Bhaskar et al., 2013; Spinelli et al., 2017). There are several factors that have been found to influence context-specific vocal modulation and understanding the finer aspects of spectro-temporal modulations in speech allows us to build better algorithms for both voice recognition and speech production. Past work in our laboratory has focused on examining the effect of autistic traits on vocal modulation in toddlers (Sharda et al., 2015). There we showed atypical modulations in speech in children with autism between 14-18 months of age. Here we examined the impact of autistic traits on vocal modulation and social communication and hypothesized that autism-related traits might constitute one such dimension of individual variability.

Similar to the approach used in Sharda et al., 2015, we used the Speech Modulation Spectrum (SMS), to study the 'articulation space' in adults in individuals. In addition, we also estimated the Autism Quotient (AQ) for all participants. Using the area of the articulation space as a measure we hypothesized that individuals high in autistic traits would exhibit reduced articulation space, indexing a reduced use of articulatory gestures.

Methods

170 healthy volunteers from three countries (43 native English speaker pairs of participants (18 Male pairs, 25 female pairs) from UK, 22 pairs (9 Male pairs, 13 Female pairs) from India and 20 pairs (10 Male pairs, 10 Female pairs) from Italy) participated in this study. Participants were asked to come along with another same-gender person, who could be either a friend or an acquaintance. All participants were also asked to fill in the Autism Spectrum Quotient (AQ) (Baron-Cohen, 2000). AQ is a self-report screening tool for quantifying autism spectrum conditions and autistic-like traits in adults. Participants were required to sit in front of each other in a silent room with a voice recording device in the middle. Once participants were comfortably seated, each participant was given one of two

coloured images (abstract paintings) printed on paper and was asked to describe the image to the other participant in as much detail as possible for around 2:30 minutes each. The experimenter then left the room. Since English is the primary language for official communication in UK as well as India, participants in these two countries spoke in English, while the Italian participants spoke in Italian.

Results

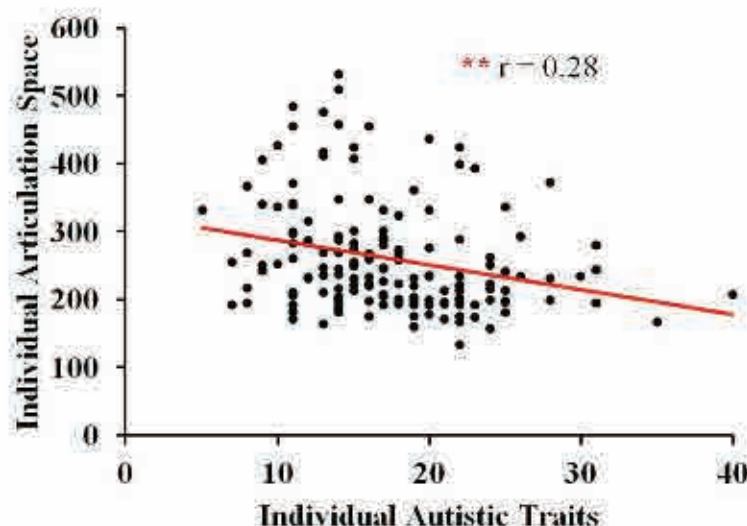
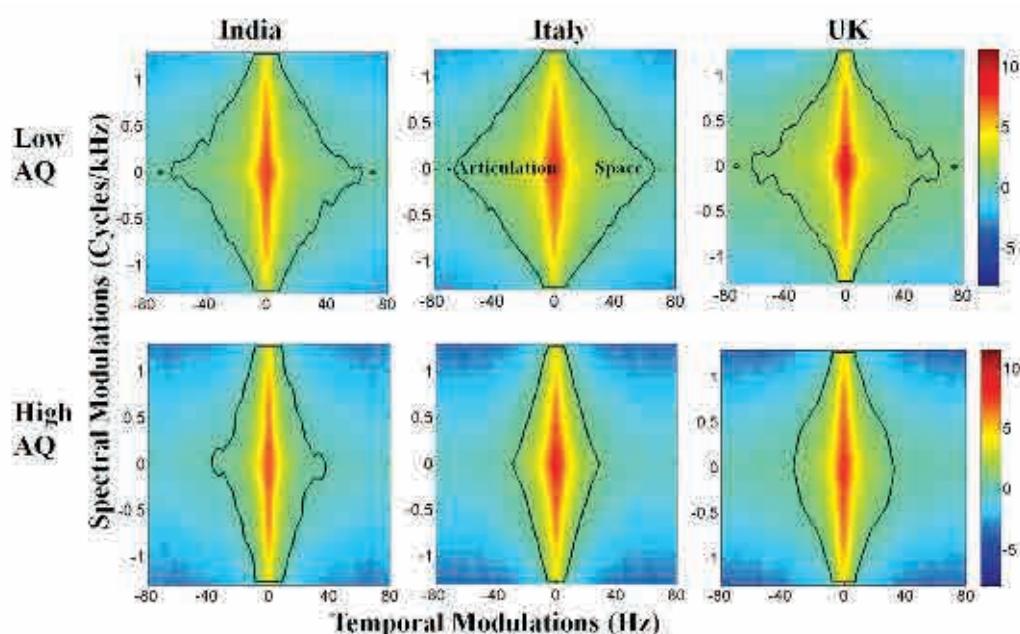
To test the role autistic traits on articulation space, a hierarchical regression was performed, after controlling for age, gender, culture, and duration of articulation. Articulation space was modelled as the dependent variable and the order of entry of the variables was as follows: age, gender,

articulation duration, culture, and autistic traits (AQ). The hierarchical regression results suggested a significant negative correlation relationship between AQ and articulation space ($r = -0.27$, $p < 0.01$) as shown in Figure 2.

Conclusions

This study found the impact of autistic traits on vocal modulation in interpersonal communication, demonstrating that the autistic related traits were associated negatively with vocal modulation, across different cultural and language settings. Future studies should extend these paradigms to include individuals with clinical deficits in social communicative abilities, such as those with ASD.

Figure 4: Representative Speech Modulation Spectra for individuals reported High and Low on Autistic traits (AQ) for all three cultures.



Scientific Reports

Publication

Singh, N. C., Rajan, A., Malagi, A., Ramanujan, K., Canini, M., Della Rosa, P. A., Raghunathan, P., Weekes, B. S., and Abutalebi, J. (2018). Microstructural Anatomical Differences between Bilinguals and Monolinguals. *Bilingualism: Language and Cognition*. 21(5):995-1008.

Funding

The neurobiology of dyslexia – combining brain and behavior. Cognitive Science Initiative, Department of Science and Technology, Government of India

Collaborators

- Prof. B. Chakrabarti, University of Reading
- M. Ingalhalikar
- Robert Zatorre
- Merav Ahissar.

Award

NASI Reliance Platinum Jubilee Award in Biological Sciences, 2018

Organization of Somatosensory and Motor Systems and the Effects of Spinal Cord Injuries



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Organization of Somatosensory and Motor Systems and the Effects of Spinal Cord Injuries

Research focus of our lab is to understand how somatosensory and motor systems of the brain are organized and processes information. Another focus of our laboratory is to study the nature, extent and mechanisms of brain reorganization following spinal cord injuries. Our laboratory studies rodent as well as non-human primate models, using multitude of technical approaches that include Magnetic Resonance Imaging (MRI), 2-photon imaging, electrophysiology, and neuroanatomy to address these research questions.

Previous work has shown that injuries to the dorsal columns of the spinal cord results in large scale reorganization of the primary (S1 or area 3b) and secondary (S2 and parietal ventral area) somatosensory cortex, and the medullary brainstem nuclei (e.g. Tandon et al., Journal of Neuroscience, 2009; Dutta et al., Brain Structure and Function, 2014; Halder et al., Cerebral Cortex, 2017), and specific changes in the movement representation in the primary motor cortex or M1 (Kambi et al., Journal of Neuroscience, 2011). Our studies in rats have also shown changes in the movement representation (Tandon et al., European Journal of Neuroscience, 2013). More recently we have established mechanisms of reorganization of the cortex using electrophysiological and anatomical approaches (Kambi et al., Nature Communications, 2014; Chand and Jain, Journal of Neuroscience, 2015).

In order to further understand the extent, time course and mechanisms of this brain-wide reorganization we added Functional Magnetic Resonance Imaging (fMRI) to our repertoire of techniques (Dutta et al., Brain Structure and Function, 2014). Our recent experiment show that that the resting state somatosensory functional network, identified from the BOLD signal fluctuation measured using fMRI, of different somatotopic body-part representations in the primary somatosensory cortex of macaque monkeys is distinct and generally mimics the underlying anatomical connectivity (see Annual Report for 2017-18). In order to determine how this brain-wide somatosensory network and the activation pattern alters after spinal cord injuries, we studied the effects of dorsal column lesions on two adult macaque monkeys. Dorsal columns were surgically lesioned unilaterally at cervical levels. Multiple resting state

fMRI scans of the monkeys were acquired before and after the lesion. In addition, BOLD signal was acquired while hand and chin underwent tactile stimulation to establish the brain reorganization.

T2 weighted MR images of the spinal cord were acquired before and after the lesions of the dorsal columns to determine the extent and level of the lesion (**Fig. 1**).

Figure 1: T2 weighted MR images of spinal cord of a monkey MK15-117 before the lesion, and of monkeys MK15-93 and MK15-117 after the lesion. The red arrows point to the opacity due to chronic partial left dorsal column lesion. The top panel shows the spinal cord in the sagittal plane, bottom in the horizontal (dorsoventral) plane.

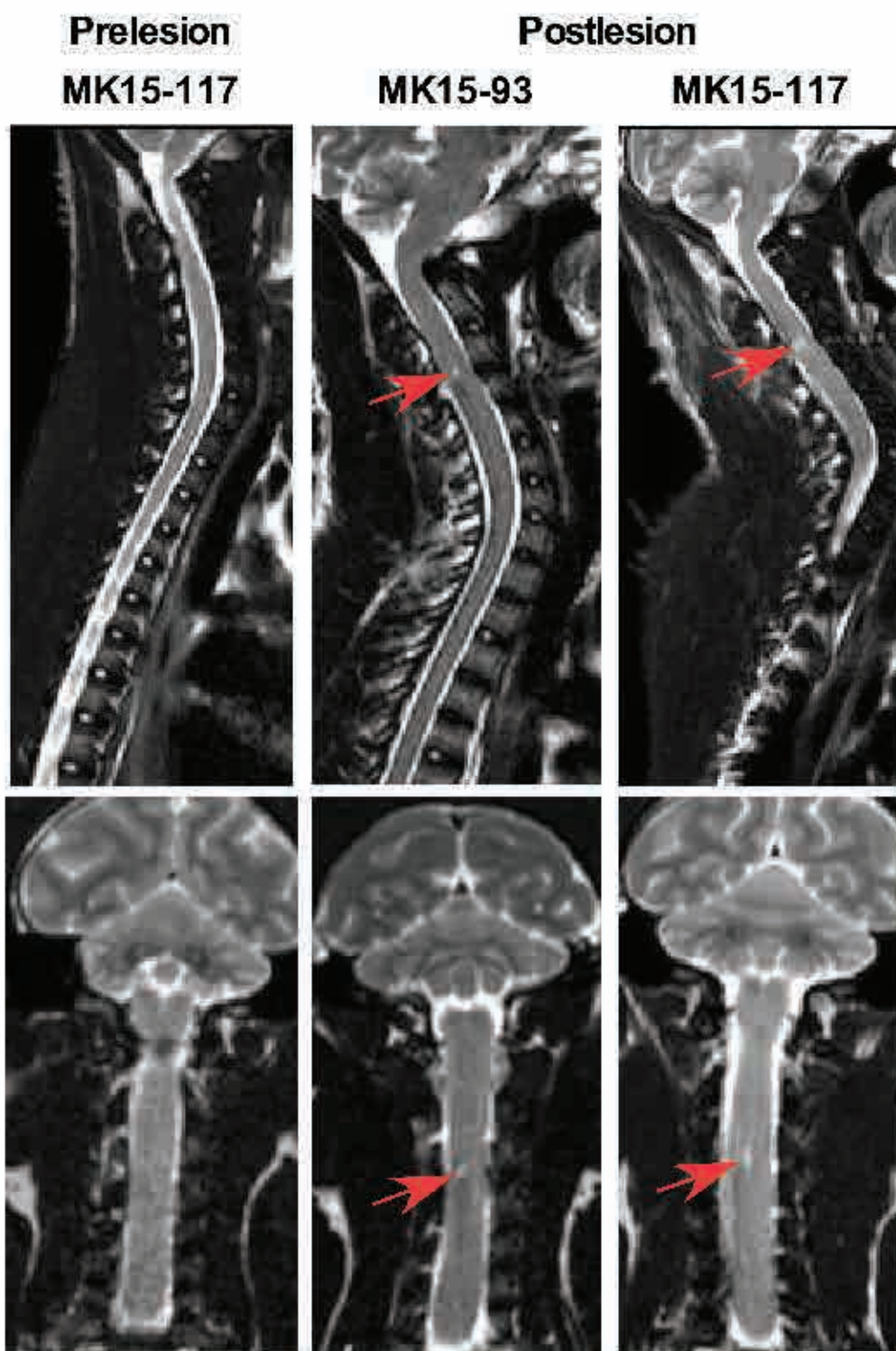
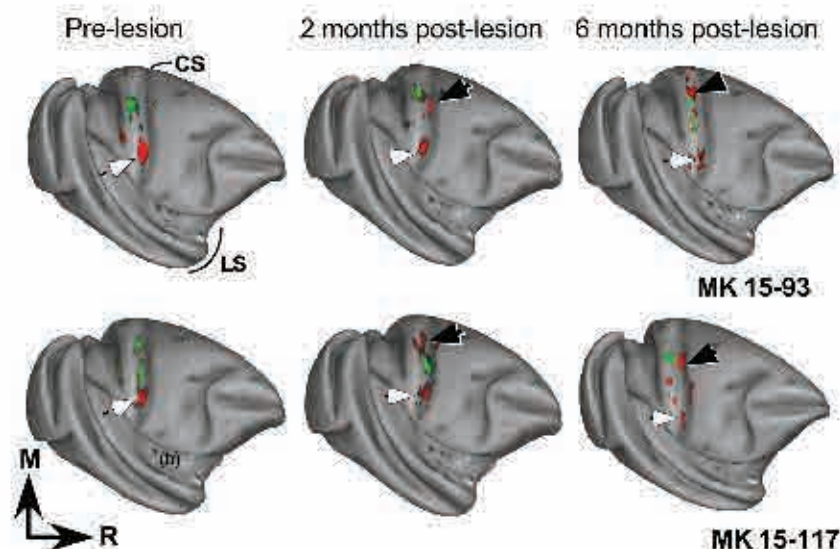


Figure 2: Reorganization of area 3b in monkeys (Top Row) MK15-93 and (Bottom Row) MK15-117 before the lesion (left column) and following lesion of the dorsal columns 2 months (middle column) and 6 months (right column) after the lesion shown on partially inflated brain surface. BOLD signal following tactile stimulation of the chin (red) and hand (green) shows that in addition to the normal responses to the chin stimulation (white arrows), post-lesion responses to chin stimulation are evoked more medially in area 3b where hand responses are expected (black arrowheads). Responses to stimulation of the hand remain after the lesion because the lesion was partial. For normal chin responses, $p < 0.005$, uncorrected; for hand responses, $p < 0.005$, uncorrected; and for chin responses in the hand region; $p < 0.02$, uncorrected.



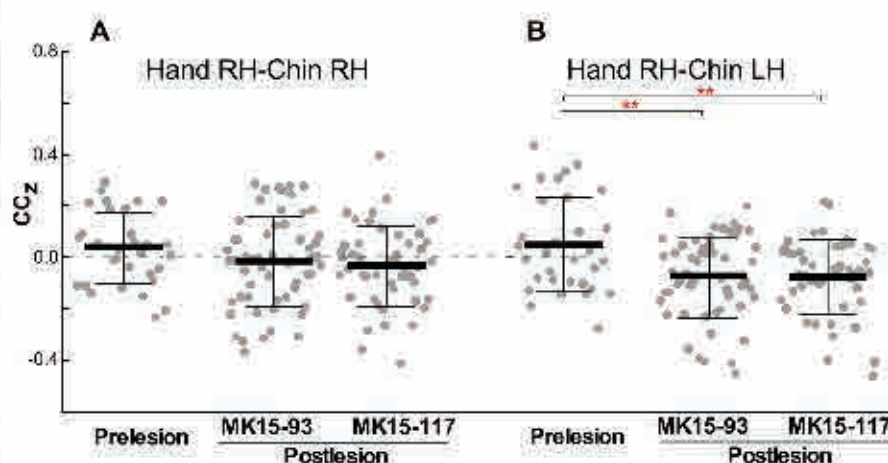
The acquired BOLD signals for both the resting state and stimulation was analysed after performing standard pre-processing steps. Data from stimulation experiments was analysed by implementing General Linear Model to localize the brain activation regions. Results showed the expected expansion of the chin representation in the deafferented hand region of area 3b in these monkeys (**Fig. 2**).

For the resting state data region-to-region correlation analysis was performed to determine functional connections between regions of interest. The results show that lesions of the dorsal columns alter the somatosensory functional network of the

brain i.e. its connectivity with other brain regions. In area 3b, connectivity of the deafferented hand region to the contralateral chin was significantly reduced postlesion, whereas the ipsilateral hand-chin connectivity was not altered (**Fig. 3**). In normal monkeys there is no significant connectivity between the hand and the ipsilateral or the contralateral chin representation. Thus expansion of the chin representation results in inhibition in the contralateral chin representation.

The other interesting result shows changes in the functional interaction between the somatosensory network and the Default Mode Network (DMN). Posterior Cingulate Cortex (PCC, a part of DMN)

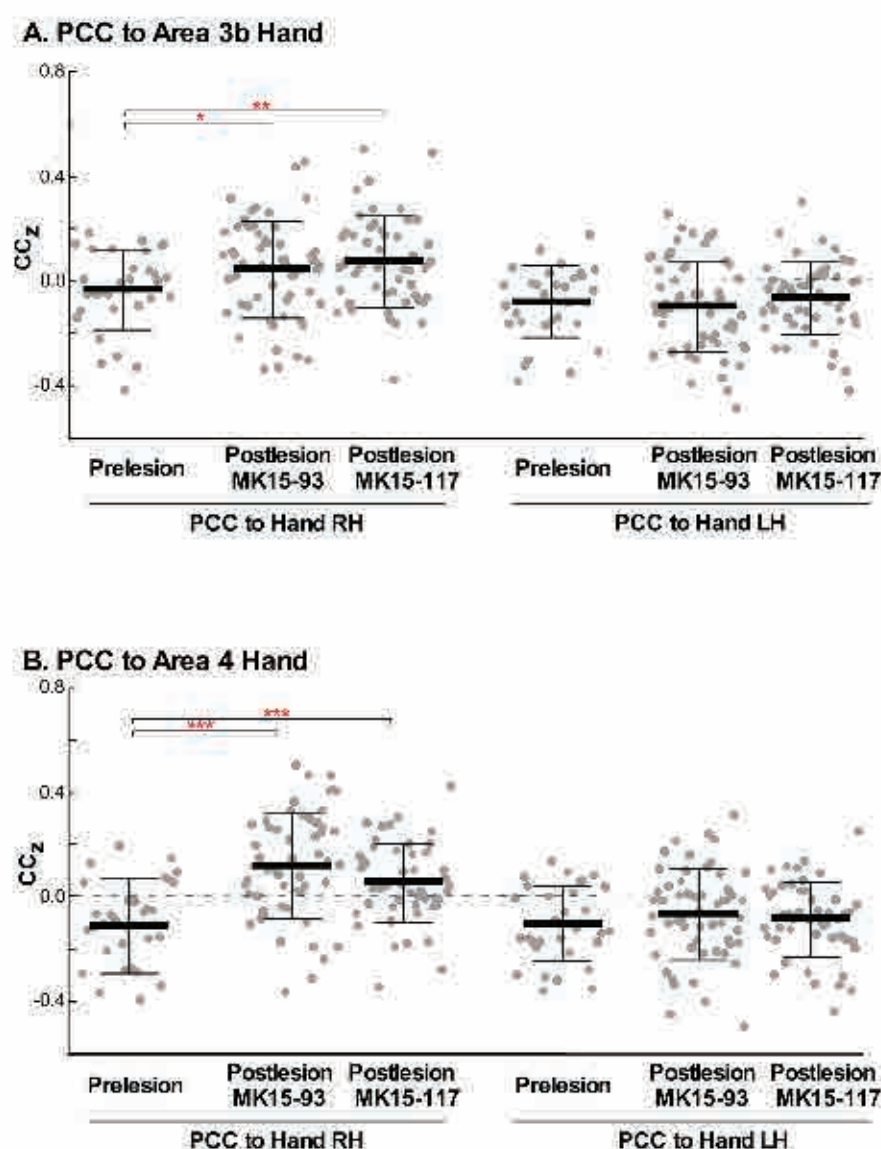
Figure 3: Resting state functional connectivity of the deafferented hand ROI of area 3b in the right hemisphere with (A) ipsilateral and (B) contralateral chin ROI before (pre-lesion) and after lesions of the dorsal columns for the two monkeys MK15-93 and MK15-117. Each dot in the scatter-plots represents Fisher Z transformed correlation coefficients (CC_z) of a resting state scanning session. No change in CC_z was observed in the hand - chin connection in the deafferented hemisphere, while the deafferented hand region to contralateral chin ROI connectivity showed a significant negative value of CC_z , i.e. an inhibition (Panel B). ** $p < 0.005$; two sample t-test.



showed increased correlations with the deafferented hand region of both somatosensory (area 3b) and motor (area 4) cortex (**Fig. 4**). This correlation was specific to the deafferented and reorganized hand representation, and was not seen for the hand representation in the contralateral hemisphere that continued to receive normal sensory inputs

after the unilateral lesion. DMN is a network that is traditionally believed to represent the correlated network which is active when no specific task is being performed by the subject. Our results suggest that the deafferented hand cortex dissociates from the somatosensory network and becomes part of DMN in the absence of active sensory inputs.

Figure 4: Resting state correlation of the Posterior Cingulate Cortex (PCC), which is a part of the Default Mode Network (DMN) to the (A) deafferented hand ROI of somatosensory area 3b, and (B) hand ROI in ipsilateral motor area 4 for monkeys MK15-93 and MK15-117. Post lesion the deafferented hand ROI of somatosensory and motor areas showed significant increase in the connectivity with PCC. However, the connectivity of contralateral hand ROI (which continues to receive normal inputs) to PCC remain unaffected. Each dot in the scatter-plot represents correlation coefficients (CCZ) of a resting state scanning session. * $p < 0.05$; ** $p < 0.005$; *** $p < 0.0001$; two sample t-test.



Publication

Halder, P., Kambi, N., Chand, P., and Jain, N. (2018). Altered Expression of Reorganized Inputs as They Ascend from the Cuneate Nucleus to Cortical Area 3b in Monkeys with Long-Term Spinal Cord Injuries. *Cerebral Cortex*. 28(11):3922-38.

Presentations

1. 'Cortical, callosal and thalamic inputs to the neck and jaw motor representations in rats'; Hisham Mohammed and Neeraj Jain. Neuroscience 2018, Annual Meeting of the Society for Neuroscience, USA. Nov 3-7, 2019; San Diego, USA.

2. 'Resting state somatosensory functional map is composed of multiple networks in macaque monkeys and humans'; John Thomas, Dixit Sharma, Sounak Mohanta and Neeraj Jain, 36th Annual Meeting of the Indian Academy of Neuroscience, BHU Varanasi, October 29-31, 2018.
3. 'Deafferentation Induced Plasticity of the Adult Somatosensory System', Gordon Research Conference (GRC): Neuroplasticity of Sensory Systems, Hong Kong, 3-8th June, 2018.
4. 'Somatosensory information processing and spinal cord injuries', 36th Annual Meeting of the Indian Academy of Neuroscience, BHU Varanasi, October 29-31, 2018.
5. 'Recent advances in technology to map brain plasticity after spinal injury', at 'Disability Inclusiveness' on Disability Day, Indian Spinal Injuries Centre, New Delhi, December 3, 2018.
6. 'Neuroplasticity and Spinal Cord Injuries' at Bhaskaracharya College of Applied Sciences, New Delhi, February 20, 2019.
7. 'Spinal Cord Injuries and Brain Plasticity' at 5th South Asian Biotechnology Conference – 2019, South Asian University, New Delhi, March 14, 2019.

Funding

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Collaborator

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Cellular and Molecular Mechanisms of HIV-1 and Zika Virus Neuropathogenesis



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The molecular and cellular events that culminate in compromised cognitive and motor functions in HIV/AIDS patients are studied under a relatively recent field called neuroAIDS. HIV-1 infection and its trafficking into human brain result in neuronal damage that causes a variety of cognitive and motor deficits that are referred to as HIV-1 Associated Neurocognitive Disorders (HAND). HAND is one of the major cause of morbidity among the HIV-1/AIDS survivors and affects up to 40% of the HIV-1/AIDS patients.

Recent research into this area suggests that most of the HIV-1 induced neuronal damage is mediated through glial cells, a cell type that is otherwise supposed to protect neuronal cells. Under physiological conditions, glial cells support optimal neuronal functions through dynamic neuron-glial interactions. We performed detailed experiments to understand how HIV-1 viral protein, Transactivator of transcription (Tat) modulated the neuron-glia interactions that may culminate into neuronal damage.

Neuron and glia, being the two primary cells, are in constant cross-talk for homeostatic functioning of the brain. Astrocytes and microglia are the two main glial cells that are permissive to the virus in the brain. The major pathway to neuronal damage and their eventual death is mainly mediated through the astrocytes that are infected and harbor the virus. These HIV-1 infected astrocytes consistently release many viral proteins, most crucial of all of them being Transactivator of transcription (Tat) which leads to loss of optimal cellular functioning. The release of these viral proteins also elicits an increase in release of cytokines and chemokines leading to neuroinflammation. Our laboratory has immensely contributed to the study of the importance of astrocytes in mediating neuronal damage and death. We continue to investigate the cellular and molecular mechanisms for astrocyte-mediated neuronal damage, the major cause of neuronal damage.

The two proteins involved in neuron-glia crosstalk are EphrinA3 and EphA4, the former being a ligand present on the astrocytes and the latter being a tyrosine kinase receptor present on the neurons. Eph receptors and Ephrins are known to play an important role in axon guidance and retinotopic mapping during embryonic development, but very

recently their importance in maintaining glutamate levels in the synapses and regulating dendritic spine morphology has come into the limelight. Interaction of EphrinA3 with its receptor EphA4 alters spine density in neurons and glutamate levels in the synapse during neuron-astrocyte interactions. We adopted human astrocyte-neuron co-culture system derived by differentiating primary human fetal progenitors and demonstrated the expression of Ephrin A3 in astrocytes and Eph A4 in neurons, thereby making our co-culture of primary cells a perfect model for this study. Interestingly, we have observed higher expression of EphrinA3 in astrocytes in the presence of HIV-1 Tat as compared to the controls. As per the hypothesis, we observed elevated levels of glutamate in the supernatant of co-cultures which is concomitant with the decrease in the levels of glutamate transporters EAAT1 and EAAT2 in the presence of HIV-1 Tat. To further corroborate the role of EphrinA3 in glutamate transportation, we performed knockdown experiments. The elevated glutamate level decreased in EphrinA3 knockdown and HIV-1 Tat co-transfected cells as compared to just HIV-1 Tat transfections. Elevated levels of glutamate led to excitotoxicity that causes neuronal damage, as documented in HIV-1 neurodegeneration. To further support our data, we are also validating the expression of these proteins in adult HIV-1 positive brain tissue sections through immunohistochemistry. Our results cumulatively point towards these proteins being potential checkpoints of therapeutics in HIV-1 neuropathogenesis. To delineate the potential microRNAs that are altered in the case of HIV-1, Next Generation Sequencing (NGS) has been performed on astrocytes transfected with HIV-1 Tat. We have then screened out those microRNAs that are downregulated and are also targeting the EphrinA3 3'UTR. Around 19 such microRNAs have been identified and their validation through qPCR is in progress.

In addition to our work on HIV-1 neuropathogenesis, our efforts are also invested into understanding the molecular mechanisms of Zika Virus (ZIKV) induced microcephaly, a condition that leads to a smaller head size which is common in infants born to mothers infected with Zika virus during their first trimester of pregnancy.

Zika Virus (ZIKV) is a mosquito-borne flavivirus. Zika Virus (ZIKV) has a positive sense RNA

genome that encodes for a polyprotein with three structural proteins and seven non-structural proteins. Structural proteins are: capsid (C), premembrane/membrane (PrM), and envelope (E protein); non-structural proteins are NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5. There has been a strong association of microcephaly in cases of babies born to mothers that acquired ZV infection during their pregnancies. ZIKV transmission has been reported in 84 countries worldwide, including India. It causes severe congenital birth defects in neonates. Due to large worldwide outbreaks and neurological complications associated with ZIKV, WHO declared public health emergency of international concern. It is largely unknown how ZIKV develops neurotropism and pathogenicity. While it has been established by others, that Zika virus infects human neural stem cells (hNSCs), but the mechanisms of its effects on hNSCs are poorly understood. We hence focused our research efforts to delineate the mechanism of ZIKV induced microcephaly, especially as ZIKV causes quiescence of NSCs thereby reducing the pool of cells in the brain. Using various cell biology and molecular approaches, we reported that E protein modulates proliferation, migration and differentiation of human neural stem cells.

To further probe into the mechanism, we performed global microRNA expression (miRNA Seq) profiling. miRNA Seq analysis delineated that E protein disrupts miRNA circuitry of fetal neural stem cells (fNSCs). We have validated a number of differentially expressed miRNAs using qPCR including hsa-miR-1273g-3p, hsa-miR-204-3p, hsa-miR-1306-5p, hsa-miR-6087, hsa-miR-23c and hsa-miR-3128 which are also involved in other viral infections.

Gene Ontology annotation for the targets of differentially expressed miRNAs revealed alterations of pathways important for proliferation, stemness of human neural stem cells and brain development. This supports our findings that E protein alters cell cycle dynamics. Further, WNT, CCKR, PDGF, EGF, p53 and notch signaling pathways are among the top enriched pathways of targets of differentially expressed miRNAs. In our ongoing studies, we have observed significant down-regulation of WNT2 and hence deregulated WNT signaling in response to E protein, this is being further looked into in detail.

We believe our findings provide novel insights into the mechanism of how Zika virus envelope (E) protein modulates the properties of fNSCs by altering miRNA profile of these cells. This

significant advancement into the field has been made at a time when basic and clinical researchers are struggling with the challenge to find therapeutic intervention for microcephaly.

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Presentations

1. Pankaj Seth: *Use of human neural stem cells as a model to understand neurodegenerative disorders*, during Luxembourg-German-India Alliance on Neurodegenerative diseases and Therapeutics (Lux-GIANT) under the Indo-German initiative, Tubingen, Germany, September 14-15, 2018.
2. Pankaj Seth: *Zika viral protein alters human neural stem cell properties by altering miRNA circuitry*, at the Section of Infections of the Nervous System (SINS), National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, USA, April 30, 2018.
3. Pankaj Seth: *Insights into HIV and Zika virus induced alterations in human progenitor cells*, at the University of Miami, Miller School of Medicine, Don Soffer Clinical Research Centre, Miami, USA, April 24, 2018.

4. Pankaj Seth: *What we know and what we need to know about HIV-1 neuropathogenesis*, at the RUSH University Medical Centre, Department of Neurological Sciences, Chicago, USA, April 17, 2018.
5. Pankaj Seth: *Friends turn foe during HIV-1 neuropathogenesis*, at the Alcohol Research Center, University of Chicago at Illinois, Chicago, USA, April 16, 2018.
6. Pankaj Seth: *Zika Virus affects human neural stem cells*, at a Two day symposia "Exploring Mind, Brain and Behaviour" at Amity University NOIDA, India, during March 28-29, 2019.
7. Pankaj Seth: *Understanding the virus induced neurodegeneration with human neural stem cells*, national symposium on stem cell technologies in neurodegenerative diseases, at Era University, Lucknow, March 9, 2019.
8. Pankaj Seth: *Cellular and molecular mechanism of zika virus induced alterations in properties of human neural stem cells*, at 3rd Symposium of Virus Diseases organized by Centre for Infectious Disease Research, Indian Institute of Science, Bangalore, India, December 13, 2018.
9. Pankaj Seth: *Molecular mechanism of HIV-1 and Zika virus neuropathogenesis*, IBRO-APRC school at Panjab University, Chandigarh, India, December 1, 2018.
10. Pankaj Seth: *Molecular mechanisms of Zika virus E protein induced alterations in human neural stem cells*, 36th Annual meeting of Indian Academy of Neurosciences (IAN-2018), at Banaras Hindu University, Varanasi, India, October 29-31, 2018.
11. Pankaj Seth: *What we know and What we need to know about Zika Virus*, at Era University, Lucknow, India, October 18, 2018.
12. Pankaj Seth: *How do viruses affect human brain – some insights*, Amity University – Haryana, Manesar, July 26, 2018.

13. Pankaj Seth: *Do's and Don'ts of Grant Writing*, in Mini-Symposium on Professional Skills in Scientific Research under aegis of UGC-UPE Focused Area-II at Interdisciplinary School of Life Sciences, Institute of Science, Banaras Hindu University, Varanasi, India July 14, 2018.

14. Pankaj Seth: *Human neural stem cells as in vitro model to study neurodegenerative diseases*, School of Regenerative Medicine, Manipal University, Bengaluru, India, May 10, 2018.

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Degree Awarded

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Predicting Novel Early Diagnostic Biomarkers for Alzheimer's Disease and Parkinson's Disease using multimodal MR based techniques and integration of data using Artificial Intelligence



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Developing early diagnostic marker for Alzheimer's Disease (AD) from multi-modal MR imaging and neuropsychological tests. To achieve this goal, we are investigating the role of major antioxidant, glutathione (GSH) and its conformational changes in various brain regions for the onset of AD in Indian population and cohorts from other continents. These GSH levels are being correlated with susceptibility of brain tissue through non-invasive techniques to test for the role of metal ion in AD. We are also testing the cognitive profile of healthy aging and cognition by testing the working memory performance and changes in deterioration of visuospatial perception. We are also conducting various neuropsychological tests that evaluate to cognitive and perceptual reserve indicators in healthy, young and old individuals as well as in AD patients.

The NeuroImaging and NeuroSpectroscopy (NINS) laboratory focuses on identifying early diagnostic biomarkers for neurodegenerative disorders such as Alzheimer's disease (AD). AD is the most common form of dementia in the world with a whopping 47.5 million individual suffering worldwide and 7.7 million additions every year (2016 WHO Dementia). An understanding of causal molecular process that transform a healthy brain to a diseased condition would help us for therapeutic advancements to cure AD.

Our lab is focusing on the study groups consisting of normal healthy control (HC), mild cognitive impaired (MCI), and AD for the following features:

- Glutathione (GSH) levels
- Brain Iron quantitation
- Working memory performance
- Visuospatial perception
- Neuropsychological test scores for cognitive reserve

This assessment of these neurochemical levels and functional performance in quantitative terms and its correlation with disease progression is the major focus of research in our laboratory.

I. Investigation for the role of oxidative stress in AD

Oxidative stress is an important event in AD process. The role of metals specifically iron as possible enhancer of oxidative stress in AD process are being investigated in our laboratory. Similarly, the antioxidants, glutathione and its role for neutralization of reactive oxygen species (ROS) are also being investigated in healthy and clinical populations.

We have found that major antioxidant glutathione exists in two conformations (Figure 1) and the potency of these conformers to scavenge the radicals is an important area to investigate with respect to AD pathology.

The brain iron levels are being detected using susceptibility weighted MRI sequence. The GSH (extended and closed) conformation levels from 5 different brain regions- Cerebellum (Cer), Left Hippocampus (LH), Left Parietal Lobe (LPC), Anterior Cingulate Cortex (ACC), Posterior Parietal Cortex (PCC) are being measured using MEGA PRESS pulse sequence with selective excitation pulses at 4.40 and 4.56 ppm respectively.

We are in the process of data collection to evaluate the difference in percentages of these conformations in healthy and AD cohorts, in order to be able to correlate them to the paramagnetic susceptibility levels, in order to test the Ferroptosis theory of AD onset. The mean levels of these GSH conformer

Figure 1: Impact of brain microenvironment on the conformational changes of GSH as evidenced by in vitro and in vivo MR spectroscopy. Tripeptide GSH is available in two forms. In the panel (A), (I and II), represents the structure of closed form of GSH and the NMR spectra. The second panel (B), (I and II), presents the extended conformation of GSH form and the NMR spectra. (A,B) (III) display MEGA-PRESS spectra of the cerebellum of the same subject for closed form of GSH (with 180° excitation pulse at 4.40 ppm and TE/TR 130/2500ms) and extended GSH form (with selective 180° pulse applied at 4.56 ppm and TE/TR 130/2500ms) using 3T Philips Achieva (National Imaging Facility, NBRC, India). The NMR spectrum of GSH (closed and extended forms) is aligned with MR spectra of GSH from human brain.

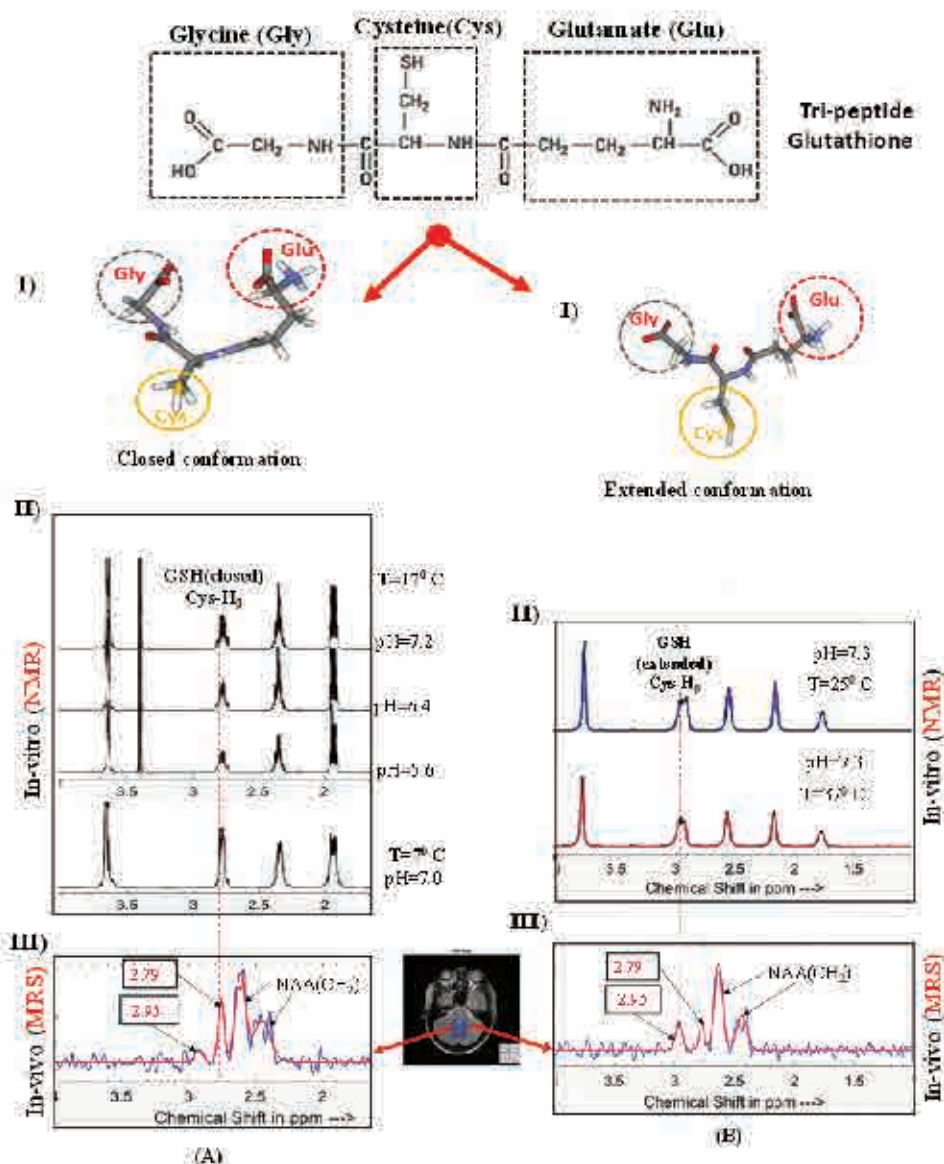
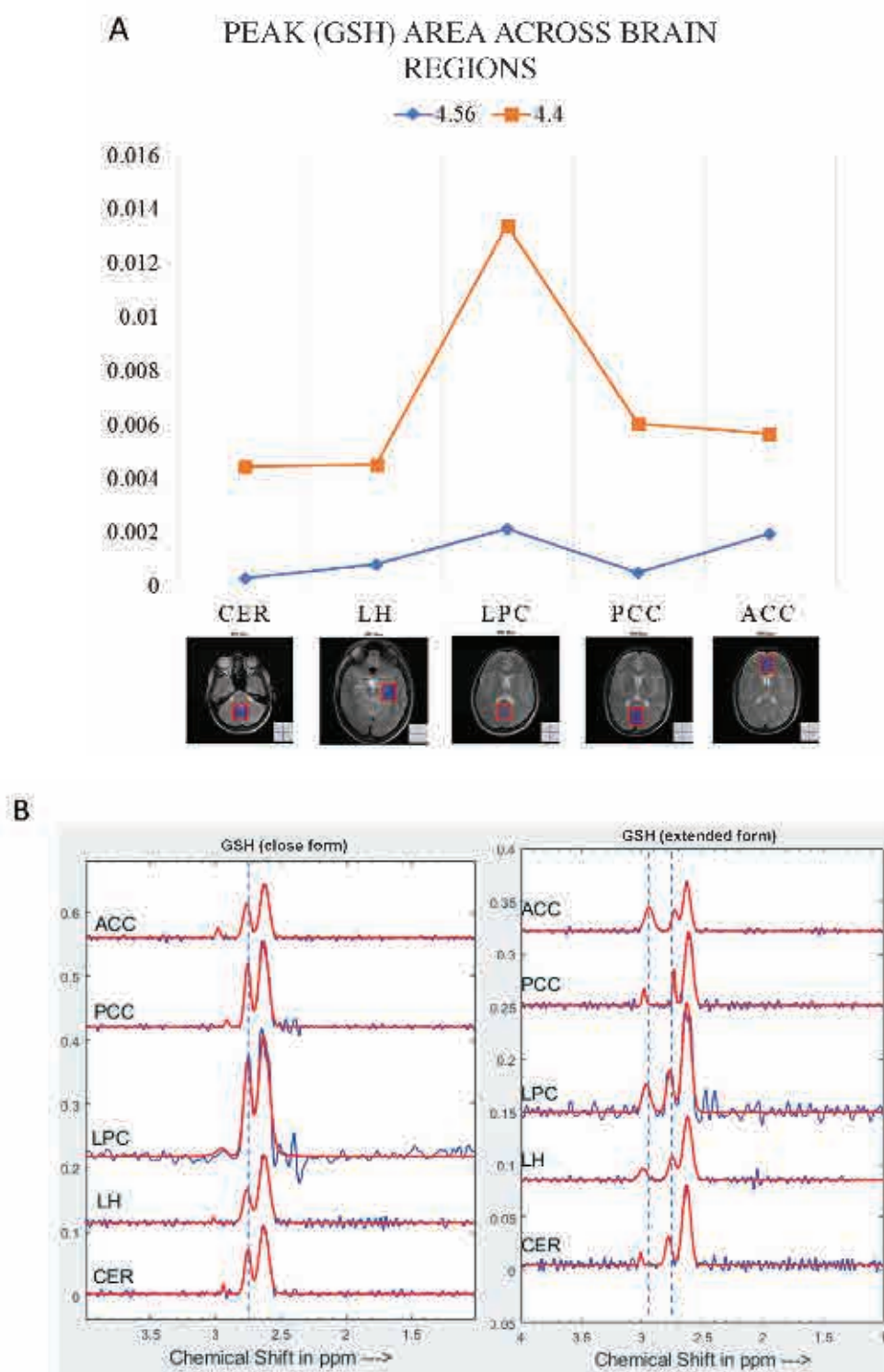


Figure 2: Panel A shows the representative of extended (4.56) and closed (4.40) conformers of GSH and Panel B shows the sample MEGA PRESS spectra in 5 brain regions Cer, LH, LPC, PCC and ACC from a single subject.



levels and sample spectra have been showed in Figure 2. We have developed a MATLAB based

pipeline for region specific iron susceptibility quantification from Susceptibility weighted Images.

II. Development of a Population-Specific MRI Brain Template for the Indian Population

Development and standardization of a robust MRI brain template for the Indian population. The population-specific template has been constructed using data from healthy adults in the range of 20-60 years.

A robust pipeline for constructing the brain template and associated segmentation maps and tissue labels has been developed, with the aim for using the constructed brain template in existing software packages used for neuroimaging analysis.

The objective of the developed brain template is also to enhance the accuracy and interpretation of structural and functional neuroimaging data acquired from Indian populations, and open new avenues for imaging based diagnosis of neurodegenerative diseases.

The differences in brain morphology across different population groups have necessitated the creation of population-specific magnetic resonance imaging (MRI) brain templates for interpretation of neuroimaging data. Variations in the neuroanatomy along with a genetically heterogeneous population, make the development of a population-specific brain

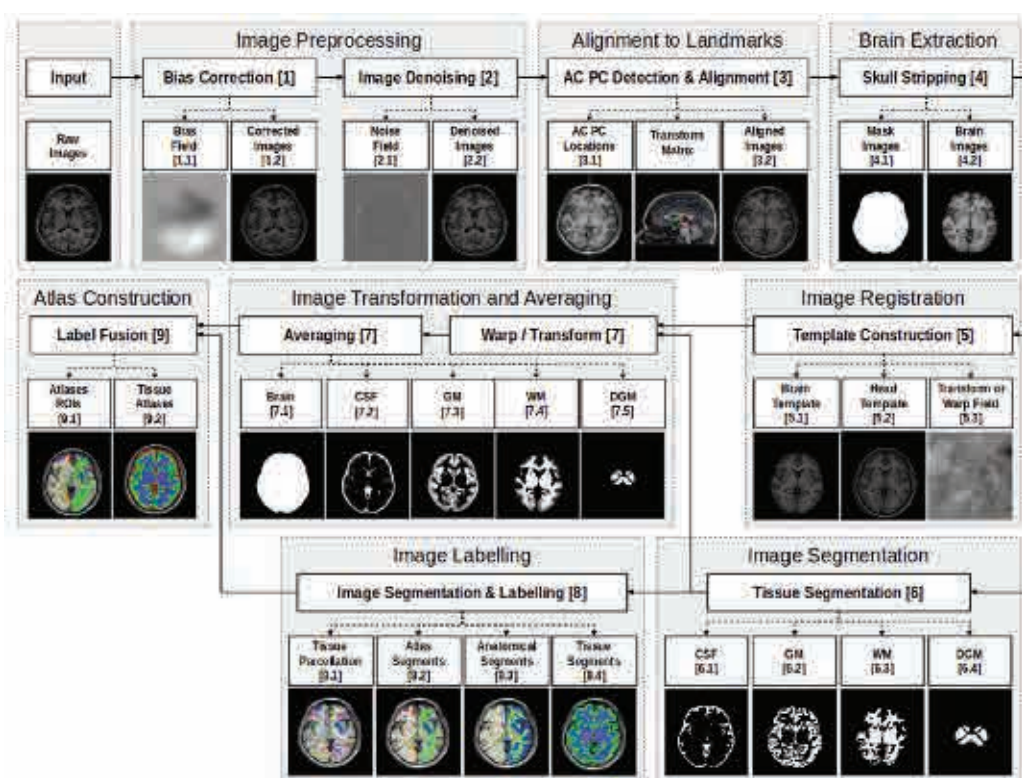
template for the Indian subcontinent imperative.

A comprehensive imaging dataset of MRI scans from normal healthy individuals hailing from different parts of India has been collected in the laboratory. An image processing pipeline for the utilisation of this dataset in template construction is being developed. This pipeline along with the developed brain template provides a sound basis for the processing of data from different MRI contrasts and the analysis of results across projects, and would be of great use for the wider neuroimaging community.

A dataset of high-resolution three-dimensional (3D) T1-weighted, T2-weighted, and fluid-attenuated inversion recovery (FLAIR) images have been acquired from a group of over a 130 volunteers for construction of T1, T2, and FLAIR templates. In addition to the MRI scans, comprehensive socio-demographic information and neuropsychological scores have also been collected from each participant.

The image processing pipeline developed for the construction of population-specific brain template is implemented through a MATLAB toolbox for simplified ease-of-use and operation. The schematic provided in Figure 3 presents the template construction pipeline along with the intermediate results obtained at each stage.

Figure 3: A schematic diagram of the steps involved in the construction of the Indian brain template, BRAHMA, along with intermediate results.



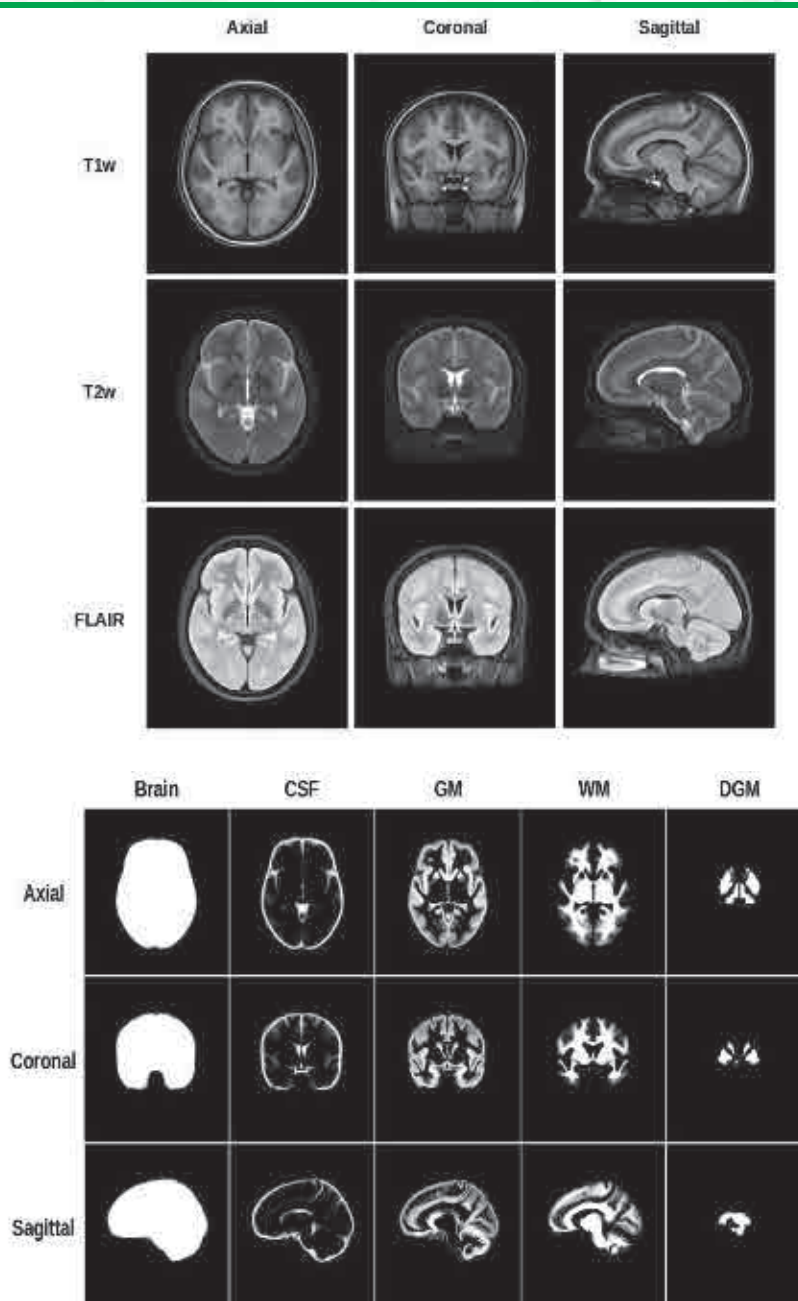
The core of the processing pipeline is formed by the steps involved in the construction of the T1-weighted brain template, with the construction of T2-weighted and FLAIR templates utilizing the results generated on T1-weighted images while skipping some intermediate steps. These intermediate steps rely on similarity in the underlying structures seen in the T1-weighted, T2-weighted and FLAIR images obtained from the individual.

A MATLAB based toolbox developed at the Neuroimaging and Neurospectroscopy Laboratory implements the processing pipeline that takes in 3-dimensional MRI images from individuals and

constructs a brain template along with a range of associated files which are listed as follows:

- I. Head Template (With Skull) and Brain Template (Without Skull)
 - i. T1-weighted
 - ii. T2-weighted
 - iii. FLAIR
- II. Brain Extraction Masks
- III. Brain Probability Map
- IV. Tissue Probability Maps
 - i. Grey Matter

Figure 4: The axial slice (80 of 160), coronal slice (120 of 240), and sagittal slice (125 of 240) of the T1, T2, and FLAIR brain templates constructed from the Indian population. The axial, coronal, and sagittal slice of the probability maps for the brain, CSF, GM, WM, and Deep GM regions of the BRAHMA template constructed from the segmentation of individual scans.



- ii. White Matter
- iii. Cerebrospinal Fluid

V. Segmentation Atlases

VI. Segmentation Priors

The constructed population-specific Indian brain template (BRAHMA) is validated through a comparison of its performance with existing templates (such as the ICBM/MNI-152) in processing structural and functional neuroimaging data. The performance of the constructed brain template is ascertained through a comparison of segmented brain volumes registered to the constructed template and existing templates. We found the segmentation volumes obtained using the IBT to be closer to the Freesurfer results in comparison to the volumes generated using the ICBM-152, with IBT based segmentation having an average absolute error of 4.32% in comparison to an error of 9.92% when using the ICBM-152 template.

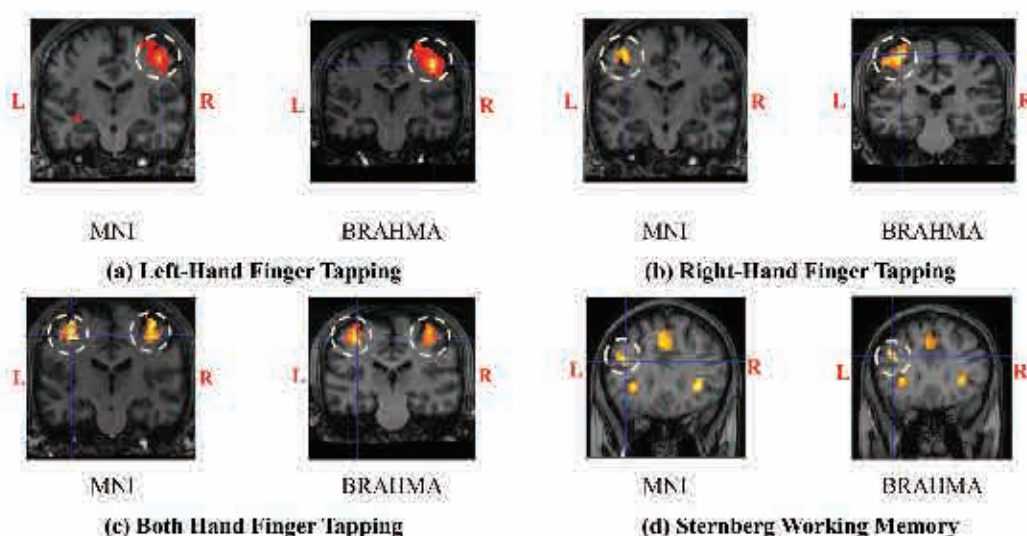
The IBT and ICBM-152 templates were utilised for processing fMRI data from two different paradigms using SPM 12. Herein, the tissue maps and segmentation atlases for the ICBM-152 template available in SPM are used for processing the functional data, followed by use of the generated IBT tissue maps and atlases. Firstly, a simple finger-tapping paradigm involving tapping of index and middle fingers with the thumb on the Left, Right, and Both hands was implemented. Thereafter, fMRI result obtained with a Sternberg working memory paradigm was tested using the two templates.

In both cases, the percentage of fMRI activation clusters lying in the relevant areas is found to increase with the use of the IBT over that obtained with the ICBM-152 template. Moreover, a greater percentage of the activation clusters is found to lie in Grey Matter (GM) regions of the brain with the use of the IBT over the ICBM-152 in fMRI processing. Figure 5 highlights the increase in activation cluster in relevant areas with the use of IBT, with Right & Left Precentral Gyrus with Left and Right Finger Tapping, and with Left Middle Frontal Gyrus for the Sternberg Working Memory.

Our results indicate that Indian brain images registered to the BRAHMA template are closer to the original scan volume than to those registered to ICBM-152. Furthermore, functional MRI data obtained using a Sternberg working memory (SWM) task was processed using the BRAHMA and ICBM-152 templates. The data processed using BRAHMA template shows a significantly higher percentage of the activation area in the region of interest, the left middle frontal gyrus.

In addition to the template images, a set of segmentation atlases with different regions of interest (ROIs) are also generated with the template images with labels for deeper brain regions such as the substantia nigra (SN) generated from the T2 and FLAIR templates. The availability of different image contrast, tissue maps, and segmentation atlases in the BRAHMA template makes it a comprehensive tool that can be readily used for multi-modal image analysis in laboratory and clinical settings.

Figure 5: The group analysed fMRI activation clusters for finger tapping paradigms [$p < 0.05$ (FWE)] with the use of the MNI and BRAHMA templates. (a) For Left Finger Tapping (b) For Right Finger Tapping (c) For Both Hands Finger Tapping (d) For Sternberg working memory paradigm.



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2. Mandal, PK, Deepika Shukla, Manjari Tripathi, Lars, Erslund (2019); Cognitive Improvement with Glutathione Supplement in Alzheimer's Disease: A Way Forward. *Journal Alzheimer's Disease*. 10.3233/JAD-181054.
3. Shukla D, Mandal, PK, Erslund L, Renate Grüner E, Tripathi M, Raghunathan P, Sharma A, Chaithya GR, Punjabi K, and Splaine C (2018). A Multi-Center Study on Human Brain Glutathione Conformation using Magnetic Resonance Spectroscopy. *Journal Alzheimer's Disease*. 10.3233/JAD-180648.
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Differential expression of CPEB alternative splice variants in mouse neuroblastoma cells, mouse brain and mouse neural stem cells



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Translational regulation of several mRNA containing poly(A) tail in their 3' UTR (untranslated region) is an important step governing stimulus dependent gene expression. These transcripts contain a conserved cytoplasmic polyadenylation element (CPE) with the consensus UUUUUUAU. Cytoplasmic Polyadenylation Element Binding Protein (CPEB), binds to CPE and regulates poly(A) addition of target transcripts for translational stimulation. CPEB not only activates translation but also keep transcripts in dormant state under basal condition (referred as translational repression). Under specific stimulus, CPEB undergoes either phosphorylation or degradation which dissociates poly(A)-specific ribonuclease and facilitate poly(A) elongation by Gld2 poly(A) polymerase and translational activation from the same shortened and dormant transcripts. This remarkable feature makes CPEBs as ideal protein in regulation of many important cellular functions like cell cycle regulation, cell signaling, cellular energetics, chromatin remodeling, synaptic plasticity for local (spatial) protein synthesis in response to external stimuli.

CPEB family has four known mammalian paralogs, such as, CPEB1, CPEB2, CPEB3, and CPEB4. These share the common functional features with unique protein specific functional variations and targets. Since CPEBs are regulators of cell cycle and cell senescence related transcripts, their participation in cancer seems highly conceivable. In addition, recent evidences point to the switch in the expression of splice variants of CPEB proteins, which are cell type specific and associated with varieties of cancer. Alternative CPEB splice variants are used in prognosis and assertion of clinical stages of cancer patients. So far, there is no report on the involvement of any CPEB in neuroblastoma, a cancerous state of neuroblast or neuroepithelial cells. Therefore, we examined the expression pattern of CPEB splice variants in a mouse neuroblastoma cell line (Neuro 2a) and compared with mouse embryonic CNS stem/progenitor cells (neurosphere) cultures and adult mouse brain employing RT-PCR (Reverse Transcription-Polymerase Chain Reaction) analysis.

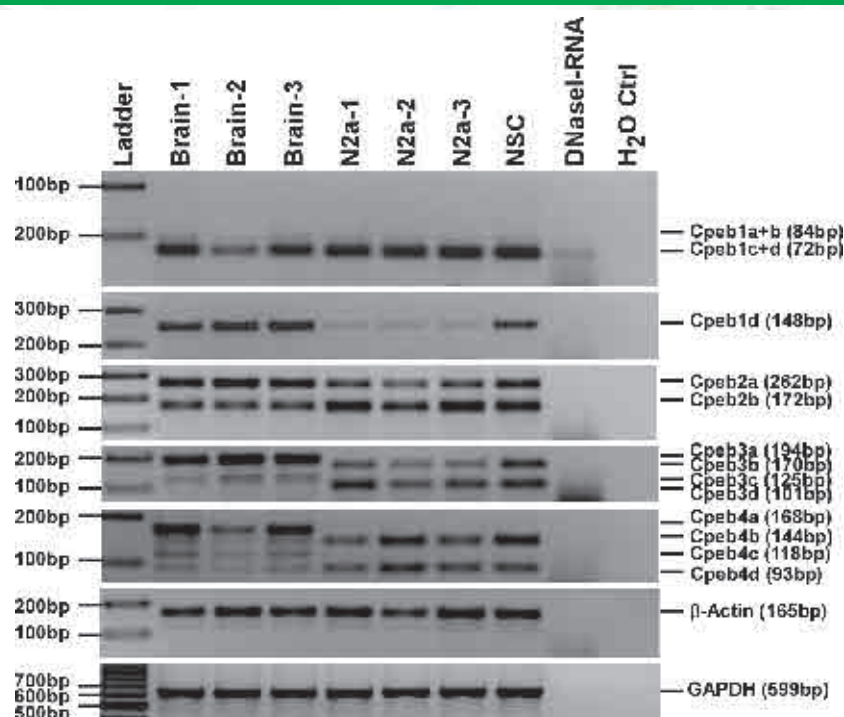
Our result shows for the first time that, Neuro 2a cells express CPEB splice variants different from both adult mouse brain and neurosphere cells (Figure-1). CPEB1-a & b isoforms are detected in adult mouse brain and neurosphere cultures but not

in Neuro 2a cells. In contrast, CPEB1-c isoforms is expressed more in neuroblastoma cells and in neurosphere culture but visibly less in adult mouse brain (Figure 1, top panel). In addition, expression of CPEB1-d isoform shows immense reduction in neuroblastoma cells than mouse brain and primary neurosphere cultures (Figure 1, second panel from top). RT-PCR analysis of CPEB2 shows CPEB2 mRNA exist mainly in two splice variants. The larger variant (CPEB2a) containing exon-3 is expressed mainly in mouse brain whereas, smaller splice variant (CPEB2b) lacking exon-3 is predominantly expressed in Neuro 2a neuroblastoma cells. However, CNS stem/progenitor cells enriched mouse neurosphere culture express both the splice variants almost at equal level (Figure 1, panel 3 from top).

RT-PCR analysis of CPEB3-4 transcripts shows, splice variants lacking B-region is highly expressed

in neuroblastoma whereas, splice variants having B-region are only present in adult mouse brain, suggesting non-B region containing splice variants are necessary for cancer cell and neural stem/progenitor cells proliferation. (Figure 1, panel 4 & 5 from top respectively). A similar role of non-B region containing splice variants of CPEB2-4 has been reported in high-grade gliomas. Furthermore, B-region containing splice variants of CPEB2-4 are be expressed in neurons suggesting their presence in adult mouse brain. However, it is not known which splice variants are expressed in glial cells of adult mouse brain. Collectively, our results for the first time suggest a link between CPEB variants expression switch in neuroblastoma cancer growth and possibly in other cancers and other brain diseases like neurodegenerative diseases, where the pathological condition might exhibit alteration of CPEB transcript variants in different cell population of diseased brain.

Figure 1: Alteration of CPEB transcript variants expression in mouse Neuro-2a, adult mouse brain and mouse neurosphere cultures by RT-PCR analysis, showing neuroblastoma cells require CPEB transcripts devoid of B-region.



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Region and cell specific expression pattern of death receptors in adult mouse brain

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P rion diseases, or transmissible spongiform encephalopathies (TSEs), are fatal neurodegenerative diseases. It comprises Kuru, Cruetzfeldt-Jakob Disease (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS) and fatal familial insomnia (FFI) in humans, scrapie in sheep, drowsy in goat, and bovine spongiform encephalopathy (BSE) in cattle. The key event in the pathogenesis 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. Spongiform degeneration, glial proliferation, and neuronal loss in brain are hallmark pathologies of prion disease. However, very little is known about the mechanism by which PrP^{Sc} mediates neurodegeneration.

There is a solo paper demonstrating the activation of caspase-8 in ME7-prion infected mice, suggesting the activation of extrinsic apoptotic pathway. Extrinsic apoptotic pathway initiates the cell death by activation of various death receptors. Except TNFR1 and FAS, nothing or very little is known about other death receptors like death receptors (DR)-3, DR5, DR6, p75NTR and RAGE in prion disease or in animal models of prion disease. In situ hybridization assay suggests both DR3 and DR6 are expressed heavily and in most parts of the normal mouse brain, and DR5 is expressed moderately in several parts of normal mouse brain. It is also unknown about the normal function of these death receptors in brain. Therefore, it is important to understand the role of all these death receptors in prion disease. Our lab at NBRC has developed prion disease in C57BL/6J and CD1 mouse brain by injecting Rocky Mountain Laboratory (RML) scrapie prion passaged in mouse. These mouse models of prion disease exhibit almost all the pathological features of prion disease. Therefore, it is now possible to study the effect of prion replication on cell death biology in vivo.

Once the neuropathology of prion diseases was established, series of western blots were performed to investigate the alteration of death receptors, such as TNFR1, FAS, p75NTR, DR3, DR5, DR6, RAGE and Caspase-8. Our results not only exhibited alteration of TNFR1 and FAS death receptors but also DR3, DR5 and p75NTR. However, it is not known, which brain region and brain cells are affected to this alteration of death receptors expression in prion disease. In order to study

this, substantial amount of time was invested to optimize the co-localization of death receptor/s with neuron, astrocytes and oligodendrocytes by immunofluorohistochemistry. To optimize immunofluorohistochemistry procedure, we utilized normal mouse brain sections and employed various types of immunofluorohistochemistry amplification technology to detect both death receptors and brain cell specific markers. In addition, techniques were optimized to detect multiple antigens using antibodies from the same species.

Results from the expression of death receptors in neuronal cells (neurofilament heavy +ve) (Figure-1A) show, TNFR1 (DR1) expression was highly observed in the olfactory lobe, frontal cortex, hippocampus, putamen and cerebellum. DR1 reactivity was also seen in neurofilament (NF) positive cells. Both NF+ve and NF-ve cells express DR1 both in cytoplasm and nucleus. DR3 expression was also observed in the olfactory lobe, frontal cortex, putamen, dentate gyrus (DG), CA3, CA2, CA1, and cerebellum. DR3 co-localised with NF+ve neuronal cells on plasma membrane, cytoplasm and rarely in the nucleus in almost all brain regions. DR5 expression was seen more in neurofilament positive cells in olfactory lobe, cortex, putamen, sub-ventricular zone, hippocampus (DG, CA3 and CA1 region) and cerebellum. DR5 expression is observed on the plasma membrane, in the cytoplasm and nucleus of NF+ve cells. Furthermore, regional expression pattern of DR5 support the ISH data reported in Allen brain atlas. DR6 expression colocalizes with NF+ve cells but appeared comparative less to other death receptors in the frontal cortex, hippocampus (DG, CA3, CA2 and CA1) and Thalamus. Neurofilament positive cells showed co-localization with DR6. DR6 expressing neurons localize DR6 mostly in nucleus and cytoplasm but less on plasma membrane. p75NTR expression was highly observed in basal forebrain and in brainstem. In addition, p75NTR, is also observed in cortex, hippocampus and cerebellum. The subcellular location of p75NTR is seen throughout the cells. RAGE expression is observed in the olfactory lobe, cortex, putamen, hippocampus (DG and few in CA3, CA2 CA1) and cerebellum. RAGE+ve/NF+ve, RAGE-ve/NF+ve and RAGE+ve/NF-ve cells are present throughout the brain suggesting, a subset of neuronal population express RAGE and non-neuronal cells also express RAGE. Furthermore, FAS expression was highly observed in the olfactory lobe, frontal cortex, putamen, DG, CA3, CA2, CA1, and cerebellum.

FAS+ve neuronal cells is seen in almost all regions of the brain. The subcellular localization of FAS is seen throughout the cell.

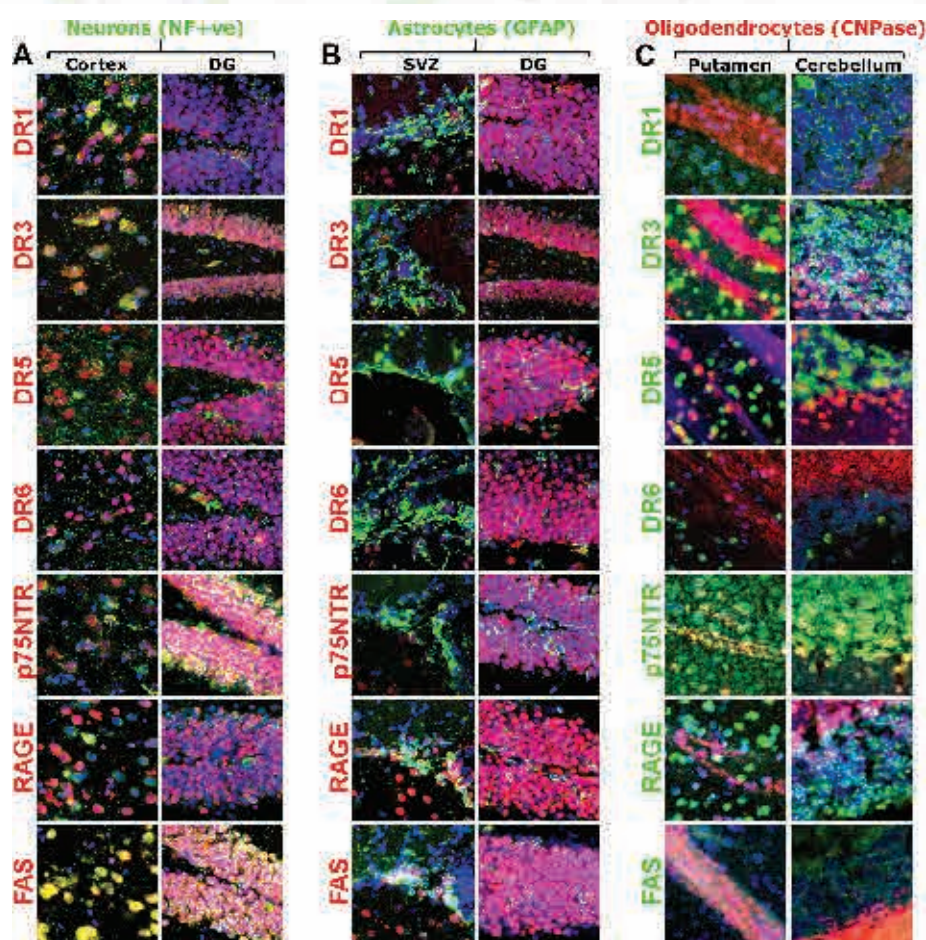
Figure-1B demonstrates the expression pattern of death receptors in astrocytes (GFAP+ve). Images from 18 different regions of mouse brain were captured for death receptor associated with astrocytes but we have presented images from sub-ventricular zone and dentate gyrus region of brain. DR1 reactivity appeared to be lesser in GFAP positive cells. However, few GFAP+ cells (astrocytes) in the olfactory lobe, cortex and cerebellum are positive for DR1 but co-localisation of DR1 and GFAP was higher in sub-ventricular zone and hippocampus region. DR3 reactivity was low in GFAP+ve cells but some regions such as sub-ventricular zone, CA1 displayed co-localization of DR3 and GFAP. DR5 reactivity is seen in few astrocytes of hippocampus, sub-ventricular zone. The subcellular localization of DR5 in astrocytes is seen on plasma membrane, in cytoplasm and nucleus. DR6 expression is observed throughout the cell. DR6 reactivity with GFAP appeared negligible in olfactory lobe, cortex, putamen and thalamus while sub-ventricular zone, hippocampus and cerebellum showed elevated reactivity with GFAP+ve astrocytes. Co-localisation of p75NTR with astrocytes is observed in few regions like sub-ventricular zone, hippocampus, CA3 and some areas of cortex. The subcellular localization of p75NTR is seen throughout the astrocytes. RAGE showed high level of reactivity with astrocytes in hippocampus, whereas, moderate expression in olfactory lobe, cortex region and very few expressions in putamen, CA1-CA3 region, thalamus and cerebellum. The subcellular location of RAGE is seen throughout the cell. FAS showed reactivity with GFAP+ve cells in olfactory lobe, hippocampus, CA1-CA3 region, thalamus and cerebellum whereas, very few expressions in cortex and putamen region. The subcellular location of FAS is seen mostly in cytoplasm and nucleus of astrocytes.

Expression pattern of death receptors in oligodendrocytes show CNPase positive cells (a marker of oligodendrocytes) co-localizes with cells in nerve fibre like structures of putamen and cerebellum (Figure-1C). Around 10 images were captured from different regions of brain with higher probability of having nerve fibre tracts but we have presented putamen and cerebellum region in this report. DR1 and CNPase dual-positive cells (oligodendrocytes) are observed in olfactory lobe,

hippocampus, putamen and corpus callosum. DR3 and CNPase dual-positive cells are observed in olfactory lobe, frontal cortex, putamen, hippocampus and cerebellum. CNPase+ve cells co-localised with DR3 in almost all brain regions. CNPase+ve cells show co-localization with DR6, p75NTR and RAGE in putamen and cerebellum. Although FAS expression is observed in the cells of olfactory lobe, frontal cortex, putamen, hippocampus and cerebellum, but less expressed in oligodendrocytes.

In conclusion, our present work not only provided ways for dual-staining of death receptors in major brain cells (neuron, astrocytes and oligodendrocytes) but also provide novel information related to death receptor expression pattern among neuronal, astrocytes and oligodendrocytes population. For example all neurons don't express DR1, DR5, p75NTR and RAGE which, itself is a new observation. Furthermore, our work in future will exhibit a detail role of death receptors in different brain cells but also their physiological role in different regions of prion diseased mouse brain.

Figure 1: Expression of death receptors in neurons (neurofilament+ve; A), Astrocytes (GFAP+ve; B) and oligodendrocytes (CNPase1+ve; C) at various mouse brain region by dual-immunofluorescence.



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Regulation of mitogen-activated protein kinase activation in Down syndrome



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My laboratory aims to understand the processes that are involved in the formation of memory, and the processes that contribute to memory impairment. The ability to make memories, and recall them when needed, is one of the fundamental features of our brain. It is evidently clear that memories are very important for our proper day-to-day functioning. Given the importance of memory in our lives, researchers have devoted considerable efforts to understand how memory is formed in the brain. Studies have shown that several molecules play critical roles in synaptic plasticity and memory. In my laboratory, we use molecular, electrophysiological and behavioral approaches to examine the processes that are involved in memory formation. This multi-disciplinary approach is likely to provide better understanding of how memory is formed than any single approach. Previously, I have presented our work on the regulation of signaling molecules by the stimuli that are relevant for synaptic plasticity and memory. In addition, I have discussed our work on long-term potentiation, a synaptic process that is widely considered to be the cellular basis of memory formation. Furthermore, I have discussed our work on memory formation using behaving animals.

With regards to the project on memory impairment, our efforts are focused on Alzheimer's disease. Memory impairment is indeed a devastating condition. It is devastating not only for the individual suffering from the disease, but also for the people around that individual. It is estimated that the prevalence of dementia is going to increase substantially in the coming years. Alzheimer's disease accounts for majority of the dementia cases. Thus, considerable efforts are directed towards understanding the processes involved in this disease. Amyloid beta, a small peptide produced by processing of a precursor protein, is considered to be the primary causative agent in Alzheimer's disease. It is clear that neuronal cell death contributes to the development of Alzheimer's disease. Hence, a lot of efforts are directed towards finding ways to protect neurons. I have previously discussed our work on identification of compounds that can protect neurons from toxicity by amyloid beta.

We have recently started our work on Down syndrome. Down syndrome is a devastating developmental disorder. It is

caused by the presence of an additional copy of chromosome 21. The individuals suffering from this disorder show several abnormal features including facial abnormalities and smaller brain size. These individuals also show cognitive deficits including dementia due to Alzheimer's disease. In order to gain insights into the pathology and processes that contribute to Down syndrome, animal models have been created. These animal models display several features that are observed in Down syndrome including deficits in memory. One animal model of Down syndrome is the trisomy 16 mouse. These animals are trisomic for chromosome 16, which is homologous to chromosome 21 in humans. Similar to human chromosome 21, mouse chromosome 16 contains gene for amyloid precursor protein. This precursor protein is processed to produce amyloid beta. The Down syndrome patients develop pathological features of Alzheimer's disease. Trisomy 16 is a good model of Down syndrome. These animals, however, die in utero. For this reason, cell lines from these animals have been created and used for studies examining the processes related to Down syndrome.

We have made considerable progress in the projects mentioned above. The research personnel who are mentioned above are working on the research projects going on in my laboratory. In addition, Mr. P. Manish has been informally working in my lab. This year, I will describe our research progress in the project that deals with the devastating condition, Down syndrome.

For the experiments relevant to Down syndrome, we used a cell line derived from the hippocampus of trisomy mouse fetus. Another cell line derived from the hippocampus of normal mouse fetus was used as a control. Both of these cell lines were obtained from Prof. Pablo Caviedes, University of Chile, Santiago, Chile. The cells were used under agreement with University of South Florida Board of Trustees, a public body corporate on behalf of the University of Chile (collectively referred to as USF/UC). We first examined the activation status of two members of mitogen-activated protein kinase (MAPK) family. The results showed that both members of the MAPK family were hyper-activated in the trisomy cells. We next asked whether the hyper-activation of the MAPK family members under study could be reduced. For this purpose, we examined the effects of a small peptide on hyper-activation of the MAPKs. The results showed that the peptide reduced activation status of the MAPKs. Given the importance of MAPKs in development, synaptic plasticity and memory, we next studied activity-dependent regulation of one of the MAPKs. We found impaired activity-dependent activation of the MAPK under study. Importantly, the peptide restored activity-dependent activation of the MAPK. These results show that basal level as well activity-dependent MAPK signaling is dysregulated in the trisomy cells. Importantly, the peptide helps in reducing the aberrant signaling. The results raise the possibility that the peptide may provide beneficial effects in Down syndrome.

Presentations

1. Shiv kumar Sharma, "India|EMBO Symposium- From synapse to memory: RNA based regulatory mechanisms", October 2018, National Brain Research Centre, Manesar, Haryana.
2. Shiv kumar Sharma, "Neuroplasticity: Role in Learning, Memory and Spinal Injury", February 2019, Bhaskaracharya College of Applied Sciences, Delhi.
3. Deepali Singh, Apurva Agrawal and Shiv K Sharma. Poster presentation in "India | EMBO Symposium- From synapse to memory: RNA based regulatory mechanisms", October 2018, National Brain Research Centre, Manesar, Haryana.
4. Kautuk Kamboj and Shiv K. Sharma. Poster presentation in "India |EMBO Symposium- From synapse to memory: RNA based regulatory mechanisms", October 2018, National Brain Research Centre, Manesar, Haryana.

Funding

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Cognition in Corvids



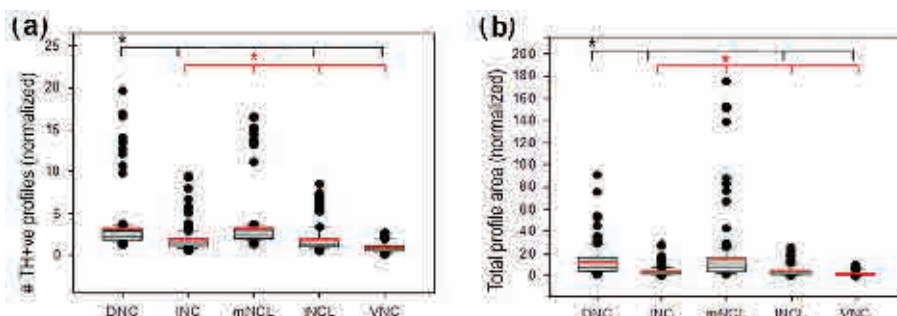
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Amongst avians, members of the genus Corvidae which include crows, rooks, starlings and jays provide interesting model systems to study advanced cognitive abilities such as attention, learning and memory, tool use and theory of mind. Some of these birds such as New Caledonian crows are known to perform tasks requiring cognition at par with non-human primates. We have been studying the dopaminergic system of indigenous species of corvids (house crows, *Corvus splendens*, as a first step to understanding the crow brain, since it is known to play an important role in cognition. We decided to study the distribution of tyrosine hydroxylase (TH), the rate-limiting enzyme in the synthesis of catecholamines as well as the expression of DARPP-32 (dopamine and cAMP-regulated phosphoprotein), which is expressed in dopaminergic neurons. As in other avian species, the house crow NCL could be differentiated from the surrounding pallial regions based on a larger number of TH-positive “baskets” of fibers around neurons in this region and greater intensity of DARPP-32 staining in the neuropil in this region. These qualitative findings were confirmed by quantification to demonstrate that the number of TH-labeled profiles (baskets and fibers) were significantly higher in mNCL (medial NCL), followed by DNC (dorsomedial nidopallium caudale) and were the least in VNC (ventral nidopallium caudale). Further, the density of TH-positive fibers was the highest in DNC across all divisions of NC, followed by mNCL (**Fig. 1**). We also found that the neuropil in NCL was significantly more immunoreactive for DARPP-32 than in surrounding regions.

Figure 1: (a) The number of TH-labeled profiles (baskets and fibers) were significantly higher in mNCL (medial part of NCL), followed by DNC (dorsomedial NC) and were the least in VNC (ventral NC). (b) The density of TH-positive fibers was the highest in DNC across all divisions of NC, followed by mNCL. Mean values, red lines; medians, black lines; significant differences indicated by black asterisks for DNC and red asterisks for mNCL.



Besides providing a method to delineate the NCL region, we also studied the expression of TH and DARPP-32 in “limbic” regions of the crow brain including the hippocampus, septum, and extended amygdala. We found that a number of TH-positive fibers and TH-labeled baskets were present specifically in the dentate gyrus and Hi2 (hippocampal area 2), in the medial part of the crow hippocampal complex. The dorsolateral corticoid area (the avian analogue of the cingulate gyrus) was also labelled with TH-positive fibers and DARPP-32-positive neurons.

The septal nuclei are a part of the limbic system which are involved in food-caching, courtship and aggression in birds. We found that both medial and lateral septal nuclei (SM and SL, respectively) were labeled with TH. Whereas DARPP-32-positive neurons were present throughout SM in house crows, TH-positive fibers were present mainly in its dorsal subdivision, suggesting that its ventral part may be functionally different. We also found that despite high levels of immunoreactivity for TH in SL, it was devoid of DARPP-32 label. These findings suggest that its functions may be modulated by catecholamines other than dopamine (**Fig. 2**).

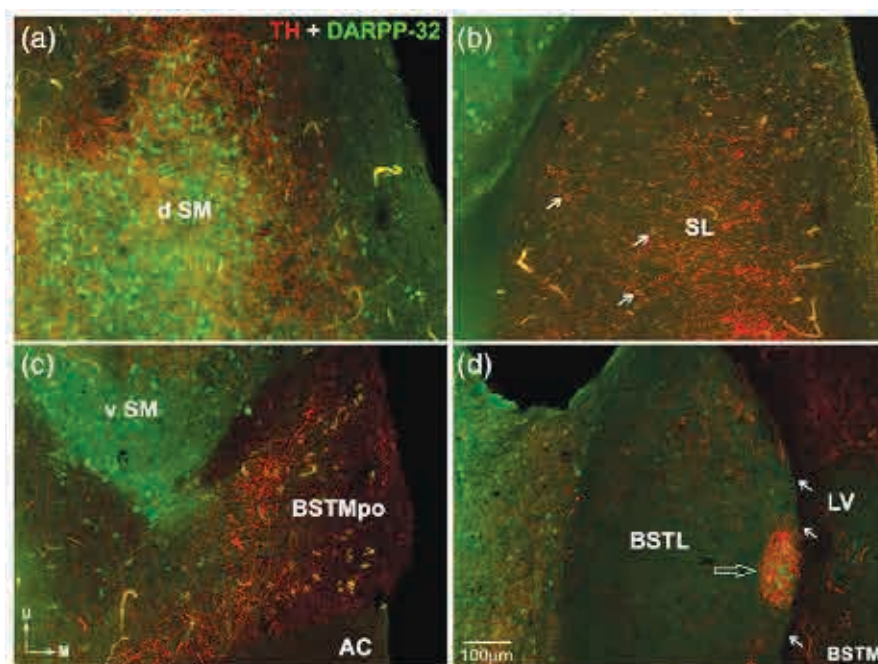
Another important limbic region is the extended amygdala in birds, which is divided into medial and central subdivisions. The medial extended amygdala (EAmE) consists of the medial amygdala (MeA) and medial bed nucleus of the stria terminalis (BSTM) and is involved in socialization related to affiliation, aggression, defensive and sexual behaviours in mammals. Since MeA in house crows was heavily labelled with DARPP-32 and also enriched in TH-positive fibers, our results showed that it was most likely modulated by dopamine. In contrast, BSTM (also part of EAmE) was likely modulated by catecholamines other than dopamine, since this region was rich in TH but not in DARPP-32 labelled neurons (**Fig. 2**). The central extended amygdala (EAcE) includes the central amygdala, the lateral bed nucleus of the stria terminalis (BSTL), the intercalated amygdalar cells and striatal capsule and is important for mediating reward, responses to fear or anxiety-causing stimuli and for modulating emotions that are important for social behaviour. However, except for a small part of intermediate BSTL, this region was not highly immunoreactive for either TH or DARPP-32. Taken together, these results suggest that house crows can be used as good model system to explore structure-function relationships of neural circuits for diverse behaviours.

Figure 1: Immunoreactivity for TH (red) and DARPP-32 (green) in the septum and extended amygdala labeled. **(a)** TH-positive axons are interspersed between DARPP-32-labeled neurons in the dorsal part of SM (d SM).

(b) Whereas SL is also innervated by a number of TH-positive fibers, DARPP-32-positive neurons in this region are sparse (arrows).

(c) In contrast, the ventral part of SM (v SM) contains a number of DARPP-32-positive neurons but few TH-positive fibers. In the same image, only TH-positive fibers can be seen in the underlying BSTMpo (preoptic division of medial part of bed nucleus of stria terminalis) which lies dorsal to the anterior commissure (AC).

(d) Scattered faintly stained DARPP-32-labeled neurons are present in dorsal BSTL (lateral part of bed nucleus of stria terminalis) which lies medial to the lateral ventricle (indicated by arrows). These neurons are surrounded by TH-positive terminals. Interestingly, a small group of neurons intensely stained for DARPP-32 and interspersed with TH-labeled fibers is present at the medial border of BSTL. However, its intermediate and ventral parts are devoid of DARPP-32 labeled neurons. Scale bar, 100µm.



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Presentations

1. S. Kumar, AN. Mohapatra, R. Narayan, UA. Singh, S. Sharma, V. Arora, N. Kambi, A. Datta, H. Sharma, T. Velpandian, S. Iyengar: The μ -Opioid Receptor System Modulates the Motivation to Sing. *International Congress of Neuroethology*, Organized by the International Society for Neuroethology; Brisbane, Australia, July 15th – 20th, 2018.
2. S. Iyengar: Neuroplasticity in the Developing Human Auditory Cortex. *IBRO-APRC School*, Organized by the Dept of Physiology, AIIMS, New Delhi, October 21st - November 4th, 2018.

3. S. Iyengar: Development of Axonal Connections in the Human Auditory Cortex. *3rd IBRO/APRC Chandigarh Neuroscience School*, Panjab University, Chandigarh, December 1st, 2018.
4. S. Iyengar: Three-dimensional reconstruction of Neurons. *3rd Asian and African Stereology Congress*, Maulana Azad Medical College, New Delhi, December 18th - 20th, 2018.

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DST Grant entitled "Effects of the δ -Opioid Receptor System on Singing and Song Learning in Zebra Finches", (EMR/2015/001422) received September 2016.

Collaborator

Prof Anton J Reiner, Department of Anatomy and Neurobiology, University of Tennessee Health Science Center and Department of Ophthalmology, University of Tennessee, Memphis, Tennessee, United States

Student's Award

Sandeep Kumar: Awarded Developing Neuroethology Awards by International Society for Neuroethology to attend the meeting

Alok Nath Mohapatra: Awarded IBRO-PERC, The Brain Prize and FENS stipend to attend the conference

Long non-coding RNA - mediated control of synaptic plasticity



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Functions of neuronal networks while performing complex cognitive functions, such as memory formation are governed by dynamic modification of synapses. Synapses undergo reversible modifications (or synaptic plasticity) through spatio-temporal control of gene expression. Thus, identifying the regulatory control point of gene expression that contributes to dynamic changes at the synapses is crucial for our understanding how impairment of these regulatory control leads to deficits in cognitive function.

Among various regulatory switches, we have focused on non-coding RNA-mediated control of gene expression at the synapses. We have particularly investigated the role of long non-coding RNAs (lncRNAs are >200 nucleotide transcripts) – an emerging class of regulatory switches implicated in the neuronal development and function. These non-coding transcripts are expressed in cell type specific manner and also function as the reversible regulators of gene expression. Therefore, we hypothesized that these non-coding transcripts are in a pivotal position to fine-tune functions of subset of synapses through the reversible control of gene expression. Of particular interest, we have focused on translational control that has been shown to make de novo changes at the synapses linked to synaptic plasticity and memory formation.

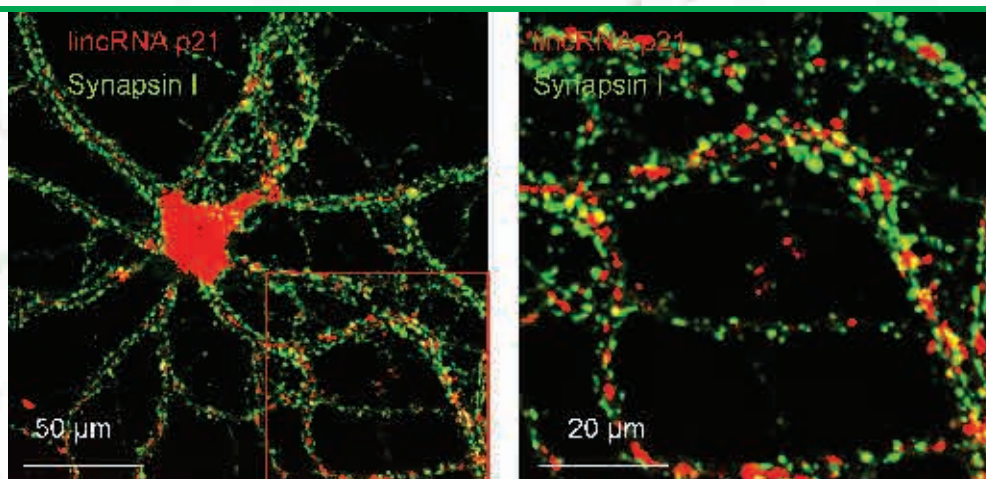
Interestingly, many of these long non-coding RNAs (lncRNAs) have been shown to express within a particular neuroanatomical region, such as hippocampus, cerebral cortex and cerebellum. Moreover, the recent study also reported sub-cellular localization of lncRNAs, such as in the neuronal dendrite. These non-coding transcripts are originated from the 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.

To explore the function of lncRNAs in synapse development and synaptic plasticity, we have employed a genome wide screen of lncRNAs expressed in hippocampal synapses. Our bioinformatics analysis identified ~151 lncRNAs (annotated lncRNAs present in both GENCODE and Ensembl data bases) that are enriched at the synaptic compartment. Among this

subset of lncRNAs, we have verified synaptic enrichment of ~10 lncRNAs by in situ hybridization. Following characterization of lncRNAs by in situ hybridization, we have focused onto synapse-localized lncRNA – linc-p21 that has been previously implicated in modulation of cell cycle.

These observations indicate that lincRNAp21 expression is dynamically regulated by neuronal activity and potentially modulate synaptic function through a spatio-temporal control of gene expression. In addition, our data also revealed that the linc-p21 is associated with the actively translating transcripts indicating its role in protein

Figure 1: Synaptic localization of linc-p21. The non-coding transcript was visualized by fluorescent in situ hybridization and synapses were visualized by Synapsin I immunostaining.



To evaluate its role in synaptic function, we performed loss of function experiment by RNAi and measured the synaptic response by whole cell patch clamp recordings. We observed that loss of lincRNAp21 function leads to significant reduction in miniature Excitatory Post Synaptic Current (mEPSC) amplitude without any alteration of frequency. This observations indicates involvement of linc-p21 in modulation of gene expression at the post synaptic compartment. Furthermore, brief stimulation (10 minute) of hippocampal neuron with glutamate and contextual fear conditioning of mice showed significant increase of its expression.

synthesis. We have investigating activity-dependent regulation of dendritic protein synthesis by lincRNAp21 using fluorescent detection of newly synthesized proteins or Fluorescent Non-Canonical Amino acid Tagging (FUNCAT). Our FUNCAT data showed significant reduction of activity-dependent dendritic protein synthesis. Furthermore, we have identified an RNA binding protein, that acts as a translation repressor, dynamically bind to linc-p21 upon neuronal activity.

We have shown that neuronal activity sequesters this RNA binding protein from translating transcripts

Figure 1: Role of linc-p21 in synaptic transmission. Whole cell-patch clamp recording was performed to measure miniature synaptic response (mEPSCs). Top trace represents synaptic activity of control neuron and bottom trace represents synaptic activity of neurons with significant knock down of linc-p21.

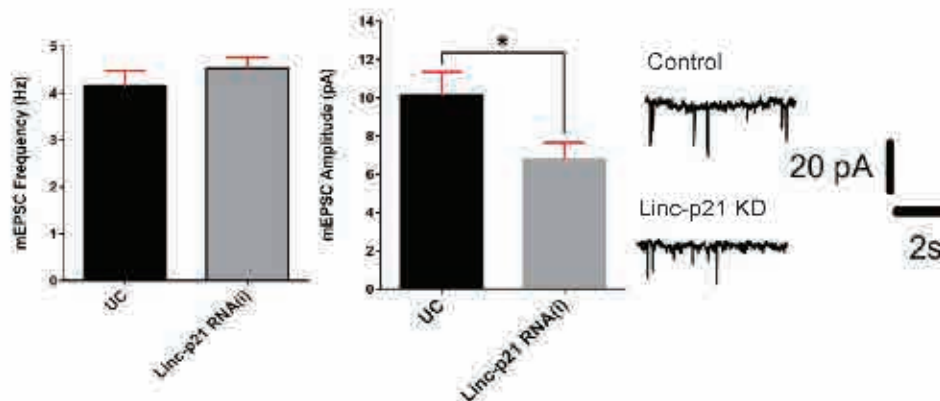
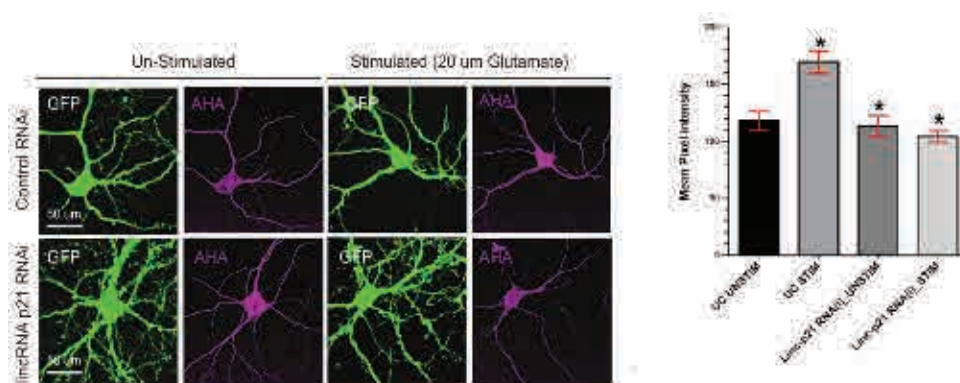


Figure 3: linc-p21 – mediated control of dendritic protein synthesis in response to synaptic activity. New synthesis of proteins from subset of dendritic transcripts following lentivirus –mediated linc-p21 knockdown was measured by FUNCAT method. A) Photo-micrograph showing newly synthesized proteins in neurons expressing shRNAs against linc-p21 or universal control shRNA without any effective knockdown. B) Quantitation of newly synthesized proteins in response to neuronal activity.



by enhancing its binding with the linc-p21 and subsequently enhance protein synthesis from a subset of dendritic transcripts. We aim to explore the importance of lincRNAp21 –mediated control of protein synthesis in hippocampus-dependent control of contextual fear memory and spatial memory formation.

Furthermore, experiments are in progress to

envisage how lncRNAs are regulated during aging. One of the key symptomatic responses of aging is sleep loss that has already been shown to impact memory formation. We will investigate how sleep deprivation will impact non-coding RNA-mediated protein synthesis in hippocampus and how altered translation contributes to memory deficits during aging.

Presentations

1. Banerjee, S. Decoding the function of long non-coding RNAs: Regulation of synaptic plasticity and memory by long non-coding RNA –mediated protein synthesis. 7th Asian forum of chromosome and chromatin biology. Jawaharlal Nehru Center for Advanced Scientific Research, Bangalore. November 2018.
2. Banerjee, S. Make in locally: Localized control of memory formation by selective miRNA decay at the synapse. Annual Meeting of Indian Academy of Neurosciences, Varanasi. October 2018
3. Banerjee S. Long non-coding RNAs at the synapse: Role in protein synthesis and memory formation. India | EMBO symposium, Manesar. October 2018.
4. Banerjee S. Non-coding RNAs in synapse development and plasticity. IBRO-APRC School, All India Institute of Medical Sciences, New Delhi. October 2018.
5. Samaddar, S., Balakumar, S., Jha, S. and Banerjee, S. Selective degradation of miRNAs at the synapse regulates dendritic protein synthesis. India | EMBO symposium, NBRC, Manesar. October 2018.
6. Balakumar, S., Samaddar, S., and Banerjee, S. Coordinated control of protein synthesis and degradation regulates homeostatic synaptic balance. India | EMBO symposium, NBRC, Manesar. October 2018.
7. Samaddar, S., Mukherjee, U., Balakumar, S., Palakodeti, D., and Banerjee, S. Activity-dependent control of dendritic protein synthesis by lincRNA-p21 and its implication in synaptic plasticity. India | EMBO symposium, NBRC, Manesar. October 2018.

Scientific Reports

8. Palanisamy, P., Mukherjee, U., Samaddar, S., Abel, T., and Banerjee, S. Sleep deprivation influences poly-ribosome association of mRNAs in mice hippocampus. India | EMBO symposium, NBRC, Manesar. October 2018.
9. Balakumar S. and Banerjee S. Molecular mechanism of feeding behaviour by high fat diet induced adult neurogenesis. EMBO workshop, NCBS, Bangalore, February 2019.

Symposium Organized

India | EMBO symposium on “From Synapse to memory : RNA based regulatory mechanisms”.

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1. Genome Engineering Grant, DBT
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3. NBRC Core Fund

Collaborators

Prof. Ted Abel, University of Iowa, USA
Dr Dasradhi Palakodeti, inStem, Bangalore
Dr James Chelliah, JNCASR, Bangalore

Student's Award

Sarbani Samaddar: Zeeshan Khan Memorial Award for the best poster presentation in Bangalore Microscopy Course 2018

Degree Awarded

Ms. Tapasya Pal (M.Sc.)

Illuminating the molecular details of neurodevelopment using human neural stem cells derived from induced pluripotent stem cells as model



Yogita K. Adlakha

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Karnika Gupta

Human brain development is a beautifully orchestrated event which involves a complex series of dynamic and adaptive processes that function in a highly constrained and genetically organized context. The utmost requirement for cognition and behavior is appropriate differentiation, maturation and function of neuronal networks as interruption of these processes can lead to neurodevelopmental disorders such as Autism, Schizophrenia, Huntington's disease or Intellectual disability. Consequently, the processes underlying neural development, including neural stem cell self-renewal and differentiation, fate specification, neuronal migration, maturation, and integration have been well established at transcriptional level. However, recently, post-transcriptional control of gene expression has emerged as an additional, and equally important, regulatory layer. In particular, microRNAs (miRNAs), an abundant class of small non-coding RNAs, have been shown to regulate neuronal differentiation, maturation and their function by modulating gene expression via mRNA translation inhibition. Although the transcriptional machinery during neural development has been studied extensively, understanding the miRNA mediated regulation of neurodevelopment remains as uncharted territory.

Several studies have revealed essential roles of miRNAs in brain development and function using animal models with deficiency in miRNA biogenesis pathway genes. For example, loss of Dicer caused impeded brain development and the abnormal phenotype of brain in zebrafish. These effects could be rescued by ectopic expression of miR-430. Further, mice deficient in Ago2 (miRNA maturation pathway gene) displayed defects in neural tube closure and mis-patterning of the forebrain. Participation of Dicer in neural stem cell proliferation and differentiation was revealed by the conditional knockout of Dicer in mouse neural stem cells which led to smaller cortex. With progress in miRNA research, it is evident that miRNAs are frequently dysregulated in neurodevelopmental disorders, suggesting a role for miRNAs in the etiology and/or maintenance of neurological disease states.

MiR-137 is one of the brain enriched miRNA that plays an important regulatory role in brain function. This miRNA

is associated with the regulation of adult neurogenesis, dendritic development and neuronal maturation as well as control of the dynamics between neural stem cell proliferation and differentiation during neural development. Emerging evidence implicates dysregulation of miR-137 with the etiology of neurodevelopmental disorders including Schizophrenia, Autism, Huntington's disease, Rett syndrome or Intellectual disability. Dysfunction of miR-137 has also been shown to contribute to neuroblastoma and glioblastoma multiforme. Owing to its significant functions in several neuropsychiatric/neurocognitive/oncological disorders, consequently, this miRNA may have great potential as a biomarker and in treatment of human diseases where dysregulation of this gene or its pathways are involved.

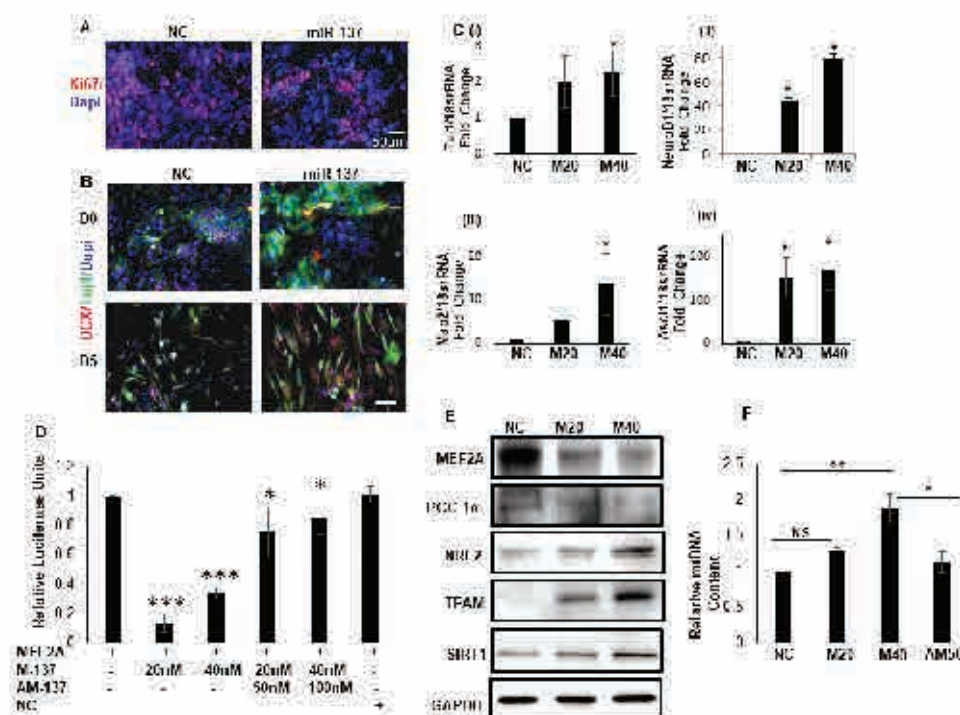
Several reports using mouse model establish that expression of miR-137 gets upregulated significantly when ESCs and neural stem cells differentiate into neuronal lineage. However, underlying molecular role of miR-137 in neural development remains elusive.

We have made some novel discoveries in understanding the molecular mechanism underlying miR-137 mediated effects as to how it regulates the dynamics of proliferation and differentiation of neural stem cells. To pursue this goal, we generated human neural stem cells (NSCs) from iPSCs which were derived from peripheral blood of healthy subjects in-house. The NSCs stained positive for neural stem cell markers i.e. Nestin and Sox2. These NSCs were differentiated into neurons using neuronal differentiation media until day 21 and neuronal population was stained positive for neuronal markers including Tuj1 and Map2. We observed enhanced expression of miR-137 in iPSCs and mature neurons as compared to NSCs. Being enriched in brain, we questioned the role of miR-137 on proliferation and differentiation of NSCs, therefore, we modulated miR-137 levels in NSCs using overexpression and knock down experiments and immuno-stained the cells with Ki67 antibody for proliferation assay. Overexpression of miR-137 demonstrated significant reduction in Ki67 positive NSCs whereas knock down of miR-137 caused increase in Ki67 positive NSCs after 24 hrs of transfection. Next, we assessed the effect of miR-137 on

NSC differentiation and observed significant increase in the transcript levels of neuronal markers i.e. Tuj1, Map2, NeuroD1 and Ascl1. Thus, miR-137 induces neuronal differentiation and reduces proliferation of NSCs. Since, neurons are metabolically highly active cells which depend primarily on mitochondrial oxidative phosphorylation for energy, hence, based on our results, we hypothesize that this miRNA might be modulating mitochondrial dynamics to achieve the necessary oxidative capacity for enhanced neuronal differentiation. Mitochondrial dynamics include the interplay between biogenesis, fusion and fission. Because miRNAs function by reducing the expression of their target gene, we analyzed the targets of miR-137 using TargetScan which illuminated Myocyte Enhancer Factor 2A (MEF2A) as target gene of miR-137. MEF2A is an upstream regulator of PGC-1 α which is the central mediator and controller of mitochondrial biogenesis. We cloned the 3'UTR of MEF2A in reporter vector and luciferase assay confirmed that miR-137 binds to 3'UTR of this gene and reduces its expression. Earlier, we observed that miR-137 decreases the transcript levels of MEF2A and PGC-1 α which is the transcriptional controller of mitochondrial biogenesis. Simultaneously, miR-137 increased NRF2, TFAM and SIRT1 transcript levels in a dose dependent manner. Thereafter, we found that miR-137 reduces the protein levels of MEF2A and PGC-1 α while increases the protein levels of NRF2, TFAM and SIRT1. Further, miR-137 increased the mitochondrial DNA content, thus confirms that miR-137 increases mitochondrial biogenesis irrespective of PGC-1 α status in NSCs. Ectopic expression of miR-137 also increases mRNA and protein levels of mitochondrial fusion gene MFN1, suggesting that miR-137 also increases mitochondrial fusion. Thus, these experiments confirm that miR-137 alters mitochondrial dynamics to achieve enhanced neuronal differentiation of NSCs. Further experiments are in process to reveal the underlying mechanisms.

These findings would help in better understanding of the molecular mechanisms underlying miR-137 mediated cellular effects and illuminate its therapeutic potential for treatment of neurodevelopmental disorders.

Figure 1: MiR-137 enhances differentiation of NSCs into neurons by altering mitochondrial dynamics. **A.** Proliferation assay with Ki67 antibody suggests miR-137 reduces proliferation of NSCs. **B.** MiR-137 enhances differentiation of NSCs into neurons as expression of Tuj1 and punctate expression of DCX increased in presence of miR-137 at day 5. **C.** Quantitative RT-PCR suggests that neuronal markers increased upon differentiation of NSCs into neurons in presence of miR-137. **D.** Luciferase activity of MEF2A cloned reporter decreased in presence of miR-137. Anti-miR-137 relieved the repression caused by miR-137. **E.** Western blots shows modulation of expression of different mitochondrial biogenesis genes after transfection with miR-137. **F.** Relative mitochondrial content was increased in presence of miR-137 and anti-miR-137 reverses the effect of miR-137.



Publications

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1. Yogita K Adlakha (2019). "Investigation of regulation of Neural Development by MiRNA" at Postdoctoral Research Conclave, Jamia Hamdard, Hamdard Nagar, New Delhi, 12th April, 2019.
2. Reshma Bhagat, Bharat Prajapati, Sonia Narwal, Yogita K Adlakha and Pankaj Seth. "Zika Virus E protein alters proliferation and differentiation of human fetal neural stem cells". Society for

Neuroscience meeting, San Diego, CA, USA, Nov, 2018.

3. Asha S C, Pankaj Seth and Yogita K. Adlakha. "MiRNA-137 regulates neural stem cell fate determination by modulating mitochondrial dynamics". Indian Academy of Neuroscience meeting, BHU, Varanasi, India, Oct, 2018.

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Collaborators

P. Seth and A. Basu, NBRC, Manesar, India.

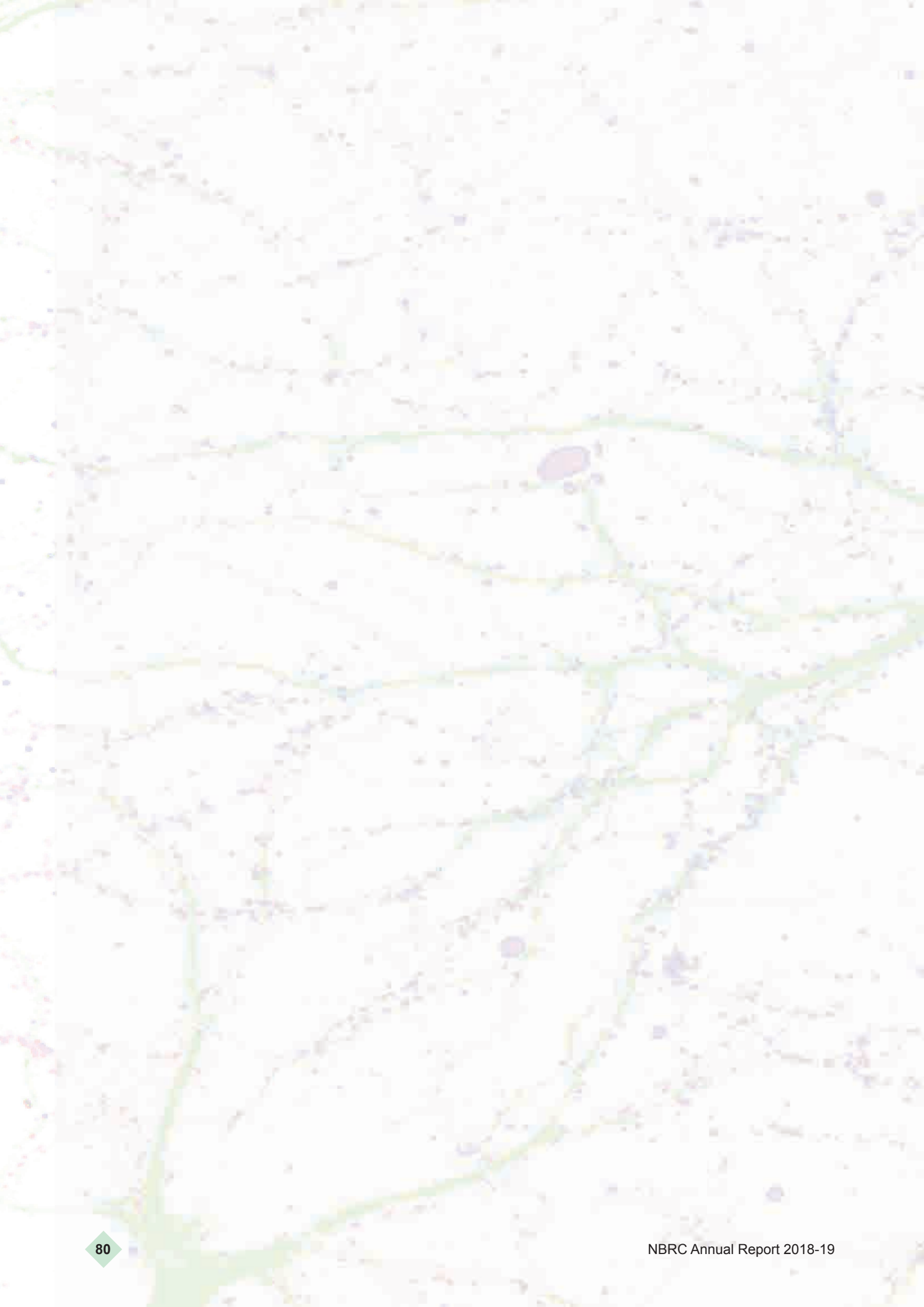
A. Swaroop, National Institute of Health, Bethesda, USA.

Award

NASI Young Scientist Platinum Jubilee Award 2018

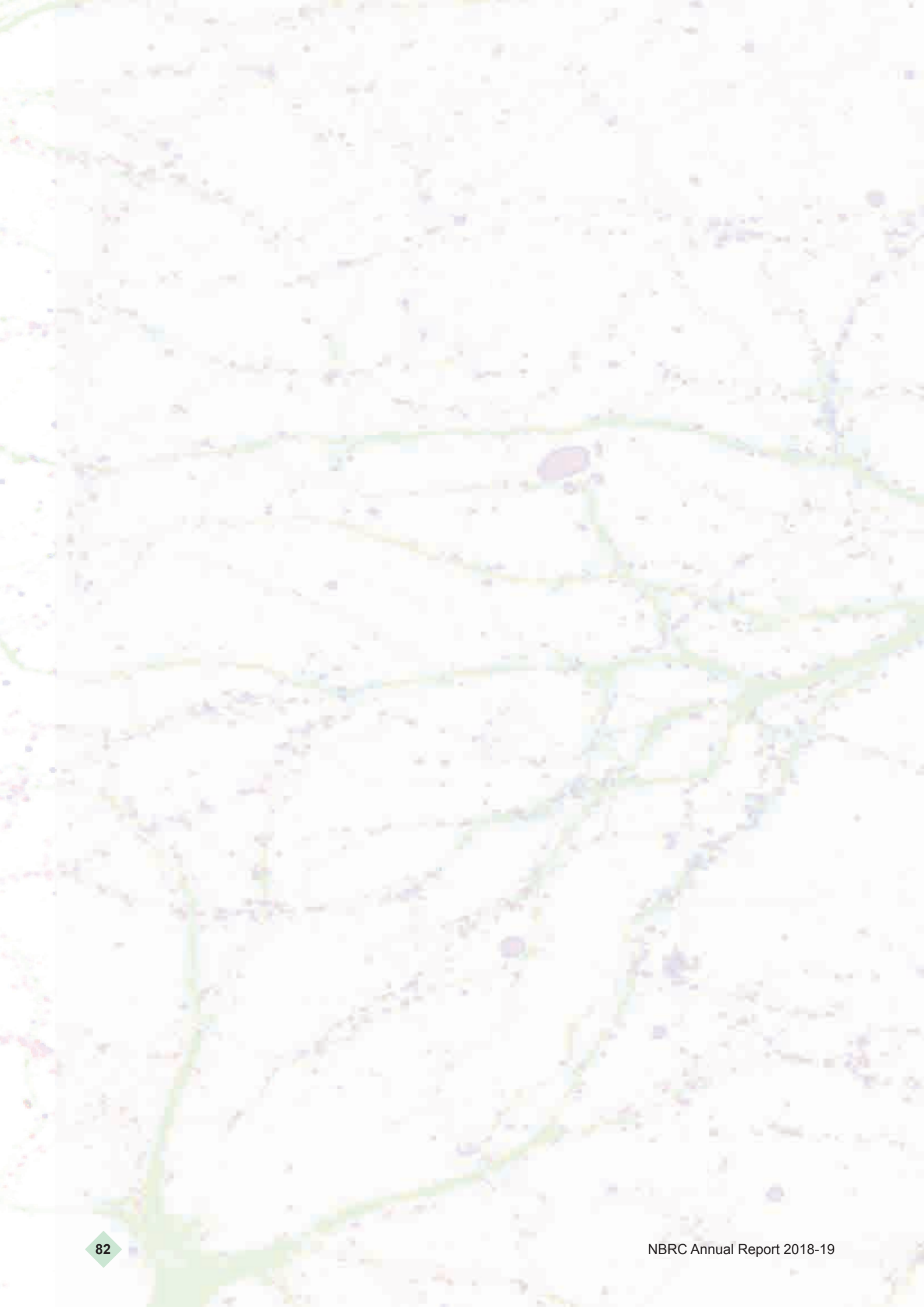
Student's Award

Asha SC : Travel Award from INA to attend INA conference 2018



The background of the page is a map of the United States, showing state boundaries and major roads. A large, solid green rectangle is centered on the page, partially obscuring the map. The text "Major Research Programs" is written in a bold, green, sans-serif font within this rectangle.

Major Research Programs



Dementia Project

Coordinator

Neeraj Jain, Director, NBRC

Co-coordinators

Shiv Sharma, NBRC

NK Arora, INCLEN Trust International

Investigators

Community Sites

NK Arora, INCLEN, New Delhi

Masaraf Hussain, NEIGRIHMS, Shillong

Suvrana Alladi, NIMHANS, Bengaluru

Hospital Sites

Atanu Biswas, BIN, Kolkata

Manjari Tripathi, AIIMS, New Delhi

PS Mathuranath, NIMHANS, Bengaluru

Ramshekhar Menon, SCTIMST,

Thiruvananthapuram

Basic Sciences

Shiv Sharma, NBRC

Pravat Mandal, NBRC

Dipanjan Roy, NBRC

Madhusudan Das, University of Calcutta

Dementia is a progressive disorder that is characterized by impairment in memory and other cognitive abilities. Alzheimer's disease, an age-associated memory impairment condition, constitutes majority of the dementia cases. Other dementia conditions include vascular dementia, fronto-temporal dementia, dementia with Lewy body etc. In coming years, the prevalence of dementia is likely to increase more in developing countries than in the developed countries. With increase in the aging population in India, the number of individuals suffering from dementia is expected to rise. This will place an increasing burden on families, caregivers and the healthcare system. Thus it is imperative to understand the disease process and identify potential window of therapeutic interventions for this devastating condition.

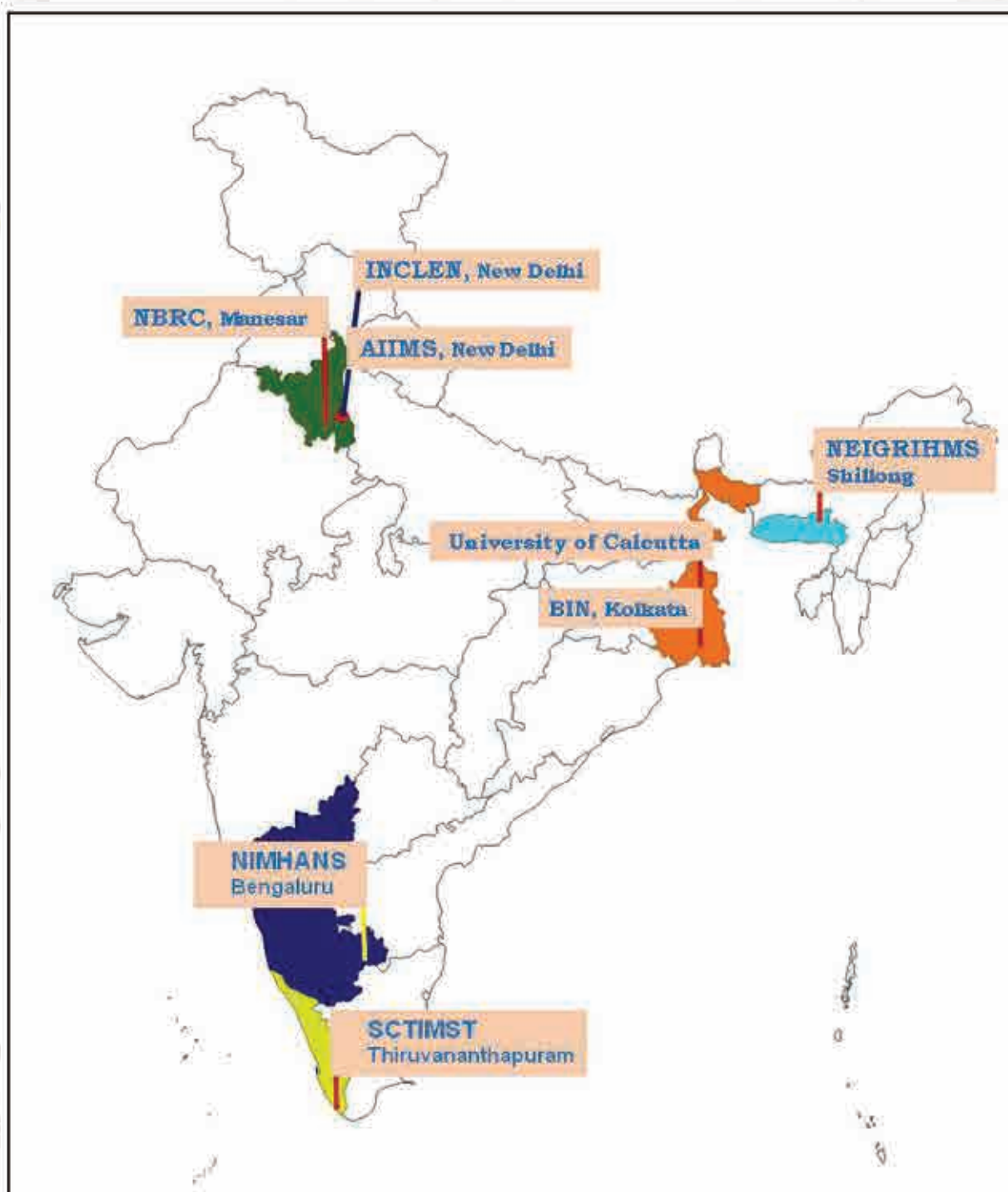
Recognizing the urgent need to understand different aspects of dementia, Department of Biotechnology has funded a research programme on dementia, Dementia Science Programme. This programme is aimed at collecting reliable data regarding prevalence, incidence, biomarkers and risk and protective factors. This is a multi-centric Programme involving researchers and clinicians from across the country. The programme aims to establish long-term population-based cohorts as well as hospital-based cohorts. These cohorts will be followed up to gain better understanding of the disease. The participating centres will use robust and uniform criteria that have been internationally accepted and validated in the Indian context for diagnosis, classification and other aspects of the disease.

The 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. The participating sites are shown on the map of India also.

- ❖ 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, Kolkata.

In order to develop uniform criteria for diagnosis, classification and other aspects of the disease that will be used by all the centres participating in the Dementia Science Programme, several discussion meetings were held with the investigators. The outcome of these meetings paved the way for development of the final Protocol.



Flagship Project: Comparative mapping of common mental disorders (CMD) over lifespan

Coordinator

Neeraj Jain, Director, NBRC

Investigators at NBRC

Arpan Banerjee

Dipanjan Roy

Shiv Kumar Sharma

Anindya Ghosh Roy

Mental health is a ticking time bomb in India. Worldwide research currently shows that mental health needs to be understood at multiple-scales of organization from molecular processes at the microscopic scale to macroscopic brain network dynamics. Furthermore, age related changes in the neurophysiological processes need to be factored in the science of mental health. Multi-scale processing of this magnitude requires insights that are shaped by genetic & cellular diversities, therefore research published elsewhere is not translatable to current Indian context. India, with a rapid increase in life expectancy in recent times is faced with an increasingly ageing population that is subjected to life in an environment drastically different from the traditional family set-up. There is an urgent need to track mental health parameters during lifespan ageing and develop interventions targeted to focus groups based on age, gender, geographical locations, lifestyle factors and cultural similarity. The end-goal of any such endeavour will be a predictive model that can incorporate the aforementioned factors and evaluate the need of intervention in a specific individual. Keeping this in mind NBRC has proposed 'Comparative mapping of common mental disorders (CMD) over lifespan' to DBT as its major ambitious flagship program.

Our proposed flagship program will involve building a "big" dataset and perform Artificial Intelligence – Machine Learning (AIML) driven analysis to address questions on 1) Lifespan ageing with construction of a normative cohort of healthy normal individuals with age range 18-80 and 2) a mental health disorder cohort affected by "common mental disorders (CMD)" comprising of Anxiety, Depression, Obsessive – compulsive disorder and Post-traumatic stress disorders (PTSD) in age ranges 18-50 and 65-80 in Phase 1. The following metrics will be tested for identifying consistent patterns that delimit the onset and developmental changes a) structural and functional network reorganization at the macroscopic level in resting brain b) inter-subject correlation among individuals during tasks c) whole-genome sequences to understand the role of gene regulation and shared pathways. These metrics will be cross-validated on a large cohort study of lifespan aging (1000 human participants) and mental health that needs to run for more than a decade. The key deliverables after completion of Phase 1 will be

- 1) Creation and dissemination of a public database that will include normative data from 200 healthy volunteers and 200 mental health affected patients. This database will include the aforementioned parameters in a deidentifiable format on which various artificial intelligence-machine learning algorithms can be implemented to discover discernible features.
- 2) Several imaging derived phenotypes (IDP) that include information about specific brain structures and their connections and correlated functional brain networks will be investigated and preliminary patterns will be identified by the end of Phase 1.

- 3) Creation of a bio-bank in Phase 1 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.

We envision this project to be a defining component of NBRC's commitment toward Department of Biotechnology's Sustainable development goals as well as the National Mission of Swasth Bharat.

Centre of Excellence (CoE) for Epilepsy Research

Investigators from AIIMS

Prof. P Sarat Chandra
Prof. Manjari Tripathi
Dr. Jyotirmoy Banerjee

Investigator from NBRC

Professor Neeraj Jain, Director

Investigator from ACBR

Dr. Aparna Dixit

Centre of Excellence for Epilepsy (COE) is collaborative project between National Brain Research Centre (NBRC) and All India Institute of Medical Sciences (AIIMS) established under the aegis of Department of Biotechnology (Government of India). This one of the few centres 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 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. These well-established epileptogenic zones surgically removed during epilepsy surgery serves as ideal model to study the mechanism of epileptogenesis in patients with DRE. Quantification of abnormalities in these tissues is performed by RNAseq/microarray analysis for gene expression abnormalities and cellular electrophysiological experiments to study the changes in the synaptic transmission and the shift of electrical properties of the neurons. Correlation of the radiological and electrophysiological parameters with the molecular/cellular properties of neurons to study epileptogenesis is the hallmark of this multi-disciplinary centre.

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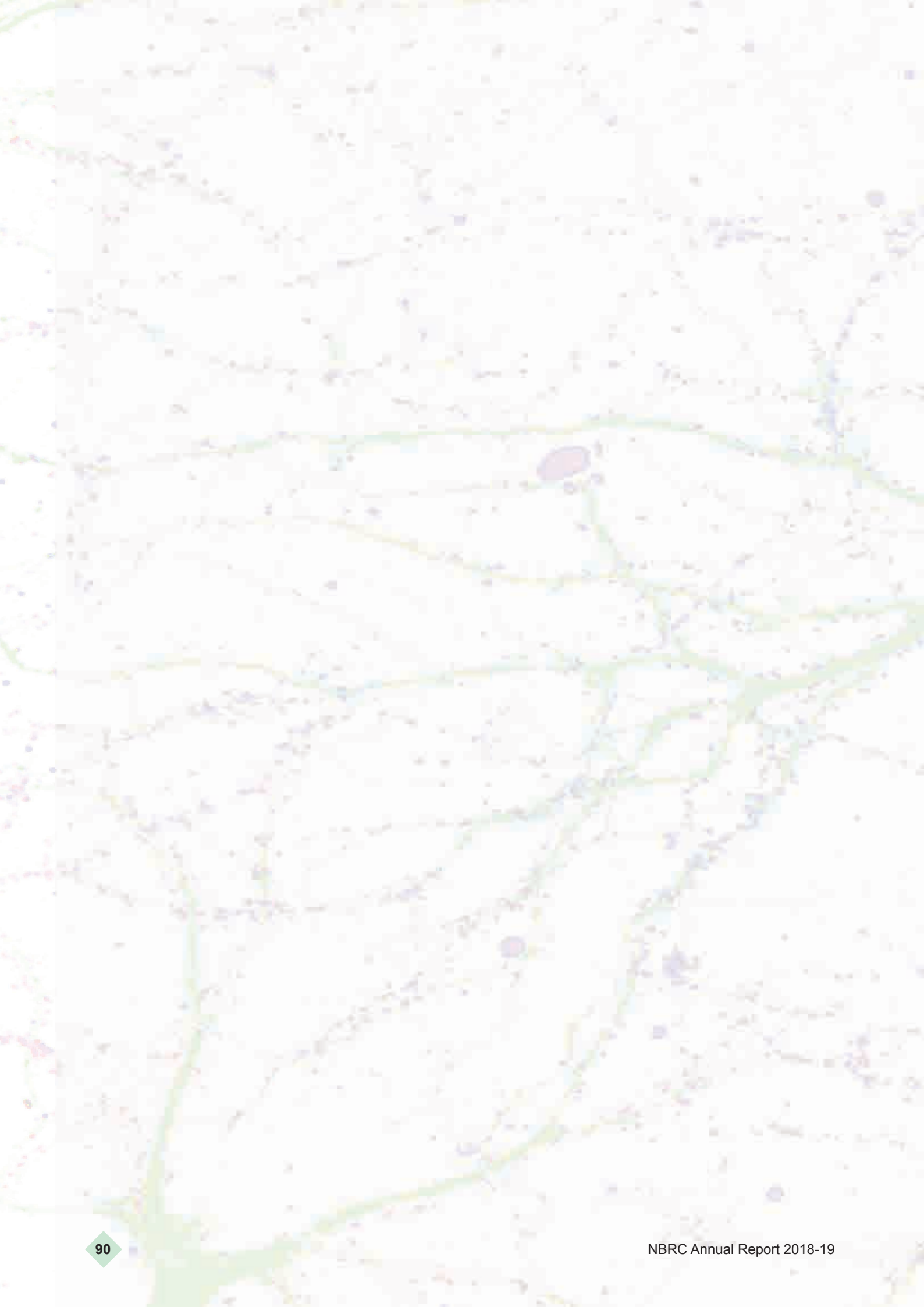
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Funding

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Publications, Patents & Presentations



Publications

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Presentations

1. Anindya Ghosh Roy: "Regulation of functional restoration after neuronal injury by miRNA pathway" in 'India | EMBO Symposium- From synapses to memory: RNA based regulatory mechanisms' 2018 between Oct 15-18, 2018 at NBRC
2. Anindya Ghosh Roy: "Femtosecond laser and live imaging meet the power of genetics in transparent nematode: Intrinsic control of axon and dendrite regeneration" 2nd RCB Bioimaging School, RCB Faridabad, 2019
3. Anindya Ghosh Roy: "C. elegans model and nerve regeneration study" at Dr. B.R. Ambedkar Center for Biomedical Research (ACBR) at University of Delhi during Summer Undergraduate Research Program (SURP) on 8th July 2019.
4. A Basu (2019) Emerging and Re-emerging Viral Diseases: Host Immunity and Therapeutic Interventions. Sonarpur Mahavidyalaya, Rajpur, South 24 Paraganas, 26th March.
5. A Basu (2019) Emerging and Re-emerging Viral Diseases: Host Immunity and Therapeutic Interventions. Lady Brabourne College, Kolkata, 25th March. (Special lecture under DBT star college program)
6. A Basu (2019) Modulation of Interleukin-1b-HSP60 axis: A potential therapeutic target to ameliorate neuro-inflammatory conditions. 32nd Annual Meeting of SNCI, Dept. of Pharmacology, JSS College of Pharmacy, Mysuru, 14th-16th March.
7. A Basu (2019) Drug repositioning/ repurposing: Promising strategy to develop therapy against viral infections. National Conference on Frontiers in Health Sciences, Institute of Medical Sciences, BHU, Varanasi, 11th-12th March.
8. A Basu (2019) Neuro-inflammation as a mechanism and therapeutic target. IBRO-APRC associate school, IGNTU, Amarkantak, 3-9th March.
9. A Basu (2019) Drug repositioning/ repurposing: Future drug developmental strategy against viral infections. CCMB, Hyderabad, 27th February.
10. A Basu (2018) Drug repositioning/ repurposing: Promising strategy to develop therapy against viral infections. Centre for Liver Research, School of Digestive and Liver Diseases, IPGME&R, Kolkata, 24th December.
11. A Basu (2018) Drug repositioning/ repurposing: Future drug development strategy against viral infections. C-CAMP, Bangalore Life Science Cluster, 14th December.
12. A Basu (2018) Modulation of Neural Stem/ Progenitor Cell response following Japanese Encephalitis Virus infection. CIDR-IIISC, 13th December.
13. A Basu (2018) Host MicroRNA: An important modulator of antiviral immunity in Japanese Encephalitis virus infection. International symposium on Infectious Diseases, RCB & Jamia Hamdard, 12-14th, November.
14. A Basu (2018) Host pathogen interaction in Japanese Encephalitis Virus infection: from bench to bedside. Frontiers in Biotechnology, Dept. of Biotechnology, St Xavier's college, Kolkata, 12th October.
15. A Basu (2018) Neurotropic Viral Infection Perturbs brain endogenous repairing mechanism. Modern Trends in Microbiology, Dept. of Microbiology, St Xavier's College, Kolkata, 11th October.

16. A Basu (2018) Interleukin -1b, a cytokine at the crossroads between infection and inflammation. International conference on Immunology, Dept. of Biotechnology, and School of Bioengineering, SRM University, Chennai, 26th -28th September.
17. A Basu (2018) Host pathogen interaction in Japanese Encephalitis Virus infection: from bench to bedside, International Society for Neuroimmunology (ISNI)-2018, Workshop 6, CNS infection, Brisbane, Australia, 27th-31st August.
18. A Basu (2018) Neural stem/progenitor cell response to Japanese encephalitis virus infection. NCR-Biotech cluster meeting, RCB, Faridabad, 29th June.
19. A Basu (2018) Host pathogen interaction in Japanese Encephalitis Virus infection: from pathogenesis to therapy. Summer Undergraduate Research Programme- 2018, ACBR, Delhi University, 1st June.
20. Arpan Banerjee Oct 2018: at Montreal Neurological Institute, Montreal Canada in Auditory processing and plasticity meeting organized by IDRC project from Israel-Canada research consortium (Invited talk)
21. Arpan Banerjee Oct 2018: at Penn State Medical Centre, Hershey Park, PA, USA, Departmental seminar in radiology (Invited talk)
22. Arpan Banerjee July 2018: at Indian Institute of Science, Bangalore, International Conference on Signal processing and communications (IEEE). (Invited tutorial)
23. Dipanjan Roy Brain Awareness week DBT STAR college program DDU Delhi "Brain Science foundations and fundamentals" March 29, 2019
24. Dipanjan Roy Invited talk at AIIMS New Delhi 10th of March one day workshop on mathematics in biomedicine.
25. Dipanjan Roy Invited plenary speaker "Aging related resting and sensory motor brain signals and their reorganization" The Department of Biomedical Engineering, SSNCE Chennai Fifth International conference on bio signals, Images and Instrumentation (ICBSII 2019)
26. Dipanjan Roy Invited speaker Brain Modes 2018 Computational modelling of Neurodegeneration and aging brain Dec 2-4, 2018 Havana, Cuba.
27. Dipanjan Roy 5th IISC Bangalore Cognition workshop Center for Neuroscience module Attention at multiple scales June 17-29, 2018.
28. Ellora Sen. Immune surveillance in cancer: Therapeutic implications. Gurudas College, Kolkata, Oct 2018.
29. Ellora Sen. TLR4 affects p53-JMJD2A-BRG1 transcriptional network to drive SOCS1 expression. Genome Architecture and Cell Fate Regulation, University of Hyderabad, Hyderabad, December 2018
30. Ellora Sen. Knowledge in Science: A philosophical Quest. DST Sponsored Faculty Development Program on Entrepreneurship Science, Technology & Management for Society, Deshbandhu College, Delhi, December, 2018
31. Ellora Sen. Bridging metabolism and inflammation in glioma: Role of genetic and epigenetic landscape. IBRO, Associate School of Neuroscience on Advances in Molecular Neurobiology Research, IGNTU, Amarkantak, March 2019.
32. Ellora Sen Metabolic modulation of immune regulatory responses in glioma. Society for Neurochemistry (India) SNCI & International Conference on Neurochemistry and Neuropharmacology: From bench to bedside Mysuru, March 2019.
33. Ellora Sen. Re-inventing oneself: Science as a Career. Department of Biochemistry, Daulatram College, Delhi, March 2019.
34. Hisham Mohammed and Neeraj Jain. 'Cortical, callosal and thalamic inputs to the neck and jaw motor representations in rats'; Neuroscience 2018, Annual Meeting of the Society for Neuroscience, USA. Nov 3-7, 2019; San Diego, USA.
35. John Thomas, Dixit Sharma, Sounak Mohanta and Neeraj Jain, 'Resting state

Publications, Patents & Presentations

- somatosensory functional map is composed of multiple networks in macaque monkeys and humans'; 36th Annual Meeting of the Indian Academy of Neuroscience, BHU Varanasi, October 29-31, 2018.
36. Neeraj Jain 'Deafferentation Induced Plasticity of the Adult Somatosensory System', Gordon Research Conference (GRC): Neuroplasticity of Sensory Systems, Hong Kong, 3-8th June, 2018.
 37. Neeraj Jain 'Somatosensory information processing and spinal cord injuries', 36th Annual Meeting of the Indian Academy of Neuroscience, BHU Varanasi, October 29-31, 2018.
 38. Neeraj Jain 'Recent advances in technology to map brain plasticity after spinal injury', at 'Disability Inclusiveness' on Disability Day, Indian Spinal Injuries Centre, New Delhi, December 3, 2018.
 39. Neeraj Jain 'Neuroplasticity and Spinal Cord Injuries' at Bhaskaracharya College of Applied Sciences, New Delhi, February 20, 2019.
 40. Neeraj Jain 'Spinal Cord Injuries and Brain Plasticity' at 5th South Asian Biotechnology Conference – 2019, South Asian University, New Delhi, March 14, 2019.
 41. Pankaj Seth (Invited Speaker) Use of human neural stem cells as a model to understand neurodegenerative disorders, during Luxembourg-German-India Alliance on Neurodegenerative diseases and Therapeutics (Lux-GIANT) under the Indo-German initiative, Tubingen, Germany, September 14-15, 2018.
 42. Pankaj Seth (Invited Speaker) Zika viral protein alters human neural stem cell properties by altering miRNA circuitry, at the Section of Infections of the Nervous System (SINS), National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, USA, April 30, 2018.
 43. Pankaj Seth (Invited Speaker) Insights into HIV and Zika virus induced alterations in human progenitor cells, at the University of Miami, Miller School of Medicine, Don Soffer Clinical Research Centre, Miami, USA, April 24, 2018.
 44. Pankaj Seth (Invited Speaker) What we know and what we need to know about HIV-1 neuropathogenesis, at the RUSH University Medical Centre, Department of Neurological Sciences, Chicago, USA, April 17, 2018.
 45. Pankaj Seth (Invited Speaker) Friends turn foe during HIV-1 neuropathogenesis, at the Alcohol Research Center, University of Chicago at Illinois, Chicago, USA, April 16, 2018.
 46. Pankaj Seth (Invited Speaker) Zika Virus affects human neural stem cells, at a Two day symposia "Exploring Mind, Brain and Behaviour" at Amity University NOIDA, India, during March 28-29, 2019.
 47. Pankaj Seth (Invited Speaker) Understanding the virus induced neurodegeneration with human neural stem cells, national symposium on stem cell technologies in neurodegenerative diseases, at Era University, Lucknow, March 9, 2019.
 48. Pankaj Seth (Invited Speaker) Cellular and molecular mechanism of zika virus induced alterations in properties of human neural stem cells, at 3rd Symposium of Virus Diseases organized by Centre for Infectious Disease Research, Indian Institute of Science, Bangalore, India, December 13, 2018.
 49. Pankaj Seth (Guest Faculty) Molecular mechanism of HIV-1 and Zika virus neuropathogenesis, IBRO-APRC school at Panjab University, Chandigarh, India, December 1, 2018.
 50. Pankaj Seth (Symposium Speaker) Molecular mechanisms of Zika virus E protein induced alterations in human neural stem cells, 36th Annual meeting of Indian Academy of Neurosciences (IAN-2018), at Banaras Hindu University, Varanasi, India, October 29-31, 2018.
 51. Pankaj Seth (Guest Lecture) What we know and What we need to know about Zika Virus, at Era University, Lucknow, India, October 18, 2018.

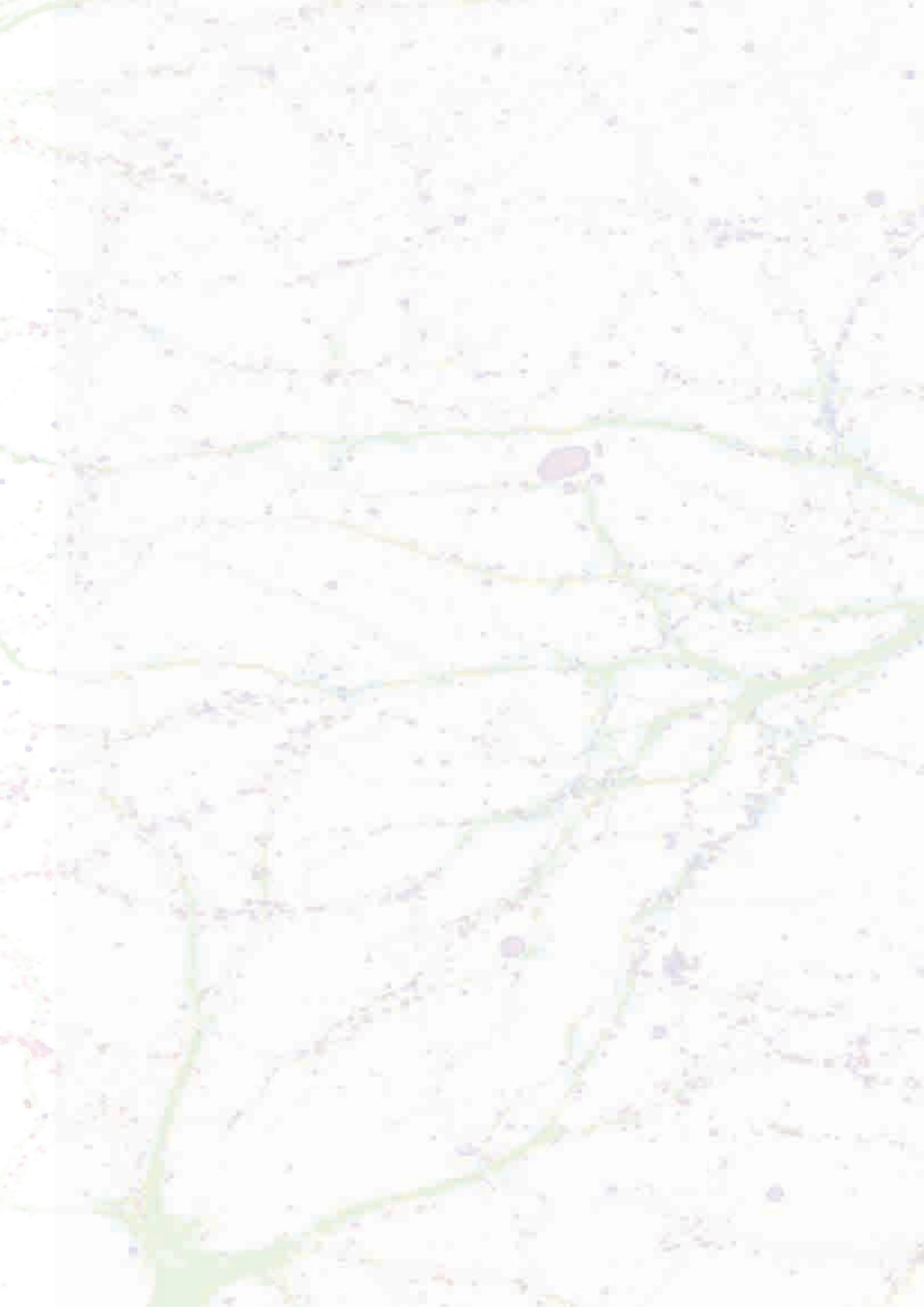
52. Pankaj Seth (Invited Speaker) How do viruses affect human brain – some insights, Amity University – Haryana, Manesar, July 26, 2018.
53. Pankaj Seth (Guest Faculty) Do's and Don'ts of Grant Writing, in Mini-Symposium on Professional Skills in Scientific Research under aegis of UGC-UPE Focused Area-II at Interdisciplinary School of Life Sciences, Institute of Science, Banaras Hindu University, Varanasi, India July 14, 2018.
54. Pankaj Seth (Invited Speaker) Human neural stem cells as in vitro model to study neurodegenerative diseases, School of Regenerative Medicine, Manipal University, Bengaluru, India, May 10, 2018.
55. Shiv Kumar Sharma Delivered a lecture in a seminar on "Neuroplasticity: Role in Learning, Memory and Spinal Injury", February 2019, Bhaskaracharya College of Applied Sciences, Delhi.
56. Deepali Singh, Apurva Agrawal and Shiv K Sharma. Poster presentation in "India | EMBO Symposium- From synapse to memory: RNA based regulatory mechanisms", October 2018, National Brain Research Centre, Manesar, Haryana.
57. Kautuk Kamboj and Shiv K. Sharma. Poster presentation in "India | EMBO Symposium- From synapse to memory: RNA based regulatory mechanisms", October 2018, National Brain Research Centre, Manesar, Haryana.
58. Shiv Kumar Sharma Delivered a lecture in "India|EMBO Symposium- From synapse to memory: RNA based regulatory mechanisms", October 2018, National Brain Research Centre, Manesar, Haryana.
59. S. Kumar, AN. Mohapatra, R. Narayan, UA. Singh, S. Sharma, V. Arora, N. Kambi, A.Datta, H. Sharma, T. Velpandian, S. Iyengar: The μ -Opioid Receptor System Modulates the Motivation to Sing. International Congress of Neuroethology, Organized by the International Society for Neuroethology; Brisbane, Australia, July 15th – 20th, 2018.
60. S. Iyengar: Neuroplasticity in the Developing Human Auditory Cortex. IBRO-APRC School, Organized by the Dept of Physiology, AIIMS, New Delhi, October 21st - November 4th, 2018.
61. S. Iyengar: Development of Axonal Connections in the Human Auditory Cortex . 3rd IBRO/APRC Chandigarh Neuroscience School, Panjab University, Chandigarh, December 1st, 2018.
62. S. Iyengar: Three-dimensional reconstruction of Neurons. 3rd Asian and African Stereology Congress, Maulana Azad Medical College, New Delhi, December 18th - 20th, 2018.
63. Banerjee S. Decoding the function of long non-coding RNAs: Regulation of synaptic plasticity and memory by long non-coding RNA –mediated protein synthesis. 7th Asian forum of chromosome and chromatin biology. Jawaharlal Nehru Center for Advanced Scientific Research, Bangalore. November 2018.
64. Banerjee S. Make in locally: Localized control of memory formation by selective miRNA decay at the synapse. Annual Meeting of Indian Academy of Neurosciences, Varanasi. October 2018
65. Banerjee S. Long non-coding RNAs at the synapse: Role in protein synthesis and memory formation. India | EMBO symposium, Manesar. October 2018.
66. Banerjee S. Non-coding RNAs in synapse development and plasticity. IBRO-APRC School, All India Institute of Medical Sciences, New Delhi. October 2018.
67. Samaddar, S., Balakumar, S., Jha, S. and Banerjee S. Selective degradation of miRNAs at the synapse regulates dendritic protein synthesis. India | EMBO symposium, NBRC, Manesar. October 2018.
68. Balakumar, S., Samaddar, S., and Banerjee S. Coordinated control of protein synthesis and degradation regulates homeostatic synaptic balance. India | EMBO symposium, NBRC, Manesar. October 2018.

Publications, Patents & Presentations

69. Samaddar, S., Mukherjee, U., Balakumar, S., Palakodeti, D., and Banerjee S. Activity-dependent control of dendritic protein synthesis by lincRNA-p21 and its implication in synaptic plasticity. India | EMBO symposium, NBRC, Manesar. October 2018.
70. Palanisamy, P., Mukherjee, U., Samaddar, S., Abel, T., and Banerjee S. Sleep deprivation influences poly-ribosome association of mRNAs in mice hippocampus. India | EMBO symposium, NBRC, Manesar. October 2018.
71. Balakumar S. and Banerjee S. Molecular mechanism of feeding behaviour by high fat diet induced adult neurogenesis. EMBO workshop, NCBS, Bangalore, February 2019.
72. Yogita K Adlakha (2019). "Investigation of regulation of Neural Development by MiRNA" at Postdoctoral Research Conclave, Jamia Hamdard, Hamdard Nagar, New Delhi, 12th April, 2019.
73. Reshma Bhagat, Bharat Prajapati, Sonia Narwal, Yogita K Adlakha and Pankaj Seth. "Zika Virus E protein alters proliferation and differentiation of human fetal neural stem cells". Society for Neuroscience meeting, San Diego, CA, USA, Nov, 2018.
74. Asha S C, Pankaj Seth and Yogita K. Adlakha. "MiRNA-137 regulates neural stem cell fate determination by modulating mitochondrial dynamics". Indian Academy of Neuroscience meeting, BHU, Varanasi, India, Oct, 2018.



Externally Funded Research Projects



Externally Funded Research Projects

Name of P.I.	Project S.No.	Name of Project	Funding Agency	Date of Sanction	Amount Received for 2018-19 (Rs. Lakh)	Sanctioned Cost (Rs. Lakh)	Date of Completion	Sanction Order No.
Amit Naskar	1	Post Doctoral Fellowship	SERB	09.12.2016	0.00	19.20	08.12.2018	PDF/ 2016/ 000378
Anindya Ghosh Roy	2	Wellcom Trust/DBT Indian Alliance	D.B.T.	01.12.2013	3.41	321.93	30.11.2019	IA/I/13/1/ 5000874
Anirban Basu	3	Deciphering NTIVIRAL Properties of Statins against Japanese Encephalitis Virus Infections	D.B.T.	26.12.2018	21.84	30.00	25.12.2020	BT/PR27796/ MED/29/ 1301/2018
	4	MicroRNA mediated regulation of neural stem/progenitor cell fate in neurotropic flaviviral infection	D.B.T.	29.12.2017	0.00	77.07	28.12.2020	No..BT/ PR22341/ MED/ 122/55/2016
	5	Understanding the therapeutic role of adult stem cell derived exosome in combating virus induced neurodegenerative disease	D.B.T.	20.03.2018	0.00	25.50	19.03.2021	No.BT/ PR15984/ MED/ 31/325/2015
	6	MicroRNAs as a potential therapeutic target in Neuro tropic viral infection (Tata Innovation fellowship)	D.B.T.	01.05.2015	8.89	45.00	31.03.2020	No.BT/HRD/ 35/01/02/ 2014
	7	Elucidating the role of long non coding RNAs (lncRNAs) in neuronal cell death during Japanese Encephalitis (JE)	D.B.T.	05.03.2019	14.20	19.00	04.03.2022	BT/PR26590/ MED/122/ 133/2017

Externally Funded Research Projects

Name of P.I.	Project S.No.	Name of Project	Funding Agency	Date of Sanction	Amount Received for 2018-19 (Rs. Lakh)	Sanctioned Cost (Rs. Lakh)	Date of Completion	Sanction Order No.
Arpan Banarjee	8	Early Diagnosis of Structural and Functional Decline in Brain Circuits Stemming from Traumatic Injuries in Professional Athletes Playing Contact Sports	MYAS	14.02.2019	40.00	100.00	13.02.2022	K-15015/42/2018/SP-V
Dipanjana Roy	9	Oscillatory network dynamics in perceptual learning	D.S.T.	23.08.2017	0.00	50.55	22.08.2020	No.SR/CSRI/21/2016(G)
	10	Role of default mode brain network in normal cognitive function	D.B.T.	26.05.2016	10.06	88.00	26.05.2021	No. BT/RLF/Re-entry/07/2014
	11	Dementia Science Program- Tissue MRI Studies	D.B.T.	18.12.2017	0.00	35.41	17.12.2020	No. BT/HRD/Dementia/2017
Ellora Sen	12	Inflammation regulated metabolic reprogramming Implications in tumor progression (UOE)	D.B.T.	30.03.2015	26.04	172.90	29.03.2020	No.BT/MED/30/SP11016/2015
Neeraj Jain	13	Mechanisms of Adult Brain Reorganization	D.B.T.	28.05.2014	15.49	89.24	27.05.2018	BT/PR7180/MED/30/907/2012
Neeraj Jain Director NBRC	14	Phase-II of the Centre of Excellence (COE) for Epilepsy	D.B.T.	27.03.2018	0.00	1498.86	31.03.2021	BT/MED/122/SP24580/2018
	15	Dist information Centre (DIC)	D.B.T.	22.12.1999	84.70	528.10	31.03.2020	BT/BI/03/012/2002
	16	Delcon (E- Library Consortia) Project	D.B.T.	18.03.2009	4,697.13	37123.67	31.03.2020	No. BT/BI/12/053/2012
	17	Dementia Science Programme	D.B.T.	14.09.2007	0.00	37.50	—	No. BT/PR-NBRC/2008
	18	Dementia Science Programme- Coordination Administration & Management setup at DBT	D.B.T.	18.12.2017	0.00	530.42	17.12.2020	No.BT/HRD/Dementia/2017

Externally Funded Research Projects

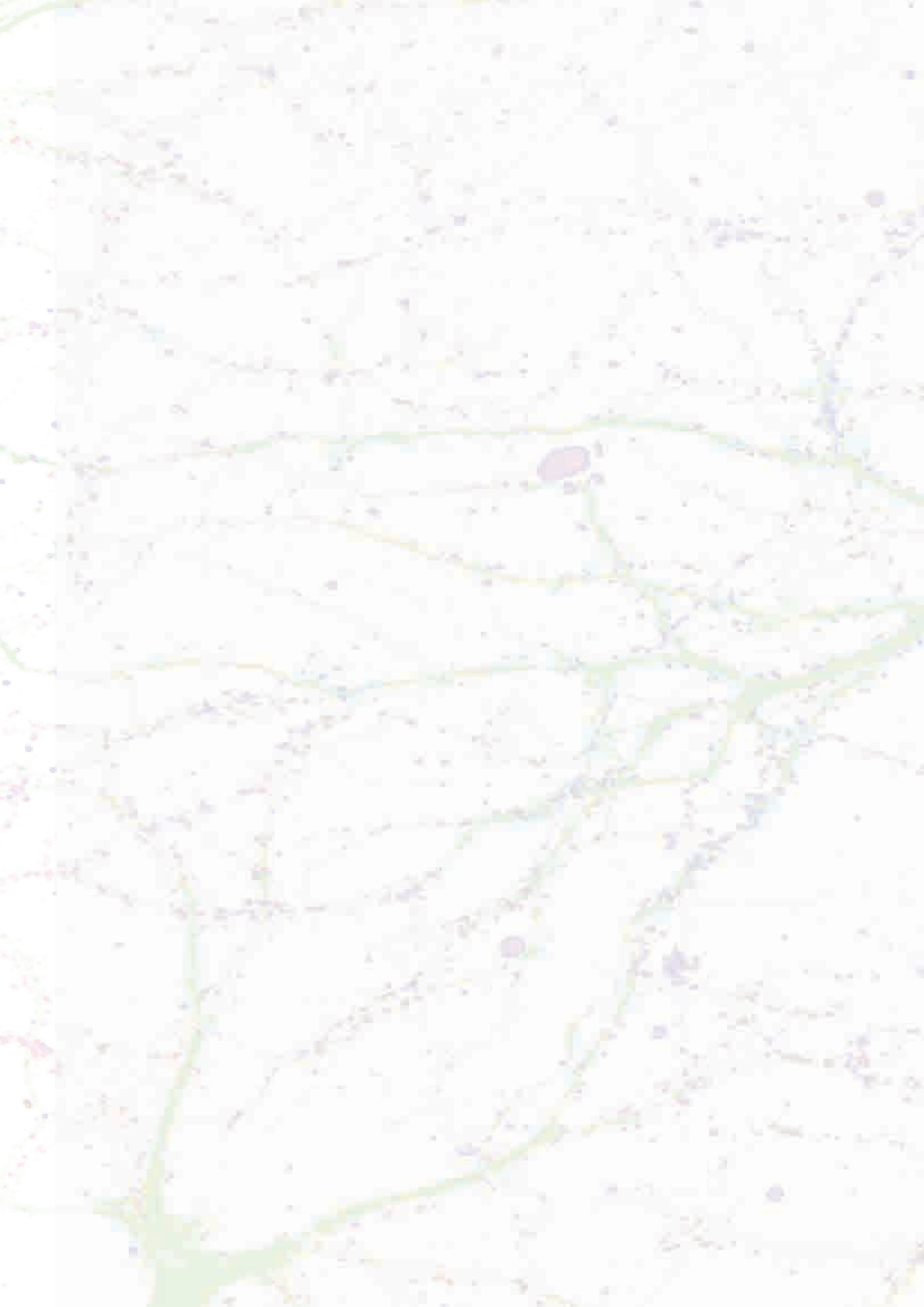
Name of P.I.	Project S.No.	Name of Project	Funding Agency	Date of Sanction	Amount Received for 2018-19 (Rs. Lakh)	Sanctioned Cost (Rs. Lakh)	Date of Completion	Sanction Order No.
Pankaj Seth	19	Differentiation of fetal neural stem cells to oligodendrocytes- a disease model to decipher the pathogenesis and devise therapeutic strategies for cerebral palsy	D.B.T.	19.03.2018	0.00	20.80	18.03.2020	No.BT/ PR17581/ MED/ 31/333/2016
	20	Insights into role of a dyslexia linked long non-coding RNA(lncRNA) in human neural stem cell	D.S.T.	18.07.2017	14.00	76.97	17.07.2020	No.SR/CSRI/ 210/2016(G)
	21	Hypoxia Induced Changes in Blood Brain Barrier	D.B.T.	12.09.2018	11.40	34.66	11.09.2021	No.BT/ PR23625/ MED/ 122/77/2017
	22	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	19.96	66.96	15.10.2023	No.BT/ PR21413/ MED/ 122/40/2016
Pravat Kumar Mandal	23	Non-invasive imaging Technology Developmnet to aid Differential Diagnosis of Alzheimer, Dementia with Lewy body and parkinson Disease from Brain Glutathione Quantiation and ph Mapping(Tata Innovation Fellowship)	D.B.T.	01.04.2015	8.91	45.00	31.03.2020	No.BT/HRD/ 35/01/05/ 2014
	24	Construction of an indian population specific brain template	C.S.I.R.	11.05.2016	8.50	26.12	09.11.2019	No. SR/ CSRI/ 229/ 2014(G)
	25	Unravelling the causes of stroke and cognitive decline in general population A cross-Cultural perspective (DBT Netherland Grant)	D.B.T.	21.04.2016	0.00	73.66	20.04.2022	No. BT/IN/ Netherlands/ 03/KP/2012
	26	Novel Imaging Diagnostics for Alzheimer's Disease	D.B.T.	24.01.2018	0.00	151.26	23.01.2021	No.BT/Indo-Aus/10/31/ 2016

Externally Funded Research Projects

Name of P.I.	Project S.No.	Name of Project	Funding Agency	Date of Sanction	Amount Received for 2018-19 (Rs. Lakh)	Sanctioned Cost (Rs. Lakh)	Date of Completion	Sanction Order No.
	27	Dementia Science Program- Imaging Studies	D.B.T.	18.12.2017	0.00	35.41	17.12.2020	No.BT/HRD/ Dementia/ 2017
Prem Chand	28	Effect of handedness on recovery of forepaw in rats with spinal cord injury	D.S.T.	11.07.2017	0.00	17.60	10.07.2019	No. SR/ CSRI/ PDF- 27/2016
Sandeep Kumar	29	Post Doctoral Fellowship	SERB	03.05.2017	9.60	19.20	02.05.2019	PDF/2017/ 001610
Shiv Kumar Sharma	30	Dementia Science Programme- Basic Biology Studies (i) Genetic Studies at lab	D.B.T.	18.12.2017	0.00	106.64	17.12.2020	No.BT/HRD/ Dementia/ 2017
Soibam Shayam-chand	31	Post Doctoral Fellowship	SERB	03.05.2017	8.50	19.20	02.05.2019	PDF/2016/ 003188
Soumya lyengar	32	Effects of the δ - opioid receptor system on singing and song learning in Zebra Finches	D.S.T. (SERB)	23.09.2016	10.00	37.43	22.09.2019	EMR/2015/ 001422
Sourav Banarjee	33	CRISPRi system : A toolbox to investigate novel regulatory mechanisms of synapse formation by long non-coding RNAs"	D.B.T.	11.01.2016	0.00	74.19	10.07.2019	No. BT/ PRI14071/ GET/119/ 36/2015
	34	Regulation of energy metabolism by miRNA-mediated control of neurogenesis	D.B.T.	21.02.2015	16.73	78.09	20.06.2019	No. BT/ PR8793/ AGR/ 36/749/2013
	35	Regulation of Fear memory formation by long non-coding RNAs and RNA binding proteins: Mechanism of combinatorial control	D.S.T. (SERB)	25.03.2019	24.04	52.39	24.03.2022	No. SERB/ F/12655/ 2018-19
Swagata Dey	36	Inspire Faculty fellowship 2019	D.S.T.	11.02.2019	22.00	112.40	10.02.2024	DST/ Inspire Faculty/ Batch-15/ 2019
Yogita K Adlakha	37	Innovation in science pursuit for inspired research (INSPIRE)	D.S.T.	01.07.2014	0.00	86.27	31.06.2019	No. DST/ INSPIRE faculty Award/ 2014/DST/ INSPIRE/ 04/2013/ 001157



Distinctions, Honours and Awards



Distinctions, Honours and Awards

Faculty

Prof. Neeraj Jain

- Elected Fellow, Indian National Science Academy

Prof. Pankaj Seth

- Nominated and Elected as Fellow, of The Indian Academy of Neurosciences, India. 2018

Prof. Anirban Basu

- Basanti Devi Amir Chand Prize (ICMR)-2017 (announced in October 2018)
- Elected to Fellowship in the American Academy of Microbiology (AAM) 2019
- Dr. Y.S. Narayana Rao Oration Award (ICMR) 2018

Prof. Ellora Sen

- Novartis Oration Award (ICMR) 2018

Prof. Nandini C. Singh

- NASI Reliance Platinum Jubilee Award in Biological Sciences 2018

DST INSPIRE Faculty

Dr. Yogita K Adlakha

- NASI Young Scientist Platinum Jubilee Award 2018

Students & Post Doctoral Fellows

Dr. Swagata Dey

- Inspire Faculty award 2019
- Early career fellowship of Wellcome Trust-DBT India Alliance 2019

Surajit Chakrabarty

- Received the best poster award in International symposium on Infectious Diseases, RCB & Jamia Hamdard, 12-14th, November

Atrayee Basu

- TNQ Inspiring Science Award 2019 Finalist for the best published scientific paper in the Life Sciences from India

Sarbani Samaddar

- Zeeshan Khan Memorial Award for the best poster presentation in Bangalore Microscopy Course 2018

Sandeep Kumar

- Awarded Developing Neuroethology Awards by International Society for Neuroethology to attend the meeting

Alok Nath Mohapatra

- Awarded IBRO-PERC, The Brain Prize and FENS stipend to attend the conference

Asha SC

- Travel Award from INA to attend INA conference 2018

Course-Work

M.Sc. 2017

Ms. Guneet Kaur : M.Sc. student, has been awarded first rank upon completion of Course-Work during the year 2017-18 and a certificate was given to her on the 15th Foundation Day, the 16th December 2018.

Mr. Azman Akhter : M.Sc. student, has been awarded second rank upon completion of Course-Work during the year 2017-18 and a certificate was given to him on the 15th Foundation Day, the 16th December 2018.

Ph.D. 2017

Ms. Vanshika Singh : Ph.D. student, has been awarded first rank upon completion of Course-Work during the year 2017-18 and a certificate was given to her on the 15th Foundation Day, the 16th December 2018.

Mr. Karthick R : Ph.D. student, has been awarded second rank upon completion of Course-Work during the year 2017-18 and a certificate was given to him on the 15th Foundation Day, the 16th December 2018.

Ph.D. Degrees Awarded

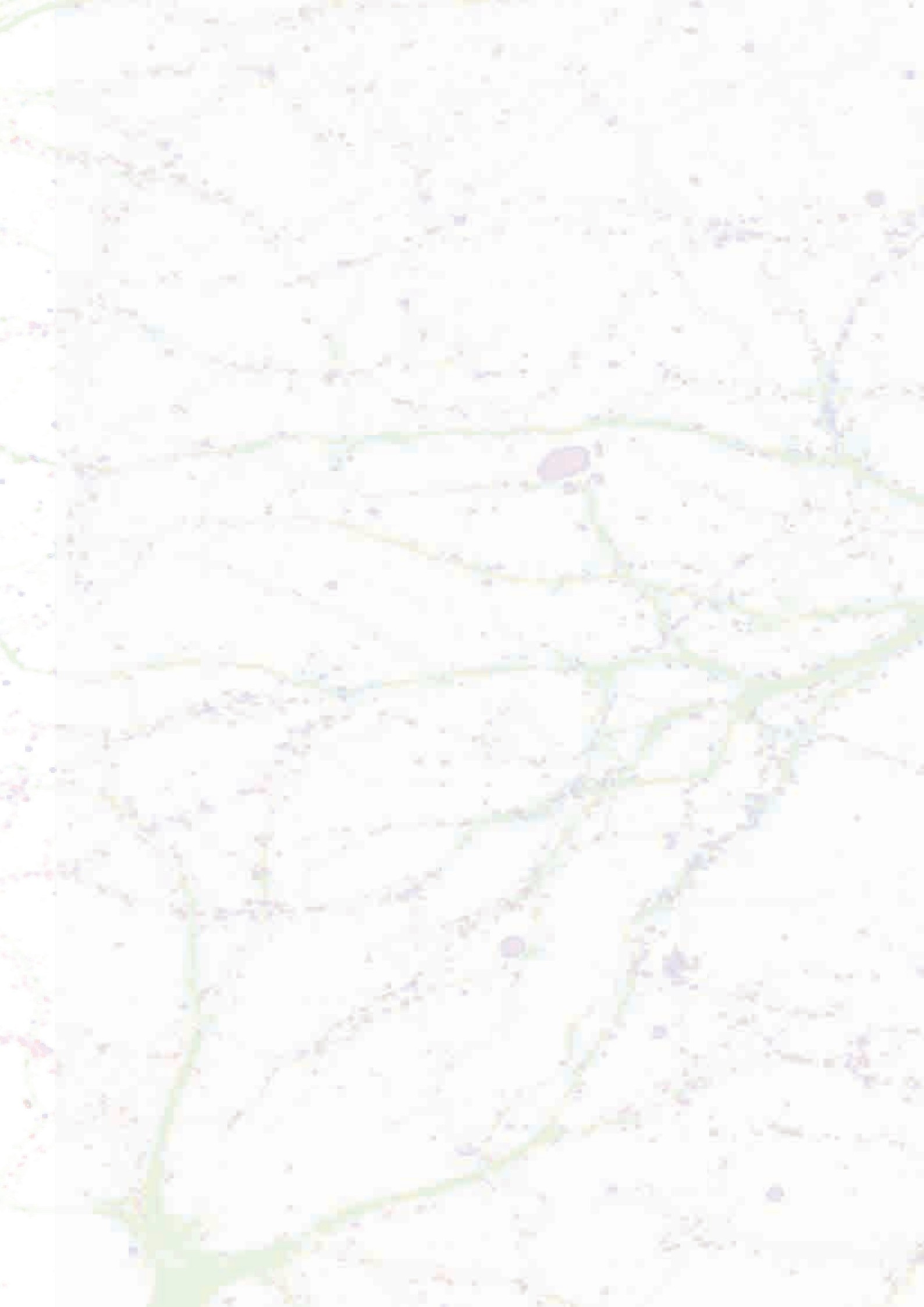
Sr. No.	Name of the Student
1.	Dr. Brijesh Kumar Singh
2.	Dr. Shalini Swaroop
3.	Dr. Abhishek Kumar Verma

M.Sc. Degrees Awarded

Sr. No.	Name of the Student
1.	Ms. Vini Tiwari
2.	Ms. Varsha Ramakrishna
3.	Ms. Tapasya Pal
4.	Ms. Sreyashi Chandra
5.	Ms. Ritu Moni Borah
6.	Ms. Kirti
7.	Ms. Kasturi Biswas
8.	Mr. Anagh Pathak
9.	Mr. Abhishek Kumar Verma

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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 the Department of Biotechnology. The 'Deemed to be university' status of NBRC has been reviewed by the Committee duly constituted by the UGC and also by an independent Committee constituted by Ministry of HRD, on completion of five years as Deemed University. The committee recommended extension of Deemed University status and placed NBRC under "A" category.

Ph.D. in Neuroscience

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

NBRC provides a fellowship of Rs. 31,000/- per month for Junior Research Fellows and Rs. 35,000/- per month for Senior Research Fellows.

M.Sc. in Neuroscience

NBRC is one of the first Institutes in the country to develop an integrated multidisciplinary teaching programme in Neurosciences. During the academic year 2015-16 NBRC reintroduced the M.Sc.

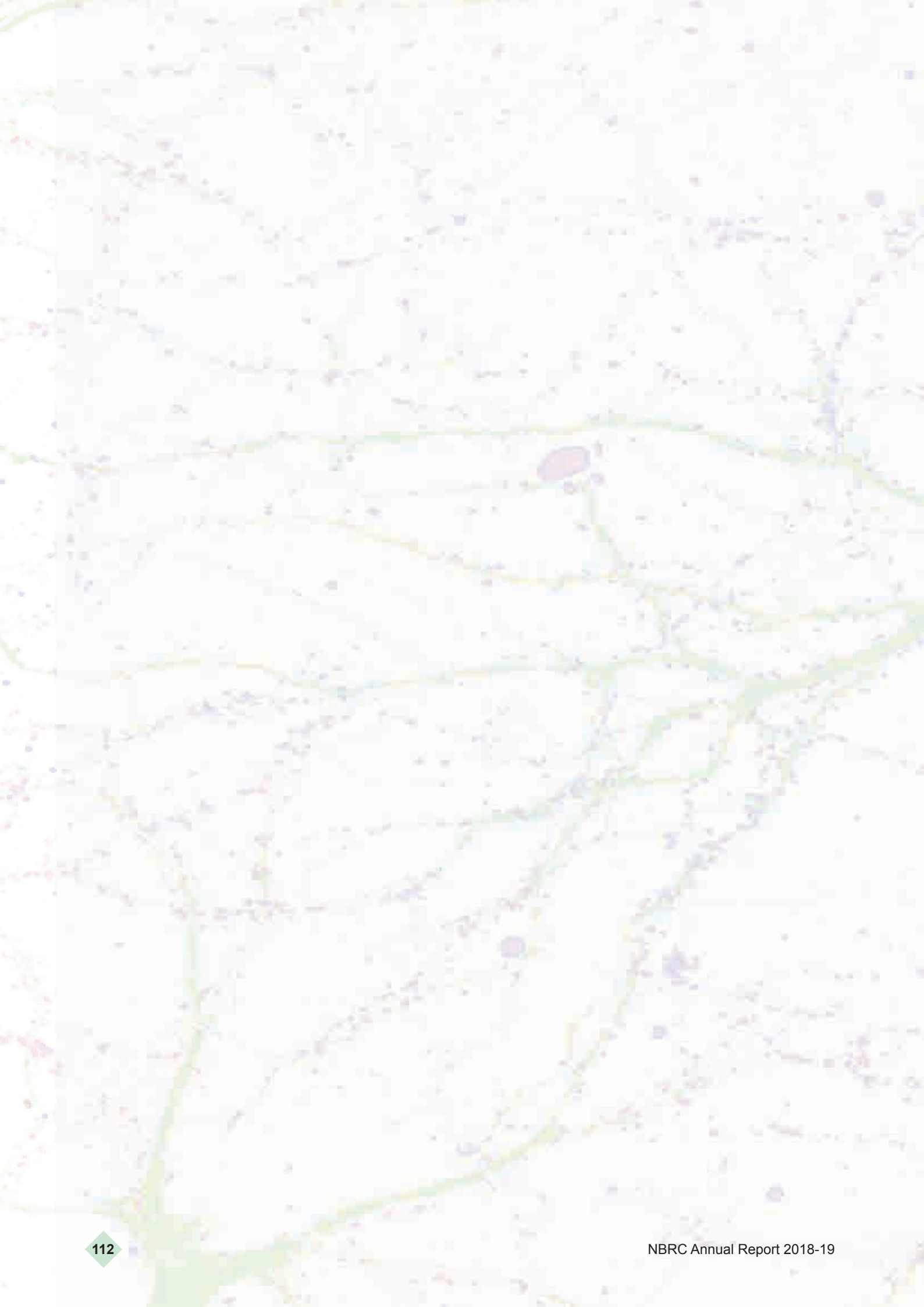
(Neuroscience) programme to develop trained manpower having a broad overview of different aspects of Neuroscience.

M.Sc. (Neuroscience) students are provided a fellowship of Rs. 12,000/- per month.

NBRC inducts students for its M.Sc. (Neuroscience) and Ph.D. programmes from diverse backgrounds having Bachelors or Masters 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.

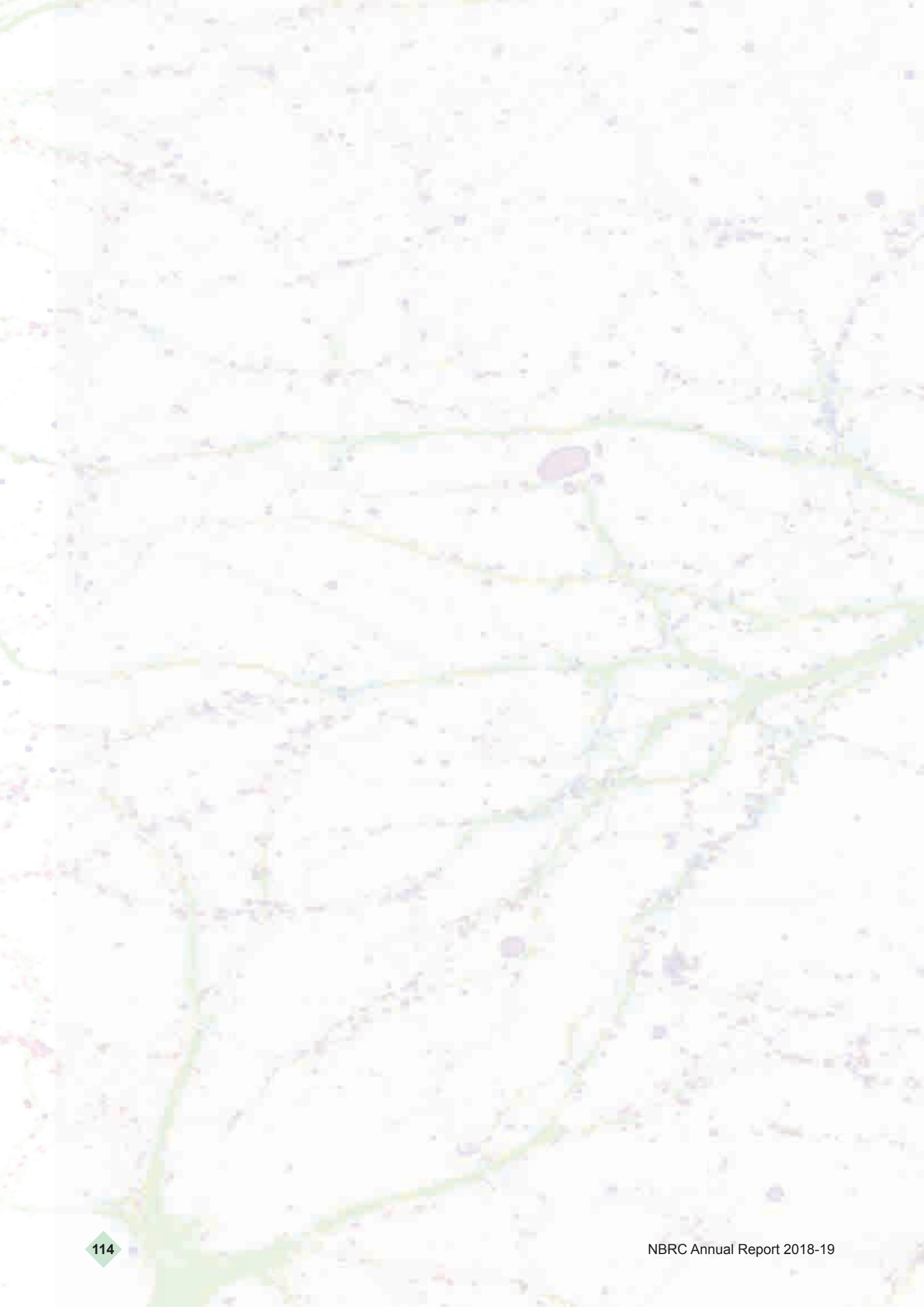
Summer Training and Short-term Programmes

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



Distributed Information Centre (DIC)

The Distributed Information Centre (DIC) of National Brain Research Centre manages the overall ICT infrastructure and communication networks of the institute apart from aiding in R&D activities. The DIC focuses on providing ICT S&T services to the scientists & researchers as well as aiding the e-Governance activities of the institute. It is also a DIC under the BTISNET initiative of Department of Biotechnology. The Centre manages the campus converged network (data and voice traffic), communications links (Network and PSTN), Institute's Datacentre, 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:

A) 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 1Gbps optical fibre link provided by BSNL, it 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.

B) IP-PBX facility

The tele-communication systems of the institute were 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.

C) Institute Core and Application Servers

The computing facility manages and maintains the server infrastructure of the institute; they are housed and maintained in the datacenter facility. In essence the institute currently has five numbers of fully utilized 42U server racks in the datacenter facility. The various service running on these server can be classified as under :

- a. Web-servers for the institute and various web-servers related to ongoing computational projects and applications of various scientific groups is also hosted and managed in the central facility. The primary webserver for official website is also running from VM's running on NIC cloud.
- b. E-mail and List servers (nbrc.ac.in) of the institute. The core employees were also provided

with email accounts on NIC mail services (gov.in/nic.in).

- c. DNS servers for the official and hosted domains which are running from NBRC datacenter as well as VM's hosted on NIC cloud.
- d. Virtualization servers for providing virtualized hardware to run various applications and service in a more managed manner and to consolidate and utilize the existing physical server infrastructure.
- e. Radius and authentication servers for access, accounting and authorization of computing resources
- f. License management servers for managing institutional site/network/concurrent licenses.
- g. Antivirus and security servers for providing protection to user end-points across the campus.
- h. Central storage servers 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.
- i. Application servers running on Windows and Linux platforms for common computing requirements of the users and also other specialized computing servers for specific data processing requirements of various laboratories.

D) Other Facilities & Services

- a. Central Documentation Facility: The central documentation facility provides round the clock availability to uses for various computational needs like facility for printing, scanning, poster-

printing etc. apart from providing data-processing computational nodes.

- b. 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 have been provided with GOV.IN email ids on NIC platform for better availability.
- c. 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.
- d. CCTV Monitoring and Management: The DIC has also installed IP-Cameras connected to the core network which are managed through central NMS device for aiding into the security of the campus. Most entry/exit points of the buildings are covered with the Central CCTV system.
- e. 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.
- f. 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.

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.

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 then 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 $22 \pm 2^{\circ}\text{C}$, relative humidity between 45-55%, 12:12 hr light-dark cycle, and 12-15 air changes per hour. The air-handling system uses 100% fresh air for each change.

All animals are procured as per 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

NBRC Facilities

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 (Hdaxon1) 62Gpb/3J (Huntington disease model)
- B6;129P2Pvalb< tm1(cre)Arbr>/J
- B6.CgGt(ROSA)26Sor<tm9(CAGtdTomato)
- B6.CgTg(Scnn1acre)3Aibs/J
- STOCK Gad2<tm2(cre)Zjh>/J
- B6.CgTg(Camk2a-cre)T29-1Stl/j
- B6.129-Rp122<tm1.1Psam>/j
- STOCK Tg(Thy1-EGFP)MJrs/J
- B6.Cg-Tg(Thy1-YFP)16Jrs/J
- B6.Cg-Tg(Thy1-YFP)HJrs/J
- B6;129S6-Tg(Camk2a-cre/ERT2)1Aibs/J
- STOCK Ssttm2.1(cre)Zjh/J
- B6.Cg-Gt(ROSA)26Sortm6(CAG-ZsGreen1)Hze/J
- B6:129X1-Gt(ROSA)26Sor<tm(EYFP)Cos>/j
- C57Bl6-Tg(Nes-cre/ERT2)Keise/j

Knock Out Mice

- UBE3A null mice (Angelman syndrome model) Mutant Mice
- CBA/J mice (Retinal degeneration model)

Rat Strains

- Long Evans
- Sprague Dawley

Non-human primates

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

Birds

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

All the mice strains are maintained by in-breeding 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.

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 1176 online journals through the DBT e-Library Consortium (DeLCON), 3 specialized journals, and 122 freely accessible online journals. It also maintains digital archives and news clips about the Centre and subscribes to Newspapers and News Letters. The collection of the NBRC Library is growing day-by-day along with new developments in research and knowledge in the field of Neuroscience and related areas. To provide optimum service to all users, the NBRC library is currently digitizing its list of collections using the LSEASE software, to which all users will have full access. A barcode technology has also been installed for accurate and speedy circulation and the management of all library documents. The new software will also help in efficient library operations viz. administration, acquisition, circulation, serial control, cataloguing and information retrieval. The Library has set up 22 Computers with Internet facility to provide services for use of researchers

and students in the NBRC Common room and has been providing electronic access to the subscribed journals through the campus portal. The NBRC Library also provides Inter Library Loan Services to NBRC's 48 networked centres all over India. Researchers at different centres send their requirement for research material or journal articles through email to NBRC Library library@nbrc.ac.in or to the Librarian Dr. D. D. Lal, ddlal@nbrc.ac.in which are then downloaded and sent to them free of cost. The library entertains an average of approximately 450 requests for articles and this number is increasing every year. The NBRC Library regularly evaluates its information services to ensure that the Institution's requirements are met. It promotes resource sharing and cooperation activities among libraries by providing an efficient and reliable means of resource sharing, that is, the inter library loan for the maximum use of resources, by providing copies of documents which are not available to researchers at centres outside the institute.

Main Activities of NBRC Library

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

DBT's Electronic Library Consortium (DeLCON)

DeLCON Consortium: A National Library Consortium For Life Sciences & Biotechnology Hosted And Administered By Nbrc And Sponsored By Department Of Biotechnology (Dbt)

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

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

In the year in 2019, the DBT added one new Institute i.e. Institute for Stem Cell Science and Regenerative Medicine (InStem) under DeLCON Consortium. Currently DeLCON has a total of 35 members. The 'DeLCON Consortium' provides current (presently 1000+ online resources) as well as archival access to more than 1176 core peer-reviewed journals and one bibliographic database (SCOPUS Database) 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), Bhubaneswar
7. Institute of Bioresources and Sustainable Development (ISBD), Imphal
8. Centre for DNA Fingerprinting and Diagnostics (CDFD), Hyderabad
9. Rajiv Gandhi Centre for Biotechnology (RGCB), Thiruvananthapuram
10. International Centre for Genetics Engineering and Biotechnology (ICGEB), New Delhi
11. National Agri-Food Biotechnology Institute (NABI), Mohali, Punjab
12. National Institute of Biomedical Genomics (NIBMG), Kalyani, Kolkata DBT's Electronic Library Consortium (DeLCON) NBRC Annual Report 2016-17 129
13. National Institute of Animal Biotechnology (NIAB), Hyderabad
14. Regional Centre for Biotechnology (RCB), Faridabad, as a part of NCR Biotech Science Cluster (BSC)
15. Translational Health Science & Technology Institute (THSTI), Faridabad, as a part of NCR Biotech Science Cluster (BSC)

16. Biotechnology Industry Research Assistance Council (BIRAC), New Delhi
17. Institute for Stem Cell Science and Regenerative Medicine (InStem), Bangalore.

North Eastern Region (NER) Institutions

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

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

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

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

List of Journals Under DeLCON Consortium

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

- ❖ American Association for Advancement of Science(AAAS)→ <http://www.sciencemag.org> → 3 Journals
- ❖ American Association for Cancer Research (AACR) → <http://www.aacr.org> → 8 Journals
- ❖ American Society for Biochemistry and Molecular Biology (ASBMB)→ <http://www.jbc.org> → 2 Journals
- ❖ American Society For Microbiology (ASM) → [http:// www.asm.org/](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](http://www.nature.com) → 36 Journals
- ❖ Oxford University Press (OUP) → <http://www.oxfordjournals.org> → 22 Journals
- ❖ Springer India → <http://www.springerlink.com> → 342 Journals
- ❖ Microbiology Society (MBS) → [http:// mic.sgmjournals.org](http://mic.sgmjournals.org) → 3 Journals
- ❖ American Society for Hematology (ASH) → [http:// bloodjournals.hematologylibrary.org](http://bloodjournals.hematologylibrary.org) → 1 Journal
- ❖ Wiley-Blackwell → [http:// www3.interscience.wiley.com/cgi-bin/home](http://www3.interscience.wiley.com/cgi-bin/home) → 86 Journals
- ❖ Elsevier Science (ScienceDirect) → [http:// www.sciencedirect.com](http://www.sciencedirect.com) → 435 Journals
- ❖ American Society of Plant Biologist (ASPB) → <http://www.aspb.org/> → 2 Journal
- ❖ American Association of Immunologist (AAI) → [http:// www.aai.org/](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](http://ovidsp.ovid.com) → 11 (Only Archives from 2009-2011)
- ❖ Marry ANN Liebert (MAL) → <http://www.liebertonline.com> → 92 (Only Archives from 2009-2018)
- ❖ American Chemical Society(ACS) → <http://pubs.acs.org>→ 47 Journals (Only Archives from 2009-2016)
- ❖ Annual Reviews (AR) → <http://www.annualreviews.org> → 23 Journals (Only Archives from 2009-2011)

- ❖ The New England Journal of Medicine (NEJM) → <http://www.nejm.org> → 1 (Only Archives from 2009-2018)

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 knowledgebase of DBT institutions has improved with increased access to international full text resources (Journals and database).
- ❖ Users have immediate access to material previously not subscribed to, at no incremental cost for accessing back files.
- ❖ It improves the existing library services and reduced the subscription cost.
- ❖ DeLCON is open so that other DBT institution can also join the DeLCON Consortium.
- ❖ DeLCON offers better terms of agreement for use, archival access and preservation of subscribed electronic resources, which would not have been possible for any single institutions.
- ❖ Members of the DeLCON Consortium have the benefit of cap on the annual increase in the rates of subscription. While the usual increase in price of e- resources is vary from 15% to 20%, but the DeLCON members enjoy a cap on increase in price ranging from 5% to 7%.
- ❖ Since the subscribed resources is accessible online in
- ❖ electronic format, the DBT institutions have less pressure on space
- ❖ requirement for storing and managing print based library resources.

Major Advantages of DeLCON for Consortium Members

- ❖ Some of the important advantages of the DeLCON

consortium provides to members as given below:

- ❖ Consortia-based subscription to electronic resources provides access to wider number of electronic resources at substantially lower cost.
- ❖ Optimum utilization of funds.
- ❖ Facilities to build up digital libraries
- ❖ Helpful in providing better library services like CAS and SDI
- ❖ Cost sharing for technical and training support
- ❖ Electronic Journals demand neither library space nor shelving costs
- ❖ The DeLCON consortium has been offered better terms of licenses for use, archival access and preservation of subscribed electronic resources, which would not have been possible for any single institution; and
- ❖ Available 24 hours a day, 7 days a week

Selection Procedures of Resources Under Delcon Consortium

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

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

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

Subject Areas of DeLCON Consortium

The DeLCON Consortium covers all the disciplines and subjects coming under Life Sciences i.e. Biotechnology, Bioinformatics, Biochemistry, Biology, Chemical Biology, Sciences, Immunology, Neuroscience, Plant Genome, Plant

Biology, Microbiology, Physiology, Psychology, Physiotherapy, Psychotherapy, Genome, Gene, Genetics, Mathematics, Physics, Chemistry, Radiology, Medicines, Computational Biology, Cell Biology, Cell Sciences, Molecular Biology, Molecular and Cellular Biology, Computational Neuroscience, System Neuroscience etc.

Operational Functionality Of Delcon Consortium

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

Nodal Centre & Head Quarter Of Delcon Consortium & Its Activities

The consortium headquarter functions under a National Steering Committee with the responsibilities of ensuring inter-institutional coordination; monitoring licenses for electronic resources, ordering and payment for subscribed

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

National Neuroimaging Facility

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

1. 3 Tesla Magnetic Resonance Imaging (MRI): Philips Achieva 3.0 T scanner
2. Electroencephalography (EEG): 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. Various imaging modalities also play important role providing crucial information which can aid to various diagnostic process. The various imaging modalities which are routinely used in National Neuroimaging facility are:

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

The 3 Tesla Phillips whole body MRI scanner at our Facility is equipped with state-of-the-art hardware, software and data processing software required for each imaging modality. The facility is being used daily for performing structural and functional MRI (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) is a test that measures and records the electrical activity of the brain. Special sensors are attached to the scalp (in a similar way as ECG) to detect brain electric activity and mV range and the signals are amplified via an amplifier that communicates and stores the information in a computer. Basic brain functions such as vision, auditory, somatosensory processing as well as higher order functions like memory, emotion, decision making and brain diseases such as epilepsy, dementia, and narcolepsy (sleeping disorder) can be studied by EEG.

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

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

Figure 1: fMRI brain activations and deactivations in visual perception and action tasks obtained from Philips 3T scanner at National Neuroimaging Facility

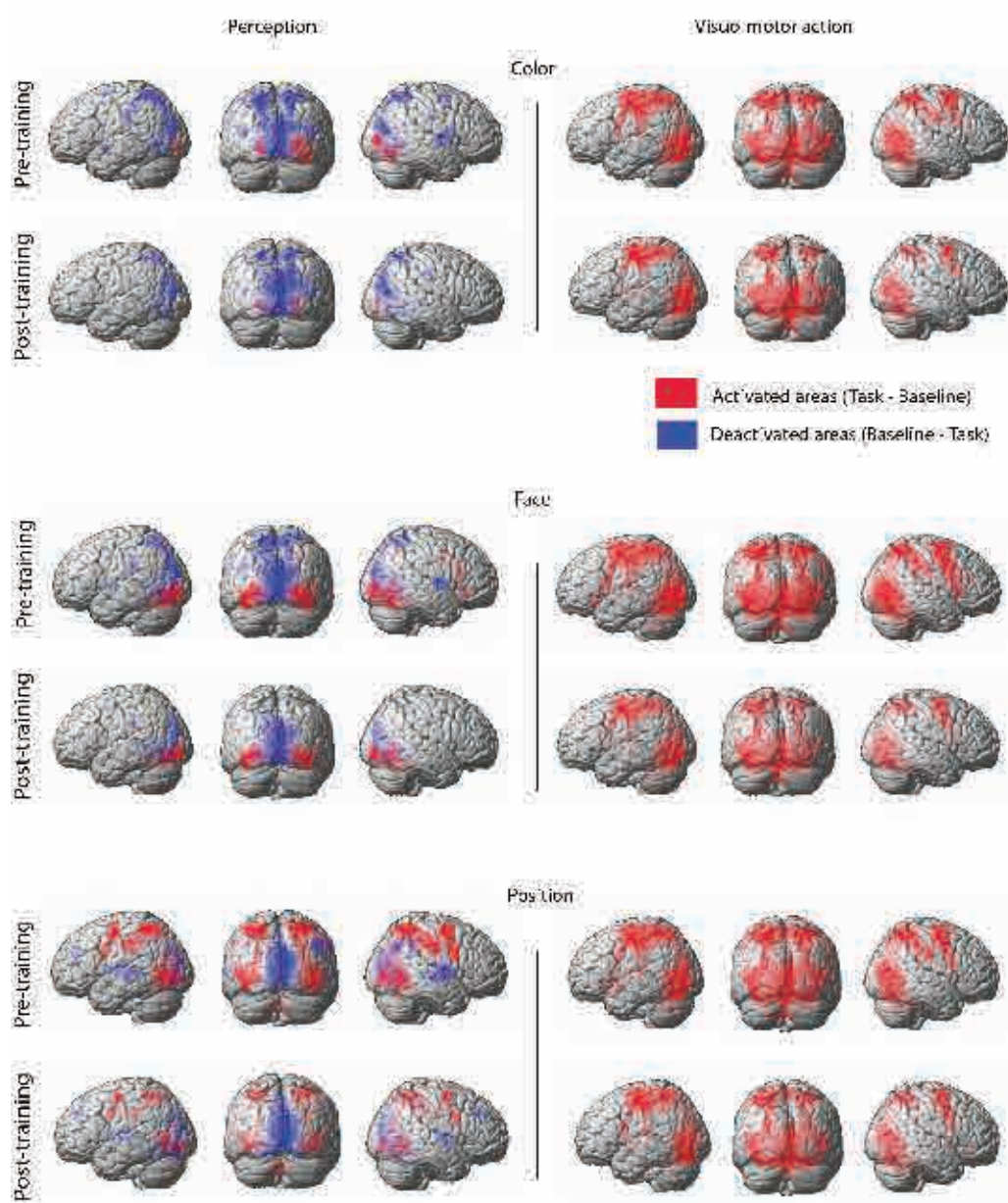
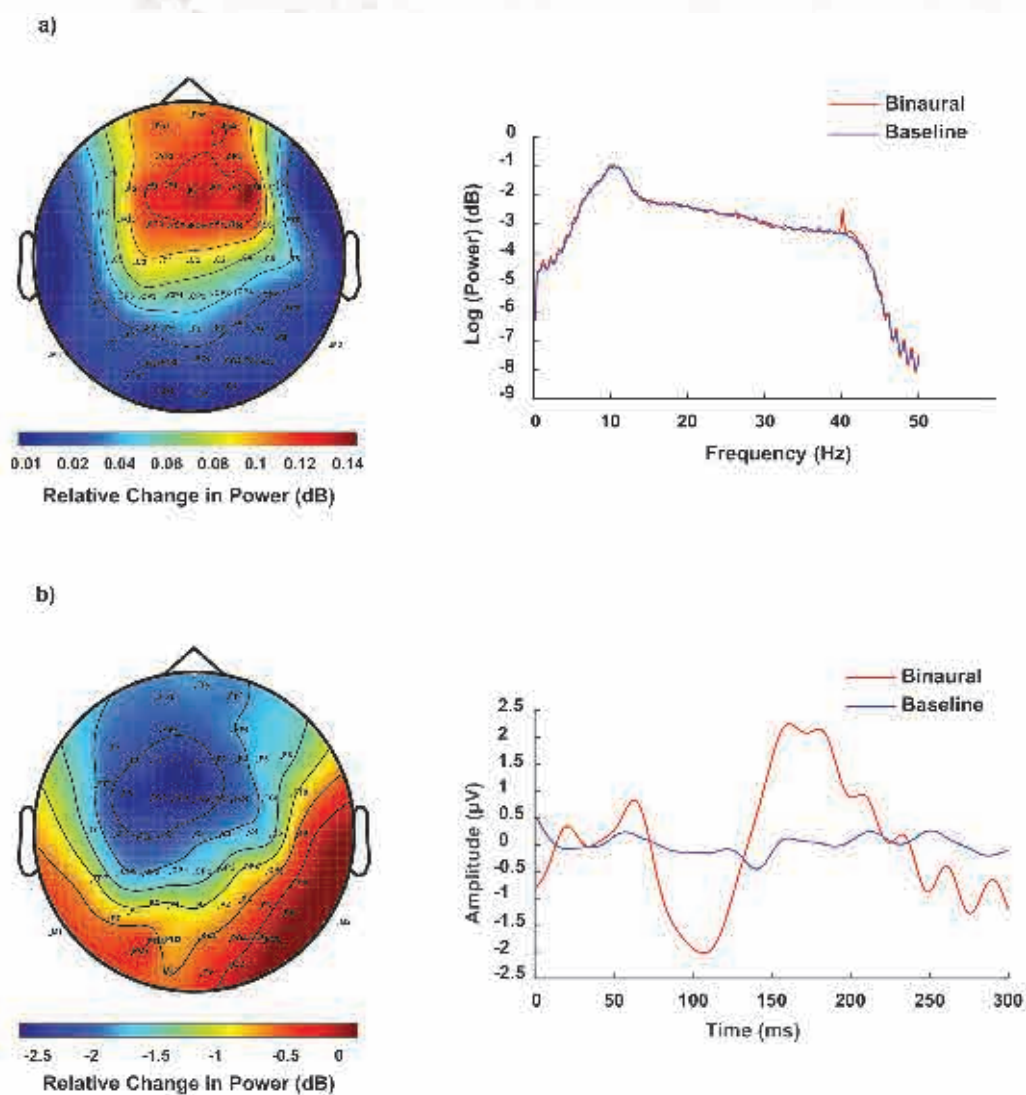


Figure 2: a) EEG scalp topography and power spectrum obtained during generation of auditory steady state response to tonal stimulus b) Scalp topography and evoked potential (N100) response to tonal auditory stimulus.



Translational & Clinical Neuroscience Unit

The National Brain Research Centre extended its reach to Government General Hospital, Gurgaon almost a decade ago by establishing the Translational and Clinical Neuroscience Unit, with an aim to provide neurology outpatient department to the government hospital and also assess the occurrence of neurological cases in this region.

The unit is located at the room number 7 of the Government General Hospital, Gurgaon that is easily accessible by the patients.

Investigation facilities:

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

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

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

- ❖ Consultant Clinical Professor, Neurology: Dr Rajnish Kumar
- ❖ Consultant Clinical Professor, Neurology: Dr. Hitesh Kumar
- ❖ Consultant Clinical Professor, Pediatric Neurology, Dr. Rakesh Jain
- ❖ Clinical Psychologist: Priyanka Kaushik
- ❖ 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

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 Pediatric 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.

As the out-patient facility is visited by patients from Gurgaon and neighbouring districts the neuro OPD is quite well attended, taking the number of patients to over 35-40 in a day. The facility also maintains a good follow up rate of patients, and about 75% of the patients return to the clinics for follow-ups. Male to female ratio is almost equal. Patients visiting the NBRC unit are of different age groups ranging from pediatric to adult and elderly patients. The Elderly or Geriatric patients mostly come for the Movement Disorders, dementia, where as that adult patients mostly come with complaints of headache, depression, tremors, dementia and pediatric patients present with symptoms of mental retardation, ASD, seizure, epilepsy.

Patients attending the OPD at Civil Hospital come from old Gurgaon township and the villages and towns in the surrounding districts of Haryana, while

NBRC Facilities

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.

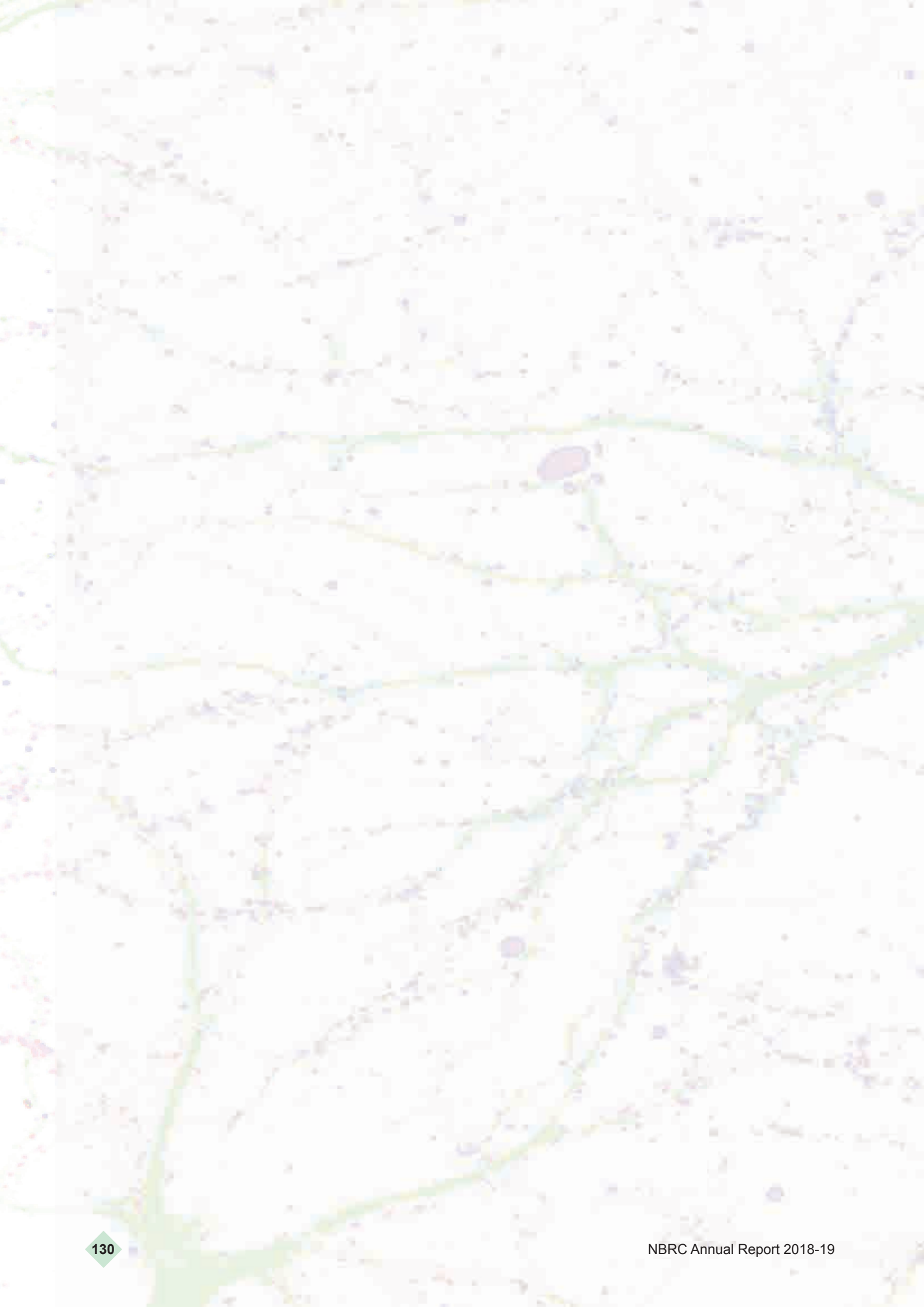
We are maintaining the medical data of new patients, to create computer database with relevant patient data along with any planned imaging/ molecular/neurophysiological studies at the NBRC labs, thus creating a well documented “clinical

window” for our research institute. 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 - Gurugram, 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.



Lectures, Meetings & Workshops



Lectures, Meetings & Workshops

Invited Speakers at NBRC

Sr. No.	Name of the Speaker	Title of the Talk	Date	Host Faculty
1.	Dr Sahil Bajaj Social, Cognitive and Affective Neuroscience (SCAN) Lab, Department of Psychiatry, College of Medicine, University of Arizona, Tucson, AZ	Blue Light Therapy Strengthens the Structural and Functional Neural Aspects Following Mild Traumatic Brain Injury	8 May 2018	Dr. Arpan Banerjee
2.	Dr Rupesh Kumar Postdoctoral Fellow Laboratoire de Neurosciences Cognitives Computationnelles, Laboratoire des Systems Perceptifs, Ecole Normale Supérieure, Paris	Persistent Encoding of Sound Categories During Task Engagement in Auditory Cortex	11 July 2018	Dr. Arpan Banerjee
3.	Dr Aprajita Mohanty Associate Professor Stony Brook University	Threat Perception and The Predictive Brain	30 July 2018	Dr. Arpan Banerjee
4.	Prof. Amitabha Chattopadhyay, SERB Distinguished Fellow Centre for Cellular & Molecular Biology, Uppal Road, Hyderabad 500 007	Cholesterol-Induced Conformational Plasticity and Oligomerization of GPCRs: Novel Insights in Health and Disease	7 August 2018	Dr. Ellora Sen
5.	Prof Krishnankutty Sathian, MBBS, PhD, FANA, FASNR Chair of Neurology Director, Neuroscience Institute Professor of Neurology, Neural & Behavioral Sciences, and Psychology, Pennsylvania State University	Sensory Grounding of Language	7 August 2018	Dr. Arpan Banerjee
6.	Dr Manasmita Das, Director, Contrast Agent and Molecular MRI Research Center for Animal MRI (CAMRI) Postdoctoral Research Associate; Department of Neurology and Biomedical Research Imaging Center (BRIC), University of North Carolina at Chapel Hill	Advanced Neural Circuit Mapping Using Optogenetic, Chemogenetic and Multimodal Molecular MRI	4 September 2018	Dr. Arpan Banerjee
7.	Dr Bela Desai Grants Adviser Wellcome Trust-DBT, India	The view from the other Side: working at a Funding Agency	7 September 2018	Dr. Arpan Banerjee
8.	Dr. Cecelia V. Williams, Ph.D, Sandia National Laboratories, USA	Laboratory Biosafety & Biosecurity	14 September 2018	Dr. Inderjeet Yadav
9.	Prof. Anthony J. Hannan, Florey Institute of Neuroscience and Mental Health, University of Melbourne, Melbourne Brain Centre, Australia	Gene-Environment Interactions Mediating Experience-Dependent Plasticity in The Healthy and Diseased Brain	26 October 2018	Prof. Pankaj Seth
10.	Prof Tadashi Isa Vice Dean, Professor of Department of Neuroscience, Graduate School of Medicine Kyoto University, JAPAN and Chair, Human Brain Research Center Kyoto, JAPAN	Large-Scaled Network Reorganization for Recovery from Spinal Cord Injury	26 October 2018	Prof. Neeraj Jain

Lectures, Meetings & Workshops

Sr. No.	Name of the Speaker	Title of the Talk	Date	Host Faculty
11.	Prof. Atsushi Nambu Division of System Neurophysiology, National Institute for Physiological Sciences, Okazaki, Japan Department of Physiological Sciences, SOKENDAI, Okazaki, Japan	Cortico-Basal Ganglia Loop and Movement Disorders	31 October 2018	Prof. Neeraj Jain
12.	Dr. Baskar Bakthavachalu	Intrinsically Disordered Domains in mRNPs Juggle Between Memory and Neurodegeneration	13 November 2018	
13.	Dr Pradeep Lal Emre Yaksi Laboratory, Kavli Institute for Systems Neuroscience/CNC, NTNU, Trondheim, Norway	Neural Circuits Underlying Sensory Processing and Behaviour in Zebrafish	16 November 2018	Dr. Anindya G Roy
14.	Dr. Nivethida Thirugnanasambandam, MBBS, MTech, PhD Extramural Research Faculty, Wellcome Trust/DBT India Alliance Clinical Research Fellow (Intermediate), Department of Neurology, National Institute of Mental Health and Neuro Sciences (NIMHANS), Hosur Road, P B 2900, Bengaluru 560 029, Karnataka, India	Looking Beyond Motor Evoked Potentials: Multimodal Transcranial Magnetic Stimulation	27 November 2018	Prof. Anirban Basu
15.	Prof Semir Zeki Professor of Neuroaesthetics, Cell & Developmental Biology, Division of Biosciences, University College of London, UK	New Insights Into The Organization of The Visual Brain	17 December 2018	Prof. Pankaj Seth
16.	Dr. V. Vatsalya University of Louisville	Effects of Varenicline in Human Laboratory Models for Screening Of Pharmacotherapeutics; And Repurposing Design of Quetiapine Fumarate XR for Alcohol Use Disorder	28 December 2018	Dr. Arpan Banerjee
17.	Dr. Anshika Srivastava Postdoctoral Fellow University of Michigan Medical School Department of Human Genetics 3703 Medical Sciences II 1137 Catherine St. SPC 5618 Ann Arbor, MI 48109-5618	ASXL3 Links Chromatin to Autism and Cardiac Hypertrophy Biology	9 January 2019	Prof. Anirban Basu
18.	Dr Arjun Ramakrishnan Research Associate, University of Pennsylvania, USA	Reward Sensitivity and How It Affects our Actions	24 January 2019	Prof. Soumya Iyengar
19.	Dr KM Sharika Post-doctoral fellow, Prof Michael Platt's lab, The University of Pennsylvania, Philadelphia, PA, USA	Understanding The Neurobiology of Social Decision-Making	25 January 2019	Prof. Soumya Iyengar
20.	Dr Vishal Bhardwaj Scientific Consultant, Centre for Cellular and Molecular Platforms (C-CAMP), GKVK Campus, Bellary Road, Bangalore 560065	Biotechnology Ignition Grant and C-CAMP	1 February 2019	Prof. Anirban Basu
21.	Dr. Vivek Tiwari Postdoctoral Fellow, University of Texas, Southwestern Medical Center, Dallas, Texas, USA	Imaging of The Neurochemicals in Healthy Brain and Gliomas in Vivo	18 February 2019	Dr. Dipanjan Roy
22.	Dr. Ananda Shankar Jayant Kalakshetra of Rukmini Devi Arundale, Founder Director of Shankarananda Kalakshetra	Rubaroo	29 March 2019	

From synapses to memory: RNA based regulatory mechanisms October 15th – 18th 2018

The thrust of the meeting was memory formation and dysfunction, with a strong focus on RNA-based mechanisms operating at the synapse that regulate memory at the molecular level. The event commenced with a keynote lecture by Thomas Carew, Dean of Faculty of Arts and Science at the Centre for Neural Science, New York University; Erin Schuman, Director, Max Planck Institute for Brain Research, Germany; Ted Abel, Director, Iowa Neuroscience Institute, University of Iowa, USA and Giovanna Mallucci, Associate Director, UK Dementia Research Institute, Cambridge University, UK. Both young as well as established scientists from India put forth their findings in the international circuit through the symposium. The meeting has served as an international platform for young Indian scientist to discuss their research on this focus topic with an international peer group across continents comprised of leading experts of the field as well as contemporary junior scientists. Young PhD scholars and post-doctoral fellows presented their work through short talks and poster presentations, which gave them an opportunity to strike a dialogue with

people from the field and receive feedback about their research findings.

The meeting discussed multi-faceted aspects of memory ranging from basic fundamental concepts to advanced understanding of how memory deficits during aging as well as neurodegenerative disorders are caused by RNA-mediated gene expression control.

The meeting took an interdisciplinary approach, and was conducted across various segments with themes ranging from non-coding RNAs in nerve regeneration to novel methodologies for RNA detection and cell type specific analysis of gene expression. The meeting also highlighted new concepts related to RNA based mechanism of memory and evolutionary perspective emerging from various model organisms including worm, fly, fish and rodents.

The meeting also hosted eminent musicians for two cultural evenings and a banquet dinner to enhance informal interactions among participants as well as speakers.



NBRC 15th Foundation Day- 16th December, 2018

The National Brain Research Centre celebrated its 14th foundation day with pomp and gaiety on 16th December 2017. The events were kicked off with an open day that saw participation of school children from various schools around NCR. The children were taken around labs in NBRC to give them a feel for neuroscience research. Student volunteers gave broad overviews of their respective labs with a view to instil scientific temper in the young children. The natural curiosity of children was on display as a lot of them posed probing questions to the student volunteers. This was followed by a science quiz which was hosted by NBRC students for the visiting schools which saw the director, faculty and the chief guest in attendance. The quiz was followed by a grand lunch for the students, staff, faculty and visiting school children. In the evening, a public lecture was delivered by the chief guest Prof. Semir Zeki, Professor of Neuroaesthetics at the division of

Biological Sciences, University College of London. The title of his public lecture was 'Neurobiology of Beauty'. His lecture enlightened audiences about what is common to all that arouses the aesthetic emotion? He made very clear that the experience of beauty, irrespective of its source (i.e. whether visual, musical or mathematical) correlates with activity in the same part of the emotional brain, field A1 of the medial orbito-frontal cortex. Activity in that part of the brain is proportional to the experience of beauty. This also shows that aesthetic judgment can indeed be quantified. The lecture was followed by an equally provocative question-answer session. The lecture was preceded by a prize distribution ceremony where the class toppers from the MSc. and PhD programme were felicitated for meritorious performance in course work. The ceremony was closed after a brief address by the president of NBRC society, Dr. P.N Tandon.

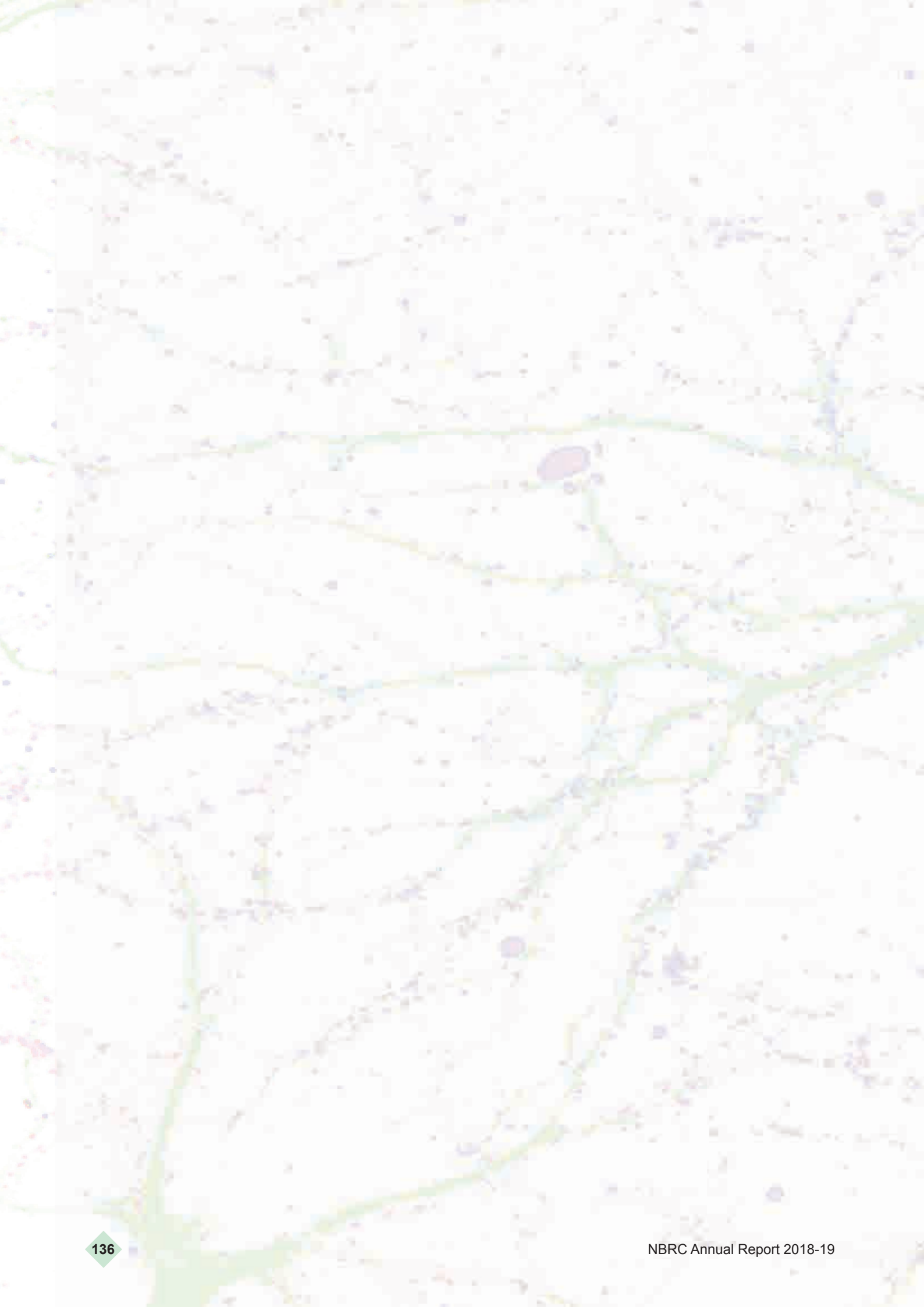


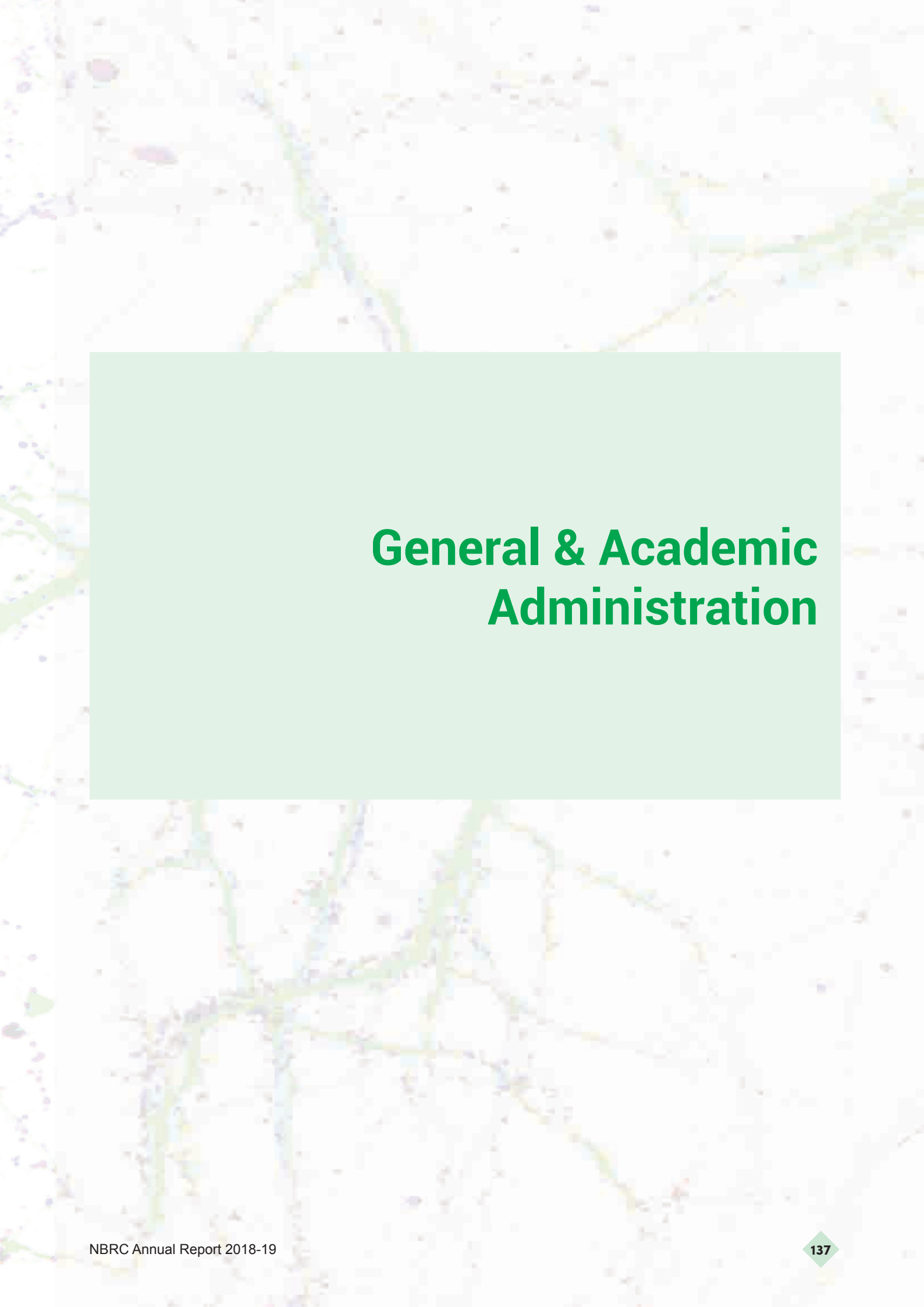
14th B. Ramamurthi Memorial Lecture

National Brain Research Centre conducted the annual B Ramamurthi Memorial lecture in memory of late Prof B. Ramamurthi. Prof. Ramamurthi is one of the founding fathers of neurosurgery in India. Notwithstanding his extremely busy professional life as a neurosurgeon, Prof Ramamurthi explored all avenues to promote neuroscience education and research in India. Prof Ramamurthi served on various committees of the highest policy-making bodies like the ICMR, DST, DBT and Health Ministry.

The 14th B. Ramamurthi lecture was delivered on 30th January, 2019, by Sunil K. Pandya, MBBS, MS, F.A.M.S. Neurosurgeon, Department of Neurosurgery, Jaslok Hospital & Research Centre. The lecture entitled - "Physicians who experimented on themselves" was highly informative about the history of physicians who fearlessly performed experiments using themselves as subject to test their hypothesis. This fascinating lecture was followed by interactive session with students and faculty of NBRC.







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. The officer is also responsible for the administration of DIC project personnel.
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, Hindi Week, Vigilance Awareness week, International Yoga Day etc. The Administration achieved excellence in execution of the following activities at NBRC:

- The annual cultural festival of NBRC, 'TANTRIKA 2018' was organized within the campus which included a variety of cultural and sports events. Students, officers, and staff of NBRC participated in the event.
- The keynote address by Professor Vaishna Narang from JNU was organized on 24th September. On 25th September, an evening of Urdu Storytelling by Fouzia was organized. We also had Ms. Subhra Priyadarshini, Chief Editor of Nature India who gave a talk on 26th September.
- Provided necessary logistics in conducting international and national conferences/seminars

organized in the campus as well outside the campus.

- Made major imports from different countries in terms of equipment and other consumables with meticulous planning and adhered to a precise schedule.
- The 15th Foundation Day of NBRC was held on 16th day of December, 2018. On this occasion, several programmes were organized within and outside the campus. The daylong celebrations included the poster presentations on ongoing research activities of NBRC. Students from various schools were invited to interact with NBRC scientists and they visited the laboratories. A quiz programme for students from local schools was also organized on this occasion. On this august occasion, Prof. Semir Zeki, Laboratory of Neurobiology, University College, London, UK delivered the lecture to the students and scientific community at India International Centre, New Delhi.

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 2018-19 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.

General & Academic Administration

Women Empowerment

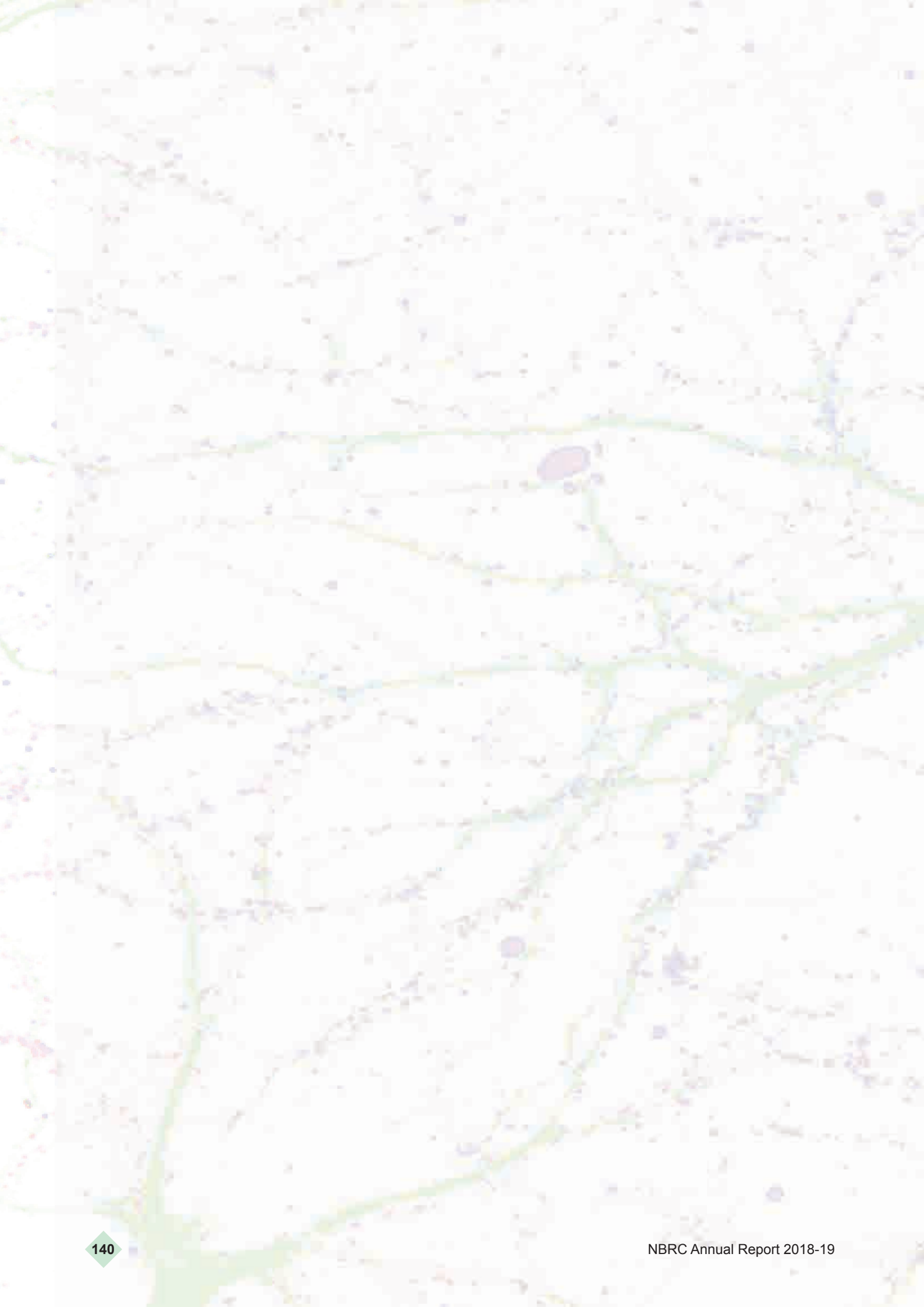
NBRC has a distinct feature of giving equal opportunity to women. The Committees, constituted to do various work of Administration, Academics and scientific activities, have women members on them which ensure fair participation and protection of women. There is a committee for redressal of complaints 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.

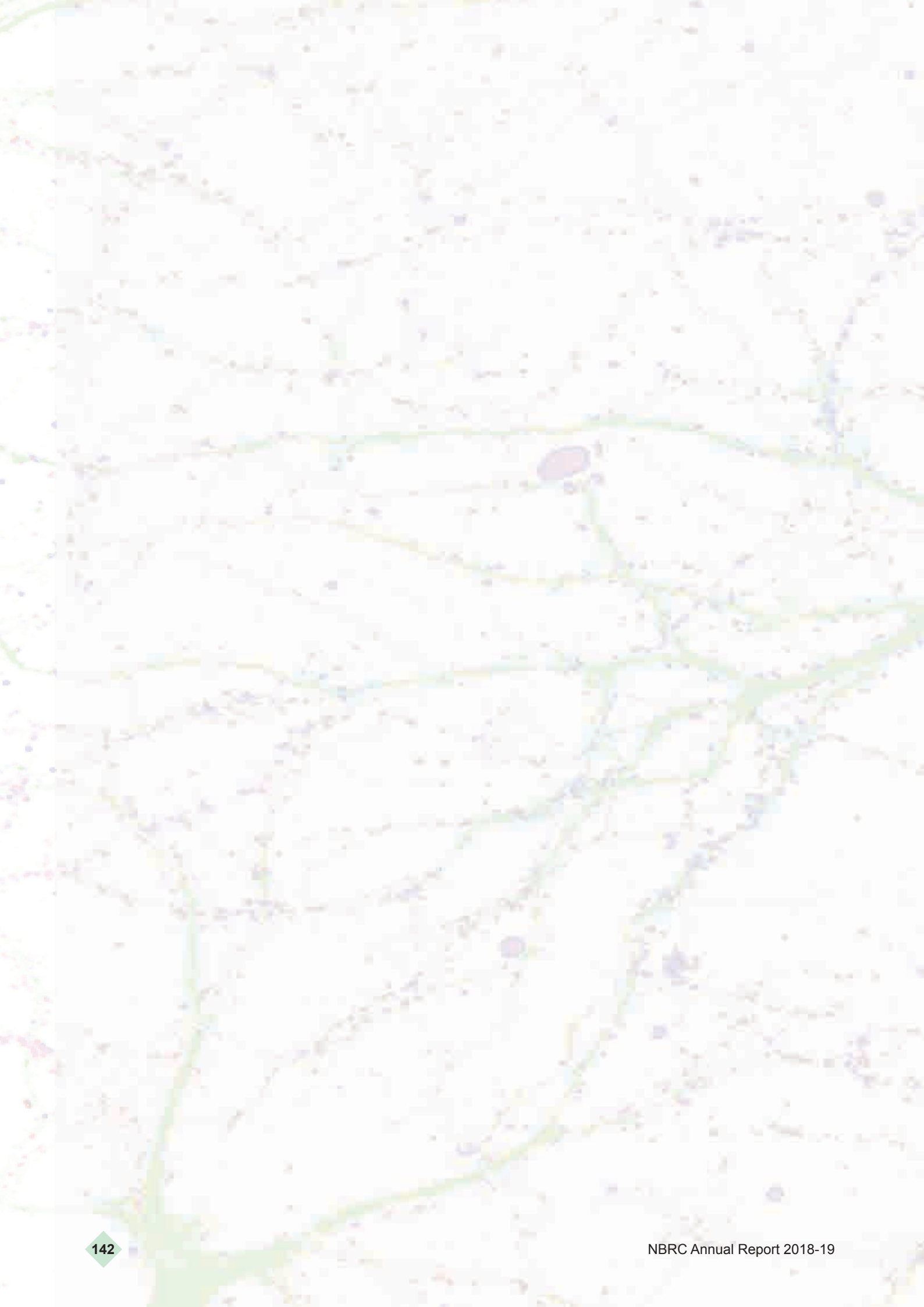
Vigilance

The Institute has a Chief Vigilance Officer. As per the guidelines of DBT, one of the scientists of NBRC has been nominated as Chief Vigilance Officer of the Centre.





Institutional Governance Structure & People at NBRC



Institutional Governance Structure & People at NBRC

Members of NBRC Society

1. Prof. P.N. Tandon (President)
Chairman (Ex-officio)
No. 1, Jagriti Enclave, Vikas Marg,
Delhi – 110 092
2. Dr. Renu Swarup Member (Ex-officio)
Secretary
Department of Biotechnology
CGO Complex, New Delhi – 110 003
3. Prof. Ashutosh Sharma Member (Ex-officio)
Secretary
Department of Science & Technology,
Technology Bhawan, New Delhi – 110 016
4. Prof. Balram Bhargava Member (Ex-officio)
Director General
Indian Council for Medical Research, V.
Ramalingaswamy Bhawan, Ansari Nagar, New
Delhi – 110 029
5. Dr. Sandip K. Basu
JC Bose Chair Professor
National Institute of Science Communication &
Information Resources (NISCAIR)
New Delhi- 110 067
6. Sh. B. Anand Member (Ex-officio)
Additional Secretary & Financial Advisor,
Department of Biotechnology
CGO Complex, New Delhi – 110 003
7. Dr. Shekhar Mande Member (Ex-officio)
Director General, CSIR and Secretary DSIR
Anusandhan Bhavan
New Delhi- 110 001
8. Dr. Sundeep Sarin, (Ex-Officio)
Scientific Coordinator of NBRC
Scientist - G
Department of Biotechnology,
New Delhi
9. Dr. M. Gourie Devi
Director (Retd.)
Flat-9, Doctors Apartment
Vasundhara Enclave
Delhi-110 096
10. Dr. L. M. Patnaik
CSA Department
Indian Institute of Science
Bangalore- 560 012
11. Dr. Kalluri Subba Rao
(INSA Hon. Scientist & Professor)
School of Medical Sciences
University of Hyderabad
Hyderabad- 500 046
12. Prof. K. VijayRaghavan
Principal Scientific Adviser to the Government
of India
Vigyan Bhavan Annexe, Maulana Azad Road,
New Delhi - 110011
13. Prof. Neeraj Jain,
Member Secretary(Ex-Officio)
Director
National Brain Research Centre,
Manesar – 122 052, Haryana

Governing Council

1. Dr. Renu Swarup, Chairperson (Ex-Officio)
Secretary
Department of Biotechnology,
CGO Complex, New Delhi – 110 003.
2. Prof. P.N. Tandon (Ex-Officio)
No. 1, Jagriti Enclave, Vikas Marg,
Delhi – 110 092.
3. Prof. Upinder S. Bhalla
Scientist
National Centre for Biological Sciences (NCBS)
Bangalore- 560 065
4. Prof. Dinakar M. Salunke
Director
International Centre for Genetic Engineering
and Biotechnology,
New Delhi – 110 067
5. Dr. A. K. Agarwal
Dean, Director, Professor & HOD (Retd.)
N-9, Green Park Main, New Delhi
6. Prof. G. Mehta, FNA, FRS
Bhartia Chair School of Chemistry,
University of Hyderabad, Hyderabad 500046
7. Dr. Chitra Sarkar
Department of Pathology,
All India Institute of Medical Sciences,
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8. Prof. Seyed E. Hasnain
Vice Chancellor
Jamia Hamdard University, Mehrauli - Badarpur
Road, Near Batra Hospital, Hamdard Nagar,
Delhi 110062
9. Prof. Ashutosh Sharma (Ex-Officio)
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Department of Science & Technology,
Technology Bhawan, New Mehrauli Road, New
Delhi – 110 016,
10. Prof. Balram Bhargava (Ex-Officio)
Director General
Indian Council for Medical Research, V.
Ramalingaswamy Bhawan, Ansari Nagar, New
Delhi – 110 029
11. Sh. B. Anand(Ex-Officio)
Additional Secretary & Financial Advisor,
Department of Biotechnology,
New Delhi – 110003
12. Nominee of UGC
13. Dr. Sundeep Sarin, (Ex-Officio)
Scientific Coordinator of NBRC
Scientist - G
Department of Biotechnology,
New Delhi
14. Sh. C. P. Goyal (Ex-Officio)
Joint Secretary (Admin)
Department of Biotechnology
CGO Complex, New Delhi – 110 003
15. Prof. Sanjeev Jain (Special Invitee)
Molecular Genetics Laboratory, Department of
Psychiatry
National Institute of Mental Health and
Neuroscience (NIMHANS)
Bangalore- 560029
16. Prof. Neeraj Jain, Member Secretary (Ex-
Officio)
Director
National Brain Research Centre
Manesar – 122 052, Haryana

Finance Committee

1. Sh. B. Anand, Chairman (Ex-officio)
Additional Secretary & Financial Advisor,
Department of Biotechnology,
New Delhi – 110003
2. Dr. Sundeep Sarin, (Ex-Officio)
Scientific Coordinator of NBRC
Scientist - G
Department of Biotechnology,
New Delhi
3. Prof. Dinakar M. Salunke,
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Vice Chancellor
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Delhi 110062
5. Prof. Neeraj Jain, (Ex-officio)
Director
National Brain Research Centre,
Manesar – 122 052, Haryana
6. Finance & Account Officer,
Non-Member Secretary (Ex-officio)
National Brain Research Centre,
Manesar-122052, Haryana

Building Committee

1. Prof. Dinakar M. Salunke (Chairperson)
Director
International Centre for Genetic Engineering
and Biotechnology
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2. Dr. Amulya K. Panda
Director,
National Institute of Immunology (NII),
New Delhi – 110067
3. Dr. S. K. Gupta
Deputy Director (Retired) & Emeritus Scientist
National Institute of Immunology (NII),
New Delhi – 110067
4. Sh. V. H. Rao
Senior Consultant Engineering,
Mehrauli - Badarpur Road,
Near Batra Hospital, Hamdard Nagar,
New Delhi – 110062
5. Mr. M. K. Gupta
Engineer-In-Charge (Civil),
IUAC, New Delhi
6. Prof. Sidhartha Satpathy
HOD Hospital Administration
AIIMS, New Delhi
7. Prof. Neeraj Jain, (Ex-Officio)
Director
National Brain Research Centre
Manesar - 122052

Scientific Advisory Committee

Prof. P. N. Tandon (Chairperson)
President, NBRC Society
No. 1, Jagriti Enclave, Vikas Marg,
Delhi – 110 092

Prof. Upinder S. Bhalla (Co-Chairperson)
Scientist
National Centre for Biological Sciences (NCBS),
Bellary Road, Bangalore - 560 065

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Principal Scientific Adviser to the Government of
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Medicine (inStem), CCMB, TIFR, GKVK, Bellary
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West Bengal - 721302

Dr. Sundeep Sarin, (Ex-Officio)
Scientific Coordinator of NBRC
Scientist - G
Department of Biotechnology,
New Delhi

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Faculty of Medicine, Department of Medicinal
Genetics, University of Geneva, 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,
California, USA 92037

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

Academic Council

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Manesar, Haryana

Prof. Sudha Bhattacharya
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Jawaharlal Nehru University,
New Delhi

Prof. Ishan Patro
School of Studies in Zoology / Neuroscience
Jiwaji University,
Gwalior

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Department of Biotechnology
Guru Nanak Dev University,
Amritsar

Prof. Soumya Iyengar
National Brain Research Centre
Manesar, Haryana

Prof. Anirban Basu
National Brain Research Centre
Manesar, Haryana

Prof. Ellora Sen
National Brain Research Centre
Manesar, Haryana

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

Prof. Nihar Ranjan Jana (Till 25/05/2018)
National Brain Research Centre
Manesar, Haryana

Dr. Yoganarasimha Doreswamy (Till 22/11/2018)
National Brain Research Centre
Manesar, Haryana

Prof. Pravat K. Mandal
National Brain Research Centre
Manesar, Haryana

Dr. Sourav Banerjee
National Brain Research Centre
Manesar, Haryana

Prof. Pankaj Seth
National Brain Research Centre
Manesar, Haryana

Dr. Arpan Banerjee
National Brain Research Centre
Manesar, Haryana

Prof. Shiv K. Sharma
National Brain Research Centre
Manesar, Haryana

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

Board of Studies

Prof. Neeraj Jain
Director,
National Brain Research Centre
Manesar, Haryana

Prof. Krishnamurthy Natarajan
Director,
Ambedkar Centre for Biomedical Research,
University of Delhi, New Delhi

Prof. Chitra Sarkar
Department of Pathology
All India Institute of Medical Sciences,
New Delhi

Prof. Soumya Iyengar
National Brain Research Centre
Manesar, Haryana

Prof. Anirban Basu
National Brain Research Centre
Manesar, Haryana

Prof. Ellora Sen
National Brain Research Centre
Manesar, Haryana

Prof. Nihar Ranjan Jana (Till 25/05/2018)
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Manesar, Haryana

Dr. Arpan Banerjee
National Brain Research Centre
Manesar, Haryana

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

DST-INSPIRE Faculty

1. Dr. Yogita Kapil Adlakha
2. Dr. Swagata Dey

Ph.D. Students

1. Mr. Apoorv Sharma
2. Mr. Sandeep Kumar
3. Mr. Bharat Prajapati
4. Mr. Brijesh Kumar Singh (Till 21/05/2018)
5. Mr. John Thomas
6. Mr. Kautuk Kamboj
7. Mr. Biswaranjan Sahoo
8. Mr. Indrajith R. Nair
9. Ms. Shalini Swaroop (Till 14/11/2018)
10. Mr. Shashi Shekhar Kumar
11. Mr. Touseef Ahmad Sheikh
12. Mr. Tushar Arora
13. Mr. S Balakumar
14. Ms. Arti Kumari
15. Mr. Dharmendra Puri
16. Ms. Mukta Kumari
17. Mr. Raghav Shankar
18. Md. Tipu Khan
19. Mr. Amit Ranjan (Till 20/09/2017)
20. Ms. Priyanka Ghosh
21. Ms. Sarbani Samaddar
22. Ms. Shruti Patrick
23. Mr. Surajit Chakraborty
24. Ms. Bindu
25. Mr. Shiladitya Laskar
26. Mr. Sibaram Behera
27. Ms. Tripti Joshi
28. Mr. Abhishek Singh Narvaria
29. Ms. Deepti Dama
30. Mr. Jithin D Nair (Till 24/09/2018)
31. Mr. Karthick R
32. Ms. Nisha Chetana Sastry
33. Ms. Shivangi Sharma
34. Ms. Sunanda Sharma
35. Mr. Sushanta Majumder
36. Ms. Vanshika Singh
37. Ms. Himali Arora
38. Ms. Meenakshi Bhaskar
39. Mr. Neeraj Kumar
40. Mr. Anil Kumar (Till 28/09/2018)
41. Ms. Dipanjana Banerjee
42. Ms. Gargi Majumdar
43. Ms. Kamakshi Garg

44. Ms. Khushboo Vinod Punjabi
45. Ms. Partika
46. Ms. Roopashi Saxena
47. Ms. Shalini Sharma
48. Ms. Stuti Mohapatra

Integrated Ph.D. Students

1. Ms. Guncha Bhasin
2. Ms. Shankhamala Sen
3. Ms. Uzma Din
4. Ms. Chitra Mohinder Singh Singal
5. Ms. Utkarsha A Singh
6. Ms. Pooja Parishar
7. Mr. Apurva Agrawal
8. Mr. Atanu Datta
9. Mr. Naman Vatsa (Till 04/04/2019)
10. Mr. Hriday Shanker Pandey
11. Mr. Abhishek Kumar Verma
12. Mr. Vikas Pareek
13. Ms. Reshma Bhagat
14. Mr. Vipendra Kumar
15. Ms. Atrayee Basu
16. Ms. Priyanka
17. Mr. Gourav Sharma
18. Ms. Harjot Kaur
19. Mr. Pruthvi S.G
20. Ms. Shelly Pal
21. Mr. Shubham Krishna

M.Sc. Students

1. Mr. Anagh Pathak (Till 31/07/2018)
2. Ms. Kasturi Biswas (Till 29/06/2018)
3. Ms. Kirti (Till 31/07/2018)
4. Ms. Ritu Moni Borah (Till 08/08/2018)
5. Ms. Sreyashi Chandra (Till 05/07/2018)
6. Ms. Tapasya Pal (Till 29/06/2018)
7. Ms. Varsha Ramakrishna (Till 14/06/2018)
8. Ms. Vini Tiwari (Till 01/08/2018)
9. Mr. Azman Akhter
10. Ms. Guneet Kaur
11. Ms. Kirti Saluja
12. Mr. Masood Ahmad Wani
13. Ms. Pallavi Singh
14. Mr. Ranjit Pradhan
15. Ms. Akanksha Goyal
16. Ms. Akanksha Gupta
17. Mr. Ankit Dhoundiyal
18. Mr. Thakar Darshit Mahesh
19. Mr. Mantosh Patnaik

20. Ms. Mishaben Parmar
21. Mr. Nitish Kumar
22. Ms. Pratibha Ahirwal
23. Ms. Rekha Singh
24. Ms. Rishika Tiwari
25. Ms. S Indira Priya
26. Ms. Sharmistha Panda
27. Ms. Shashwati Tripathi
28. Ms. Surbhi
29. Ms. Vinsea A V Singh

Project Assistant

1. Mr. Alok Nath Mohapatra (Till 29/06/2018)
2. Ms. Kanza Saleem (Till 22/06/2018)
3. Ms. Hajare Nilambari Anil (Till 25/06/2018)
4. Mr. Shrey Dutta (Till 14/09/2018)
5. Mr. Archith Rajan (Till 13/11/2018)
6. Ms. Titash Mukherjee (Till 01/11/2018)
7. Mr. Irshad Akbar (Till 22/02/2019)
8. Ms. Hymavathy B. (Till 21/08/2018)
9. Mr. Siddharth Talwar (Till 31/01/2019)
10. Ms. Anuradha Mehta (Till 23/08/2018)
11. Ms. Sujata Dev (Till 31/01/2019)
12. Ms. Revathi M (Till 22/05/2018)
13. Mr. Nandu Raj P (Till 28/02/2019)
14. Ms. Asha S C (Till 27/02/2019)
15. Ms. Keerthana. P
16. Mr. Vivek P. Krishnan (Till 21/12/2018)
17. Ms. Sonia (Till 01/08/2018)
18. Mr. Subhajit Jana
19. Mr. Utsav Mukherjee
20. Ms. Vaishali (Till 28/05/2018)
21. Dr. Fahd M Yasin
22. Ms. Ritu Nayak
23. Ms. Madhura S Rao (Till 17/07/2018)
24. Ms. Nicky Singh (Till 05/07/2018)
25. Mr. Bikash Chandra Sahoo (Till 29/06/2018)
26. Ms. Aditi Charak (Till 20/06/2018)
27. Mr. Jayakrishnan U (Till 10/09/2018)
28. Dr. Bahaar Meera Jain
29. Ms. Divya Dwivedi
30. Ms. Fathima Murshida P
31. Ms. Priya Maddhesiya
32. Mr. Vivek Sharma
33. Ms. Karnika Gupta
34. Ms. Smriti Bhardwaj
35. Mr. Devashish Arvind Pande
36. Mr. Paritosh Jaiswal
37. Mr. Sharan G

Research Associates

1. Dr. Chetan Kumar Yadav, Research Associate-3
2. Dr. D. Subhashree, Research Associate-2
3. Dr. Jeffrey Michael Valla, Research Associate-2 (Till 07/09/2018)
4. Dr. Sandeep Kumar, Research Associate-2 (Till 05/04/2018)
5. Dr. Bibhabasu Hazra, Research Associate-3 (Till 31/05/2018)
6. Dr. Dipanjan Ray, Research Associate-3
7. Ms. Deepali Singh, Research Associate-2
8. Mr. P. Premkumar, Research Associate-2 (Till 07/12/2018)
9. Dr. Sonika, Research Associate-1 (Till 22/10/2018)
10. Dr. Romita Thounaojam, Research Associate-1 (Till 23/07/2018)
11. Dr. Navinder Kumar, Research Associate-1 (Till 30/04/2019)
12. Dr. Moumita Das, Research Associate-2
13. Mr. M. Gopi
14. Dr. Kanu Megha
15. Dr. Sonika

Research Associate (Project)

1. Dr. Prem Chand
2. Dr. Bibhabasu Hazra
3. Dr. Amit Naskar
4. Dr. Soibam Shyamchand Singh
5. Dr. Shubham Kumar
6. Dr. Rituparna Chaudhuri

SERB-National Post Doctoral Fellowship

1. Dr. Akansha Jalota (Till 02/04/2018)
2. Dr. Suvadip Mallick (Till 31/08/2018)
3. Dr. Amit Naskar (Till 31/07/2018)
4. Dr. Ashok Kumar Datusalia (Till 03/04/2018)
5. Dr. Soibam Shyamchand Singh (Till 02/05/2019)
6. Dr. Sandeep Kumar

Research Fellows

1. Ms. Reshma Bhagat (From 30/04/2019 till 29/10/2019)
2. Mr. G. Vinodh Kumar (From 01/02/2019 till 31/07/2019)

Project Employees

1. Ms. T. Ammaponnu Sumathi, R&D Engineer (Project)
2. Mr. Kamal Bharti, Technologist (MEG Project) (Till 15/03/2019)
3. Mr. Vibhin V., Technologist (MEG Project) (Till 09/08/2018)
4. Ms. Km. Ruchika Mittal, Data Entry Coordinator (Project) (Till 22/11/2018)
5. Mr. Manjit, Lab Attendant (MEG Project)
6. Mr. Rakesh Yadav, Nursing Orderly (MEG Project)
7. Mr. Ashok Kumar, Nurse (MEG Project)
8. Mr. Gaurav Singh, Technician (MEG Project)
9. Mr. Vivek Singh, Technician (MEG Project)
10. Mr. Om Prakash Jakhar, Nurse (MEG Project)
11. Ms. Mini Mohan, Nurse (MEG Project) (Till 22/03/2019)
12. Mr. Arun E V R, Senior Research Fellow (Project)
13. Mr. Sukhvir Singh Pundir, Technical Associate (Computer / IT)
14. Mr. Prem Chand, Accounts Administrative Assistant (DeLCON Project) (Till 28/11/2018)
15. Ms. Ragini, Lab Technical Assistant (Project) (Till 01/02/2019)
16. Ms. Ankita Sharma, R&D Scientist (Project) (Till 22/11/2018)
17. Ms. Deepika Shukla, Research Scientist (Project)
18. Mr. Krishan Sharma, Technician A (Project)
19. Mr. Dixit Sharma, Junior Research Fellow (Project)
20. Ms. Teesta Naskar, ICMR-SRF (Project)
21. Ms. Sriparna Mukherjee, DST-INSPIRE Fellow Senior Research Fellow (Project)
22. Dr. Uday Pratap, Research Associate (Project) (Till 02/04/2018)
23. Mr. Prasann Jeet, Lab Attendant (Project)
24. Mr. Ibrahim Olabayode Saliu, DBT-TWAS Sandwich Postgraduate Fellowship (Till 13/03/2019)
25. Ms. Shalini, Technical Assistant (Project)
26. Mr. Neeraj Kasana, Technical Assistant (Project)
27. Ms. Lahoti Ritu Kamalkishor, R&D Engineer (Project) (Till 24/07/2018)
28. Ms. Khushboo Vinod Punjabi, R&D Engineer (Project) (Till 31/07/2018)
29. Dr. Anwesha Banerjee, Senior R&D Engineer (Project) (Till 08/06/2018)
30. Dr. Gueyraud Rolland Kipre, Doctoral Fellowship (Till 05/07/2018)
31. Ms. Shallu, Neuropsychologist (Project)
32. Ms. Anshika Goel, Senior R&D Engineer (Project)
33. Mr. Praful P Pai, R&D Engineer-1 (Project)
34. Ms. Tripti Goel, Research Scientist (Project) (Till 26/06/2018)
35. Ms. Kanika Sandal, Research Manager (Project)
36. Mr. Saurav Roy, R&D Engineer (Project)
37. Mr. Chen Chongtham, Research Assistant (Project)
38. Mr. Jayakrishnan U, R&D Engineer (Project)
39. Mr. Ritwick Mishra, Clinical Coordinator (Project)
40. Ms. Radhika Shivhare, Senior R&D Engineer (Project)
41. Ms. Avinash Kalyani, R&D Engineer (Project)
42. Mr. Prem Chand, Manager (Project)
43. Mr. Jatin Rathore, Technologist (Project)
44. Ms. Priya Shrivastav, Nurse (Project)
45. Ms. Ruchika Mittal, Programmer (Project)
46. Mr. Rajnish Kumar, Technical Assistant (Project)

Scientific Staff

1. Prof. Neeraj Jain, Director, (w.e.f. 21/12/2018)
2. Prof. Nihar Ranjan Jana, Scientist – VII, (Relieved on 25/05/2018)
3. Prof. Pravat Kumar Mandal, Scientist – VII
4. Prof. Pankaj Seth, Scientist – VII
5. Prof. Shiv Kumar Sharma, Scientist – VI
6. Prof. Nandini C. Singh, Scientist-VI (Working at UNESCO, New Delhi on deputation basis)
7. Dr. Ellora Sen, Scientist – VI
8. Prof. Soumya Iyengar, Scientist – VI
9. Prof. Anirban Basu, Scientist – VI
10. Dr. Ranjit Kumar Giri, Scientist – VI
11. Dr. Yoganarasimha Doreswamy, Scientist – V, (Relieved on 22/11/2018)
12. Dr. Sourav Banerjee, Scientist – V
13. Dr. Arpan Banerjee, Scientist – V
14. Dr. Anindya Roy Ghosh, Scientist – V
15. Dr. Dipanjan Roy, Scientist - IV
16. Mr. Mahender Kumar Singh, Information Scientist

Technical Staff

1. Mr. Rajbir Singh, Superintending Engineer
2. Mr. Sanjeev Kumar Choudhary, Assistant Engineer
3. Dr. D.D. Lal, Technical Officer
4. Mr. Jitender Ahlawat, Technical Officer – B
5. Mr. Arvind Singh Pundir, Technical Officer – B
6. Dr. Inderjeet Yadav, Veterinarian
7. Mr. Kedar Singh Bajetha, Computer Operator
8. Ms. Seepika, Computer Operator
9. Mr. Sachin Kumar, Computer Operator
10. Mr. Amit Kumar Gaurav, Computer Operator, (Relieved on 31/05/2018)
11. Ms. Tarnnum Mansoori, Computer Operator
12. Mr. Sanjeev Bhardwaj, Computer Operator
13. Mr. Kanhaiya Lal Kumawat, Technician-C
14. Mr. Shankar Dutt Joshi, Technician-C
15. Mr. Sumit Kumar Sinha Mahapatra, Technician-C
16. Mr. D. Narender, Technician-C
17. Mr. Sanjay Kumar, Technician-B
18. Mr. Mithlesh Kumar Singh, Technician-B
19. Mr. Ankit Sharma, Technician-B
20. Mr. Yunis Khan, Technician-B
21. Mr. Durga Lal Meena, Technician-B
22. Md. Irshad Alam, Technician-B
23. Mr. Manish Kumar, Technician-B
24. Mr. P. Manish, Technician-B
25. Mr. Dil Bahadur Karki, Technician-A
26. Mr. Rammehar, Technician-A
27. Mr. Hari Shankar, Technician-A
28. Mr. Mahendra Singh, Technician-A
29. Mr. Sanjay Kumar Singh, Technician-A

Administrative Staff

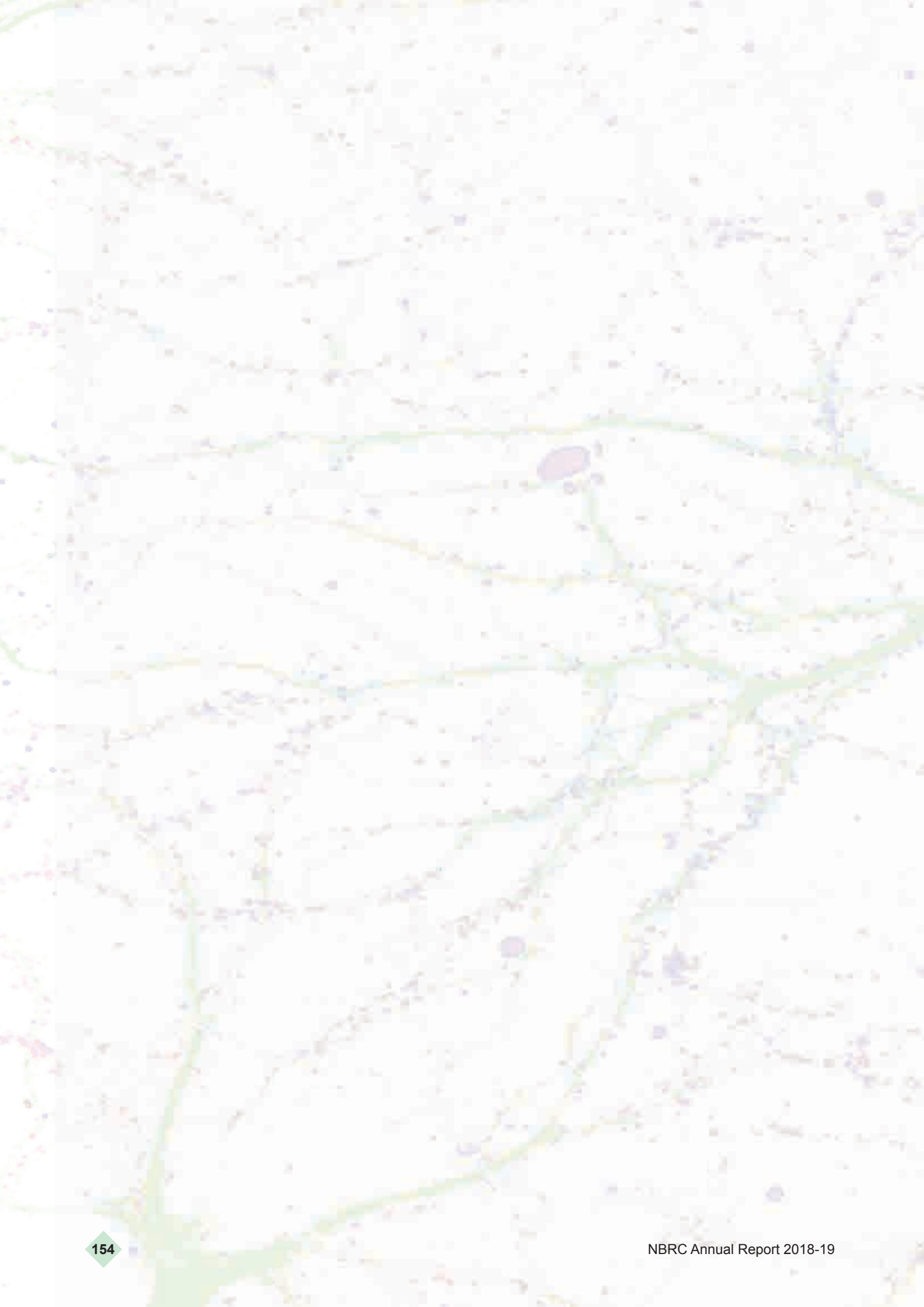
1. Mr. Tanmoy Bhattacharyya, Chief Administrative Officer
2. Mr. Santosh Kumar Choudhary, Deputy Finance Officer
3. Mrs. Pooja Gosain, Administrative Officer
4. Mr. Ravinder Pal, Stores & Purchase Officer
5. Mr. Suraj Bhan, Office Assistant
6. Mr. Rakesh Kumar Yadav, Office Assistant
7. Mr. Himanshu Mal, Office Assistant (Working at ICSSR, New Delhi on deputation basis)
8. Mr. Ajay Kumar Dehariya, Office Assistant (Working at DTU, New Delhi on deputation basis)
9. Mr. Parmander Singh Rawat, Office Assistant
10. Mr. Jitendra Kumar Meena, Office Assistant
11. Mr. Sanjay Kumar Gupta, Office Assistant (w.e.f. 01/03/2019)
12. Mr. Kailash Chandra Khuntia, Office Assistant (On Lien vacancy & Relieved on 27/02/2019)
13. Mr. Bhupender Pal Sharma, Driver
14. Mr. Satish Kumar, Driver
15. Mr. Surender Kumar, Driver (Relieved on 31/07/2018)

DIC Project Staff

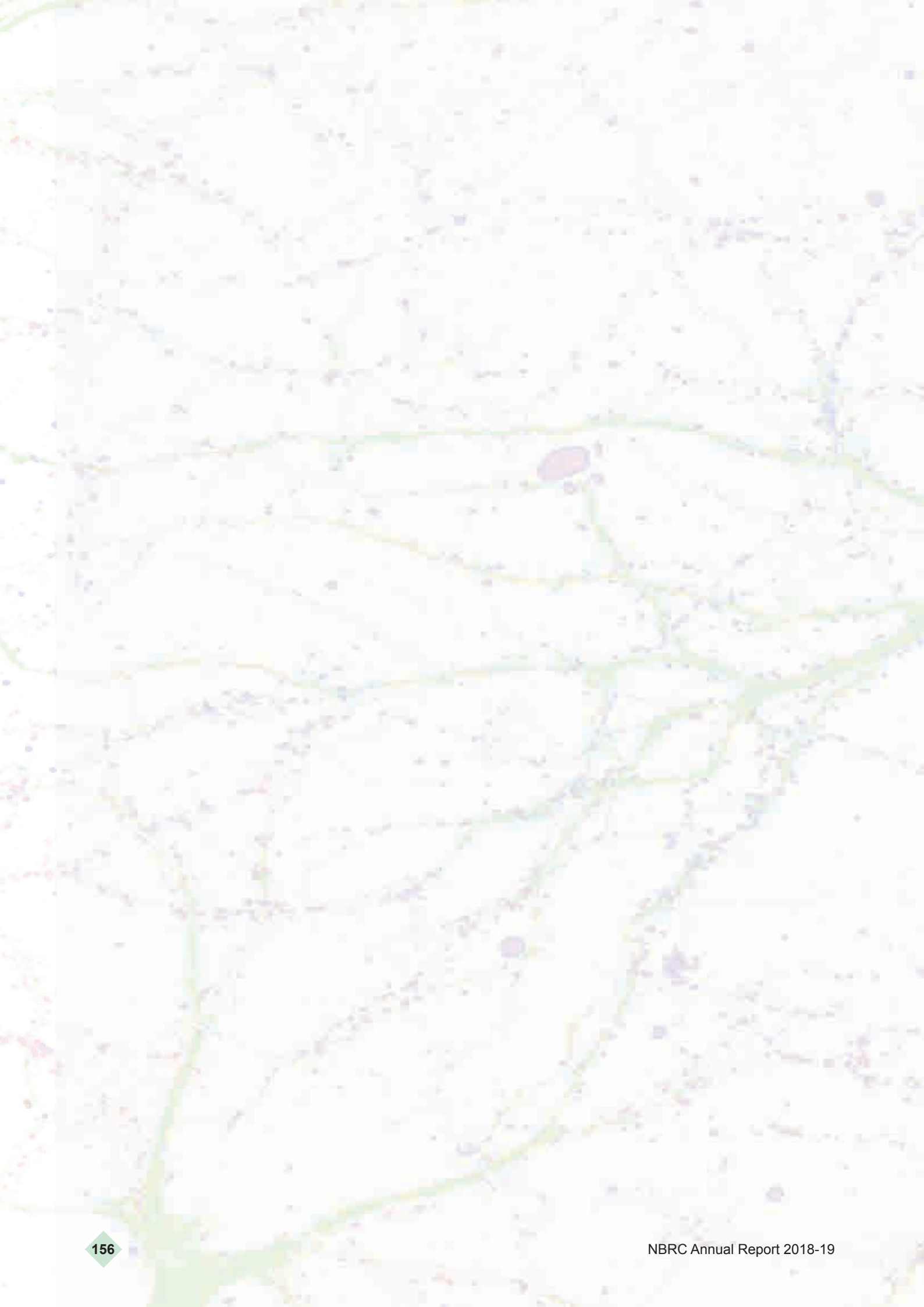
1. Mr. Sanjay Kumar Gupta, Assistant Administrative Officer (Relieved on 28/02/2019)
2. Ms. Reema Saxena, Computer Operator
3. Ms. Sunita Yadav, Computer Operator
4. Mr. Amit Kumar, Computer Operator
5. Mr. R. Ganesh Gurumoorthy, Computer Operator

Contract Employees

1. Dr. Rema Velayudhan, Sr. Consultant (w.e.f. 19/12/2018)
2. Dr. P. Raghunathan, Consultant
3. Dr. Karan Singh, Veterinarian at the level of application specialist (Relieved on 02/05/2018)
4. Mr. Hanish Kumar Sauda, Management Assistant (Admin.) (Relieved on 12/03/2019)
5. Mr. Mukesh Chauhan, Management Assistant (Acad.)



Annual Financial Statements



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, 2019, 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 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, 2019;

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 two cases which are 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.

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)

Date: 09th September, 2019
Place: New Delhi

(PARTNER)
CA ABHISHEK GOEL
M. NO. 412467

National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Balance Sheet As At March 31,2019

(Amount-Rs.)

CORPUS / CAPITAL FUND AND LIABILITIES	Schedule	Current Year	Previous Year
Corpus/Capital Fund	1	1,373,502,000.00	1,243,502,000.00
Reserve and Surplus	2	(79,172,116.60)	(114,354,308.02)
Earmarked/Endowment Funds	3	911,744,376.60	1,187,969,732.29
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	43,017,897.24	46,763,755.48
Total (Liabilities)		2,249,092,157.24	2,363,881,179.75
ASSETS			
Fixed Assets	8	1,172,321,456.15	1,228,875,012.89
Investments - From Earmarked/Endowment Funds	9	0.00	0.00
Investments-Others	10	29,112,172.79	31,526,119.18
Current Assets, Loans, Advances etc.	11	1,047,658,528.30	1,103,480,047.68
Miscellaneous Expenditure (to the extent not written off or adjusted)		0.00	0.00
Total (Assets)		2,249,092,157.24	2,363,881,179.75
Significant Accounting Policies	24		
Contingent Liabilities and Notes on Accounts	24		

Santosh Kumar Choudhary
Dy. Finance Officer & Offg. F&Ao

Prof. Neeraj Jain
Director

As Per Our Separate Report Of Even Date Attached

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

Abhishek Goel
Partner
Membership No. 412467
Date: 09.09.2019
Meerut

National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Income And Expenditure Account For The Year Ended March 31, 2019

(Amount-Rs.)

INCOME	Schedule	Current Year	Previous Year
Income from Sales/Services	12	0.00	0.00
Grants/ Subsidies (Revenue) from DBT	13	320,000,000.00	277,100,000.00
Fees/Subscriptions	14	3,316,303.00	2,045,382.78
Income from Investments (Income on Invest. From earmarked/endow. Funds transferred to funds)	15	939,240.00	2,950,807.80
Income from Royalty, Publication etc.	16	0.00	0.00
Interest Earned	17	20,982,261.64	19,673,173.88
Other Income	18	4,701,483.00	2,153,676.84
Increase/(decrease) in stock of Finished goods and work-in-progress	19	0.00	0.00
Total Income (A)		349,939,287.64	303,923,041.30
EXPENDITURE			
Establishment Expenses	20	86,294,023.00	88,310,380.00
Other Administrative etc.	21	189,055,416.22	167,298,103.71
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)		32,446,934.00	33,002,815.00
Total Expenditure (B)		307,796,373.22	288,611,298.71
Balance being excess of Income over Expenditure (A-B)		42,142,914.42	15,311,742.59
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		42,142,914.42	15,311,742.59
Significant Accounting Policies	24		
Contingent Liabilities and Notes on Accounts	24		

Santosh Kumar Choudhary
Dy. Finance Officer & Offg. F&Ao

Prof. Neeraj Jain
Director

As Per Our Separate Report Of Even Date Attached

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

Abhishek Goel
Partner
Membership No. 412467
Date: 09.09.2019
Meerut

National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Receipts And Payments For The Year Ended March 31, 2019

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	273,188.00	160,755.00		i)	Establishment Expenses	10,418,602.00	8,891,249.00
	b)	Bank Balances				ii)	Administrative Expenses	2,972,287.21	3,533,097.21
		i) In Deposit Accounts	-	-	II.	Payment Made Against Funds For Various Projects			
		ii) Saving Accounts	1,083,725,273.34	494,595,608.23		i)	Recurring / Capital expenditure	91,895,138.46	29,284,337.05
		iii) CPF Investments	31,526,119.18	19,537,816.00		ii)	Capital Grant Refunded to DBT		
						iii)	Refund to RCGB		
II.	Grants Received					iv)	Refund of Interest	26,633,897.00	
	a)	From Government of India			III.	Maintenance Cost			
		Plan				i)	Lab Maintenance Expenses	24,885,124.56	40,613,374.66
	i)	Recurring Income	320,000,000.00	277,100,000.00		ii)	Office Maintenance	44,280,071.00	47,799,068.00
	ii)	Non-Recurring Income	130,000,000.00	150,000,000.00		iii)	Vehicle Running & Maintenance	689,915.00	638,975.00
		Plan (Recurring)			IV.	Investment and Deposit Made			
	b)	Fellowship Grant	2,346,598.00	1,085,280.00		i)	Out of Earmarked/ Endowment funds	37,253,214.00	1,316,018.00
	c)	Delcon Projects (Including Interest)	477,350,987.00	765,764,625.00	V.	Expenditure of Fixed Assets & Capital Work-in-progress			
III.	Receipt made against funds for various projects					i)	Purchase of Fixed Assets	9,587,767.99	6,167,743.00
	i)	Recurring Receipt/ Capital Grant (Including Interest)	45,566,732.16	262,855,562.00	VI.	Training Expenses		2,054,761.93	2,934,731.06
	ii)	Bank Deposits	269,250,000.00	-	VII.	Other Payments (Specify)			
IV.	Interest Received					i)	Advances to Supplier	13,005,547.75	6,310,579.14
	i)	On Bank Deposits		-		ii)	Advances to Staff	3,733,709.87	3,701,364.00
	ii)	Savings Account	12,652,633.10	19,295,909.00		iii)	Leave Encashment/ LTC/ Bonus	676,169.00	403,019.00
	iii)	On CPF Fund	7,298,493.41	1,278,245.59		iv)	Security Deposit Paid	378,656.00	527,296.00
	iv)	Other Interest	140,382.00	149,980.00		v)	EMD Refunded	2,883,827.00	3,673,353.00

V.	Any Other Receipt				vi)	TDS Paid	60,056,240.00	62,288,998.00
	Indirect Income				vii)	Imprest	181,991.00	180,121.00
	i)	Advance to Supplier Received	219,668.00	12,307.00	viii)	Payment of Current Liabilities	789,065,434.17	673,391,300.33
	ii)	Advance to Staff Received	1,194,170.00	2,129,640.46	ix)	Prepaid Insurance	296,064.00	890,114.00
	iii)	Sale of Tender Documents	404,771.00	30,000.00	VIII.	Closing balances		
	iv)	Misc. Receipts.	1,471,729.00	793,583.00	a)	Cash in Hand	148,195.00	273,188.00
	v)	Earnest Money Deposit Received	1,673,964.00	4,944,980.00	b)	Bank Balance		
	vi)	Sale of Scrap	2,000.00	14,000.00	i)	In Deposit Accounts	719,100,000.00	-
	vii)	Guest House Charges	289,650.00	248,400.00	ii)	Saving Accounts	557,226,483.43	1,083,725,273.34
	viii)	Hostel Deposit	446,000.00	510,000.00	iii)	CPF Investments	9,112,172.79	31,526,119.18
	ix)	CPF Fund Received	12,750,556.00	5,669,097.00				
	x)	Library Deposit	140,000.00	162,000.00				
	xi)	Current Liabilities Rec.	8,555,587.86	785,076.56				
	xii)	Other Receipts	63,650.00	66,484.00				
		TOTAL	2,407,342,152.05	2,007,189,348.84		TOTAL	2,407,342,152.05	2,007,189,348.84

Santosh Kumar Choudhary
Dy. Finance Officer & Offg. F&Ao

Prof. Neeraj Jain
Director

As Per Our Separate Report Of Even Date Attached

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

Abhishek Goel
Partner
Membership No. 412467
Date: 09.09.2019
Meerut

National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 1-Corpus/Capital Fund:			(Amount-Rs.)	
1		Current Year		Previous Year
	Grant-in-Aid - Balance as at the beginning of the year		1,243,502,000.00	1,093,502,000.00
	Add: Contribution towards Corpus/ Capital Fund	130,000,000.00		150,000,000.00
	Add/(Deduct): Balance of net income/(expenditure) transferred from		130,000,000.00	150,000,000.00
	Balance as at the year end		1,373,502,000.00	1,243,502,000.00

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Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 2 - Reserves And Surplus:			(Amount-Rs.)	
		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 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
4 General Reserve				
As per last Account	(114,354,308.02)		(215,139,232.61)	
Addition during the Year	-		85,473,182.00	
Surplus during the year (as per I&E A/c)	42,142,914.42		15,311,742.59	
Less : Deductions during the year (deficit)	6,960,723.00	(79,172,116.60)	-	(114,354,308.02)
Balance as at the year end		(79,172,116.60)		(114,354,308.02)

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National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 3 - Earmarked/Endowment Funds		FUND-WISE BREAK UP					(Amount-Rs.)
		Project Fund	Fixed Assets Fund (Project)	Contributory Provident Fund	Delcon E-library Consortium		
a) Opening Balance of Project Fund		237,903,169.20	341,316,133.60	18,510,585.00			587,608,056.49
b) Additions to the Funds:							
i. Donations/grants/Additions to Fund	34,134,227.40	10,529,027.54		2,750,556.00	469,712,865.00		
ii. Income from investments made on account of funds	0.00	0.00		0.00	0.00		
iii. Other additions (Interest Earned)	4,825,616.00	0.00	10,529,027.54	255,908.00	7,638,122.00		477,350,987.00
Total (a+b)		276,863,012.60	351,845,161.14	21,517,049.00			1,064,959,043.49
c) Utilisation/Expenditure towards objectives of funds							
i. Capital Expenditure							
Fixed Assets (net)	10,389,135.54	0.00		0.00	139,892.00		
Others	0.00	0.00	0.00	0.00	0.00		
Total	10,389,135.54	0.00	0.00	0.00	139,892.00		139,892.00
ii. Revenue Expenditure							
-Salaries, Wages and allowances etc	20,613,850.00	0.00		0.00	1,132,002.00		
-Rent	0.00	0.00		0.00	0.00		
-Others	87,995,988.96	0.00		7,253,214.00	626,485,578.13		
-Depreciation	0.00	52,062,017.00		0.00	0.00		
Total	108,609,838.96	52,062,017.00	7,253,214.00	7,253,214.00	627,617,580.13		627,617,580.13
Total (C)	118,998,974.50	52,062,017.00	7,253,214.00	7,253,214.00	627,757,472.13		627,757,472.13
Net Balance As At The Year-End (A+B-C)	157,864,038.10	299,783,144.14	14,263,835.00	14,263,835.00	437,201,571.36		437,201,571.36

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

Santosh Kumar Choudhary

Dy. Finance Officer & Offg. F&AO

Prof. Neeraj Jain

Director

As Per Our Separate Report Of Even Date Attached

For Maheshwari PA & Associates

Chartered Accountants

(FRN-012023C)

Abhishek Goel

Partner

Membership No. 412467

Date: 09.09.2019

Meerut

Contd....

National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Balance Sheet As At March 31, 2018

FUND-WISE BREAK UP							(Amount-Rs.)	
Schedule 3 - Earmarked/Endowment Funds								
	Endowment Fund for Building		Donation		Current Year		Previous Year	
a) Opening Balance of Project Fund	0.00		2,631,788.00		1,187,969,732.29		897,493,189.08	
b) Additions to the Funds:								
i. Donations/grants/Additions to Fund	0.00	0.00			517,126,675.94		1,038,790,822.31	
ii. Income from investments made on account of funds	0.00	0.00			0.00		0.00	
iii. Other additions (Interest Earned)	0.00	0.00	0.00		12,719,646.00		3,332,143.00	
Total (a+b)		0.00	2,631,788.00		1,717,816,054.23		1,042,122,965.31	
c) Utilisation/Expenditure towards objectives of funds								
i. Capital Expenditure								
Fixed Assets (net)	0.00	0.00			10,529,027.54		7,901,610.19	
Others	0.00	0.00	0.00		0.00		0.00	
Total		0.00	0.00		10,529,027.54		7,901,610.19	
ii. Revenue Expenditure								
-Salaries, Wages and allowances etc	0.00	0.00			21,745,852.00		21,603,921.00	
-Rent	0.00	0.00			0.00		0.00	
-Others	0.00	0.00			721,734,781.09		662,357,019.91	
-Depreciation	0.00	0.00			52,062,017.00		59,783,871.00	
Total		0.00	0.00		795,542,650.09		743,744,811.91	
Total (C)		0.00	0.00		806,071,677.63		751,646,422.10	
Net Balance As At The Year-End (A+B-C)		0.00	2,631,788.00		911,744,376.60		1,187,969,732.29	

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National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule-7 Current Liabilities And Provisions			(Amount-Rs.)	
A.	Current Liabilities	Current Year		Previous Year
	1. Acceptances		0.00	0.00
	2. Sundry Creditors			
	-For Goods	0.00		0.00
	- Others	802,155.00	802,155.00	753,403.00
	3. Advances Received		3,627,996.42	3,562,962.42
	4. Interest accrued but not due on:			
	-Secured Loans/borrowings	0.00		0.00
	-Unsecured loans/borrowings	0.00		0.00
			0.00	0.00
	5. Statutory Liabilities:			
	-Overdue	0.00		0.00
	-Others (TDS payable)	168,081.50		254,136.50
			168,081.50	254,136.50
	6. Others current Liabilities		29,311,362.52	32,745,466.76
	Total (a)		33,909,595.44	37,315,968.68
B.	Provisions			
	1. For Taxation		0.00	0.00
	2. Gratuity		6,137,980.00	6,468,960.00
	3. Superannuation/Pension		0.00	0.00
	4. Accumulated Leave Encashment		2,970,321.80	2,978,826.80
	5. Trade Warranties/Claims		0.00	0.00
	6. Others (Specify)		0.00	0.00
	Total (b)		9,108,301.80	9,447,786.80
	Balance as at the year end (a+b)		43,017,897.24	46,763,755.48

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National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 8 - Fixed Assets/ Depreciaton													
DESCRIPTION		Rate of Dep.	Cost /valuation As at beginning of the Year	GROSS BLOCK			Deductions during the Year	Cost /valuation As at end of the Year	As at the beginning of the Year	DEPRECIATION		NET BLOCK	
				Additions during the Year		On Deductions during the year				Depreciation for current year	Total Depn. Upto 31.03.18	As at Current year-end	As at Previous year-end
A.	Fixed Assets:		More than 6 Months										
	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	0.00	0.00
	b)	On Leasehold Land	71,887,574.00	0.00	0.00	0.00	71,887,574.00	32,730,094.67	3,915,748.00	0.00	36,645,842.67	35,241,731.33	39,157,479.33
	c)	Ownership Flats/Premises	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	d)	superstructures on land	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	not belonging to the entity		0.00				0.00						
	3	Plant Machinery & Equipment	320,973,551.13	2,964,912.05	7,754,127.67	0.00	331,692,590.85	187,539,236.02	21,041,444.00	0.00	208,580,680.02	123,111,910.83	133,434,315.11
4	VEHICLES	2,086,342.00	2,058,692.00	0.00	0.00	4,145,034.00	1,597,033.80	382,200.00	0.00	1,979,233.80	2,165,800.20	489,308.20	
5	FURNITURE, FIXTURES	40,178,952.00	291,770.00	0.00	0.00	40,470,722.00	22,008,326.84	1,846,240.00	0.00	23,854,566.84	16,616,155.16	18,170,625.16	
6	OFFICE EQUIPMENT	45,463,510.95	1,242,868.00	429,707.00	35,000.00	47,101,085.95	21,895,975.65	3,748,539.00	0.00	25,644,514.65	21,456,571.30	23,567,535.30	
7	COMPUTER/PERIPHERALS	8,019,585.81	54,438.00	2,381,627.00	0.00	10,455,650.81	6,548,310.59	1,086,611.00	0.00	7,634,921.59	2,820,729.22	1,471,275.22	
8	ELECTRIC INSTALLATIONS	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
9	LIBRARY BOOKS	0.00	0.00	268,721.00	0.00	268,721.00	0.00	53,744.00	0.00	53,744.00	214,977.00	0.00	
10	TUBEWELLS & W. SUPPLY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
11	OTHER FIXED ASSETS (Patents & Copyrights)	5,182,914.00	47,999.00	124,730.00	0.00	5,355,643.00	3,803,646.75	372,408.00	0.00	4,176,054.75	1,179,588.25	1,379,267.25	
	TOTAL OF THE CURRENT YEAR	493,792,429.89	6,660,679.05	10,958,912.67	35,000.00	511,377,021.61	276,122,624.32	32,446,934.00	0.00	308,569,558.32	202,807,463.29	217,669,805.57	
B	CAPITAL WORK IN PROGRESS (Building)	669,889,073.73	0.00	0.00	158,225.00	669,730,848.73	0.00	0.00	0.00	0.00	669,730,848.73	669,889,073.73	
C	PROJECT EQUIPMENTS	772,267,242.44	998,936.95	9,530,090.59	0.00	782,796,269.98	430,951,108.85	52,062,017.00	0.00	483,013,125.85	299,783,144.13	341,316,133.59	
	TOTAL (A+B+C)	1,935,948,746.06	7,659,616.00	20,489,003.26	193,225.00	1,963,904,140.32	707,073,733.17	84,508,951.00	0.00	791,582,684.17	1,172,321,456.15	1,228,875,012.89	

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

Schedule 9 - Investment From Earmarked/Endowment Funds: NIL

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Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 10 - Investments-Others		(Amount-Rs.)
	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)	29,112,172.79	31,526,119.18
Total	29,112,172.79	31,526,119.18

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Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 11 - Current Assets, Loans, Advances Etc.				(Amount-Rs.)	
A.	Current Assets	Current Year		Previous Year	
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 Debots:				
	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)		148,195.00		273,188.00
4	Bank Balances:				
a)	With Scheduled Banks:				
	-On Current Accounts	0.00		0.00	
	-On Deposit Accounts (includes margin money)	469,020,130.00		0.00	
	-On Savings Accounts	557,226,483.43		1,083,725,273.34	
			1,026,246,613.43		1,083,725,273.34
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)		1,026,394,808.43		1,083,998,461.34

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National Brain Research Centre
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Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 11 - Current Assets, Loans, Advances Etc. (Contd.)				(Amount-Rs.)	
B.	Loans, Advances And Other Assests	Current Year		Previous Year	
1	Loans:				
	a) Staff	6,830,166.87		5,737,970.00	
	b) Other Entities engaged in activities/ objectives similar to that of the entity	0.00		0.00	
	c) Other (Imprest)	55,922.00		63,983.00	
			6,886,088.87		5,801,953.00
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)	1,169,770.00		890,114.00	
	C) Other - Advance to Parties	1,892,638.75		2,246,662.22	
	- Other Advances	5,762,502.45		5,762,502.45	
			8,824,911.20		8,899,278.67
3	Income Accrued:				
	a) On Investments from Earmarked/ Endowment Funds	0.00		0.00	
	b) On Investments-Others	920,860.00		145,628.87	
	c) On Loans and Advances	0.00		0.00	
	d) Others (SB A/C)	6,628.00	927,488.00	89,156.00	234,784.87
b)	(includes income due unrealised- Rs.....)				
4	Claims Receivable (TDS Receivable)		4,625,231.80		4,545,569.80
	Total (B)		21,263,719.87		19,481,586.34
	Total (A+B)		1,047,658,528.30		1,103,480,047.68

Santosh Kumar Choudhary
Dy. Finance Officer & Offg. F&Ao

Prof. Neeraj Jain
Director

As Per Our Separate Report Of Even Date Attached

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

Abhishek Goel
Partner
Membership No. 412467
Date: 09.09.2019
Meerut

National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 12 - Income From Sales/Services		(Amount-Rs.)
	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 Received)	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	320,000,000.00	277,100,000.00
5 International Organisations	0.00	0.00
6 Others (Specify)	0.00	0.00
Total	320,000,000.00	277,100,000.00

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Schedule Forming Part Of Balance Sheet As At March 31, 2019

Schedule 14 - Fees/ Subscriptions		(Amount-Rs.)
	Current Year	Previous Year
1 Entrance Fees	478,205.00	790,917.78
2 Annual Fees/Subscriptions	717,500.00	491,000.00
3 Seminar/Program Fees	0.00	0.00
4 Consultancy Fees	0.00	0.00
5 Others (Fellowship Grants)	2,120,598.00	763,465.00
Total	3,316,303.00	2,045,382.78

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National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Balance Sheet As At March 31, 2018

(Amount-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	Dividends:				
	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	358,250.00	348,941.00
4	Others (Project Receipts)	0.00	0.00	580,990.00	2,601,866.80
	Total (B)	0.00	0.00	939,240.00	2,950,807.80
Transferred To Earmarked/Endowment Funds					

Schedule 16 -Income From Royalty, Publication Etc.

NIL

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Director

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Abhishek Goel
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National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Income And Expenditure For The Year Ended March 31, 2019

Schedule 17 -Interest Earned		(Amount-Rs.)
	Current Year	Previous Year
1 On Term Deposits:		
a) With Scheduled Banks	17,128,965.54	0.00
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	3,712,914.10	19,460,697.88
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	140,382.00	212,476.00
4 Interest on Debtors and Others Receivables	0.00	0.00
Total	20,982,261.64	19,673,173.88

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National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Income And Expenditure For The Year Ended March 31, 2019

Schedule 18 -Other Income		(Amount-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	4,701,483.00	2,083,352.00
5	Prior Period Income	0.00	70,324.84
	Total	4,701,483.00	2,153,676.84

Schedule 19 -Increase/(Decrease) In Stock Of Finished Goods & Work In Progress

NIL

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National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Income And Expenditure For The Year Ended March 31, 2019

Schedule 20 -Establishment Expenses		(Amount-Rs.)	
		Current Year	Previous Year
a) Salaries and Wages		60,969,873.00	67,493,373.00
b) Allowances and Bonous		0.00	0.00
c) Contribution to Provident Fund		1,374,949.00	2,560,398.00
d) Contribution to Pension Scheme		0.00	0.00
e) Staff Welfare Expenses		9,428.00	114,330.00
f) Expenses on Employees Retirement and Terminal Benefits		0.00	0.00
g) Others - Children education reimbursement		1,478,970.00	427,450.00
- Leave encashment		358,134.00	390,035.00
- LTC expenses		1,010,082.00	318,216.00
- Medical reimbursement		1,325,658.00	1,060,163.00
- NPS(employer subscription)		3,679,120.00	3,921,146.00
- overtime allowance		0.00	7,194.00
- Skilled manpower		14,277,821.00	10,844,610.00
- Medical insurance (Staff)		1,084,544.00	665,245.00
- Office expenses		725,444.00	508,220.00
Total		86,294,023.00	88,310,380.00

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Abhishek Goel
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National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Schedule Forming Part Of Income And Expenditure For The Year Ended March 31, 2019

Schedule 21 -Other Administrative Expenses		(Amount-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	38,758,940.00	36,420,887.00
5	Water Charges	0.00	0.00
6	Insurance	443,596.00	503,007.00
7	Repairs and maintenance	49,738,583.39	28,125,094.16
8	Excise Duty	0.00	0.00
9	Rent (Lease Rent), Rates and Taxes	1,405,777.00	1,191,336.00
10	Vehicles Running and Maintenance	94,287.00	268,584.00
11	Postage, Telephone and Communication Charges	644,650.00	760,126.00
12	Printing and Stationary	1,732,733.00	1,761,738.00
13	Travelling and Conveyance Expenses	5,871,140.61	7,034,209.00
14	Expenses on Seminar/Workshops	1,177,531.00	852,433.00
15	Subscription Expenses	2,225,055.82	1,660,355.93
16	Expenses on Fees	0.00	0.00
17	Auditor Remuneration	24,000.00	34,981.00
18	Hospitality Expenses	318,176.00	131,477.00
19	Professional Charges	1,354,118.00	929,795.00
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	2,082,547.32	1,106,638.00
26	Foreign Exchange Fluctuation Loss/Gain	-8,840.52	79,368.56
27	Prior Period Expenses	7,599,724.96	6,454,048.00
28	Others - Bank charges	5,834.80	1,623.21
	- Misc. expenses	229,161.00	268,070.75
	- Books and Periodicals	112,217.00	183,000.00
	- Honorarium (others)	315,112.00	313,000.00
	- Petrol, Diesel & CNG etc.	717,443.00	587,388.00
	- Manpower	12,767,226.00	6,844,930.00
	- Horticulture	3,627,188.00	2,691,761.00
	- Training and networking expense	30,659,449.00	29,153,386.00
	- Laboratory & Animal Consumables	27,159,765.84	39,940,867.10
Total		189,055,416.22	167,298,103.71

SCHEDULE 22 - EXPENDITURE ON GRANTS, SUBSIDIES ETC.

NIL

SCHEDULE 23 - INTEREST PAID

NIL

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Prof. Neeraj Jain
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As Per Our Separate Report Of Even Date Attached

For Maheshwari PA & Associates
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(FRN-012023C)

Abhishek Goel
Partner
Membership No. 412467
Date: 09.09.2019
Meerut

National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
**Significant Accounting Policies & Notes on Accounts Forming Part of The Balance Sheet As At 31st March,
2019 and Income & Expenditure Account for The Year Ended 31st March, 2019**

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 historical cost. 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..
- 3.5 NBRC has entered into a Memorandum of Understanding (MOU) with DC&SEM for construction of NBRC's Building at Manesar, Gurgaon. As per the MOU with the DC&SEM, NBRC is depositing funds with DC&SEM from time to time to be utilized by DC&SEM for construction. Total amount deposited with DC&SEM is Rs. 44,46,52,000.00 till 31st March 2018 , out of which Rs. 1,58,225.00 has been Received back from DC&SEM in F.Y : 2018-19 and remaining amount yet to be settled.

4. Depreciation:

- 4.1 Depreciation provided for current year on the fixed assets of Project for Rs. 5,20,62,017 (previous year Rs. 5,97,83,871.00) 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. 3,24,46,934.00 for current financial year (Rs. 3,30,02,815.00 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, which results in increase in profitability.

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 / subsidy 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. Foreign Currency Transactions/ Grants:

- 7.1 Transactions denominated in foreign currency are accounted at the exchange rate prevailing at the date of the transaction.
- 7.2 Current assets, foreign currency loans and current liabilities are converted at the exchange rate prevailing as at the year end and the resultant gain/loss is adjusted to cost of fixed assets, if the foreign currency liability related to fixed assets, and in other cases is considered to revenue.

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 rent. 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 maximum number of employees joined New Pension Scheme (NPS) and as they had joined NBRC before 01st January, 2004.
- 9.2 The NBRC has not made any provision for gratuity and leave encashment during financial year 2018-2019 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. 29,70,321.80 respectively already exists on 31st March, 2019, (Rs. 64,68,960.00 and Rs. 29,78,826.80 respectively as on 31st March, 2018) against provision made earlier.

10. Taxation:

In view of the tax exemption status of the National Brain Research Centre, no provision for Income Tax had been considered necessary. As The institute is registered under 12A vide Reg no : CIT/FBD/TECH/07-08/12A/80/11/ Dated 14/06/2007

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.

12. Bank Balance:

All Banks accounts have been reconciled till 31st March, 2019.

13. Prior Period Items:

Accounting Standard-5 Issued by Institute of Chartered Accountants of India (ICAI), Prior Period items are income or expenses, which arises, in current period as a result of error or omission in the preparation of financial statement of one or more prior periods. In the current year, the Prior Period items recognized, related to expenditure i.e. Rs. 75,99,724.96 for the financial year 2018-19 (previous year 2017-18 Rs. 63,83,723.16.00) that was omitted in that year.

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, 2019, 28 Projects have already closed which amount of Rs. 66.66 Lakh (Debit Balance) is still pending and 13 Projects closed which amount of Rs. 1.41 Cr is 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. 54,12,350.00 (Previous year Rs. 51,62,410.00) which is under representation before the concerned authorities. Further, TDS deducted and to be received as refund amounts to Rs. 46,25,231.80 out of which Rs. 40,30,217.52 is pending since 2008-09.

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).

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 for Rs 17,12,895.17 and Equipments for Rs. 56,90,336.72/-

20.2 Expenditure in foreign currency:

- a) Travelling charges Rs. 8,12,848/-.
- 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. 34,981.00).

21. Legal Cases:

As on 27th April, 2015, a cheque of Rs. 92,625.00 drawn on A/c No. 056010100453998 of NBRC with M/s Axis Bank was issued in favour of M/s Golden Feeds Pvt. Ltd., 894/8, Mehrauli, New Delhi-110030, against their invoice No. 2591 dtd. 09th March, 2015. The said cheque was dispatched via speed post (India Post Ref. No. EH643251489IN) to the said recipient. However, it was subsequently brought to notice of NBRC by said recipient that envelope received contained only payment advice. The cheque was found later to have been credited to some Mr. Bhagirath Chauhan's account, at Bank of India, Rajnagar Extn., Ghaziabad (Uttar Pradesh). The matter had been reported to police authorities, Haryana for further investigation and action which is pending since.

A case with DC&SEM is pending against the amount paid Rs 66,97,30,848.73/- for construction of the building in Manesar, the work was partially left by the contractor against which NBRC has filed an civil suit against them.

22. 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. 8,01,636.00 (previous year Rs. 7,53,403.00).

22.2 Schedules 1 to 24 along with Annexures 1 to 59 are annexed to and form an integral part of the Balance Sheet as at 31st March, 2019 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 policies not referred to otherwise be consistent with Generally Accepted Accounting Principles (GAAP).

Santosh Kumar Choudhary
Dy. Finance Officer & Offg. F&Ao

Prof. Neeraj Jain
Director

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

Place: New Delhi
Date: 09th September, 2019

CA Abhishek Goel
Partner
Membership No. 412467

National Brain Research Centre
NH-8, Nainwal More, Manesar, Gurugram, Haryana
Annexure Of Project Grants And Expenditure For The Year Ended 31.03.2019

S. No./Annex. No.	NAME OF PROJECT	Opening Balance as on 01.04.2018	Grants received during the year 2018-19	Interest earned during the year 2018-19	Capital Exp. during the year 2018-19	Revenue Expenditure during the year 2018-19			Refund of Unspent Balance	Closing Balance as on 31.03.2019
						Manpower	Others	Total Expenditure		
1	Distributed Information Centre	42,952.33	8,470,000.00	42,355.00	2,123,450.00	3,442,555.00	379,926.00	3,822,481.00	0.00	2,609,376.33
2	Dementia Meeting	2,123,562.00	0.00	0.00	0.00	0.00	145,943.00	145,943.00	0.00	1,977,619.00
3	INDO-US & NIH RO1 - Dr. Pankaj Seth	129,407.58	-761,236.00	0.00	0.00	0.00	0.00	0.00	0.00	-631,828.42
4	Epilepsy Project of NBRC	6,708,604.30	0.00	0.00	0.00	473,721.00	840,000.00	1,313,721.00	0.00	5,394,883.30
5	Multi Disciplinary System of Parkinson Disease - Dr. Nandini C. Singh	1,189,000.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1,189,000.00
7	FICCI Millenium Alliance- DR. NANDINI C. SINGH	2,307,400.00	0.00	0.00	0.00	0.00	0.00	0.00	2,307,400.00	0.00
8	DBT ITPAR Grant-Dr.Nandini C. Singh	556,234.89	0.00	0.00	0.00	0.00	0.00	0.00	0.00	556,234.89
9	Neurobiology of Dyslexia Brain & Behavior-Dr.Nandini C.Singh	1,860,000.00	0.00	37,167.00	0.00	0.00	5,032.00	5,032.00	0.00	1,892,135.00
10	Wellcom Trust/DBT Indian Alliance -Dr. Anindya Ghosh Roy	1,461,468.56	340,863.00	0.00	0.00	492,000.00	1,815,702.56	2,307,702.56	0.00	-505,371.00
11	Neuro -Cognitive Networks Underlying Dr. Arpan Banerjee	236,204.00	0.00	8,473.00	0.00	226,239.00	0.00	226,239.00	0.00	18,438.00
12	Autism Behavior and Diffusion Tensor Imaging - Dr. Nandini C. Singh	140,356.00	0.00	0.00	0.00	0.00	79,882.00	79,882.00	0.00	60,474.00
13	Characterizing biomarkers of Alzheimer's disease Dr. Pravrat Mandal	1,457,171.50	0.00	0.00	0.00	1,184,724.00	177,405.00	1,362,129.00	0.00	95,042.50
14	National Programme on Preception Engineering - Phase II- Dr. Pravrat Kumar Mandal	1,566,102.60	315,749.40	21,884.00	0.00	992,650.00	437,098.00	1,429,748.00	452,104.00	21,884.00
15	National Programme on Preception Engineering - Prof. P.K. Roy	744,464.00	0.00	0.00	0.00	0.00	0.00	0.00	744,464.00	0.00
16	CSIR Japanese Encephalities - Dr. Anirban Basu	-	2,184,500.00	0.00	0.00	0.00	66,376.00	66,376.00	0.00	2,118,124.00
17	Vision Guide Speech Percepton- Dr. Arpan Banerjee	592,020.00	0.00	0.00	0.00	0.00	6,748.00	6,748.00	0.00	585,272.00
18	National Bioscience Award- Dr. Ellora Sen	432,856.09	0.00	0.00	0.00	0.00	374,341.80	374,341.80	0.00	58,514.29
19	Tata Innovation Fellowship- Dr. Nihar Ranjan Jana	511,908.91	845,185.00	0.00	0.00	75,000.00	177,150.00	252,150.00	795,185.00	309,758.91

20	Innovation In Science Pursuit For Inspired Research (INSPIRE) - Dr. Yogita Mehanisms Of Adult Brain Reorganisation - Dr. Neeraj Jain	688,843.22	1,549,000.00	0.00	0.00	341,950.00	1,058,391.00	93,873.00	1,152,264.00	0.00	-814,005.82
21	Tata innovation fellowship award - Dr. Anirban Basu	115,445.81	889,000.00	5,819.00	0.00	0.00	529,723.00	450,443.00	980,166.00	0.00	1,257,677.22
22	Implications in tumor progression - Dr. Ellora Sen	891,207.19	2,604,000.00	0.00	0.00	849,260.95	496,558.00	635,971.75	1,132,529.75	0.00	1,513,416.49
23	Tata innovation fellowship Award - Dr. Pravat Mandal	138,860.00	891,072.00	8,764.00	0.00	0.00	0.00	963,926.00	963,926.00	0.00	74,770.00
24	Crispi System - Dr. Sourav Banarjee	1,209,929.48	0.00	0.00	0.00	46,023.00	395,204.00	706,753.80	1,101,957.80	0.00	61,948.68
25	DBT Mima Meditate Control - Dr. Sourav Banarjee	225,147.97	1,673,483.00	22,291.00	0.00	60,913.95	104,646.00	1,239,795.05	1,344,441.05	0.00	515,566.97
26	A CROSS-CULTURE PERSPECTIVE/DBT-NETHERLANDS) - Dr. PRAVAT MANDAL	47,556.00	692,321.00	19,510.00	0.00	0.00	430,490.00	52,771.00	483,261.00	0.00	276,126.00
27	SPECIFIC BRAIN TEMPLATE DST Dr. PRAVAT K MANDAL	68,241.50	850,000.00	0.00	0.00	0.00	554,898.00	317,371.00	872,269.00	23,108.00	22,864.50
28	S&S LEARNING IN ZEBRA FINCHES Dr.SOUMYA IYENGAR	111,606.00	1,000,000.00	4,224.00	0.00	0.00	343,839.00	333,729.00	677,568.00	0.00	438,262.00
29	PDF-SERB(AKANSHA JALOTA)	153,808.00	0.00	0.00	0.00	0.00	55,000.00	98,808.00	153,808.00	0.00	0.00
30	PDF-SERB(AMIT NASKAR)	345,972.00	0.00	0.00	0.00	0.00	313,000.00	161,907.00	474,907.00	0.00	-128,935.00
31	PDF-SERB(SUVADIP MALLICK)	72,183.00	380,754.00	0.00	0.00	0.00	330,000.00	122,937.00	452,937.00	0.00	0.00
32	J.C.Bose Fellowship(PROF. SUBRITA SINHA)	101,720.58	0.00	0.00	0.00	0.00	0.00	2,000.00	2,000.00	99,720.00	0.58
33	PDF-SERB(Ashok Datusalia)	243,424.00	0.00	0.00	0.00	0.00	58,536.00	138,968.00	197,504.00	0.00	45,920.00
34	PDF-SERB(Soibam Shyamchand)	357,237.00	850,000.00	0.00	0.00	0.00	754,838.00	100,000.00	854,838.00	0.00	352,399.00
35	DST- Inspired Fellow (Sriparna Mukherjee)	69,904.00	415,201.00	0.00	0.00	0.00	397,083.00	27,734.00	424,817.00	0.00	60,288.00
36	TWAS-DBT (Saliu Ibrahim)	257,262.00	123,930.00	0.00	0.00	0.00	275,000.00	85,470.00	360,470.00	0.00	20,722.00
37	C V Raman Interest Income Fellow(Dr. Rolland Kipre)	224,355.00	0.00	0.00	0.00	0.00	166,452.00	18,457.00	184,909.00	12,903.00	26,543.00
38	Dementia Science Programme	92,835,000.00	0.00	854,177.00	0.00	0.00	69,677.00	60,860,000.00	60,929,677.00	0.00	32,759,500.00
39	DST-CSRI (Dr. Prem Chand)	569,193.00	0.00	17,425.00	0.00	0.00	656,451.00	196,776.57	853,227.57	17,425.00	-284,034.57
40	MicroRNA mediated Reg. of Neural stem(Dr. Anirban Basu)	4,201,989.00	0.00	58,689.00	0.00	3,231,914.64	0.00	1,373,191.57	1,373,191.57	0.00	-344,428.21
41	Therapeutic Role DBT(Dr. Anirban Basu)	814,783.65	0.00	759.00	0.00	0.00	0.00	838,185.00	838,185.00	0.00	-22,642.35
42	Oscillatory network dynamics DST(Dr. Dipanjan Roy)	3,283,742.00	0.00	95,837.00	0.00	2,710,843.00	338,861.00	26,543.00	365,404.00	0.00	303,332.00
43	BRICS Research Project(Dr. Nihar Ranjan Jana)	1,385,000.00	0.00	49,168.00	0.00	0.00	0.00	0.00	0.00	1,434,168.00	0.00
44	Dyslexia Linked RNA(Dr. Pankaj Seth)	2,495,042.46	1,400,000.00	23,525.00	0.00	1,000,000.00	330,551.00	700,572.08	1,031,123.08	35,290.00	1,852,154.38
45	Fetal neural stem cells to oligodendrocytes (Dr. Pankaj seth)	1,004,000.00	0.00	8,334.00	0.00	0.00	0.00	1,390,866.00	1,390,866.00	0.00	-378,532.00
46	Novel Imaging Dignostics Indo-Aus grant(Dr. Pravat Kumar Mandal)	7,278,000.00	0.00	128,306.00	0.00	0.00	1,334,747.00	872,199.00	2,206,946.00	0.00	5,199,360.00

48	Dementia Tissue MRI studies(Dr. Dipanjan Roy)	1,709,000.00	0.00	45,801.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1,754,801.00
49	Dementia Basic Biology(Dr. Shiv Kumar Sharma)	5,108,000.00	0.00	136,894.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	5,244,894.00
50	Dementia Imaging studies(Dr. Pravat Kumar Mandal)	1,709,000.00	0.00	45,801.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1,754,801.00
51	Centre for Excellence for Epilepsy(Phase-II)	82,197,000.00	0.00	3,177,861.00	24,780.00	4,137,183.00	6,624,518.00	10,761,701.00	0.00	0.00	0.00	0.00	74,588,380.00
52	Workshop & Conference(NBRC)	-649,620.78	1,520,290.00	0.00	0.00	0.00	1,332,955.00	0.00	0.00	0.00	0.00	0.00	-462,285.78
53	Programme of Co-Operation Between India and Syria Project	3,558,649.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3,558,649.00
54	DBT Tata Innovation Fellowship -Dr PK Roy	-207,575.40	875,000.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	667,424.60
55	Influence of social cues on spatial cognition - Dr. Chetan Yadav	-3,128.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1,745.00	0.00	-4,873.00
56	InRNAs-Dr Basu	0.00	1,420,000.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1,420,000.00
57	Mechanism of Combinational Control- Dr Sourav	0.00	2,404,000.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	2,404,000.00
58	Effect of Hypoxia on different Neural-Dr Pankaj Seth	0.00	1,996,000.00	0.00	0.00	0.00	898,898.70	898,898.70	0.00	0.00	0.00	0.00	1,097,101.30
59	Hypoxia Ind. Change in Blood Brain- Dr Pankaj Seth	0.00	1,140,000.00	0.00	0.00	0.00	720,950.00	720,950.00	0.00	0.00	0.00	0.00	419,050.00
60	Ramalinga Swamy - Dr. Dipanjan Roy	0.00	1,006,686.00	12,552.00	0.00	0.00	853,194.74	853,194.74	0.00	0.00	0.00	0.00	166,043.26
61	Early Diagnostics of structural and functional- Dr Arpan Banerjee	0.00	4,000,000.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4,000,000.00
62	PDF-SERB-Dr Sandeep Kumar	0.00	960,000.00	0.00	0.00	595,833.00	156,053.00	751,886.00	0.00	0.00	0.00	0.00	208,114.00
63	Understanding the Signaling Circuitries - Dr. Ellora Sen	-575,915.39	0.00	0.00	0.00	0.00	2,114.00	2,114.00	0.00	0.00	0.00	0.00	-578,029.39
64	Multifactorial Risk Factor - Prof. V. Ravindranath	-29,346.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-29,346.00
65	Func. Magnetic Resonance Imaging - Prof. V. Ravindranath	-355,435.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-355,435.00
66	COGNITIVE NEUROSCIENCE DBT-PROJECT- (Dr. Aditya)	-437,464.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-437,464.00
67	Mole. Role of Transc. Factors - Dr. Prabodha Kumar Swain	-644,021.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-644,021.00
68	Comp. Analysis of Speech Imp. - Dr. Nandini C. Singh	-547,567.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-547,567.00
69	Spinal Cord Plasticity ILTP - Dr. Neeraj Jain	-31,869.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-31,869.00
70	Study of Mole. Mechanism - Dr. Anirban Basu	-68,830.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-68,830.00
71	Cellular & Mole. Basis - Dr. Pankaj Seth	-34,974.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-34,974.00

72	Est. of Translational Res. Unit - Prof. P.K. Roy	4,307,442.00	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	4,307,442.00
73	BBNSC - Dr. Renu	1,809,628.00	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	1,809,628.00
74	BBNSC - Dr. Shyamala	-392,947.00	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	-392,947.00
75	BBNSC - Dr. Neeraj	296,937.00	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	296,937.00
76	BBNSC - Dr. Ellora	-403,419.00	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	-403,419.00
77	Ramalinga Swamy - Dr. Ranjit Kr. Giri	-68,440.70	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	-68,440.70
78	CSIR - Project Dr. Nihar Ranjan Jana	73,089.50	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	73,089.50
79	Motivated Behaviour in Male Zebra Finches - Dr. Soumya Iyengar	73,194.65	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	73,194.65
80	Collaboration for Trans. & Clin. Res. (GLUE) - Prof. P.K. Roy	-344,006.00	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	-344,006.00
81	CSIR - II Study the Role of Neural Immune Response - Dr. Anirban Basu	-168,365.93	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	-168,365.93
82	DST Autism Spectrum Disorder - Dr. Nandini C. Singh	82,849.00	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	82,849.00
83	DST Cognitive Science Research Initiative (CSI) - Dr. Chaitra Rao	-324,000.00	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	-324,000.00
84	DIT McGILL Linkage (NKN) - Prof. Prasum Kumar Roy	-596,926.84	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	-596,926.84
85	DBT National Bioscience Award 2010 - Dr. Anirban Basu	585.65	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	585.65
86	DBT CSI Development and Validation of Screening Tools - Dr. Nandini C Singh	-303,509.00	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	-303,509.00
87	National Initiative On Glia Cell Research Project - Dr. Pankaj Seth	92,588.71	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	92,588.71
88	DBT BIRAC Under CRS Scheme Project Grant - Dr. Ranjit Kr. Giri	56,991.50	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	56,991.50
89	DST PDF Project Under CSI - Dr.D Subhashree	-21,941.00	0.00	21,941.00	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
90	DST Inspire Faculty Award -Dr. Supriya Bhavani	-12,189.65	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	-12,189.65
91	DBT INCRE Grant (NBRC)	1,799,153.00	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	1,799,153.00
92	National Institute Glial Cell Research - Shiv Kumar Sharma	-84,803.61	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	-84,803.61
93	Chandipura Virus Infection - Dr. Anirban Basu	-22,580.53	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	-22,580.53
94	Chromatin Remodelers in regulating associated - Dr. Ellora sen	-2,562.16	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	-2,562.16
95	Deregulation of micro RNA in cell and animal models of Huntington's disease Dr. Nihar Ranjan Jana	-2,999.59	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	-2,999.59

96	Women Scientist Scheme DST -Dr. Sayali Ranade	-131,556.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-131,556.00
97	IYBA DBT 2013- Dr. Supriya Bhavanani	49,737.51	0.00	0.00	0.00	0.00	0.00	0.00	0.00	49,737.51
98	DST Inspired Faculty Award- Dr. Deepashri	1,900,000.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1,900,000.00
99	PDF-SERB(POONAM MEENA)	-149,358.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-149,358.00
100	Material Malnutrition - Dr. Shyamala	-579,048.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-579,048.00
101	Multifaceted Kinase CDK5 - Dr. Aparna Dixit	-354,315.93	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-354,315.93
102	Neural Network Mechanism - Dr. Yoganarashimha	(1,070,838.61)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-1,070,838.61
103	A critical assessment of the dual stream models of visual information processing- DST - Dr. Dipanjan Ray	220.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	220.00
104	EBM Including Alzheimer Disease - Dr. Vijaylaxmi Ravindranath	-230,717.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-230,717.00
	Total (A)	237,903,169.20	40,057,739.40	4,825,616.00	10,389,135.54	20,613,850.00	87,995,988.96	108,609,838.96	5,923,512.00	157,864,038.10
6	DELCON E-LIBRARY CONSORTIUM (B)*	587,608,056.49	469,712,865.00	7,638,122.00	139,892.00	1,132,002.00	626,485,578.13	627,617,580.13	0.00	437,201,571.36
	Grand Total (A+B)	825,511,225.69	509,770,604.40	12,463,738.00	10,529,027.54	21,745,852.00	714,481,567.09	736,227,419.09	5,923,512.00	595,065,609.46
	Note:- Projects from Sl. No 64 to 104 are closed and non operational.									

Santosh Kumar Choudhary

Dy. Finance Officer & Offg. F&Ao

Prof. Neeraj Jain

Director

As Per Our Separate Report Of Even Date Attached

For Maheshwari PA & Associates

Chartered Accountants

(FRN-012023C)

Abhishek Goel

Partner

Membership No. 412467

Date: 09.09.2019

Meerut

Compiled and Edited by Anindya Ghosh Roy and Kedar Singh Bajetha
Front Cover: "Surface AMPA receptor at synapses on hippocampal neurons expressing green fluorescent protein"
- Sourav Banerjee
Back Cover: i) "PVD neuron labeled with Green Fluorescent Protein in the nematode *C. elegans*"
- Anindya Ghosh Roy,
ii) NBRC panorama view: Sourav Banerjee



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