NIBMG

ANNUAL REPORT 2022-23

राष्ट्रीय जैवचिकित्सा जीनोमिक्स संस्थान

NATIONAL INSTITUTE OF BIOMEDICAL GENOMICS

Accelerating Genomics for Health

Content

FROM THE DIRECTOR'S DESK

It is an honor and privilege to take over the role of the Director of National Institute of Biomedical Genomics (NIBMG) about a year back. And it gives me real happiness to write a short note for the Annual Report 2022-2023.

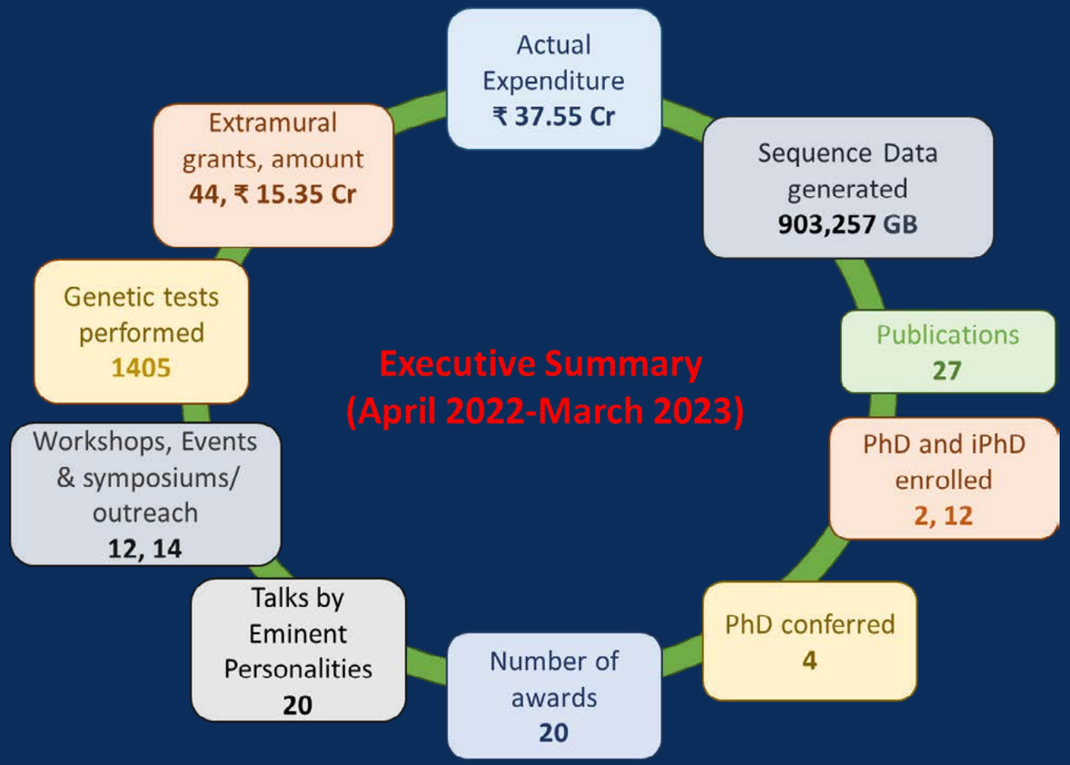
It has been a challenging time for the country in recent past and only now we are getting back into normalcy. It is well known in the scientific community how NIBMG has contributed in the response of Government of India towards the detection and control of the pandemic. In the present Annual Report, we have tried to capture how we have tried to answer to the call of the country and helped the local and scientific community. We have also done a significant amount of outreach activity in the recent past – interacting with school and college students, conducting workshops and of course contributing to the advancement of science not only on genome research but also on other aspects of biology.

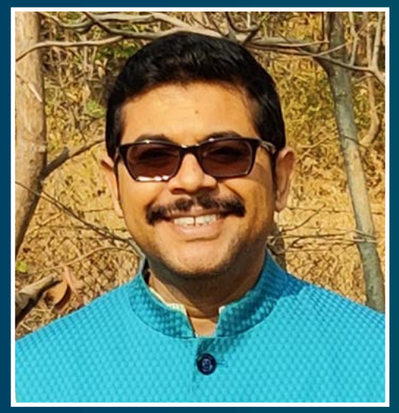
The strength of any institute lies in its scientific achievements. NIBMG has taken significant steps in this direction also. It has published cutting edge scientific papers on multiple aspects of research. Our faculty and students have travelled far and wide – disseminating and also gathering knowledge and talking about the scientific achievements. We are in the process of enhancing our capabilities by establishing newer facilities. For example, the institute is on the verge of setting up radioactivity facility, a bacteriological facility, a BSL-3 facility, a state-of the art proteomics and metabolomics facility.

Hence – through the Annual Report I invite you to join NIBMG in the next part of its journey. I promise it will be an exciting and enriching journey – not only for every member of the NIBMG scientific, technical and administrative staff but also for the NIBMG Fellows, post-doctoral researchers, students and trainees who make up this unique institute. And I invite the brightest and best who are not yet part of this journey – to join us as we move forward to the next phase.

Sagar Sengupta

EXECUTIVE SUMMARY





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Research Focus

Estimation of contributing genetic and environmental factors in complex diseases is challenging since effect of a single factor might be confounded by others. Nonetheless, the genetic dissection of complex diseases can be aided by distinctions between gene identification and gene effect characterization. My research is focused specifically on investigating genetic underpinnings in a) complex traits associated with visual neurodegeneration and b) rare diseases with neuronal defects. I use an integrative approach of high throughput omics backed with functional investigations. I believe this would lead to a deterministic path of genomic medicine and be beneficial to patients suffering from these diseases.

Research Highlights

a) Comprehensive genomic analyses of primary angle closure glaucoma

i) A comprehensive genome wide association to functional study reveals the role of CNTNAP5 in glaucomatous neurodegeneration in primary angle closure glaucoma.

Primary angle closure glaucoma (PACG) is one of the major leading causes of blindness. In India, ~30% of people show a narrow iridocorneal angle (<15°), but out of these only 0.5-1% people develop PACG. Two comprehensive case-control GWAS identified eight loci where heterogenous controls were included with respect to anatomically predisposed parameters. To exclude heterogeneity, we conducted an haplotype based age-agnostic model of progressive angle closure GWAS early-onset PACG patients (PACG:age ≤50years) compared with anatomically predisposed (narrow angle) non-glaucomatous older individuals (PACS:age ≥60 years). Further, we performed dual-luciferase assay for functional follow-up of risk variants of *CNTNAP5* and subsequently the role of *CNTNAP5* figured out in ocular development and morphology of the retinal nerve in zebrafish. In our GWAS cohort (PACG=148 and PACS=92), we identified 13 SNPs of *CNTNAP5* that were associated with PACG. Subsequently, the prioritized SNP rs780010 of *CNTNAP5* was significantly (*P*=0.0024) associated with higher cup-to-disc ratio (CDR), which is a clinical parameter directly correlated with glaucomatous neurodegeneration. We further validated the sentinel SNP rs780010, with *P* =2.131e-06, in a separate replication cohort (PACG=50; PACS=39) and observed a significant association (odds ratio=2.307, *P*=0.012). According to Hi-C database, the associated genic region shows higher retinal neuronal expression of *CNTNAP5* with active enhancer marks; these were subsequently validated using a dual-luciferase assay. Additionally, immunofluorescence analyses showed significant eye size reductions and retinal nerve thinning in zebrafish upon morpholino mediated knockdown of *CNTNAP5*. Our GWAS results not only indicate a genomic association of the *CNTNAP5* with PACG but also imply that it might play a role in glaucomatous neurodegeneration. Further, post-GWAS functionalization led us to believe that *CNTNAP5* is an important player to perturb the development of the neural retina and neurodegeneration that further leads to retinal nerve thinning, thereby increasing the risk of PACG-associated vision loss.

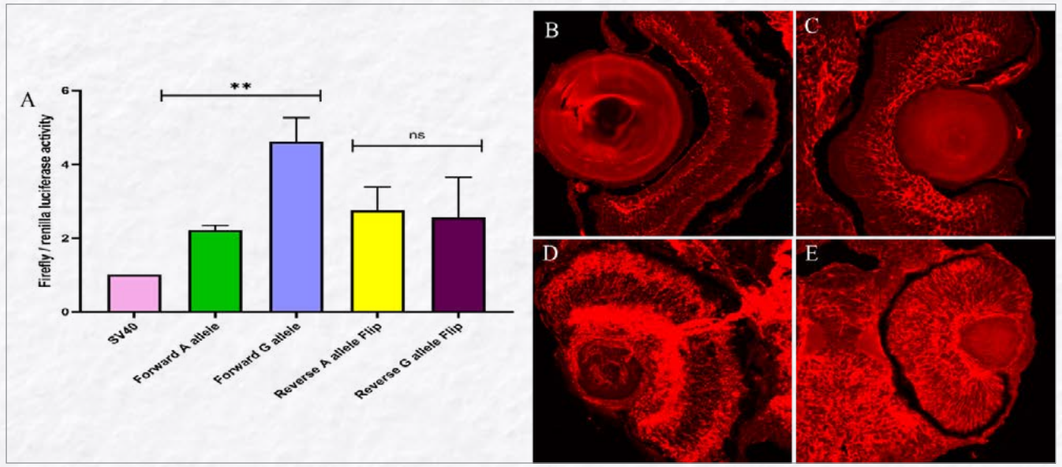

Figure 1: **A**. Lucifrease assay showing enhancer activity of the *CNTNAP5* sentinel SNP rs780010. The risk allele G shows significantly higher luciferase activity corroborating with higher enhancer mark in the SV40 promoter background. The reverse fragments, however, show no such activity. **BCDE**: immunostaining of sagittal section of eyes of zebrafish embryos with acetylated tubulin (red), stains for axonal tracts hence can be used to visualize neuronal layers in retina. **B&C** injected with mismatch control morpholino and **D&E** injected with zebrafish cntnap5 a and b double morphants. Relative to the mismatch morphants, eye size were severely reduced and dysregulated retinal layer organization were observed in **D&E**.

ii) Functional investigation of TNF-α promoter SNP rs1800629 associated with glaucomatous neurodegeneration

To understand the genetic underpinning and risk factors involved in primary angle closure glaucoma (PACG), a case-control genome wide association study (GWAS) was conducted. It included Primary angle closure glaucoma (PACG) patients (cases) and Primary angle closure suspects (PACS, controls) where *TNF-α* promoter SNP rs1800629 was found to have statistically significant association along with 10 other SNPs. The association was replicated in a separate replication cohort with significant p-value. After going through the literature survey and bioinformatic prioritizations, we considered rs1800629 for post-GWAS functional validation. Bioinformatic analysis including epigenomic signature analysis (CTCF, DNase, H3K4Me3 and H3K27Ac Z-scores from ENCODE database), chromosomal architecture using 3C based databases, motif discovery and transcription factor binding site analysis using MEME suite and TOMTOM were performed for further prioritization. To study the allele specific regulatory of the rs1800629, dual luciferase reporter assay was performed. For the reporter assay, two different constructs of 400bp and 800 bp length were created spanning the region of rs1800629. The luciferase activity was also checked in presence of TNF stimulation. The bioinformatic investigation of the TNF-α promoter region indicated strong allele specific regulatory effects and suggested presence of no long-range interaction of rs1800629. Luciferase assay showed that the promoter activity was significantly increased for the constructs containing the allele A in comparison to the allele G in HEK 293T cells. This enhancement in expression was found to be increased in presence of TNF stimulation. Further investigation needs to be done to decipher the pleiotropic role of TNF-α in the context of PACG pathogenesis.

b) Investigating the role of WT1 transcription factor in the pathogenesis of developmental glaucoma spectrum disorders.

Morpholino knockdown experiments- *wt1* and *pax6* genes were intended to be knocked down in zebrafish to look into the specific eye related phenotypes during development. Zfin database was used to select a splice site blocker morpholino i.e., MO6-WT1a, WT1b (Tomar et al) which can knockdown both *wt1a* and *wt1b* genes in zebrafish. For knocking down *pax6a* we used the translation blocker morpholino MO2-PAX6a (Royo et al). 5ng of each of *wt1a/b* and *pax6a* morpholinos were injected in single celled stage zebrafish embryos. We also injected mismatched control oligos that served as the negative control. Both *pax6a* and *wt1a/b* morphants were observed under the microscope in 24 hours intervals. On the fifth day post fertilization, the pax6a fish showed severe developmental eye defects (aniridia, reduced eye diameter etc.) along with abnormally curved body and a bent tail. The diameter of eyes was measured on the 5 dpf and 10 dpf stage and it was found to be significantly reduced in morpholino injected than the mismatched controls and un-injected fish. In case of *wt1* morphants, gross morphological deformations were observed in the morpholino injected fish in 6 to 7 dpf stage. There were some serious deformations near the head and eye region, curved body, and prominent heart edema. The diameter of the eyes was measured, and significant reductions were observed.

To check the genetic interaction the morpholino dose was reduced by performing serial dilution to a point where the phenotypes are no longer observed. This subeffective dose for both the genes *wt1a/b* and *pax6a* were combined and injected in the zebrafish embryos at single cell stage. Five days post fertilization it was observed that the fish have various phenotypic abnormalities like severe iris deformations, curved tail, bent body and heart edema. The combination dose injected morphants were fixed and sectioned and thin sections were stained using hematoxylin and eosin and toluidine blue for deep phenotyping. The reappearance of *wt1* as well as *pax6* phenotypes when the two subeffective doses are combined and injected together in the zebrafish embryos, infer the genetic interaction between *wt1* and *pax6*.

c) Investigating genomic signatures causal to rare congenital muscle disorders.

i) Functional characterization of mutations causal to rare muscle disorders in genes encoding intermediate filament proteins

Desmin and lamin intermediate filaments (IFs) coded by *DES* and *LMNA* genes play an important role in the formation of cytoskeleton and nucleoskeleton in the muscle cells, respectively. Mutations in these genes have been known to cause desminopathy and laminopathy. Functional characterization of novel c.448C>T;p.(Arg150*) mutation in the *DES* gene leading to formation of truncated desmin protein at 150th amino acids and the missense mutation c.590T>C;p.(Leu197Pro) in the *LMNA* gene were undertaken to understand its pathological role in these diseases. Primary human skeletal muscle cells (HSkMC) were used as an *in vitro* model to electroporate with wild-type and mutant constructs of desmin and lamin. Confocal microscopy imaging was performed to elucidate the formation of aggregates which is a determinant of pathogenicity. Large clumps of truncated desmin aggregates on the periphery of the nucleus were observed altering the positioning as well as distribution of organelles such as golgi and endoplasmic reticulum in the cells. The aggregates also interfere with the actin filament assembly leading to improper bundling and collapsed structure of actin-binding protein network such as fimbrin and alpha-actinin. On the other hand, c.590T>C change in the *LMNA*

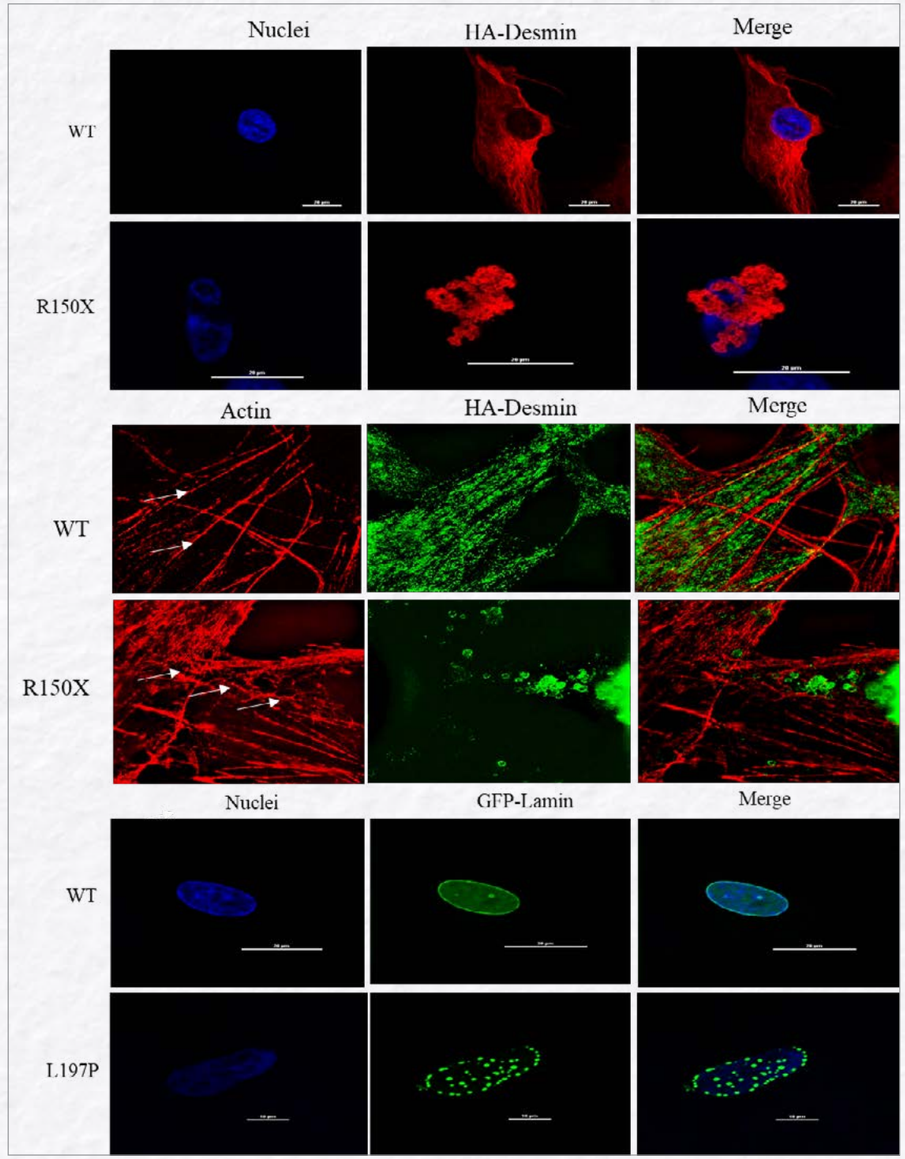

Figure 2: **Top**: Primary human skeletal muscle cells (HSkMC) show of wild type (WT) and mutant (R150X) desmin in red. R150X shows clear aggregate formation. **Middle**: Changes in actin (red) cytoskeleton structure depicted in white arrows in cells expressing R150X mutant desmin (green). **Bottom**: wild type lamin (green) forms rim like structure around nucleus while mutant lamin (L197P) forms aggregates in the nucleus. In all cases nuclei were stained in Dapi (blue).

gene also show formation of aggregates and disappearance of laminar rims in the nuclei of the muscle cells. The formation of such aggregates might disturb the cellular homeostasis and organelle positioning including the nucleus and mitochondria thereby impacting the mechano-transduction signalling and leading to oxidative stress and cell death.

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Group photo of the Lab

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Research Focus

My research has two distinct components: method development and data-driven inference. My major focus has been in deciphering human population diversity and evolution with special emphasis to South and South-East Asia. Utilizing the knowledge about ancestry, population structure, ancient and recent migration patterns in human populations, I look for an evolutionary viewpoint in connecting genotype with phenotype. I also work towards development of appropriate statistical and computational approaches, especially where no 'off-the-shelf' solution is available, to provides new insights into biological data. My recent interest has been in analysis of longitudinal data and application of Artificial-Intelligence (AI) in genomic data driven clinical inference.

Research Highlights

The GenomeIndia Project is a Multi-Centre, Multi-Institutional Project which aims at performing whole genome sequencing (WGS) of at least 10,000 individuals representing the Indian population to create a detailed catalogue of genetic variations. It will be an immensely valuable scientific resource for the country and the international scientific community as it moves to the future into the realm of genomics driven health sciences.

Progress of work during the current reporting year (2022-2023)

Sample Collection: Ground work pertaining for the workflow of sample collections, DNA isolation and sequencing has been set up. We have collected 1395 blood samples from the populations of Santhal (n=189), Namasudra (n=170), Kudmi Mahato (n=271),Mahishya (n=256), Rarhi Brahmin (n=210), Kayastha (n=104) and Karn Kayastha (n=37), Rajbanshi (158) respectively. We have received ethcal approval from the Orrisa Biotechnology Council for the collection of Karn Kayastha samples and have all the necessary paperwork done to collect the amples with the help of ICMR-RMC-Bhubaneshwar. The measurement of various biochemical parameters were completed for 1,395 samples from the above mentioned populations.

Samples Received: Additionally, we have received 1625 blood samples from different partner institutions, including MZU (Rabha, n=148, Mizo, n=148, Khasi, n=148, Hajong, n=148), ILSB (Namasudra population (161), Gond (150), Juang (122) , Oriya Brahmin (218) and IBSD (Meitei population, n=370). The sensitization of the targeted populations for Karn Kayastha have been completed through various stakeholders that includes ICMR and state government agencies. Sample collection halted due to unavailability of funds.

Genotyping & Sequencing: The DNA isolation of 3008 blood samples have been completed. The array based whole genome genotyping (GSA chip) for 2910 individuals as well as the whole genome sequencing of 1619 individuals have been completed during the study period.

Quality-Control: We have completed whole genome sequencing of 5 'round-robin' samples and analyzed them in conjunction with the 'Genome in a bottle' samples to assure high and uniform quality of variant calling between different centers (Data analysis groups).

Data-Transfer: We have copied 800 uBAM files and sent them to method development centers of Genome India Project. 800gVCF transferred to CBR.

Primary Analysis:

Data Analysis: The array based whole genome genotyping (GSA chip) for 2910 individuals completed and screened for sequencing unrelated individuals and for Trios. We consider the unrelated individuals atleast for first cousin level. We have developed a graph-theory inspired computer algorithm to identify and select the maximum number of 'unrelated' individuals for sequencing. The algorithm also enables us to identify the nature of relationship between the related samples and confirm the self-identified trios from their genetic data.

We have generated 1000gVCF through DRAGEN version 4.0.3. Joint variant calls have been performed for 750 individuals where around 5 million novel variants were identified. We have now installed a beta version of the variant caller which is being developed by the DRAGEN team and we are in the process of quality testing along with the DRAGEN team.

We have done the analysis of novel variants. As expected, the burden of novel variants is high in the Indian populations. In total we found 32588729 Single Nucleotide Variants or SNVs. Out of these 4927330 variants are novel and are not found in any global database (genomeAD, dbSNP). However, the commonality of these novel variants across the populations is very little. Altogether there are only 33 of these novel-variants which are shared among all the 7 populations that we have sampled which indicates that these novel variants, most of which have arose recently while some of which are in high frequency and private in certain populations; are very rarely shared. This underscores the importance of wider and deeper sampling and sequencing in Indian populations to establish a catalogue of variants. The genetic diversity of Indian populations has been complex. Previous genetic studies on Indian populations had reported four different ancestries in populations of mainland India. It has also been shown that there is clustering inspite of the evidence of wide admixture. Although the population clusters are confounded with language, geography as well as social hierarchy, neither of them is a sufficient determinant of the ancestry of an individual.

In correspondence to the clusters identified from previous studies, we at NIBMG, have samples from three of them. We have genotype data from the following populations: Rahri Brahmins, Oriya Brahmins, Karan Kayastha, Kayastha, Mahisya, Namasudra, Kudmi Mahato, Gond, Santal, Juang, Hajong, Khashi, Rabha, Maitei Mizo. Of which Rahri Brahmins, Oriya Brahmins, Karan Kayastha, Kayastha, Mahisya, Namasudra, Kudmi Mahato are Indo-European language speakers and are sampled mostly from river basins and plainlands of Orissa and West Bengal. Gond, Santal, Juang are Austro-Asiatic speaking (Mundari branch) tribal people from the Chotonagpur plateau area of West Bengal and Odisha. It may be noted here that the language and the folklore of the Kudmi Mahato has strong association with the Mundari speaking tribal people. There is also a geographic proximity with the Mundari speakers. Hajong, Khashi, Rabha, Maitei and Mizo are populations from North-East India of which all speak the Tibeto-Burman languages except Khashi who speak an Austro-Asiatic language (Khashi Khumeric branch).

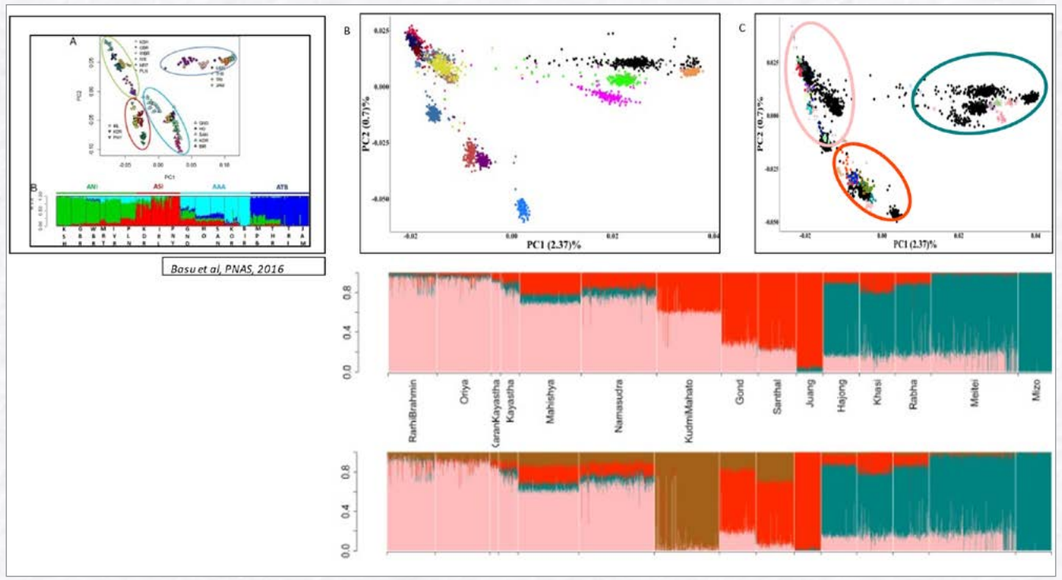

Figure 1. We determined the axes of human genomic variation using principal-components analysis (PCA), as implemented in EIGENSTRAT. Using a dynamic programming-driven unsupervised clustering algorithm, ADMIXTURE, we determined the genomic admixture at the individual level, by partitioning the genome of an individual into *K* components contributed by hypothetical ancestors and then estimating their relative contributions. An unsupervised clustering algorithm, ADMIXTURE, was run on our high-density dataset to explore global patterns of population structure varying the number of ancestral clusters ($K = 2$ through 6) and were successively tested. As LD can adversely affect the inferences of ADMIXTURE, the program was run on multiple datasets after pruning SNPs at LD. Cross-validation errors for each *K* was calculated. PCA was applied using EIGENSOFT 4.2 and plots were generated using R 4.2.2 (https://www.r-project.org/). We have also run the Admixture analysis among 2738 individuals along with the Genome Asia population. Here we have restricted the Genome Asia data to include the mainland Indian populations only.

The scatterplot of the Individuals shows a clustering pattern similar to what has been reported earlier in Indian populations (Figure 1B). The Indo-European speaking caste populations, namely Rahri Brahmins, Oriya Brahmins, Karan Kayastha, Kayastha, Mahisya, Namasudra, Kudmi Mahato, cluster together (indicated also by the pink circle in Figure 1C). The Austro-Asiatic speaking tribal populations of Chotonagpur Plateau, namely Gond, Santal and Juang also cluster together as indicated in the red oval in Figure 1C. In accordance to the oprevious nomenclature the pink cluster corresponds to Ancestral North-Indians (ANI), the red corresponds to Ancestral AustroAsiatic (AAA) and the Green corresponds to Ancestral Tibeto-Burman (ATB). It is to be noted here that the Kudmi Mahato population is close to the Austro-Asiatic speaking populations. We have also plotted the GenomeIndia population along with the GenomeAsia data which is available for the corresponding clusters (Figure 1C). In this figure all GenomeIndia populations are coloured black. As we can see the GenomeIndia populations encompass all the genetic variation that was covered by the previous studies.

The ADMIXTURE analysis recapitulated the findings of PCA. When we ran ADMIXTURE with 3 hypothetical ancestries (i.e K=3), the Cross-Validation (CV) error drastically reduced and the populations which were identified with the ancestries also corresponded with the clusters in the PCA plot. We have used the same set of colours for the ancestries which predominates in the corresponding cluster (Pink, Red and Green) in the PCA plot. When K was increased from 3 to 4 the CV remained identical whereas the new ancestry (coloured brown) was predominantly identified among the Kudmi Mahatos.

Publications

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Research Focus

There is a rapid transition happening in developing countries, where an upward trend in the prevalence of NAFLD is evident, perhaps as a result of economic prosperity and lifestyle changes. Epidemiological studies suggest prevalence of NAFLD is around 17% to 32% of urban population in India, and 9% in rural population, with higher prevalence in those with obesity and Type 2 diabetes. NAFLD is often a progressive disease with worsening prognosis, which is largely asymptomatic in the initial stages. It is therefore important to identify and validate the clinical and molecular changes involved in NAFLD development and progression, which will provide therapeutic targets.

Research Highlights

Topic 1: Characterization of MAFLD among Indians

NAFLD is associated with other diseases and risk factors such as dyslipidemia, obesity, T2DM, insulin resistance, and is also known as hepatic manifestation of metabolic syndrome.

In 2020, Prof. Eslam and his colleagues proposed to rename NAFLD to metabolic-associated fatty liver disease (MAFLD). The argument was that the current definition of NAFLD does not take into account the actual heterogeneity of the disease. Diet, ethnicity, age, sex, metabolic health, genetic and epigenetic factors and the microbes from the gut all dynamically interact with each other and is responsible for fatty liver pathogenesis.

So, in this study we wanted to investigate the actual differences in pathophysiological phenotypes between the NAFLD individuals (from the previous definition) and the fatty liver individuals with metabolic dysregulation (MAFLD). To address this question our first objective was to identify the clinicopathological risk factors of MAFLD in comparison with NAFLD among Indians. Within this objective we compared anthropometry, diabetic profile, lipid profile, blood pressure and inflammation markers among our different study groups. We also characterized body fat and muscle composition among the study groups. Then we investigated the pathophysiological risk factors of metabolic dysregulation separately for the obese and non-obese individuals, along with disease severity.

Our second objective was to study genetic predisposition of the individuals with MAFLD (individual who has fatty liver along with metabolic dysregulation (FLD+/MeS+)) compared to Healthy individuals (FLD-/MeS-).

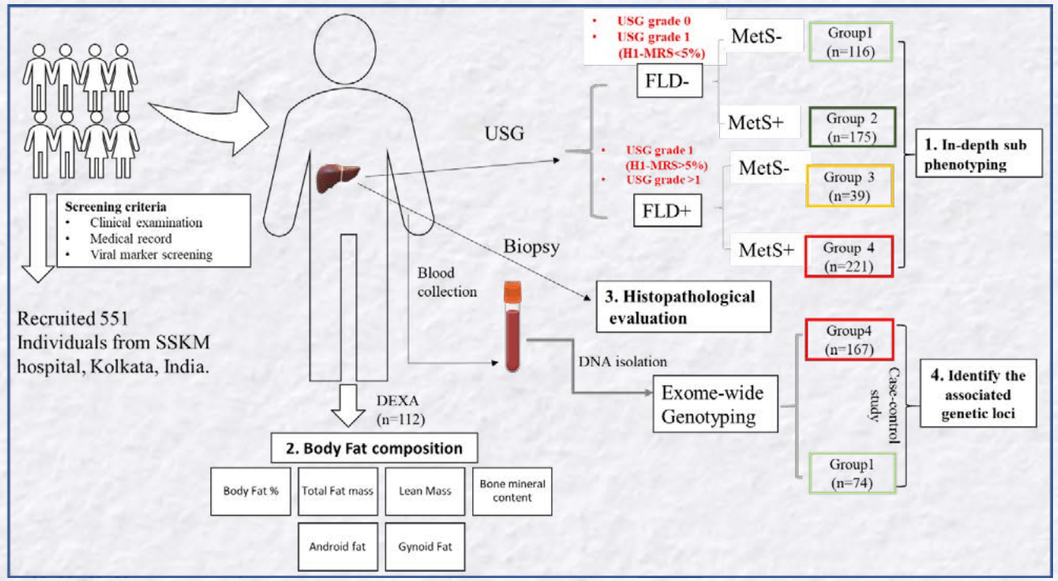

Figure 1: Study Design

We had recruited 551 non-alcoholic individuals from the SSKM hospital after clinical examination, medical report and viral screening. Here, we divided 551 individuals into two groups based on presence or absence of fatty liver confirmed by ultrasonography and we named the groups: FLD- (absence of fatty liver) and FLD+ (presence of fatty liver) (Figure 1). Then we subdivided these two groups into two additional groups: MeS- and MeS+ based on the definition of metabolic dysregulation. Thus, we have formulated four groups such as Group4FLD+/MeS+ or MAFLD individuals, N=221;

Group3FLD+/MeS- or NAFLD individuals, N=39; Group2FLD-/MeS+ or MeS individuals N=175; and Group1FLD-/MeS- or Healthy individuals N=116. Then we identified the clinicopathological risk factors of MAFLD in comparison with NAFLD among the four groups and we also identify the genetic loci associated with MAFLD compared healthy individuals.

Among 551 individuals, 47.2% had fatty liver (FLD+) and 71.87% had metabolic dysfunction (MES+). Compared to Group3 (FLD+/MeS-), Group4 (FLD+/MeS+) patients had significantly higher age, higher adiposity, severe diabetic profile, lipid profile, liver damage marker, CRP, low bone mineral content and higher liver damage, both among the obese and the non-obese. Non-obese Group2 (FLD-/MeS+) patients had significantly higher serum TG and lower HDL compared to obese Group3 (FLD+/MeS-) patients, while obese Group3 (FLD+/MeS-) patients had higher liver damage markers compared to Group2 (FLD-/MeS+) patients. Additionally, we also showed that in our population, Group (4FLD+/MeS+) patients carried risk alleles in rs3761472-G (SAMM50, OR=2.9(2.0-4.1); p=0.002), rs738409-G (PNPLA3, OR=2.8 (1.9-4.07) p=0.003), rs58542926-A (TM6SF2, OR=2.7(1.9-3.9) p=0.021), rs35665085-A (CECR5, OR=2.7 (1.9-3.9) p=0.038), rs471364-G (TTC39B, OR=3.1 (2.1-4.5) p=0.001), rs2800-G (SLC9A9, OR=3.1 (2-4.5) p=0.028), rs7200543-A (PDXDC1, OR=2.261 (1.1-4.8) p=0.031) compared to Group1FLD-/MeS-. To conclude, fatty liver patients with metabolic dysfunctions possess higher risk of disease progression compared to other subtypes.

Topic 2: Study on the relationship of dysregulated Insulin signaling and Non-Alcoholic Fatty liver Disease

A) Aims and Objectives

a) To investigate the changes in clinicopathological characteristics of NAFLD patients with increasing severity of Type 2 Diabetes and to understand the genomic underpinnings behind it.

b) Investigating the role of PI3K-Akt pathway in NAFLD etiology

B) Materials And Methods

Categorizing NAFLD patients based on Diabetic status:

Consenting individuals were recruited from outpatient wing of the SSKM hospital, Kolkata, India. Individuals were screened for any history of alcohol intake and those who had secondary causes of steatosis (including autoimmune hepatitis, Wilson's disease, α-1-antitrypsin deficiency, viral hepatitis, hemochromatosis) were excluded from the study. All the study participants, after exclusion, were categorized into two major groups: NAFLD and no-NAFLD based on the Ultrasonography (USG) grade and hepatic fat content detected by Magnetic Resonance spectroscopy (MRS). Individuals having a USG grade 0 and USG grade 1 with MRS fat percent less than 5% were included into the no-NAFLD group and the individuals having a USG grade 1 or higher with MRS fat percent greater than 5% were included into the NAFLD group. Each of these two groups were further sub-categorized into non-diabetic (ND), prediabetic (PD) and diabetic (D) groups based on fasting blood glucose (FBG) levels and percentage of glycated hemoglobin (HbA1c) according to the American Diabetic Association (ADA) norms. Individuals having a FBG <= 100 mg/dL and/or HbA1c <= 5.7% were included in the non-diabetic group, those having FBG 101-124 mg/dL and/or HbA1c 5.8-6.3% were categorized in the prediabetic group and those having FBG > 124 mg/dL and/or HbA1c > 6.3% were included in the diabetic group.

Clinicopathological characteristics and Liver Histopathology Scores

Clinicopathological characteristics like lipid profile (triglyceride, total cholesterol, HDL, LDL, VLDL), liver damage markers (ALT, AST, GGT, ALP), Albumin, Globulin, Creatinine, Fasting Insulin were compared between the ND, PD and D Groups in NAFLD as well as no-NAFLD group using pairwise t-test and ANOVA to see how the clinicopathological characteristics change in NAFLD patients with increasing severity of Type 2 Diabetes. Liver histopathology scores like Steatosis Score (SS), Hepatocyte Ballooning Score (HBS), Lobular Inflammation Score (LIS), Fibrosis Score (FBS), Portal Inflammation Score (PIS) and NAFLD Activity Score (NAS) were compared to check the proportion of individuals showing higher severity of liver damage based on histopathology scores, among the different subgroups, to assess the disease severity of NAFLD with increasing severity of Type 2 Diabetes.

Exome-wide Association Analysis using GGT as a Quantitative trait

To identify the genetic loci associated with non-alcoholic fatty liver, we used a quantitative trait locus (QTL) mapping strategy using GGT (Gamma-Glutamyl Transferase) as the quantitative trait. Exome-wide association with GGT was studied among the 223 participants. Prior to QTL analysis, the distribution of GGT (n = 223) was tested for normality using the Shapiro-Wilk's test. Since the distribution of GGT significantly deviated from normality, Box-Cox transformation was applied to induce normality. Standardized residual values, after eliminating the effects of significant covariates on GGT, were used for further analysis. Normality of the residual values was confirmed with Q-Q plot and Shapiro-Wilks's test.

Quantitative trait association analysis was done with the residual values using linear regression, assuming an additive genetic model, using PLINK v1.07. Benjamin-Hochberg FDR (BH-FDR) procedure was used for multiple testing correction. Manhattan and QQ plots were constructed with "qqman" Bioconductor package.

C) Observations

Lipid profile including LDL, HDL, VLDL and Total cholesterol did not show discreet differences among non-diabetic, prediabetic and diabetic NAFLD patients and among the most established liver damage markers such as ALT (Alanine Amino Transferase), AST (Aspartate Amino Transferase).)). Only GGT (Gamma Glutamyl Transferase) showed discreet difference among the groups with a mean GGT level of 42.61± U/L in non-diabetic NAFLD group, 46.16 U/L in prediabetic NAFLD group and 62.34 U/L in diabetic NAFLD group. The different Histopathology Scores showed an increased proportion of individuals belonging to severe disease phenotypes in the diabetes group in NAFLD. Using GGT as a QTL among patient group the SNP (single nucleotide polymorphism) rs362949 in the GRM1 gene was found to be significantly associated with GGT levels after multiple testing correction.

d) Conclusion

Diabetic group in NAFLD showed more disease severity as compared to the other groups as evident from the different liver histopathology parameters such as steatosis score, fibrosis score, hepatocyte ballooning score, NAFLD Activity Score (NAS) and lobular inflammatory score. In our study, among the most established liver damage markers, only GGT showed marked difference among the non-diabetic, pre-diabetic and diabetic NAFLD individuals as opposed to the no-NAFLD individuals. Using GGT as a quantitative trait, a SNP (rs362949) belonging to GRM1 gene was found to be significantly associated with GGT levels after multiple testing correction.

Group photo of the Lab

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Research Focus

Our group mainly works on two broad areas: (a) Chronic Disorders – Thalassemia, rare disorders (b) Cancer Immunity. *In the area of Thalassemia*, utilizing multi – omics system, we are investigating the severity factors, pursuing drug development through *ex vivo* RBC maturation. We are also working on NGS based thalassemia test panel. Our group is also looking towards CRISPR based gene editing technology and autologous therapy in the coming days.

In the area of Cancer Immunity, we are investigating the cross talk between cancer cells and immune cells and modulating the immune checkpoint molecules for cancer therapy.

Research Highlights

Broad Area: Chronic Diseases – Monogenic disorder - Thalassemia

Broad Area: Chronic Diseases – Monogenic disorder - Thalassemia

A] **Integrated Transcriptome and Proteome investigation to identify severity markers for the progression of Thalassemia**

Depending on the extent of the anemia, and transfusion requirements, thalassemia is of two types: Severe type, and non-severe type. After initial screening, thalassemia patients having same beta globin compound genotype of IVS 1-5 G>C and CD 26 G>A, of severe category needing regular blood transfusion (TDT) and non-severe or Non-Transfusion Dependent Thalassemia patients (NTDT), were selected for omics investigation. Age-sex matched 5 normal subjects were also included as control subjects. Peripheral blood was collected for PBMC and RBC isolation. Total RNA was extracted from PBMCs and RBCs and bulk RNA sequencing-based transcriptome study was undertaken. Proteome based investigations were also carried out from RBCs, depleting high abundant globin proteins.

Finding commonality between PBMC and RBC transcriptome signature

Correlation matrix analysis revealed that twenty-five genes were significantly correlated between PBMC and RBC transcriptome. Metascape analysis further showed that these genes were associated with anemia, hemoglobinopathy, and thalassemia-related disorders and others (Fig 1).

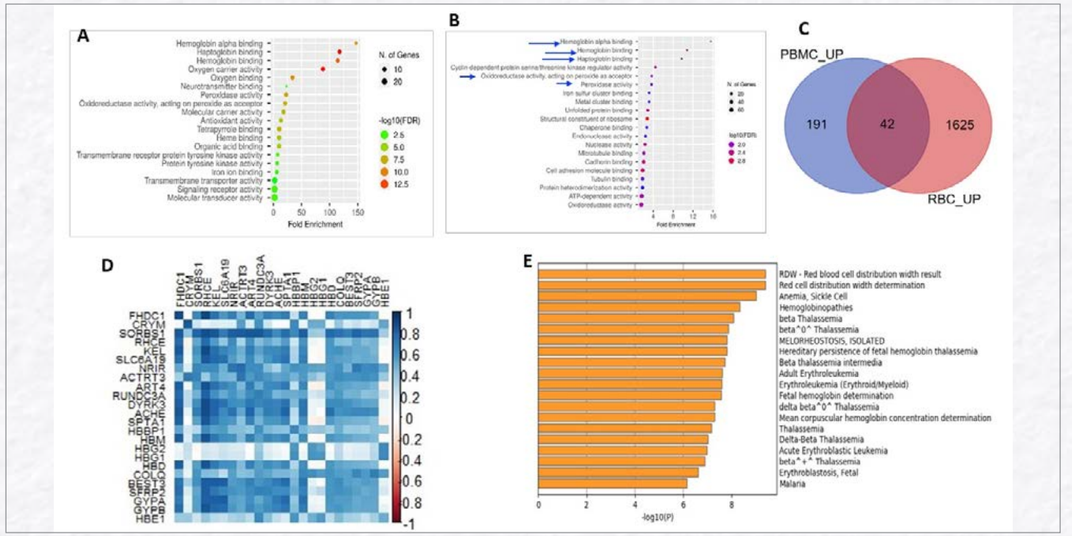


Fig 1. Convergence of PBMC and RBC transcriptome. A) Gene Ontology Molecular function (GO:MF) of upregulated genes of PBMC; B) GO:MF of upregulated genes of RBC, C) Identifying common upregulated genes between PBMC and RBC transcriptome, D) Correlation matrix of 25 good correlated genes, r>5, E) Metascape analysis showing disease association of correlated genes.

Integration of RBC transcriptome and proteome signature uncovers molecular pathways defining clinical pathology of Thalassemia

Integrated analysis of RBC transcriptome and proteome revealed the dysregulation of molecular chaperones, actin binding, antioxidant activity, which are linked to ineffective erythropoiesis in thalassemia (Fig 2)

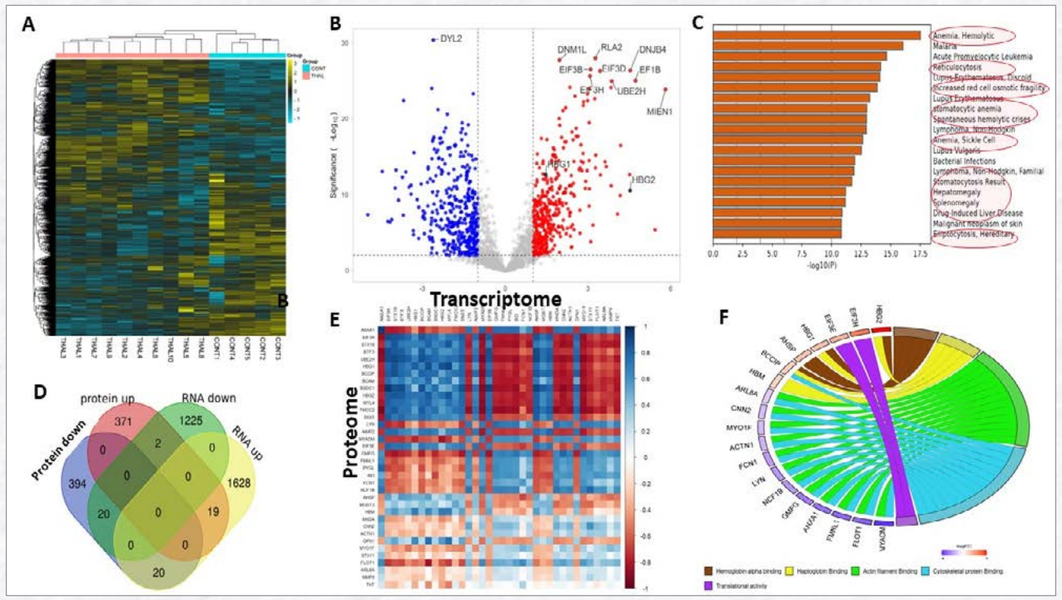
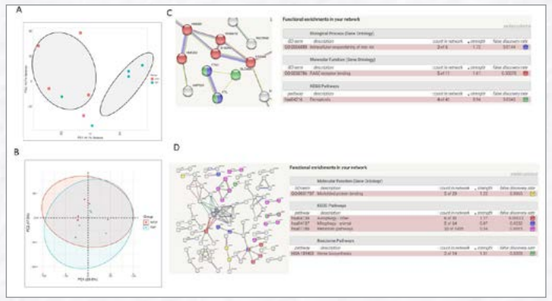


Fig 2. RBC proteome and transcriptome analysis. A) Cluster Heatmap analysis of Proteome data between control and patient group, B) Volcano plot showing high and low abundant proteins in Thalassemia patients , C) Metascape analysis showing disease association of low abundant proteins. (D-F) Integration of RBC transcriptome and Proteome data, D) Identifying common dysregulated genes, E) Correlation matrix of common expressed genes between RBC transcriptome and proteome F) GO:MF_ Circos chord plot showing the most enriched genes of molecular functions from RBC transcriptome and proteome analysis.

Findings of dysregulated genes associated with severe type or transfusion dependent thalassemia.

On comparing the severe and non-severe types of Thalassemia patients having same HBB genotype (IVS 1-5 G>C and CD 26 G>A), many dysregulated genes were identified. Down regulated or low abundant proteins were associated with autophagy related pathways (Fig 3) in severe type or transfusion dependent patients. It was further revealed that autophagy related genes ATG2B and ATG4B, were downregulated in most of the severe patients than in non-severe patients (Fig 3E).



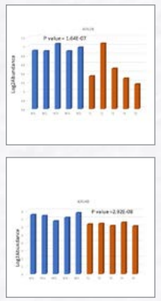


Fig. 3. Differentially expressed genes between Severe (TDT) and Non-Severe (NTDT) patients. A) PCA analysis, for TDT and NTDT of RBC transcriptome data. B) PCA analysis, for TDT and NTDT of RBC proteome data. C) STRING analysis of high abundant proteins of TDT group D) STRING analysis of low abundant proteins of TDT group, E) Relative abundance of the ATG2B and ATG4B in the TDT and NTDT patients from proteome data.

Discovery of the dysregulation of snRNAs and snRNP complexes in severe form of Thalassemia

On comparing the RBC transcriptome data, it was found that there was upregulation of snRNAs in Thalassaemic subjects than in control subjects. On the other hand, from the proteomics data, it was also observed that 5 snRNP proteins: RU17, RUXF, SMD2, HEXIM1 and LSM1 had higher abundance in severe group of patients. In recent studies, it has been shown that higher expression of HEXIM1 is correlated with ineffective erythropoiesis. Higher expression of HEXIM1 in severe patients, which is a part of RNA polymerase II transcription regulation complex, justify the severe anemia.

B] **Development of One Stop NGS based Thalassemia Gene test Solution**

Around 540 mutations, comprising point mutations and large deletion in HBB, are responsible for thalassemia. No single conventional gene test method can able to cater the diagnosis. Thus, we have developed a targeted Next Generation Sequencing (NGS) panel and workflow that enables the detection of both single nucleotide variants (SNVs) and copy number variations (CNVs) within the beta globin gene cluster in a single run. [*Manuscript under review*]

C] Identifying the pathogenic Indian variants and genes for diagnostic accuracy of a Rare Genetic Disorder

We have identified a novel Homozygous Missense Mutation in the *CLDN16* Gene for the diagnosis of Familial Hypomagnesemia with Hypercalciuria and Nephrocalcinosis in an Indian Family. Through exome sequencing, FHHNC type 1 was confirmed by discovering a novel homozygous missense mutation in the *CLDN-16* gene (Exon 2, c.374T>C) which causes altered protein structure with F55S. Associated clinical, biochemical, and imaging findings also corroborated the final diagnosis. [*in Press,* **"Calcified Tissue International"]**

Broad Area: Cancer Immunology

A] Interaction of Cancer epithelial cells and Macrophage in Breast Cancer (BC) progression

The interaction between Macrophage and Breast Cancer cells was studied. TCGA data was examined to observe the survivability. It was found that, OS was negatively associated with high expression of CD68. It was further observed that high expression of CD68 in HER2+ subclasses had an improved OS while in case of luminal and triple negative subtypes, higher expression of CD68 correlated with poor survival (Fig 4).

In the in-direct *co*-culture system, human monocyte THP1 derived, macrophage cells were allowed to differentiate in the presence of human breast cancer cell (T47D, MCF7 and MDMB231) derived conditioned media. The T47D and MCF7 cells influenced M1 like morphology and on the contrary the MDAMB23 influenced the M2 like morphology (Fig-4). Different types of cancer cell treated macrophage cells were activated differently to express different pro/ anti-inflammatory cytokines (Fig. 4).

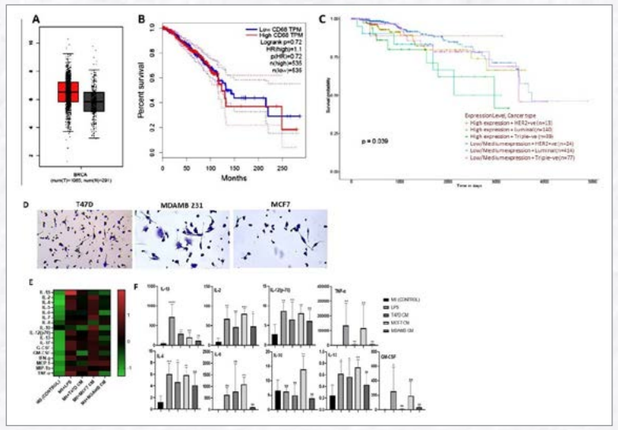


Fig 4. Cancer cell and Macrophage Interaction (A) The boxplot shows the mRNA expression of CD68 in breast tumor (n=1085) and normal breast tissue (n=291) using GEPIA2 web tools based on TCGA and GTEx database. (B-C) As per TCGA data, overall survival estimates in relation with CD68 expression levels in BC patients. (C) overall survivability in relation with CD 68 expression in HER2+, luminal, Triple negative (TNBC) subtypes of BC respectively. (D-F) Effect of breast cancer cell derived conditioned media (CM) on THP1 cell derived MO macrophages. (D) Microscopical images showing morphological changes of macrophages after treatment with conditioned media from the different subtypes of BC cells (T47D, MCF7 and MDAMB231) (E) Heatmap generated from the cytokine expression of the macrophage cells after treatment with conditioned media from different cancer cells and Lipopolysaccharide (F) Bar graph showing expression of pro-inflammatory (IL-1β, IL-2, IL-12(p-70), TNF-α) and anti-inflammatory cytokines (IL-4, IL-6, IL-10, IL-13, GM-SF) from macrophage treated with BC condition media.

B) Blocking of the immune check point receptor - *TIM3* reduces the FOXP3 expression on mice breast tumor model.

4T1 cell derived tumor bearing BALB/C mice were treated with anti-TIM3 antibody for 30 days. There was substantial reduction of FOXP3 expression in the CD4+T cells in treated group. FOXP3 plays a key role in CD4+ T cell function and represents a specific marker of immunosuppressive factor within the tumor-microenvironment. Further investigation is ongoing for this interesting finding (Fig 5).

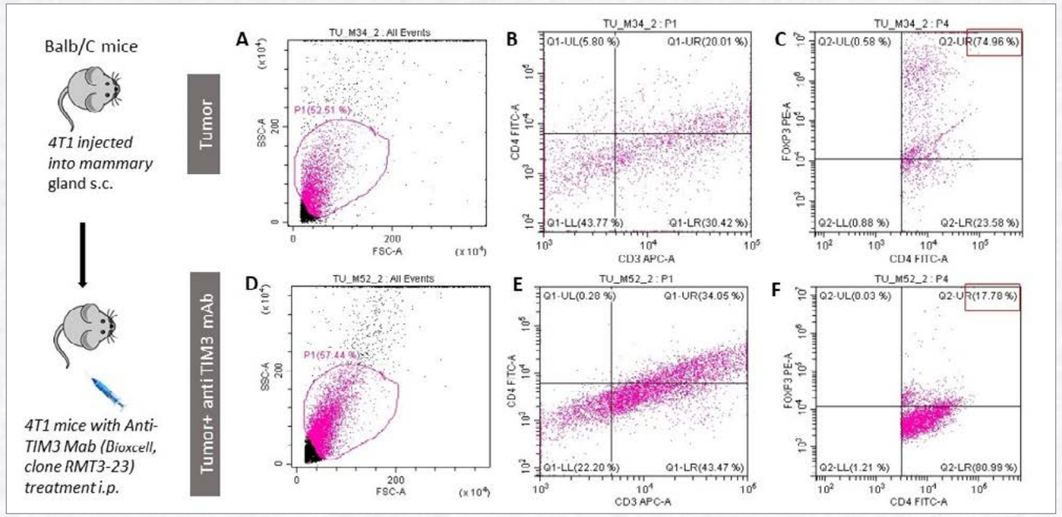


Fig 5. Flow cytometry analysis for the expression of FOXP3 in 4T1 treated mice tumor. (A-C). Mice Tumor tissue injected with saline, B) Relative abundance of CD 3+ and CD 4+ cells, C) FOXP3 expression by CD4+ T cells, (D-F) Mice tumor tissue injected with anti-TIM3 monoclonal antibody. E) Relative abundance of CD 3+ and CD 4+ cells, F) FOXP3 expression by CD4+ T cells.

Group photo of the Lab

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Research Focus

The long-term goal of research in my group is to accelerate the process of a) comprehensive identification of genetic factors conferring susceptibility to complex diseases, b) deciphering of gene-gene and gene-environment interactions and c) understanding the causal mechanisms behind disease progression. Currently, our focus is to develop statistical methods and software tools that can enable an 'integrative genomics' workflow, where investigators can utilize prior biological knowledge and evidence from omics data such as transcriptome, epigenome to efficiently discover novel variants, mediating genes, their crosstalk with the environment and potential regulatory mechanisms involved in pathogenesis of complex diseases.

Research Highlights

Towards the goal of 'integrative genomics' for complex diseases using the large public genomic databases, we made significant progress on the following directions, this year.

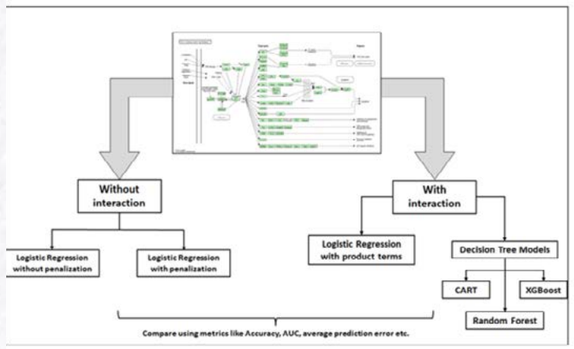
Predicting Causal Variants & Mechanisms using Machine-Learning Techniques: We are building algorithms to predict causal variants and genes based on GWAS data on genomic regions by incorporating prior knowledge from various annotations and functional genomics datasets such as ENCODE and GTEx. We have employed Machine Learning approach (XGBoost) to generate 'mechanism-specific' as well as 'mechanism and tissue specific' decision trees for 5 mechanistic categories based on location of SNP with respect to Gene: 1) Proximal upstream regulation 2) Distal (upstream/downstream) regulation 3) Splicing regulation 4) Coding variation and 5) 5'/3' UTR. These ML models can generate 'prior predictions' for causal SNP/Gene identification from GWAS and omics datasets that can be then used to enhance the accuracy of causal variant identification and colocalization analysis tools.

Formula-s for joint posterior inclusion probabilities (PIP-s) accounting for mechanism-specific regulation have been derived. The likelihood of causal variants (without any prior) is being approximated based on PIP-s derived using fine-mapping algorithms. Once joint posterior probabilities are derived, the posteriors of 1) Causal Variants 2) Mediating Genes and 3) Mechanism of Regulation are being obtained by marginalizing. When GTEx data from relevant tissue are available, eQTL colocalization is one way of prioritizing mediating genes. We have also started to use our ML predictions to prioritize eQTL-colocalization results. In future, we plan to incorporate chromatin-interaction data to further improve the ML models.

Gene-Gene Interaction Effects in Complex Traits:

Gene-Gene and Gene-Envrionment interactions are notoriously difficult to detect from GWAS data, due to the low power resulting from small effect sizes and rarity of specific categories of 'combined risk genotypes at two or more loci'. We are characterizing the best scales and models where genetic interactions are statistically detectable and provide improvements in prediction. Some of the common scales of measurement are risk-difference (or additive scale) where is regressed on presence of genetic and environmental factors, risk ratio (multiplicative scale), where is the outcome in the regression, and odds ratio (multiplicate scale), where is used as the outcome in regression.

We have started to quantitively assess (e.g., see Figure 1) the interaction among GWAS SNPs in complex traits using various parametric models (logistic and log-linear models) with or without LASSO penalization and various Machine Learning models (e.g., CART, XGBoost, Random Forest).



Machine Learning Model	Prediction Accuracy
Logistic Regression	51.06 %
Logistic Regression via penalized maximum likelihood (glmnet)	52.60 %
CART	52.42 %
Random Forest	**53.24 %**
XGBoost	52.62 %

Enrichment Analysis with 1000 permutations

Enrichment Measure	Scale	P-value
Average Z-Score	Multiplicative	**4.89 e-05**
	Additive	**6.95 e-05**
Tail Strength	Multiplicative	**1.96 e-05**
	Additive	**4.48 e-06**

Figure 1. Enrichment of G-G interactions and prediction of risk in Myocardial Infarction GWAS data. Top left shows ML models being compared for prediction and bottom left shows some preliminary prediction accuracy results from 130 selected SNPs (without environmental factors). QQ-plots (top right) show enrichment of pairwise interactions among the SNPs measured in multiplicative and additive scales. QQ plot inflation is quantified by Average Z-score or Tail-strength and p-value assessed using permutations (bottom right).

Research Further, we have analyzed Psoriasis GWAS SNP-s based on UK Biobank dataset, in situations where biological interactions are expected. The conclusion emerging from our analyses is that interactions are present ubiquitously among common variants (more among genes directly connected in disease pathways). The power to detect individual interactions is quite low, but collectively they are detectable using enrichment methods. Further, prediction accuracy can also improve on accounting for interactions, although the improvement is limited if environmental factors (such as Age) and their interactions are ignored.

Role of Transcription Factors in Complex Traits:

We have initiated this objective, where our goal is to study regulatory variants from GWAS data whose contribution in disease-etiology is mediated by a transcription factor (TF). These include cis-eQTL SNPs that alter binding of a TF and regulate a neighboring gene which may be directly involved in the disease process. We aim to identify TF-s which control several such genes through cis-eQTL SNPs based on GWAS summary data using Enrichment methods and understand their possible tissues of action. This will help in elucidating the pathways and processes involved in the disease as well as the contribution of the important TF-s. We have started with some candidate TF-s relevant for glucose metabolism and insulin signaling and are quantifying their enrichment in specific tissues. For this, we are using JASPAR and ENCODE databases to derive TF motifs and target genes, GTEx data to derive tissue-specific cis-eQTLs. Some preliminary tissue and cell-type enrichments have been derived for candidate TF-s as shown in Figure 2.

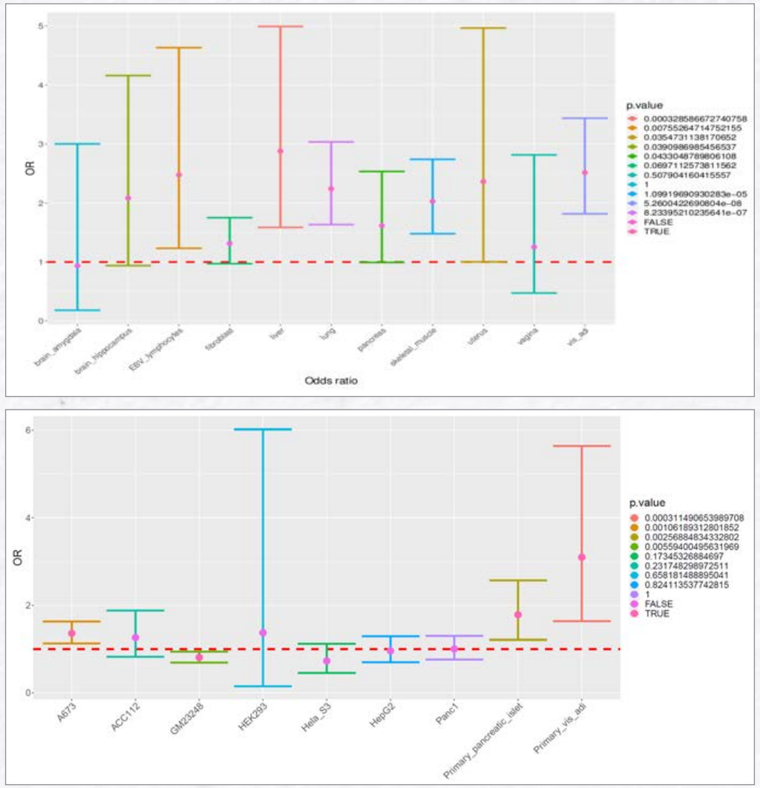


Figure 2. Enrichment p-values for Type-2 Diabetes SNPs for transcription factor SREBF1 in selected GTEx tissues (left) based on ENCODE CHIP-seq data and in some ENCODE cell-lines based on enhancer-marks (histone acetylation).

Database and Web-Portal for Integrative Genomics:

We have made significant progress in implementing an integrative genomics portal for scalable mining of large-scale public genomics datasets. The portal has 3 major parts the 'back-end storage', 'computational modules' and 'interactive front-end'. Various public genomics data are being centrally curated in the 'back-end' storage in our server in a uniform structure with indexing suitable for fast real-time access (e.g., UKBB summary data, GTEx v8 summary data, 1000 genomes data, Gene Ontology database etc. are already curated). The server also has 'computational modules' for various common tasks such as manhattan plot, fine-mapping, enrichment analysis, that employ standard R packages and our in-house scripts. Gradually more analysis and integration modules will be added to the portal. Finally, the front-end is an interactive browser-based interface is being developed using R/Shiny, where the user can select datasets, explore, analyse and visualize using the available modules. Based on preliminary tests, as shown in Figure 3, this infrastructure allows considerable reduction in computational time for basic analysis tasks such as genotype extraction, cis-eQTL data extraction from GTEx and enrichment analysis based on Gene Ontology database.

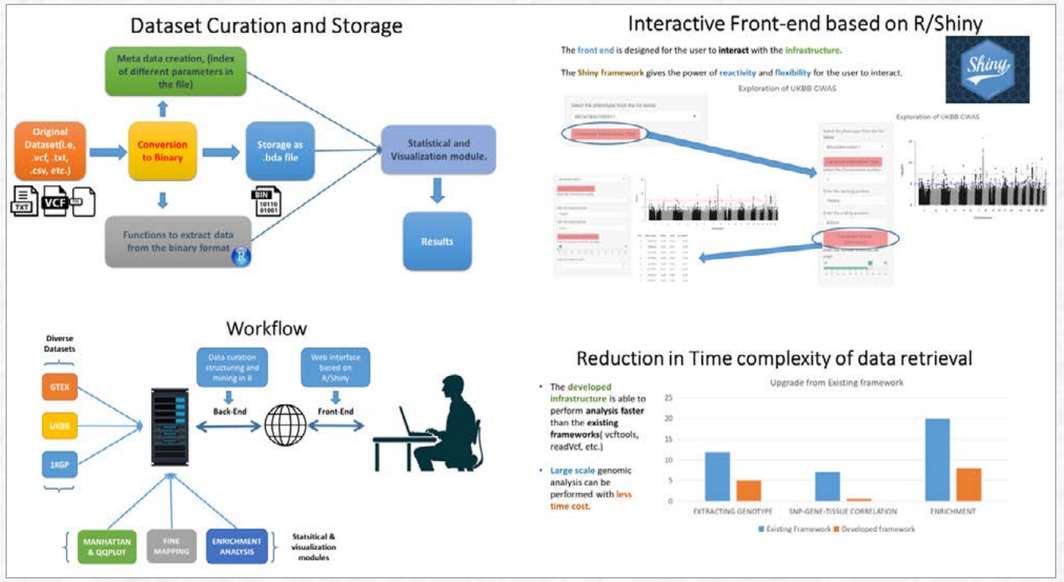


Figure 3. Workflow for Integrative Genomics portal showing how data storage and access in binary mode can make basic analysis tasks faster.

Statistical Methods to Analyse Correlated and Comorbid Phenotypes:

In this newly initiated objective, we are developing statistical methods for cross-phenotype integration. This includes testing for correlated traits in families and identifying common genetic factors underlying them. We are considering a test for genetic pleiotropy among two comorbid traits that is able to distinguish between horizontal pleiotropy (e.g., a SNP showing associations with type-2 diabetes and hypertension which could be related to obesity or inflammation) and mediated pleiotropy (e.g., a SNP associated with type-2 diabetes and hence also diabetic retinopathy). A custom likelihood ratio test was devised to test the proposed composite null hypothesis. We are currently refining the method and running simulations to test its type-1 error and power properties.

Collaborative Research: I have been engaged in several collaborative projects. With Dr. Mainak Sengupta's group at University of Calcutta we have identified cis-regulatory variants including promoter SNPs in ALDH3B1 and RAD52 genes associated with lung cancer and chemotherapy

response in tobacco smokers from Eastern India. In a collaboration with NIMHANS, Bengaluru we studied severe mental illnesses using Whole Exome Sequencing on Indian families, we identified several deleterious rare variants and found pleiotropic action of genes involved in monogenic CNS syndromes. In a collaboration with Dr. Moulinath Acharya's group at NIBMG we have conducted a QT-GWAS of ocular lens thickness in narrow-angle PACG suspects and identified a novel risk locus at PTPRM gene.

Publications

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Research Focus

Major focus of our laboratory is to - (i) identify drivers of oral precancer initiation and molecular progression towards frank cancer, (ii) understand tumour immune heterogeneity in oral cancer, both within and between patients (iii) develop fast and flexible data-analytics pipelines for rapid processing of genomics bigdata for mission-mode projects like ICGC-India project, GenomeIndia project, Indian Breast Cancer Atlas project, etc, and others, (iv) the pandemic has motivated us to utilize our bioinformatics strengths and expertise to help track SARS-CoV-2 lineages across India and the world.

Research Highlights

Our overall research effort is structured into the following two components: (i) *Cancer genomics* - identifying the drivers of oral cancer initiation, progression, recurrence and metastasis, and (ii) *Data informatics* - development of tools and databases to accelerate large-scale multi-omics (genomics, transcriptomics and methylation) data processing. The following are our current ongoing objectives:

A. Genomic underpinnings of oral cancer initiation and progression

- *Comparing and contrasting the early molecular changes in the oral precancerous lesions that are predictive of disease progression. This study would help us build a stepwise evolutionary model for oral cancer pathogenesis.*
- *Profiling somatic copy-number alteration landscape in Indian OSCC-GB patients using genome-wide approach and identification of novel genomic vulnerabilities which are therapeutically targetable.*
- *Understanding the evolutionary perspective of lymph-node metastasis in oral tumours through multi-regional sequencing.*

B. Development of tools, databases and resources for accelerating multi-omics analysis

To develop tools, methods and pipelines that can rapidly process large-scale multi-omics (WGS, WES, RNAseq, Single-cell, spatial) datasets in a high-performance supercomputing setup.

- *To track, monitor and alert temporal fluctuations of SARS-CoV-2 lineages by doing rapid analysis of virus sequencing data to identify emerging lineages and help the national public health agencies combating ongoing pandemic.*

Comparing and contrasting the early molecular changes in the oral precancerous lesions that are predictive of disease progression

Oral squamous cell carcinoma of the gingivobuccal region (OSCC-GB) accounts for most cancer-related deaths in Indian males and sixth-most among Indian females (GLOBOCAN, 2020). Early diagnosis and detection of OSCC-GB is extremely important to improve both life expectancy and quality of life of the patient. Previous studies from our group have characterized genomic, transcriptomic and epigenetic drivers of OSCC-GB in great detail (ICGC-India Team, Nat. Commun., 2013; Biswas NK *et al*., Nat. Commun., 2014; Das D *et al*., Clin. Epigen., 2019). Our recent study (Ghosh A *et al*., J. Path., 2022) identified the genomic underpinnings of oral cancer progression from oral precancerous lesions (leukoplakia). In this study we have identified mutational and transcriptional events associated with the malignant transformation in patients where leukoplakia and frank tumour both were present. During the malignant transformation process, besides accumulation of pathogenic mutations in driver genes, we show immune evasion (depletion of cytotoxic T cells and dendritic cells) with concomitant increase in inflammation (infiltration of MO macrophages and mast cells) played a major role. In order to deeply characterize the stepwise oral cancer progression, tissues from patients with - (a) precancerous lesions without tumour, (b) precancerous lesion with concomitant presence of tumour, (c) early stage tumours and (d) late stage tumours - need to be profiled. Moreover, to understand the tumour initiating events, the precancerous lesions (oral dysplasia), without presence of a frank tumour need to be profiled for genomic, transcriptomic and immune landscape from a relatively large set of patients.

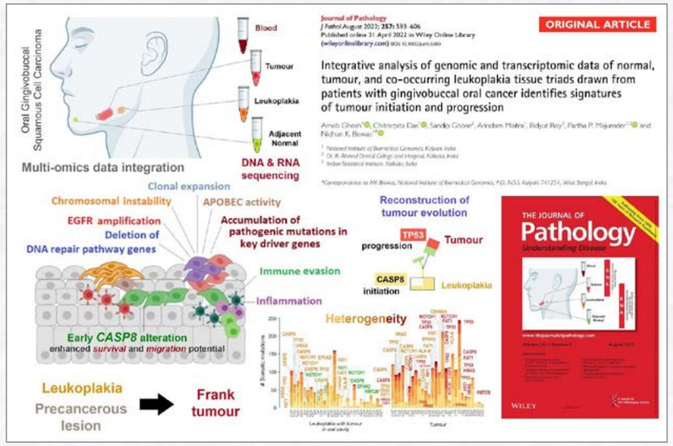

Figure legend: Infographic showing series of molecular events that are associated with tumour progression. [Our manuscript got a mention in the cover page of the journal]

Therefore, as a natural extension to this work, we are presently aiming to capture the complete spectrum of genomic changes resulting in OSCC-GB pathogenesis in a substantial cohort utilizing deep omics-driven methods. We have recruited patients with only leukoplakia (n=50) without frank cancer, patients with both leukoplakia and co-occurring tumors (n=50), early stage OSCC-GB (n=75) and late-stage OSCC-GB (n=100) patients, both through collaborations with a number of clinical organizations (TMH, Mumbai, RADCH, Kolkata) and as part of a past ongoing studies. Multi-omics data was generated for these patients. Integrative data analysis is in progress to understand the progression landscape on a much finer detail. Knowledge about these molecular markers may help improve the efficiency of existing cancer therapy. In addition, we have started validating some of our omics-driven molecular leads favoring OSCC tumorigenesis and aggressive phenotype in suitable oral cancer cell line models.

Software pipelines, databases and data interpretation for viral bioinformatics

Our team has developed an interactive platform called XPLORECoV2, aimed at helping scientists investigate and identify SARS-CoV-2 variants of concern (VOCs). This innovative tool is a response to the need for rapid identification and characterization of VOCs, which is crucial for adapting public health measures to combat emerging threats. XPLORECoV2 sources its genomic sequence data from the Indian Biological Data Center (IBDC). Our team has developed a state-of-the-art pipeline to analyze this data, generating plots that summarize the raw information. These plots provide insights into the prevalence, demographic and geographic distributions, and the common mutations associated with specific variants. The data is presented in a simple-easy-to-use format, allowing users to visually assess dynamics across different divisions and identify trends or anomalies. The pilot website offers a user-friendly interface for researchers and healthcare professionals to evaluate the available SARS-CoV-2 sequencing data, along with its accompanying metadata. By providing up-to-date figures and tables based on shared data, XPLORECoV2 delivers a wealth of information to its users, fostering better understanding and management of the virus. One of the standout features of XPLORECoV2 is its flexible search capability including Pangolin assigned lineage, Nextclade assigned lineage, Clade, State, amino acid and nucleotide mutation. This unique functionality allows users to query variants even before they are designated by a lineage nomenclature system, providing early insights into potential VOCs and their associated mutations. Built using cutting-edge technology, the frontend of XPLORECoV2 is a single-page Nuxt/Vue application written in JavaScript. This robust framework enables seamless interaction with the platform, ensuring a smooth user experience. The platform retrieves data from REST APIs, which are constructed using the popular web framework Django, known for its scalability and versatility.

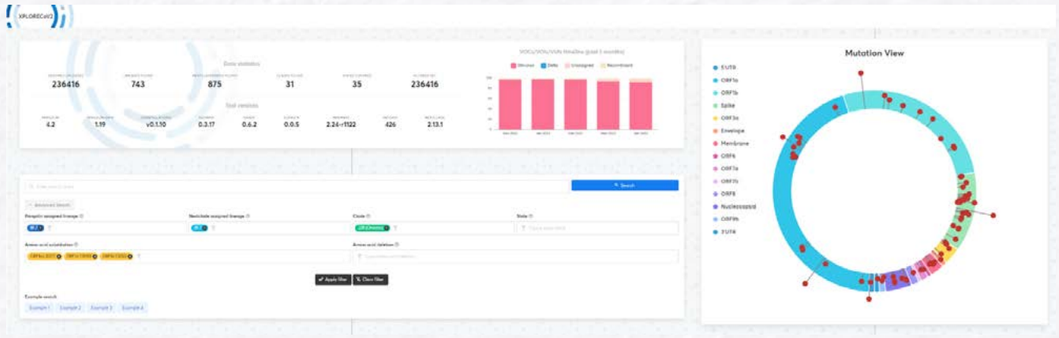

Figure legend: XPLORECoV2 dashboard showcasing the flexible mutation search and data visualization for SARS-CoV-2 genomic analysis

Publications

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Research Focus

Type III interferons (IFN) (or IFN-Ls) are the newly discovered IFNs that have protective as well as inflammatory roles at barrier surfaces. Human IFN-L4 is expressed only in a subset of people who possess the ΔG allele at the dinucleotide variant rs368234815 (TT/ ΔG). My focus is to understand the function of human IFN-L4 in human health and disease using genetic, molecular, immunological, and epidemiological methods. By using molecular and omics methods we seek to define the immunomodulatory roles of IFN-Ls; we also strive to gain a better understanding of the important SNPs in the *IFNL* region.

Research Highlights

The human interferon (IFN) lambda locus has four duplicated genes *IFNL1-4*. The locus is evolutionarily important as several single nucleotide polymorphisms (SNP) have been identified that show strong signatures of selection during human evolution. These SNPs have been known to regulate the expression and/or activity of at least two IFNL genes, i.e., IFN-L3 and IFN-L4 (or IFN-λ4). The dG allele of the dinucleotide variant rs368234815 (dG/TT) gives rise to a new open reading frame (ORF) that is disrupted by the recently evolved TT allele. The *IFNL4* promoter is known to be weak, but the transcriptional regulation of the gene is complex that involves generation of more than ten different splicing variants and also intron retention variants. The significance of such a complex gene expression regulation is not known. In our recently published work (Bharatiya et al, 2023) we have shown the expression of a novel isoform from the TT allele that resembles the active p179 variant of the IFN-λ4 active isoform only in the C-terminus but not at the N-terminus.

When we tested the IFN-λ4 expression in PBMCs, we unexpectedly saw that PBMCs from individuals with the TT/TT variant also made proteins that reacted with a C-terminal specific IFN-λ4 antibody. We verified that these proteins did not come from the *IF1IC2* gene (IFNL4 paralog), which is a pseudogene in humans. We discovered that the new protein from the TT variant uses the same start and stop codons that are used to translate IFN-λ4 from the ΔG allele. However, this protein did not trigger any IFN-related gene expression. Our experiments suggest that this protein likely arises from an alternative splicing event that leads to a frame shift (Fig. 1). The new isoform which we call p179+(TT) is secreted better than p179 protein and is also glycosylated. We also show that the ΔG allele might also produce a similarly frame-shifted isoform (p123+ΔG). It remains to be seen what the possible functional implications for these closely related isoforms of human IFN-λ4 could be.

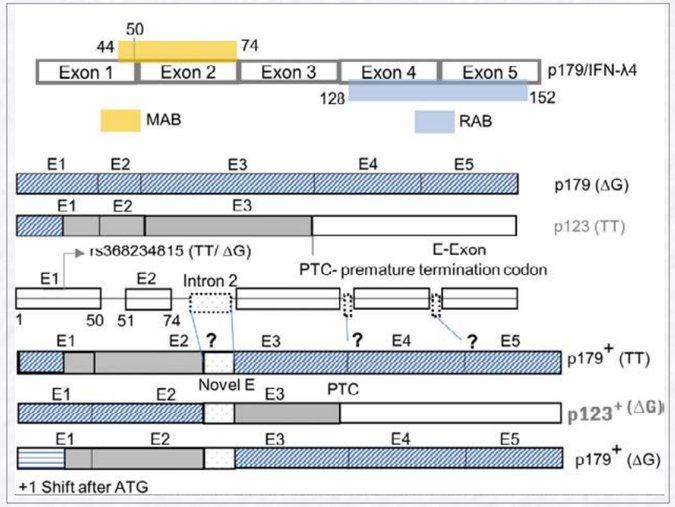


Fig. 1: A possible explanation for the alternate splice event that generates a novel TT allele isoform is schematically depicted; the binding sites for the two antibodies are shown as amino acid positions; exon 1 ends at 50th amino acid position. The novel isoform could be generated due to the splicing in of a novel exon (novel E) from the introns leading to a frame restoring change and expression of p179+ from the TT allele. A similar event happening with the ΔG allele will lead to p123+, but this may lead to nonsense-mediated decay due to the generation of a premature termination codon as a result of the frameshift. The potential expression of p123+ from the ΔG allele can be tested by constructing a +1 frameshift that generates a p179+ in the ΔG allele background as shown.

In another study, based on a cross-sectional population-based design, we have examined the association of the IFNL genetic variants with 30 different blood phenotypes in healthy individuals (N=552) recruited from around the city of Kalyani. We have found that indeed the IFNL genetic variants associate with multiple phenotypes, in a sex-specific manner. While selecting the participants for the study, we applied strict exclusion criteria to rule out any chronic diseases or any viral diseases in the previous 6 months, since viral infections can induce IFNs and could potentially influence the results. We were able to select about 552 self-declared healthy individuals to be included in the study. The age of the participants ranged from 11 to 94 years (median 32 years) with similar distribution in both the genders. The cohort had 268 females and 284 males. We genotyped them at rs12979860 and rs28416813 (both regarded as *IFNL3* SNPs) using competitive allele-

specific PCR assays and tested their association with the blood profiles under dominant and recessive models for the minor allele. IFN-λ4 variants rs368234815 and rs117648444 were also genotyped or inferred

While we saw no association in the combined cohort under either of the models for any of the phenotypes, when we stratified the cohort based on gender, we saw a significant association only in males with monocyte ($p=1\times10-3$) and SGOT ($p=7\times10-3$) levels under the dominant model and with uric acid levels ($p=0.01$) under the recessive model (Fig. 2).

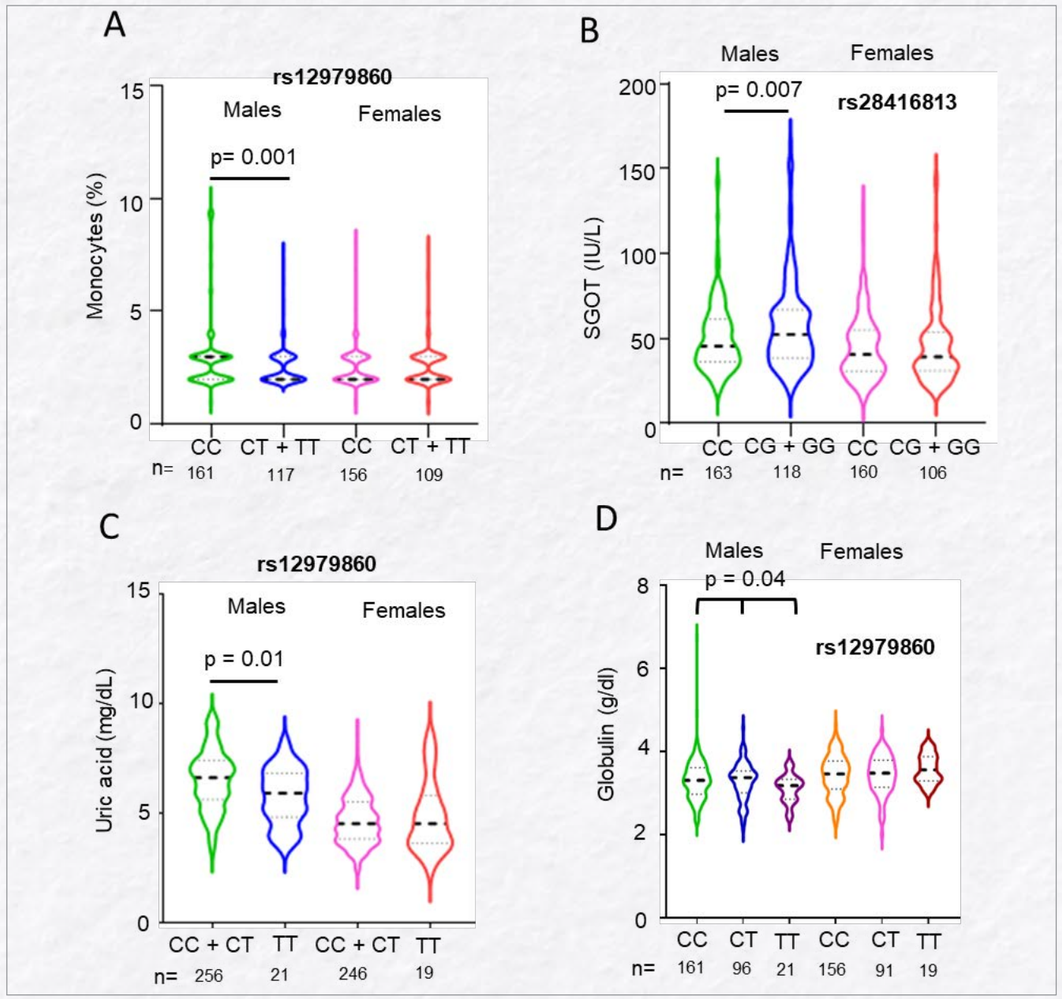
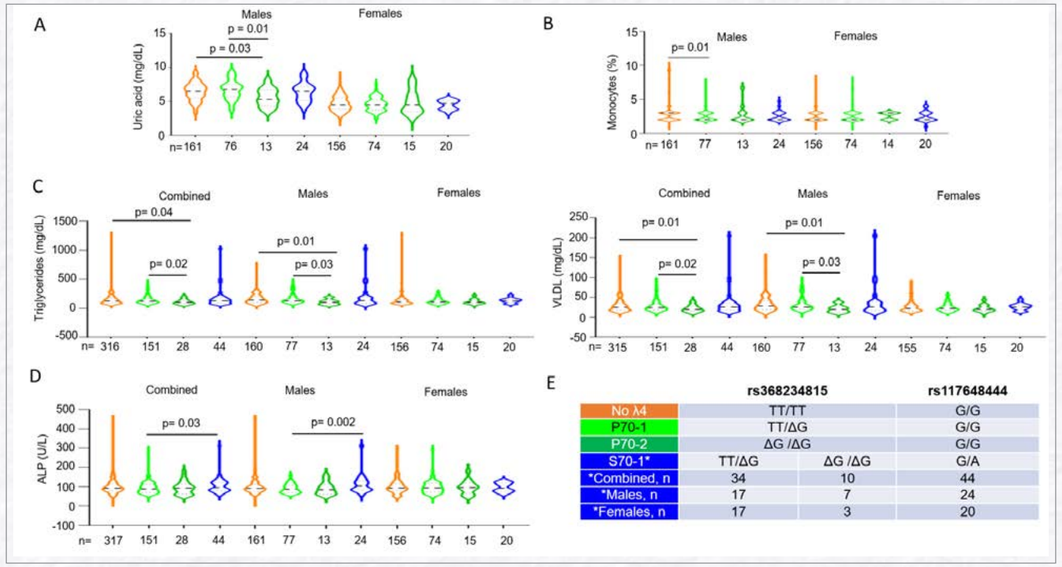


Fig. 2: Multiple phenotypes significantly associate with IFNL SNPs only in males. (a–d) Violin plots showing the distribution of data around the median (thick dashed line shows median and the two thin lines separate the quartiles in each half) for the phenotypes shown according to the IFNL SNPs under the dominant or recessive models (for a, b and c) or additive model (for, d) for the minor allele as shown. All the p-values shown were for the effect of the individual SNPs derived from multiple linear regression analysis with age as the covariate. No significant effect of the SNPs was seen in the female sub-cohort.

When we tested the IFN-λ4 activity modifying variant within groupings based on absence or presence of one or two copies of IFN-λ4 and on different activity levels of IFN-λ4, we found significant ($p<0.05$) association with several phenotypes like monocyte, triglyceride, VLDL, ALP, and uric acid levels, only in males (Fig. 3). All the above significant associations did not show any confounding when we tested for the same with up to ten different demographic and lifestyle variables.



	rs368234815		rs117648444
No λ4	TT/TT		G/G
P70-1	TT/ΔG		G/G
P70-2	ΔG /ΔG		G/G
S70-1*	TT/ΔG	ΔG /ΔG	G/A
*Combined, n	34	10	44
*Males, n	17	7	24
*Females, n	17	3	20

Fig. 3: Multiple phenotypes significantly associate with groupings based on IFN-λ4 copy number and status. (a–d) The IFN-λ4 status was inferred from the genotype information at rs12979860 and rs117648444 for each individual before grouping them in to one of the four groups- no-IFN-λ4, P70-1 (having a single copy of the high activity P70 variant of IFN-λ4), P70-2 (having two copies of the high activity P70 variant of IFN-λ4) and S70-1 (while the large majority of the individuals in this group had one copy of the low-activity S70 variant of IFN-λ4, a minority of them had diplotypes, i.e. had one copy of the high activity P70 variant as well, as explained in e). (e) The IFN-λ4 status based on copy number and activity level explained. A minority (10 in the combined, 07 in the males and 03 in the female sub-cohort) of individuals had both a high activity and low-activity IFN-λ4 variant in them; this grouping strategy allowed us to have more power in testing the S70 variant due to the low MAF of rs117648444 SNP (see Figure 1b). All the p-values shown were for the effect of the individual SNPs/variants derived from multiple linear regression analysis with age and gender (for the combined cohort) or age only (for the individual gender sub-cohorts) as the covariate(s).

The simultaneous availability of genome-wide genotype information from GWAS and large number of phenotypes in health cohorts have augmented genotype–phenotype correlation studies that have been carried out as phenome-wide association studies (pheWAS) and the summary statistics are made available in public databases. The GBE (McInnes et al., 2019) that has PheWAS data from the UK biobank is one such resource. We searched the GBE for all the phenotypes that are associated with any of the known IFNL genetic variants (Figure 4). We saw that the IFNL genetic variants with varying MAFs were associated with multiple phenotypes similar to our results. While the p-values are comparable, the significant association in our study was only from the male sub-cohort, while the gender-stratified data in the GBE was not accessible.

Further, when we compared the effect size of the IFNL SNPs with that of the top SNPs in the UK biobank (top ten SNPs were selected: top five for effect sizes and top five for significance levels), we found the effect sizes we report for the male sub-cohort were better than at least six of the top ten SNPs in the UK biobank for monocyte counts and SGOT. These results have been recently published as Roy DG et al, 2023, *Mol. Gen. And Genomic Med.*

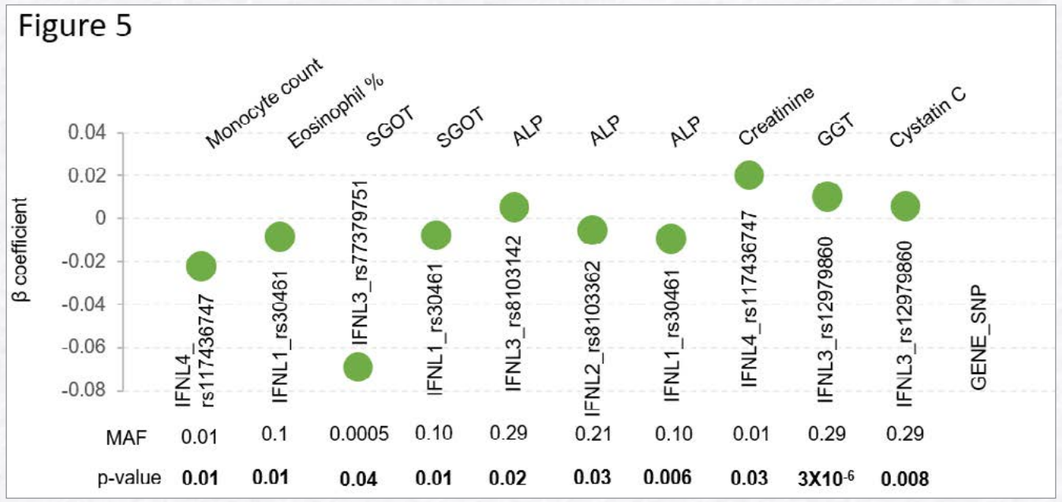


Fig. 4: Summary statistics of PheWAS data derived from the UK biobank showing the multiple cross-phenotype association of IFNL SNPs. The data was accessed from Global Biobank Engine (GBE), Stanford, CA (URL: http://gbe.stanford.edu) [June–July 2023]. MAF-minor allele frequency.

Publications

Bharatiya S, Agarwal A, Chinnaswamy S. A Novel Inactive Isoform with a Restored Reading Frame Is Expressed from the Human Interferon Lambda 4 TT Allele at rs368234815. J Interferon Cytokine Res. 2023 Sep;43(9):370-378. doi: 10.1089/jir.2022.0199. Epub 2023 Mar 7. PMID: 36880961; PMCID: PMC10517323.

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Research Focus

Major challenges in breast cancer are understanding the reasons for tumor recurrence and having therapeutics to treat resistant tumors. Our laboratory employs molecular, cellular and genomic technologies to identify novel long non-coding RNAs, studies the contribution of serum derived exosomes, and cancer relevant proteins such as Jumonji domain containing protein 6 (JMJD6) and Y-box binding protein 1 (Y-box1) towards tumor behavior and microenvironment. Our integrative efforts may yield an improved understanding of cancer and identify molecular probes/tests for improved prediction of cancer progression and recurrence.

Research Highlights

JMJD6 and Tamoxifen Insensitivity:

Our lab has shown that high JMJD6 expression associates with poor prognosis in breast cancer patients and it induces cell proliferation and motility in cancer cells. JMJD6 also participates in ER regulated gene expression using its enzymatic activities and by enhancing hormone induced ER transcriptional activity by releasing RNA pol II from paused or stalled sites. JMJD6 demethylates Arg residues in ER to promote its nuclear localization to regulate its non-genomic actions. JMJD6 appears to positively regulate ER action in the presence of hormonal stimulus. However, despite Tamoxifen mediated blockade of ER in breast cancer patients, higher JMJD6 expression associated with poorer prognosis in women undergoing endocrine therapy. Our preliminary observations from Gene set enrichment analysis (GSEA) of microarray data in MCF7 cells overexpressing JMJD6 showed similarity with patterns found in Tam resistant cell lines and in tumors from xenograft studies. This suggests that JMJD6 appears to have functions beyond that of promoting ER target gene expression in ER+ breast cancer. Therefore, we explored the transcriptional program elicited by JMJD6 in MCF7 cells (JOE cells) and its contribution to endocrine therapy response. 76% of differentially expressed genes (DEGs) overlapped with ER target. However, JMJD6 decreased ER target gene expression prompting us to check ER levels. A significant decrease in both ESR1 and ER levels was observed in JOE cells that was reversed by JMJD6 siRNA transfection. Additionally, JOE cells showed increased RET and ERK1 expression, events that are associated with resistance to endocrine therapy. Accordingly, JOE cells displayed lower sensitivity and survived better at higher doses of 4-hydroxy tamoxifen (Tam) as compared to parental MCF-7 cells. We developed two models of Tamoxifen resistance that are derived from parental MCF7 cells; tamoxifen resistance (TAMR) cells and LongTermEstrogen Deprived cells. LTED cells have different sensitivity to estrogen (LTED-20/LTED-Q quiescent; LTED-40/LTED-H hypersensitive; LTED-80/LTED-I Independent). Our premise was that if JMJD6 associated with Tam insensitivity, TAM R and LTED-80 cells would show higher JMJD6 expression, and both did. Further, JMJD6 siRNA treatment decreased growth and improved Tam sensitivity in both cells. Comparison of JOE DEGs with known Tam signature genes showed a substantial overlap. We further demonstrated that several TAMR signature genes were found to be regulated by JMJD6. These data indicate that JMJD6 maybe an upstream regulator of multiple genes that are associated with Tam resistance. The data from this study is summarized in the graphical abstract below (Figure 1).

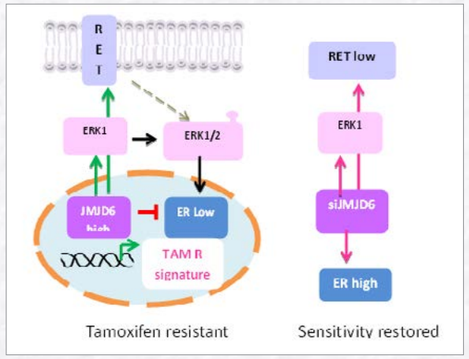


Figure 1: Graphical abstract of JMJD6 mediated Tam insensitivity

We propose that JMJD6 levels may perform well as a valuable marker to determine response to endocrine therapy and the use of iJMJD6 as a viable therapeutic strategy to treat ER+ breast cancers involving high expression of JMJD6.

To determine the common pathways to Tam insensitivity we have performed RNA-seq analysis of parental MCF7, TAMR, LTED-20 and LTED-80 cells. Analysis of these datasets identified 1104 genes that were differentially regulated in TAMR, JOE and LTED-80 cells that were insensitive to tamoxifen (Tam set). We compared the tumor versus normal gene expression data from Firehose Legacy and Pan-cancer TCGA breast cancer datasets to determine the differentially expressed genes. Next, we checked the profile of Tam set of genes in tumors. Of the 1104 genes, 331 were differentially expressed in TCGA breast cancer data when compared to Adjacent normal. We found that genes that were commonly regulated in the Tam set were oppositely expressed in the TCGA data. The RNA profiling of these samples has been carried out at diagnosis, however the follow-up data to determine the outcome of Endocrine therapy is unavailable. Nevertheless, we propose that reversal of direction of expression of these genes could be responsible for endocrine therapy resistance. The common genes mostly belonged to the cell proliferation cluster of genes as shown in figure 2. However, more in-depth analysis of these data is required to find genes that can inform Tam insensitivity at diagnosis.

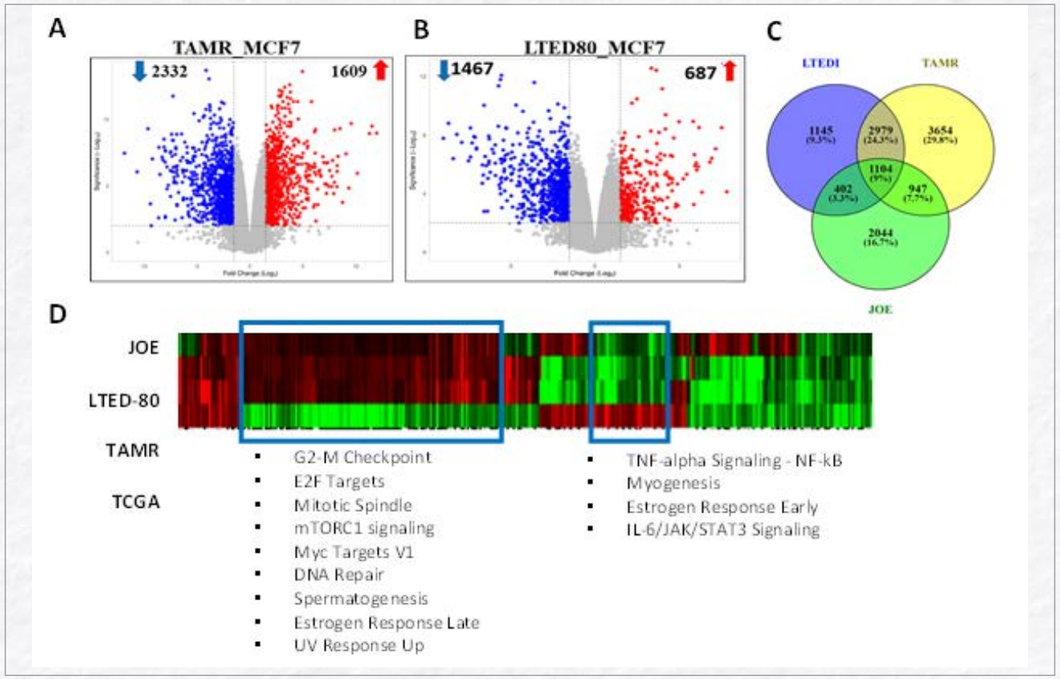
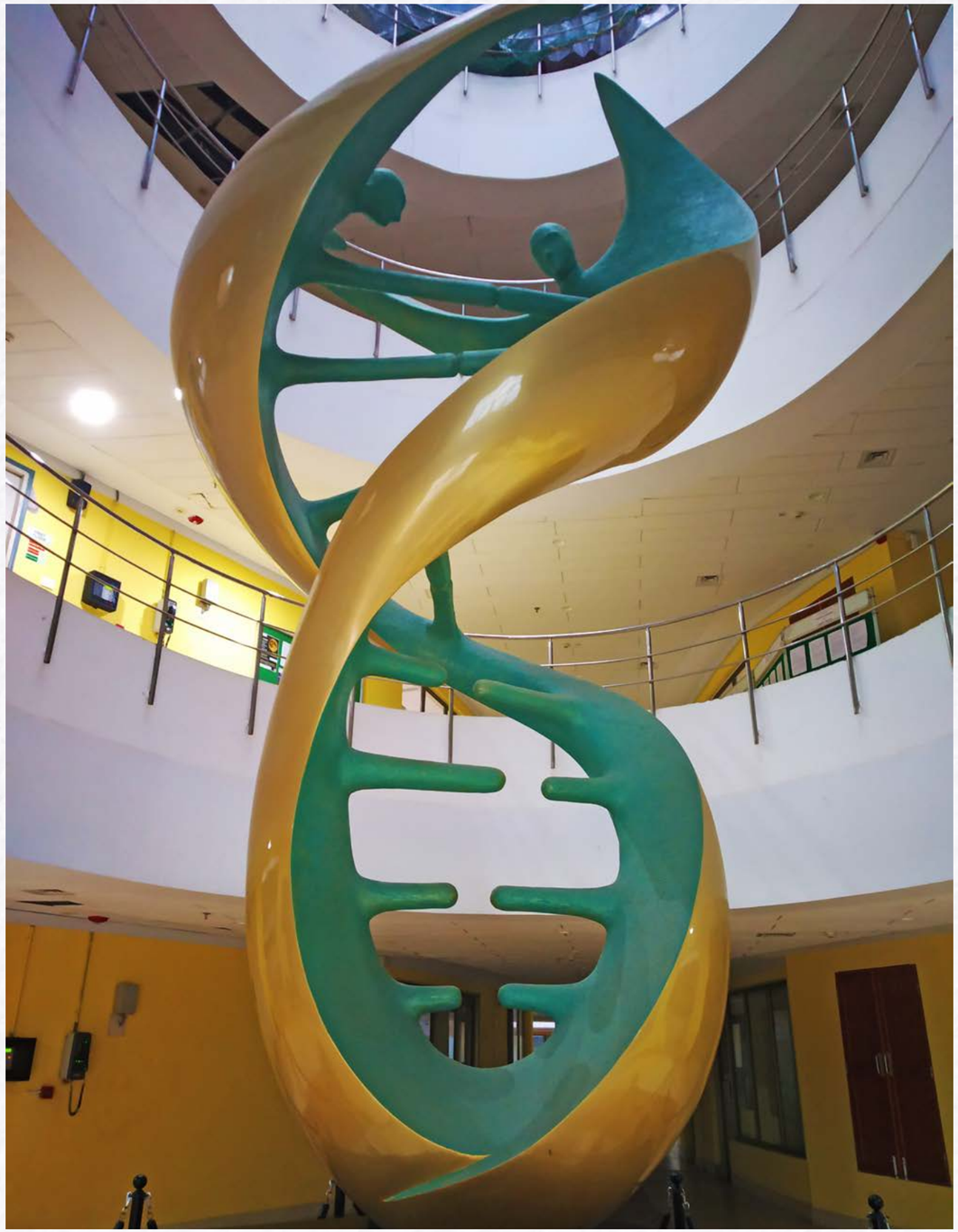


Figure 2: Volcano plots of TAMR and LTED80 cells are shown in figure A and B respectively. C) Venn diagram showing 1104 genes that are common in the 3 cell lines and may contribute to Tam insensitivity. D) Comparison of TCGA ER+ tumor gene expression and pathways that may require alteration to execute endocrine therapy resistance.

Publication

Das P, Gupta A, Desai KV. JMJD6 orchestrates a transcriptional program in favor of endocrine resistance in ER+ breast cancer cells. Front Endocrinol. 2022 Nov 7: 13 PMID: **36419768**

Group photo of the Lab



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Research Focus

Chronic Pancreatitis (CP) is a disease of the pancreas where progressive inflammation of the organ ultimately leads to the destruction of both exocrine and endocrine part of pancreas resulting in high morbidity and mortality. The disease also increases the susceptibility of the patients to develop pancreatic ductal adenocarcinoma (PDAC) representing a classic model of progression of chronic inflammation to malignancy. Our main research focus is to perform comprehensive analysis and integration of transcriptomic and methylome data and functional characterization of key leads to have a mechanistic idea of development of PDAC from chronic inflammatory disease of the pancreas.

Research Highlights

Title: Exploring functional role of malignancy specific genes in pancreatic cancer

Funding Agency: DST-SERB, Govt. of India and Intramural, NIBMG

Description:

We hypothesize that:

OSBPL3 and DLGAP5 genes play significant role in both development and progression of pancreatic ductal adenocarcinoma.

In order to test the hypothesis, we shall focus on the following specific objectives:

Specific Objective 1: To find out the exact contribution and molecular mechanism of action of OSBPL3 and DLGAP5 in pancreatic ductal adenocarcinoma.

Specific Objective 2: To explore the molecular mechanism underlying the deregulation of OSBPL3 and DLGAP5 in pancreatic ductal adenocarcinoma.

In the FIRST Specific Objective, we plan to interrogate the functions of these genes in the disease. In order to do that, we need to follow specific experimental procedures.

(a) To find out the expression of OSBPL3 and DLGAP5 in pancreatic cancer cell lines:

We have compared the expression of OSBPL3 and DLGAP5 in pancreatic cancer cell lines to normal ductal cells. Both the genes were upregulated in all the cell lines. MIAPACA-2, BxPC-3 and PANC-1 carry TP53 mutation along with CDKN2A and SMAD4 mutation. AsPC-1 carries both KRAS and TP53 mutations. AsPC-1 is metastatic cell line.

(b) Cloning of the genes:

We amplified the respective cDNAs from the cells having the maximum expression. Subsequent steps were followed to clone both the genes in pcDNA-3.1(+) mammalian expression vector.

(c) Knockdown of the genes:

In order to perform knockdown of the genes, we decided to design shRNAs against the relevant target regions of both the genes, which were decided based on published literatures.

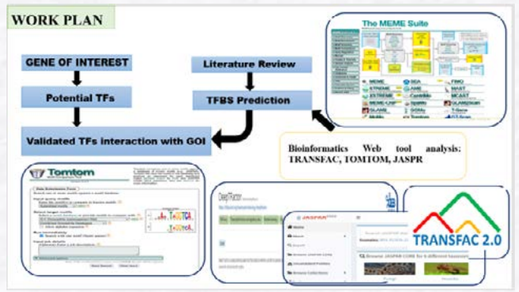

Figure-1: Workflow for bioinformatic prediction of Transcription Factors

Oligonucleotides for the required sequences were ordered, annealed and cloned into pLKO.1 vector following necessary steps. Clones were confirmed through differential digestion by AgeI and EcoRI.

(d) Cellular assays:

We have in the process of using overexpression and knockdown plasmids and perform proliferation, invasion and apoptosis assays to find out the role of these to genes in pancreatic cancer.

In the SECOND Specific Objective, we plan to find out how the expression of these two genes are regulated. The regulation could be either transcriptional or post-transcriptional. To find out what are the possible transcription factors possibly binding to the promoters of these genes we took help of bioinformatic means. MEME discovers novel, un-gapped motifs (recurring, fixed–length patterns) in input sequences and TOMTOM compares one or more motifs against a database of known motifs (e.g., JASPAR) (Figure-1).

Interestingly, the prediction resulted in identification of FOXM1 as an important Transcription Factor binding to the promoter of DLGAP5. FOXM1, or transcription factor Forkhead box M1, was also a candidate identified in our transcriptome analysis and was part of the five-gene signature for malignancy specific genes. So, the prediction suggested that there could be possible cross-talk between them. In order to have more information on their possible interaction, we looked at STRING

database and also explored the correlation of expression between them in pancreatic cancer patient samples. STRING result showed interaction between them and their expression in pancreatic cancer patient samples were also very highly correlated. At present, we are in the process of validating these bioinformation predictions.

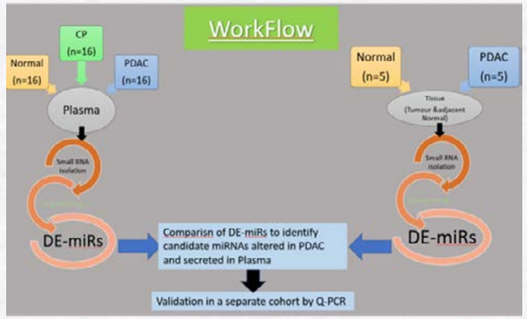

Figure-2: Overall work-flow of the study

Title: To explore the alterations in circulating small RNAs between Pancreatic Cancer and Chronic Pancreatitis patients

Funding Agency: WB-DSTBT

Description:

Confirmed cases of chronic pancreatitis and pancreatic ductal adenocarcinoma were recruited in the study and blood was collected from them. Total RNA was isolated from blood plasma and small RNA sequencing library preparation was performed using Illumina® TruSeq® Small RNA Library Prep Kit according to the manufacturer's instructions. Subsequently reverse transcription and amplification were performed to generate a cDNA library. Single read 1 x 50 bp sequencing of these pooled libraries were performed in Novaseq-6000 (Illumina).

In addition to that, we have also collected pancreatic cancer and adjacent normal tissue samples from patients and performed similar small RNA sequencing (Figure-2).

We have received the sequencing data and at present, we are in the process of analyzing them.

Group photo of the Lab

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Research Focus

Mass-spectrometry based proteomics and metabolomics: Pathophysiology of oral, pancreatic and colon cancer. Our primary research is to study cellular metabolism implicated in colon, pancreatic and oral cancer. In the recent past, cellular metabolism has emerged as an integral process in generating key signaling molecules associated with various cellular processes including immune response, cell growth, apoptosis, and redox homeostasis. Often, cancer cells adopt dramatic metabolic phenotype to support proliferation and progression. Our lab is interested in understanding cancer and stages specific metabolic phenotype as well as the factors that influence/regulate proteome and metabolome remodelling that would aid in developing potential therapeutics.

Research Highlights

Proteomic profiling of $CD44^{positive}CD24^{positive}$ and $CD44^{positive}CD24^{negative}$ tumour derived cell lines to understand the principles underlying proteomic remodelling in defining the role of CD24 during EMT (Epithelial-mesenchymal transition).

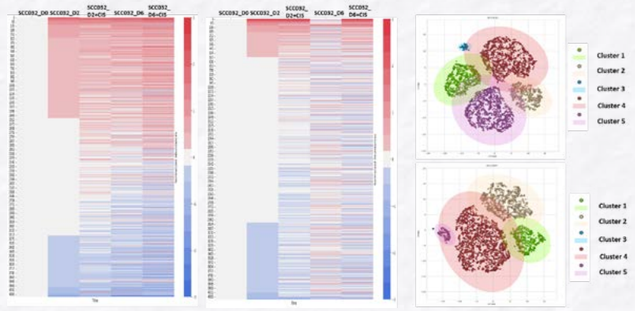

Figure 1. Comparative quantitative proteomics analysis of SCC032 and SCC084. A total of 4477 proteins were quantified across all samples. Further, the proteome was analyzed, and k-means clustering was performed derived from t-SNE.

Summary:

The preliminary data obtained from proteomic analysis suggests that the SCC032 ($CD44^{positive}CD24^{negative}$) cells are sensitive to cisplatin in comparison with SCC084 ($CD44^{positive}CD24^{positive}$) cells in a TGF- β induced model system. Dramatic proteomic remodeling was observed in SCC032, where SCC032 cells are adopting OXPHOS metabolic phenotype on 6^{th} day of TGF-β induction in comparison with SCC084 cells. Taken together, our data suggests that CD24 status plays a central role during EMT in OSCC cells. However, further validation in clinical oral cancer tissues studies needs to be performed.

Group photo of the Lab

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Research Focus

Oral Cancer

We are elucidating the transcriptomic diversity of oral cancer at the single-cell level, to understand how it impacts inter and intra tumour heterogeneity and treatment outcome.

Maternal and Child Health

We are unravelling the genomic, epigenomic and transcriptomic factors and unique placental cell types contributing to preterm birth, to identify at-risk women and develop targeted interventions.

Gene Regulation

Our investigations seek to identify cell-type and cell state specific expression quantitative trait loci (eQTLs) in peripheral blood, shedding light on diversity of gene regulation across individual cell types and states diverse in immune responses and defining the healthy state.

Research Highlights

Cellular heterogeneity in OSCC ecosystem

We conducted single-cell RNA sequencing (scRNA Seq) of 52,393 cells isolated from tumours of 12 treatment-naïve OSCC-GB patients. We developed an in-house method by distance-profiling to malignant cells, which exhibited predominantly patient-specific clustering why non-malignant cells showed clustering by cell type.

Specific malignant cell clusters exhibited gene expression signatures of partial epithelial-mesenchymal transition (pEMT) and fetal cellular reprogramming. We observed a mutually exclusive relationship between these two gene programs, with cells expressing pEMT gradually transitioning towards those expressing fetal cell-like genes.

We also observed considerable heterogeneity among non-malignant cells, evident both within individual patients and across the patient cohort. Of particular significance were the DNT (double-negative) T cells, likely to be pivotal in the anti-tumor immune response in OSCC-GB. Myeloid cells emerged as the second-largest immune cell population within the OSCC-GB ecosystem, emphasizing their role in disease progression. These macrophages exhibited a dual pro-inflammatory and pro-tumorigenic nature, indicating a transitional phase in the tumor microenvironment that may favor malignant cell proliferation.

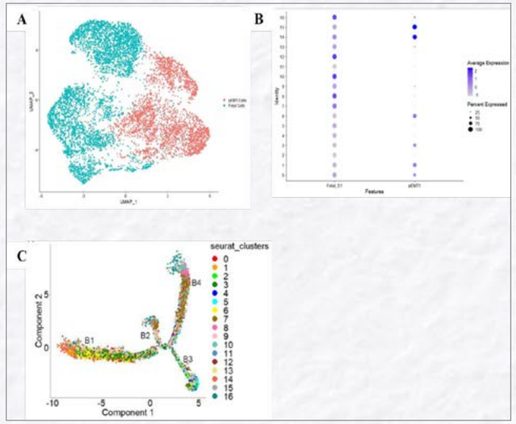

Figure 1(A): Malignant cells show one of two dominant gene expression programs- pEMT or Fetal Cellular Genes **(B)** Dot plot of the average expression of pEMT and fetal cell type signatures across malignant cell clusters **(C)** Pseudo-time analysis exploring the development trajectories of malignant cells.

Multi-omic study of spontaneous pre-term birth (sPTB)

We are conducting our studies on spontaneous preterm birth (sPTB) on pregnant women from GARBH-Ini (interdisciplinary Group for Advanced Research on BirtH outcomes- DBT India INitiative) cohort located in Haryana, India. We performed a GWAS on 6,211 pregnant women at 700,604 genomic loci. Our analysis identified 38 SNPs associated with PTB outcome, and one highly significant genome-wide SNP, rs57480735 (OR=1.57, p=2.78e-8), which also showed an association with gestational length (β=-0.16, p=2.05e-3). Out of these 39 SNPs, 15 were also linked to early sPTB (<33 weeks). Furthermore, in a cross-ancestry meta-analysis combining our data with 23andMe sPTB GWAS results (conducted on European women), we identified 199 transethnic SNPs, with rs35760881 being the most significant (p=8.45e-7).

We found that rs57480735 is a cis-eQTL of *NR2F2*, a gene involved in maternal placental development, which was highly expressed in maternal-origin cell types. Women harbouring the minor allele of rs57480735 who delivered preterm had elevated uterine arterial pulsatility index, indicating suboptimal placental development. The transethnic SNP, rs35760881, was a cis-eQTL of *AKIP1*, potentially triggering early myometrial contractions via inflammation, with high expression observed in placental immune cells. Women carrying the minor allele of rs35760881 and delivering preterm exhibited high systemic mean arterial pressure, suggesting early myometrial

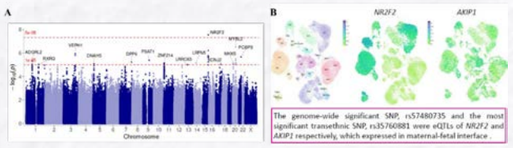

Figure 2 (A) Manhattan plot showing results on genome-wide significant SNPs associated with preterm birth. **(B)** Expression of specific genes which are regulated through eQTL effects of genome wide significant and most significant trans-ethnic SNPs, in placenta single-cell RNA sequencing dataset.

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contractions. Our findings suggest that inflammation may contribute to sPTB across populations, whereas poor placental development may be specific to certain populations.

In the epigenomics arm of our study, we conducted DNA methylation profiling at 865,918 CpG sites, involving longitudinal sampling at three stages of pregnancy and delivery. Using a linear mixed model analysis, we identified CpG sites exhibiting significant differential methylation (Bonferroni-adjusted p-value < 0.01) which fell into two distinct temporal methylation trends: those increasing during gestation and those decreasing. Genes associated with increasing methylation were linked to T cell activity, while those with decreasing trends were related to solute transport and cell structure organization. The consistent DNA methylation patterns observed in maternal peripheral blood underscore the crucial role of T cells in supporting a healthy pregnancy. Moreover, the coordinated cellular remodelling, as indicated by these methylation trends, appears pivotal for a successful full-term delivery.

Previous findings from our laboratory suggested the involvement of telomere homeostasis in pregnancy outcomes. We undertook a targeted analysis of the genomic and epigenomic factors specifically in telomere homeostasis genes identified genomic variants in the *TNKS* gene and to be significantly associated with sPTB. Notably, the minor allele (C) of rs78731121 correlated with hypermethylation at TSS-200 of *TNKS* at 11-14 weeks and hypomethylation in the gene body of *TNKS* at 26-28 weeks of gestation. We also identified a CpG site, cg00538229, located in the TSS-1500 region of the *TAL1* gene, which was significantly hypomethylated in sPTB delivering women at 11-14 weeks of gestation.

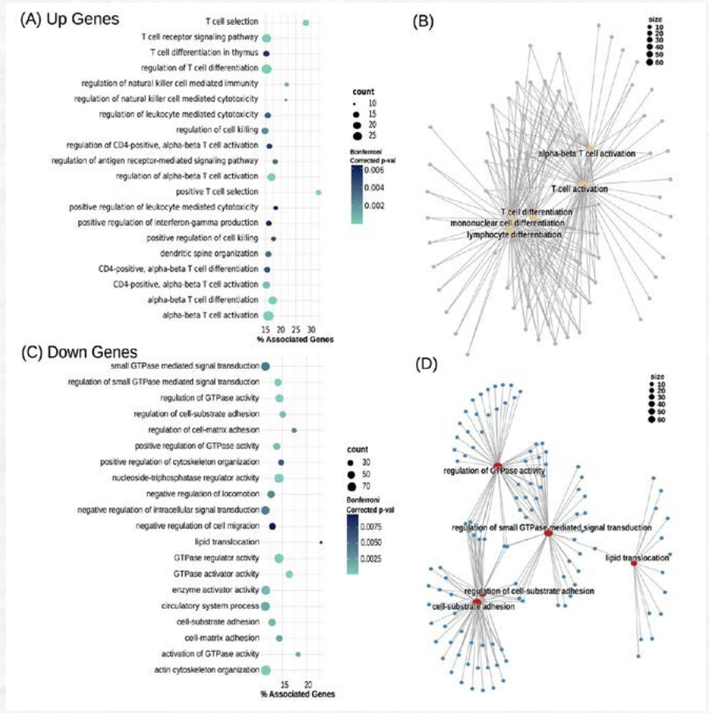

Figure 3(A, C)- GO Biological Processes enriched by Up and Down trending genes. **(B, D)**- Gene Concept Network using all the significant Up and the Down trending genes respectively, which shows the major Biological Processes.

Investigating gene regulation across cell types and cell states

Our objective is to determine cell-type-specific eQTL effects in the peripheral blood immune cells of healthy individuals, which might govern the regulation of various immune responses. We conducted scRNA-seq and genome-wide genotyping of PBMCs in a cohort of 8 healthy adult individuals. We sequenced about 25,000 peripheral blood immune cells and identified major cell-types and cell-states and selected five candidate cis-eQTLs (SNP-gene pairs), which have been previously reported in bulk studies or eQTL databases (GTEx, eQTLGen) and for which all three genotypes of the SNPs were available in our dataset. Significant associations were observed exclusively in CD8+ effector T cells for two cis-eQTLs: rs6547705-*CD8A* (p=0.0229), and rs10806425-*BACH2* (p=0.035) and only in dendritic cells (DCs) for rs7298416-*CLEC12A* (p=0.018). Additionally, rs9442372-*HES4* exhibited a weak effect in plasma B cells, and rs2223286-*SELL* in CD14+ monocytes.

Our findings offer initial evidence supporting the presence of cell-type-specific eQTLs, indicating the potential for refining the characterization of the healthy immune state through such investigations.

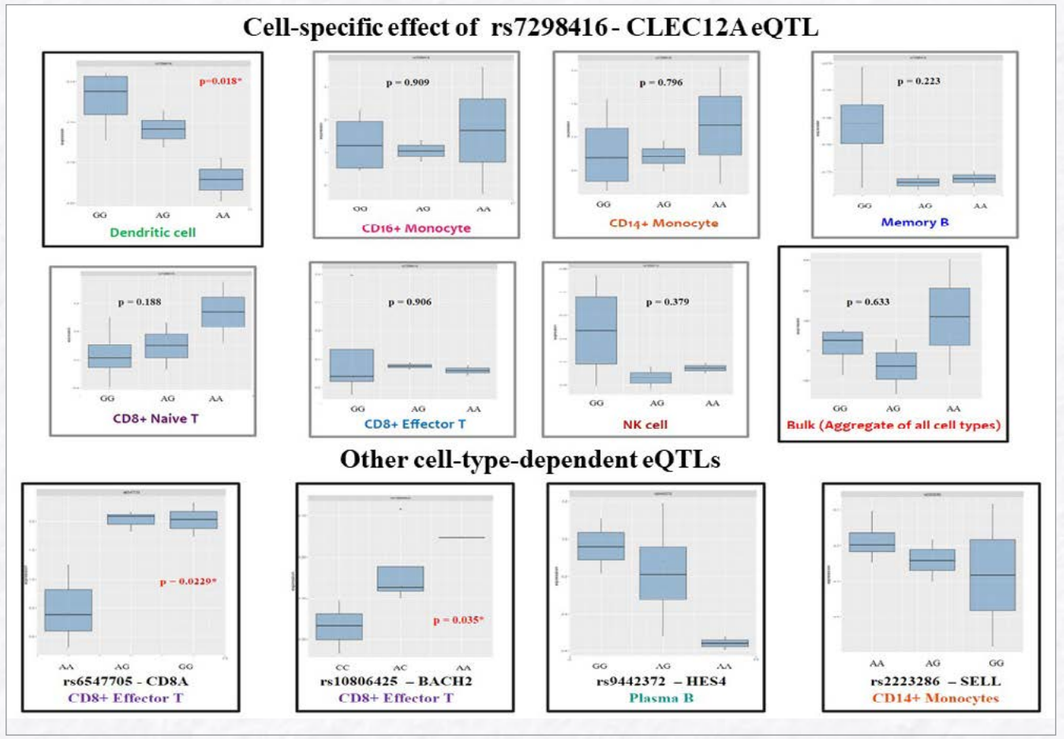

Figure 4: Determination of cell-type-specific effects of eQTLs across different peripheral blood immune cell types and cell states.

Publications

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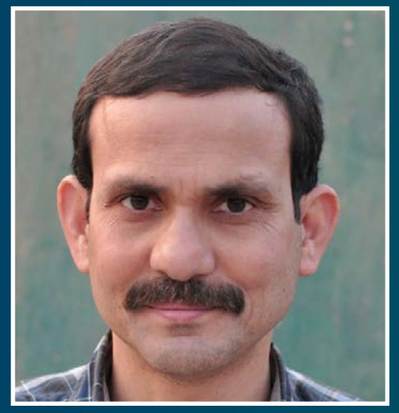
Pradhan A, Aneja A, Ghosh S, Devvanshi H, C D, Sahu R, Ross C, Kshetrapal P, **Maitra A**, Das S. Association of exosomal miR-96-5p and miR-146a-5p with the disease severity in dengue virus infection. J Med Virol. 2023 Mar;95(3):e28614.

Group photo of the Lab



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Deepshikha Shaw

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Research Focus

Neonatal Sepsis:

A Preventable Tragedy with Genomic Promise

Sepsis, a life-threatening infection, is a leading cause of preventable neonatal death. Genomic technologies will revolutionize the diagnosis and treatment of sepsis in newborns.

Nanopore sequencing enables rapid and targeted antibiotic therapy in the Neonatal ICU, improving patient outcome.

Systems biology provides new insights into the underlying mechanisms of Disseminated Intravascular Coagulation (DIC) in sepsis. These insights can be used to identify new therapeutic targets.

Our lab aims to save lives and improve the quality of life for survivors by developing innovative ways to identify and target the disease-causing mechanisms of sepsis.

Research Highlights

Project 1:

Molecular characterization, immunopathology, and optimized therapy of *Klebsiella pneumoniae*: A comprehensive approach to understanding and combating a major pathogen. [Manisha Rout]

Klebsiella pneumoniae is a Gram-negative bacterium that is a leading cause of sepsis. This project aims to comprehensively understand the key players of *K. pneumoniae* pathogenicity and develop optimized therapies to maximize efficacy and minimize toxicity. We will investigate antibiotic resistance, virulence, and host immune responses to *K. pneumoniae infection.* This will involve using molecular techniques to identify the genes and proteins involved in these processes, as well as studying the interactions between *K. pneumoniae* and the host immune system. We will also explore different antibiotic combinations to develop optimized therapies for *K. pneumoniae* infections. This will involve screening antibiotic combinations for synergy against *K. pneumoniae* strains. By taking a comprehensive approach to understanding and combating *K. pneumoniae*, we hope to develop new and innovative strategies to improve patient outcomes.

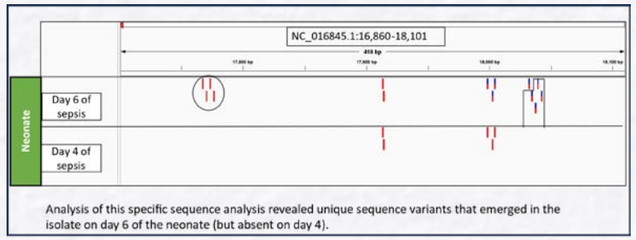


Figure 1: SNPs and Indels in region between 16860 and 18101 of the sample from day 6 but not of sample from day 4 of sepsis.

To identify the pathogen factors responsible for the hypervirulent nature of *Klebsiella pneumoniae*, we extended our previous analysis to include 6 more samples collected from sepsis patients in collaboration with the SCB Medical College & Hospital, Cuttack, Odisha. In total, 12 (3 hypermucoviscous and 9 classical strains) isolates of *K. pneumoniae* from various human clinical biospecimen were subjected to bacterial whole-genome sequencing.

We established a pipeline to generate annotated variant call files as well as genome assemblies to identify the sequence type, antimicrobial resistance genes, and virulence genes. We observed many sequence variants uniquely present in the virulent group of isolates, affecting lipoprotein and capsule polysaccharide biosynthesis.

We were particularly interested in identifying the genomic differences between isolates from the same neonate on different days. Analysis of the genomic regions revealed many SNPs and Indels in the region between 16860 and 18101 of day_6 sample but not of day_4 sample. Variant attributes for these SNPs and Indels suggested that the gene KPHS_00120 (ribose operon repressor RbsR) was affected in all mutations (Figure 1). Functional annotation suggested potentially disruptive changes in terms of polarity and charge content of the amino acids in the gene product. RbsR has previously been associated with virulence in *Aeromonas hydrophila* and *Staphylococcus aureus*, where it has been shown to promote increased survival by increased adherence and capsule production. Mutations in this downstream gene variant may have generated a gain of function or increased expression leading to the hypermucoviscous phenotype observed in sample collected from the patient on day 6. The association of virulence with the RbsR gene and its effect on hypermucoviscosity has not been reported in *Klebsiella pneumoniae* to date. Our findings suggest that RbsR may be a novel virulence factor associated with hypermucoviscosity in *Klebsiella pneumoniae*.

We have established new collaborations and continue to strengthen our old collaborations. Currently we have been actively collecting bacterial isolates from AIIMS Bhubaneswar, SCB Medical College Cuttack, and SRM University, Sonepat. These isolates shall be subjected to phenotypic and genomic characterization.

Project 2:

Exploration of Neutrophil Extracellular Trap formation (NETosis) in human bacterial sepsis [Deepshikha Shaw]

Sepsis is defined as life-threatening organ dysfunction caused by the dysregulated host response to infection. One of the dysregulated host responses is Disseminated Intravascular Coagulation (DIC), which is a major complication of sepsis and a strong predictor of multi-organ failure and death. Recent research, including from our lab, has shown that the formation of Neutrophil Extracellular Traps (NETs) can lead to the activation of intravascular coagulation pathways.

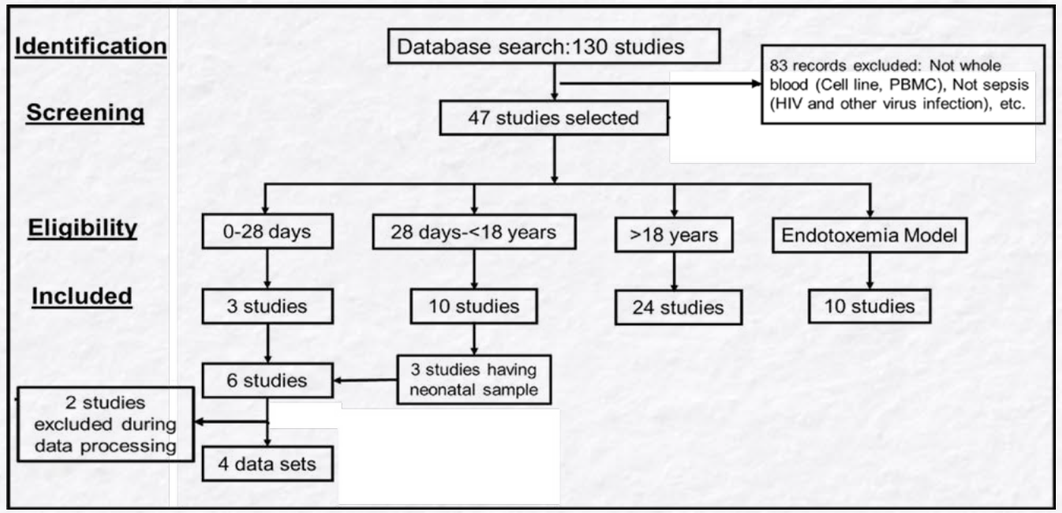


Figure 2: Flow-chart for selection of transcriptomic data sets

We sought to establish the association of NETosis with bacterial sepsis by re-analyzing published transcriptomic data sets retrieved from the NCBI GEO database (Figure 2). We identified 47 data sets and divided them into four groups: neonates (n=3), children (n=10), adults (n=24), and human endotoxemia model (n=10). We selected four neonatal data sets for further analysis. The mean gene expression of NETosis and complement-coagulation pathways was estimated for the data set GSE26440 (n=16). We observed a strong correlation between the two pathways. We also observed a monotonic increase in the expression of genes functional in NETosis across the control, sepsis survivor, and sepsis non-survivor groups. This work is being extended to a comprehensive analysis that tests the association of NETosis with coagulopathy and poor outcome in neonatal sepsis.

In addition to the data analysis, we have performed induction of NET formation in healthy neutrophils using PMA as a trigger. This experimental set-up shall enable us to test any biologically relevant trigger, such as cytokines, in induction of NETosis.

Project 3:

Evolution of Vancomycin Intermediate *Staphylococcus aureus* [Prerona Ghosh]

Staphylococcus aureus is a high-priority pathogen as defined by the WHO. Vancomycin Intermediate *Staphylococcus aureus* (VISA) is believed to be produced by mutations of genes leading to a thicker cell wall and hence antibiotic sequestration, while Vancomycin Resistant *Staphylococcus aureus* (VRSA) is due to the presence of *van* gene clusters and hence target modification. Therefore, it was hypothesized that the conversion of VISA to VRSA should not be possible. We tested this hypothesis in the following manner.

Eight Vancomycin Sensitive *Staphylococcus aureus* (VSSA) isolates (Minimum Inhibitory Concentration (MIC) ≤ 2 µg/ml) lacking van genes were grown in the presence of vancomycin for 60 days. Four isolates (V26, V29, V30, and B11) converted to VISA (MIC = 4-8 µg/ml), and then 1 of these 4 (B11) converted to VRSA (MIC ≥ 16 µg/ml). This suggests that VISA may not only be an intermediate in terms of its phenotypic resistance but also possibly an intermediate stage in the conversion of VSSA to VRSA.

The VRSA obtained had an MIC that was 8-fold higher than that of its sensitive counterpart. Hence, it was interpreted as a heteroresistant isolate. This was followed by growing the 3 VISAs (V26, V29, V30) and 1 VRSA (B11) in the absence of vancomycin for 30 days. On removal of vancomycin pressure, V26 reverted to VSSA while V29 and V30 remained stable. B11 VRSA also spontaneously reverted to VSSA, indicating an inability to detect them by routine diagnostic testing resulting in strains being misclassified as susceptible.

Based on further transcriptomic analysis and candidate gene qRT-PCR, we propose a model connecting cell wall stress to peptidoglycan biosynthesis and hence increased cell wall thickness leading to the VISA phenotype (Figure 3).

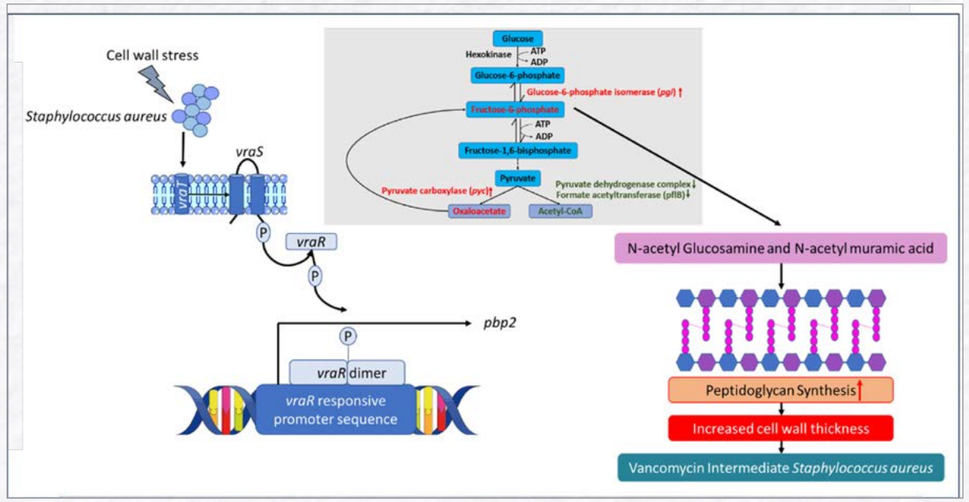
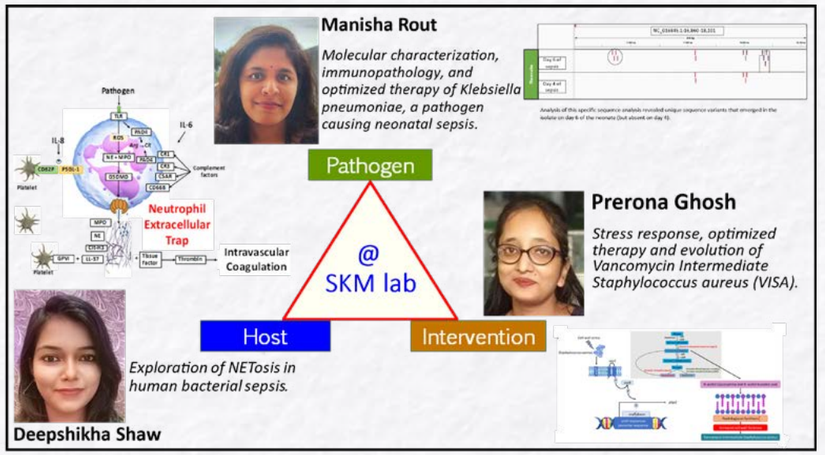


Figure 3: Model showing how cell wall stress can lead to increase in peptidoglycan biosynthesis and hence increased cell wall thickness leading to VISA phenotype. [vraTSR three-component system senses any cell wall stress and upregulates the expression of peptidoglycan biosynthesis genes like pbp2. The increased peptidoglycan synthesis increases the demand for NAG and NAM, which is fulfilled by metabolic reprogramming]

We also observed that the resistance profile of the samples changed after their conversion to VISA or VRSA, with additional resistance to some of the antibiotics to which they were previously sensitive. This indicates that vancomycin treatment for a prolonged period in *S. aureus* can lead not only to reduced susceptibility to vancomycin but also to other antimicrobials.



Group photo of the Lab

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Research Focus

Host-Microbiome Interactions in Human Health and Disease: The aim of our lab is to unravel specific host genomic and metagenomic interactions for sustenance of human health and characterize the perturbations that eventually leads to chronic, complex disorders and development of antibiotic resistance in humans. Human microbiome performs essential functions and are co-evolving with humans by interacting with host immune system thus providing an excellent opportunity for quantifying gene-environment interactions. Hence, we have focused on two major research areas to dissect the role of host-microbiome interactions in: (a) Chronic inflammatory skin disorders and non-healing wounds and (b) Maternal and Child health.

Research Highlights

Project-I: Probiotic Approach to Control Dry Skin: Host Genomic Alterations and Metagenomic Profiles of Skin Microbiome Associated with Atopic Dermatitis (529 words)

Atopic dermatitis (AD) is a chronic autoimmune inflammatory skin condition characterized by an altered composition of the skin microbiome. Previous study from our lab has revealed a significant increase in the abundance of *S. aureus* and its associated microbial pathways, which can degrade the skin barrier protein Filaggrin, resulting in AD lesions (Nath *et al.,* Front Cell Infect Microbiol. 2020). Studies have demonstrated that bacteriophages may hold promise for treatment of AD, particularly due to their ability to specifically target the pathogenic bacteria without affecting beneficial microbes. As antibiotic resistance poses an escalating global challenge, exploring alternative therapies like phage therapy becomes imperative for advancing our treatment options for chronic skin conditions such as AD.

During this period, we developed analysis pipeline for analysing the shotgun metagenomic sequencing data from 23 AD cases (adult and paediatric) and 31 adult controls for the identification of bacteriophages that are significantly enriched in the AD lesions along with their virulence properties, if any.

In brief, the raw FASTQ files were subjected to initial QA/QC and human reads were removed. The filtered paired-end reads were then used for K-mer based contig preparation using IDBA-UD algorithm. These contigs were predicted for potential bacteriophages with the help of VIRSORTER and phage taxonomy classification was done on only those contigs whose length is ≥ 5kb using BLAST by aligning to NCBI Viral RefSeq database. Further toxin genes or virulence factors were identified by PATHOFACT.

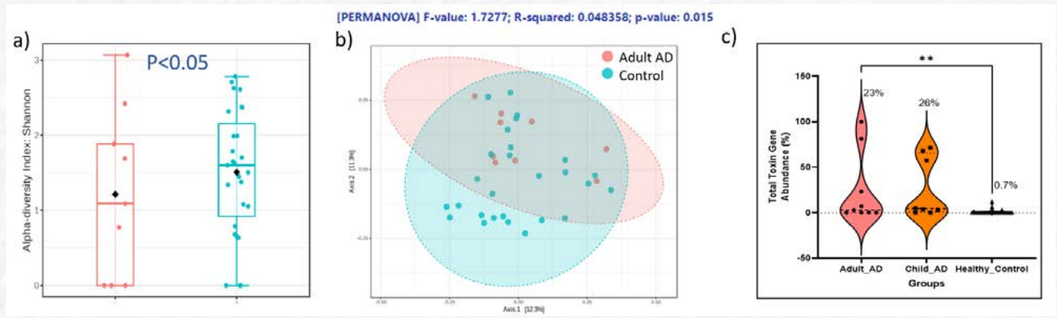

Figure 1: Phage diversity comparison between adult AD patients and healthy controls. a) Shannon diversity is significantly high in controls compared to AD patients b) Principal Coordinate analysis showing significantly higher inter-individual diversity in controls based on Bray-Curtis dissimilarity index compared to AD patients c) Total toxin gene abundance is significantly high in Adult AD patients

In total 462 bacteriophages were obtained and among them 65% were uniquely present in healthy control samples compared to AD patients. This was also supported by significantly higher intra and inter-individual phage diversity among healthy controls compared to adult AD patients (Figure 1).

We have observed temperate phage *Staphylococcus phage StB12* to be significantly enriched in adult AD patients compared to controls (AD: 7.34%, Control: 0.36%). The host bacteria for this phage is *Staphylococcus hominis*. Deghorain *et al.*, J Bacteriol 2012 have shown that although *Staphylococcus phage StB12* is a temperate phage (lysogenic), it carries 7 tail proteins that has human as well as bacterial cell wall lytic activity and in stressed condition this *StB12* can use these tail proteins to enter into lytic cycle. This explains our previous observation of significantly lower abundance of *S. hominis* in AD lesions (AD: 0.20%, Control: 16.43%) which can be mediated by the lytic activity of *StB12* under stressed condition in AD lesions.

We also observed several toxin genes harboured by other *S. aureus* specific temperate (lysogenic) bacteriophages in AD skin lesions such as *Phietavirus_B122* (AD: 6%, Control: 0%), *Staphylococcus phage CNPH82* (AD: 4.57%, Control: 0%), *Staphylococcus phage_37* (AD: 3.03%, Control: 0%), *SA780ruMSSAST101* (AD: 1.22%, Control: 0%). Hence, our observations clearly indicate that *S. aureus* specific lysogenic

phages are the reservoir for toxin genes in AD patients and the secretory toxin proteins expressed by these genes are integrated in *S. aureus* genome that can impair the skin barrier integrity in AD. Currently, the data analysis pipeline for resistome mapping from the shotgun data is being optimized for identification of AMR genes in the AD metagenome.

Project-II: Inter-Institutional Program for Maternal, Neonatal and Infant science (GARBH-Ini- Phase II): Role of Vaginal Microbiome in Pregnancy Outcome with Special Emphasis on Preterm Birth

Preterm birth is the birth of a baby at less than 37 weeks of pregnancy, and is one of the leading causes of neonatal mortality worldwide. India contributes the highest number of preterm birth every year (3.5 M preterm babies are born out of 27 M babies). Multiple factors can be a potent contributor for indicative preterm birth but the reason behind "spontaneous preterm birth" is still under investigation. The literature suggests that "vaginal microbiome" is one of the contributing factors among the complex scenario responsible for preterm birth. Previous studies provided evidence that the vaginal microbiome community structure is not similar for all the ethnicities, as well as prevalence of preterm birth is also not same for all.

For understanding the difference in vaginal microbiome composition between preterm and term pregnancies in Indian women, swabs from high vaginal site were collected from pregnant women in different trimesters of pregnancy for microbiome study. Only mothers having spontaneous childbirth with no twin pregnancy, preeclampsia, maternal diabetes etc. were included in this study. In the previous year, the association of vaginal microbiome with preterm birth was investigated in a discovery cohort of 20 term and 20 preterm samples for all the three trimesters (n=120) (Kumar *et al.,* Front Cell Infect Microbiol. 2021). In this year, the findings were validated in a larger sample size, the validation cohort. Total, 600 biospecimens (DNA samples) from 140 term and 60 preterm mothers were sequenced for 16S study in HiSeq 2500 platform. The FASTQ files obtained after sequencing were analysed by integrating QIIME2 and DADA2 using R based software packages. The quality passed reads were clustered into ASV (Amplicon Sequence Variant) based on 100% sequence similarity. Each ASV was assigned for taxonomy using SILVA and NCBI microbial database.

In the validation cohort, total 25 microbial phyla and 618 genera were identified. A total of 5 phyla and 17 genera were selected as core taxa comprising of >96% of total microbial reads. Core taxa were those which were present in any of the two groups (i.e., TB or PTB) and at any of the three trimesters (V1/V2/ V3): with (a) average relative abundance ≥ 0.1%, and (b) present in ≥ 50% of women in that group. Firmicutes was the most abundant phylum in both the groups (TB: 78%, PTB: 74%) followed by phylum Actinobacteria (TB: 11 %, PTB: 11%). By comparative analysis it was observed that phylum Fusobacteria (TB:1%, PTB: 3%) was significantly higher (p value: 0.001, Wilcox. Test) in PTB compared to TB at 1st trimester of pregnancy.

Out of 17 core genera, *Lactobacillus* was the highest abundant taxa in both TB (74.16%) and PTB (68.83%) groups followed by *Gardnerella* (TB: 5.24%, PTB: 6.85%), *Atopobium* (TB:2.98%, PTB:4.05%) and *Sneathia* (TB:1.17%, PTB:2.94%). In species level, a total of 26 species belonged to the *Lactobacillus group*. By Linear Discriminant Analysis (LDA) we identified the most discriminant taxa in TB and PTB. *Lactobacillus crispatus* was the most discriminating taxa in TB whereas *Lactobacillus iners* was the most discriminant taxa in PTB samples at the 1st trimester. Besides the *Lactobacillus* taxa, some non-Lactobacillus vaginal flora was also found to be significantly higher in PTB samples, such as *Atopobium*

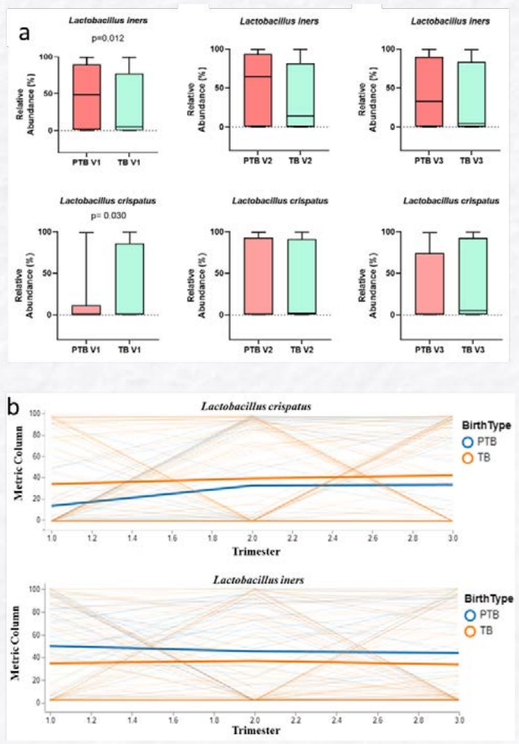

Figure 2: (a) The significantly different taxa between TB and PTB. (b) Longitudinal shift of *Lactobacillus crispatus* and *Lactobacillus iners* during pregnancy

vaginae, *Sneathia sanguinegens*, *Prevotella corporis* and *Pseudomonas sp.*, at the 1st trimester. By correlation study we found that *L.crispatus* is strongly negatively correlated with *L.iners*. *L. crispatus* was also found to be negatively correlated with non-Lactobacillus taxa in both the groups. Linear Mixed Effect (LME) analysis was performed to understand the longitudinal shift of the vaginal taxa from 1st to 3rd trimester of pregnancy (Fig 2). As a result, we could find a distinct signature of high abundance of *L. iners* throughout the pregnancy in PTB delivering women whereas high abundance of *L. crispatus* was maintained in vagina who delivered term birth (manuscript under preparation).

By 16S study we could reveal the community structure of microbiome based on their existence and abundance value but we could not characterize their gene families and pathways. To identify differentially enriched gene families and pathways in the vaginal microbiome of term or preterm mothers, we next performed shotgun whole genome sequencing in a subset of samples. The library preparation was done for shotgun sequencing using Nextera XT DNA library Prep Kit and sequencing was done using NovaSeq 6000 platform with 2x250 base paired end chemistry. The FASTQ files thus generated are now being analysed by the shotgun sequencing data analysis pipeline optimized in our lab during that period.

Publications

Gupta N, Chhibber-Goel J, Gupta Y, **Mukherjee S**, Maitra A, Sharma A, Tandon R. Ocular conjunctival microbiome profiling in dry eye disease: A case control pilot study. Indian J Ophthalmol. 2023 doi: 10.4103/ijo.IJO_1756_22. PMID: 37026304; PMCID: PMC10276740. (in press during the period Apr 2022-Mar2023)

Pratap Singh R, Kumari N, Gupta S, Jaiswal R, Mehrotra D, Singh S, **Mukherjee S**, Kumar R. Intratumoral Microbiota Changes with Tumor Stage and Influences the Immune Signature of Oral Squamous Cell Carcinoma. Microbiol Spectr. 2023 doi: 10.1128/spectrum.04596-22. PMID: 37409975; PMCID: PMC10434029. (accepted during the period Apr 2022-Mar 2023)

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Research Focus

Host genetic factors, and it's role in the determination of susceptibility to various infectious diseases as well as their clinical courses upon infection has now been unequivocally proved. Our research focus is to understand the genetic basis of an individual's susceptibility to tuberculosis and outcome of an infection, with an initial emphasis on genes of the innate immune system. To understand the host genomic modulators of disease, development of drug resistant state of the pathogen in host after prolonged exposure, we have chosen to study genomics, transcriptomics, cytokine, metabolite signature in circulation of TB infected individuals and corresponding household contacts.

Research Highlights

Project A: Genomics-driven Dissection of Susceptibility and Drug Resistance to Pulmonary Tuberculosis, with a Geographical Focus on North East Region

OBJECTIVE: *Exploratory studies on host genomics and functional genomics to dissect tuberculosis susceptibility and host genetic basis of emergence of drug resistance.*

This project is being done in collaboration with Mizoram University and many other institutes in India. This project aims to have a comprehensive picture of genomic analysis of tuberculosis disease in the state of Mizoram exploring both host and the pathogen. To test the hypothesis that host genomic constituents may have role in development of drug resistant within host we have enrolled 27 pair of samples (tuberculosis cases and their household contacts) with all demographic information. Tuberculosis cases visiting hospitals were first enrolled who fulfill all our selection criteria. Presence of infection was tested using sputum culture and CBNAAT. Rifampicin resistance was determined from CBNAAT results. Out of 27, 6 patients harbour rifampicin resistance Mycobacterium. 3 ml blood was collected from cases with informed consent and questionnaire. Contacts with previous history of infection and treatment were not recruited. Same volume of blood was collected from household contacts after 3 months (allow sufficient time for exposure), provided they do not acquire tuberculosis. One part of the blood was used for genomic DNA isolation. The other part was used for plasma isolation which was used for Vitamin D and cytokine assay. Vitamin D assay was done in-house using ELISA. Cytokine assay was done using multiplex assay using Bioplex200 system. Genomic assay was done using Infinium GSA-24+ V3.0 DISEASE chip (Illumina). Preliminary quality control on the data was done using GenomeStudio and subsequent probe and sample level quality control and association testing was done using PLINK.

Cytokine Expression in TB cases and Controls

We aimed to explore differential cytokine expression among drug resistant and sensitive cases. On analysis of multiple cytokines, five cytokines IL6, IP10, IL12p70, IL5, TNFα were significantly high in TB cases compared to controls. Four of them IL6, IP10, IL5, TNFα were higher among rifampicin resistant cases. Representative picture is shown below.

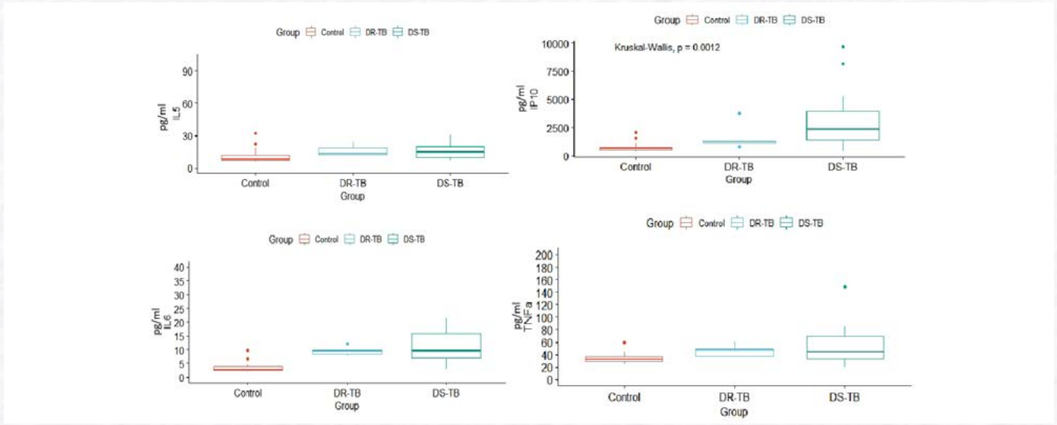

Figure 1:Expression of IL5, IP10, IL6 and TNFα in Rifampicin Resistant TB cases, Drug Sensitive TB Cases and Controls.

We attempted to correlate with one of the metabolite vitamin D which is known for its protective effect in tuberculosis. Drug resistant cases showed lower Vit D level (mean 23.7 ng/ml) compared to drug sensitive (26.3 ng/ml) cases and controls (30.89 ng/ml). However, this difference was not statistically significant.

Host Genomic Analysis of pulmonary TB Cases and Controls

We have performed genome wide genotyping using Infinium Global Screening Array-24 v3.0 with the aim to perform genotype phenotype correlation. Out of 654027 probes, 326511 had a MAF>1% while 300800 probes remained monomorphic. Remaining polymorphic probes were checked for HWE and other quality control parameters using Plink (V 1.9). 75 variants were observed to be deviating from HW equilibrium using the Hardy Weinberg Exact test and were filtered out. Overall genotyping rate for all samples was >99%.

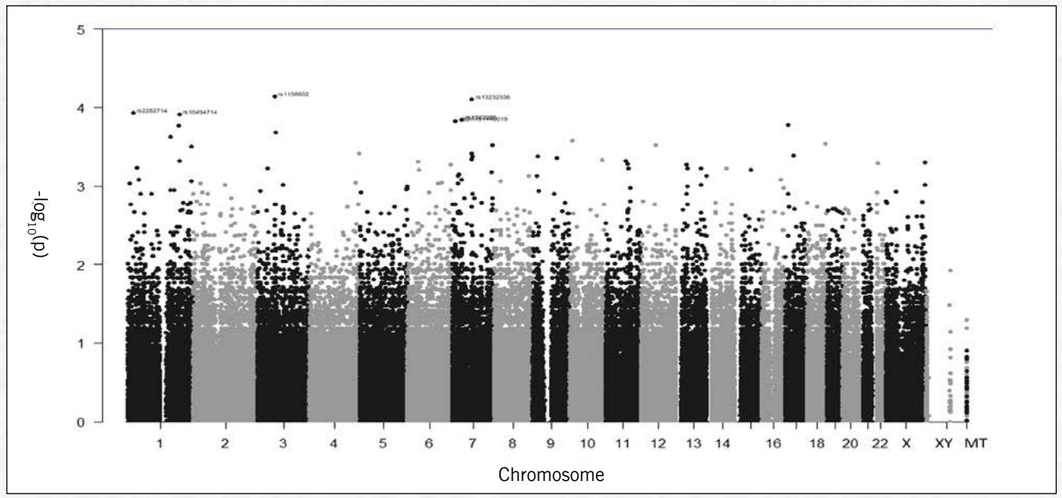

Figure 2: Manhattan Plot of SNPs associated with TB susceptibility

Analysis of the genomic variants identified SNP rs1158602 (p=7.22E-05) on chromosome 3 to be associated with TB susceptibility (Table 1). rs1158602 is an intronic SNP located on the genes LOC107986094 which is an uncharacterized noncoding RNA gene. For the 15 top variants identified to be associated with TB susceptibility, the RegulomeDB rank and scores were assessed. It was found that most, if not all variants were either located within a DNase sensitive site, or had evidence of a transcription factor binding motif (highlighted in red, Table 1).

Table 1: Functional characterization of the top associated SNPs

CHR No.	SNP	P (unadjusted)	OR	Hap IoReg				RegulomeDB Rank
				Variant Type	Gene	Nearest Mapped Genes (5')	Nearest Mapped Genes (3')	
3	rs1158602	7.22E-05	6.235	Intronic	LOC107986094		MAGI1	5
7	rs13232536	7.89E-05	5.269		CLDN4			If
7	rs1440019	0.0001487	5.788			VWDE		3a
17	rs4791961	0.000165	4.886				MYH13	
1	rs2131384	0.0001708	4.929			U6	KCNT2	5

CHR No.	SNP	P (unadjusted)	OR	Hap IoReg				RegulomeDB Rank
				Variant Type	Gene	Nearest Mapped Genes (5')	Nearest Mapped Genes (3')	
10	rs4749774	0.0002653	8.5			RP11-543F8.2	GATA3	6
18	rs11660894	0.0002868	4.738			RP11- 510D19.1, LOC100505776		5
7	rs12538729	0.0002991	5.371			SMARCD3		4
7	rs10274751	0.000381	4.375		CALN1			3a
4	rs12505423	0.0003835	4.957	Intronic	LOC339975		RP1 1-91J3.2	5
7	rs477387	0.0004605	4.156	Intronic	CALN1			3a
1	rs10921667	0.0004738	4.643			AL357932.1	KCNT2	4
1	rs163393	0.0004738	4.643			AL357932.1	KCNT2	5
6	rs302613	0.0004909	4.086			DAAM2		5
X	rs686328	0.0004957	17.73	Intronic	MAMLD1			5

SNPs rs477387 and rs163393 have been shown to be present within DNase-seq peaks in Th-2 cells, an important effector T cell population involved in protective immunity against TB. These data points towards the regulatory potential of these variants.

The genomic neighbourhood for these SNPs was assessed. It was observed that most variants were present in intergenic regions while four variants were intronic (Table 1). Nearby genes towards the 5' and 3' end of each SNP was found from HaploReg. Enrichment analysis of these genes using DAVID was carried out. With an input list of 14 genes thus identified, KEGG pathway for "Tight junctions" was enriched with a FDR corrected p value of 0.0028. Tight junctions are known to be involved in maintaining the barrier function of the lung epithelium. The heteromeric protein complexes of Tight junction seal the interface between adjacent epithelial cells. The damage of TJ is the major cause of epithelial barrier breakdown during lung inflammation. Furthermore, rs13232536 is an eQTL for genes WBSCR27 and ABHD11 while rs1440019 is an eQTL for the gene *VWDE* von Willebrand factor D and EGF domains. This information was queried from GTEx Database. Presently implications of these genes in immune system or resistance against pathogen is not known. Further functional exploration of these genes will be carried out.

Project B: Characterization of variants on IL32 gene and their association with tuberculosis: A functional approach

IL32 is an important candidate gene associated with protection against M.tb infection. Following sequencing of the IL32 gene, two TB associated SNPs rs11860424 and rs9927163 were validated in a cohort of 191 TB cases and their asymptomatic household contacts. In the validation set (191 pairs of cases and controls), rs9927163 was found to show a significant dominance effect in association with tuberculosis disease (OR 1.57, p value=0.03647). Similar dominance effect for this SNP has been previously reported for susceptibility to acute lung injury (ALI). This adds to the effect of this SNP in diseases of the lung and also makes it a suitable candidate for functional validation. The protective G allele of rs9927163 was shown to have significantly greater enhancer activity compared to T allele by luciferase assay in Jurkat E6-1 (p= 0.0059). The SNP rs99271163 is an eQTL of IL32 gene itself. We evaluated the genotype-associated expression of different isoforms of IL32 genes in PBMC from unexposed healthy controls (N=25), stimulated with TB antigens ESAT6 and CFP10. TT (risk) genotype (mean dCt=7.8275, p=0.038), showed higher IL32δ expression compared to GG. We speculate that higher expression of IL32δ which is a less pro-inflammatory isoform, is associated with TB susceptibility.

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Research Focus

Overall research focus of our laboratory is to understand and gain insights for; Why some individuals respond with severe threatening diseases, whereas others remain asymptomatic following stimulus challenge applications. Our lab is keenly interested in unraveling genetic or cellular/molecular mechanisms underlying in unstudied or update regarding mechanisms by which inflammasome activation (NLRP3) and regulate immune cells profiles to decipher mechanistic events of pulmonary disease (COPD). Studies will focus and findings will demonstrate unexplored heterogenic prototype study of Indian population (*East and South cohorts*) to decipher mechanistic events of pulmonary disease (copd)/related pulmonary disorders or chronic disease(diabetic foot ulcers) progression and disease-regulation.

Research Highlights

Project 1:

UNDERSTANDING THE MOLECULAR MECHANISM IN CERAMIDE INDUCED PATHOLOGICAL ACTIVATION OF NEK7 / NLRP3 – INFLAMMASOME

NLRP3, a critical component of the innate immune system that forms the NLRP3 inflammasome, an intracellular molecular platform that drives caspase-1 activation and the secretion of biologically active IL-1beta and IL-18. In addition to its protective role in innate immunity, aberrant activation of the NLRP3 inflammasome contributes to the pathogenesis of several inherited and acquired inflammatory disorders, such as COPD, Diabetes, Atherosclerosis, CAPS, Crohn's disease, Alzheimer's etc. NIMA related kinase 7 (NEK7) is a kinase that is an upstream regulator of NLRP3 inflammasome. More recently few studies report that NIMA-related kinase 7 (NEK7) is necessary for NLRP3 inflammasome activation during potassium efflux. However, the expression of NEK7/NLRP3 inflammasome pathway is unclear. Ceramide is the central molecule of sphingolipid metabolism pathway that acts as pro-apoptotic second messenger. Studies reveal that high levels of ceramide in inflammation is positively correlated with the presence of apoptotic marker in the caspase mediated signaling, leads to pathogenesis of pulmonary or lung complications like COPD, bronchitis, lung inflammation etc.

Ceramide also plays an important role in the cellular processes like apoptosis and cell cycle arrest. Ceramide is also known as an inducer of NLRP3 inflammasome. Although a few recent studies have highlighted the significance of NEK7/NLRP3 inflammasome pathway, ceramide mediated molecular mechanism leading to NLRP3 inflammasome activation remains elusive and are yet to be explored. Hence, we intend to unveil the molecular mechanism involved in Ceramide-NEK7-NLRP3 axis.

Here, we aim to identify molecular mechanisms of ceramide mediated NEK7/NLRP3 inflammasome activation via NF-kB and Calcium-Calmodulin pathway. Overall, we are particularly interested in characterizing the dose dependent ceramide responses mediated in the NEK7/NLRP3 inflammasome activation validating through WT and Mutant in-vitro studies to define and establish the ceramide-anchored sphingolipid metabolism. Also, the influential role in cellular physiology or homeostasis and indicate therapeutic potential to treat NLRP3 inflammasome associated inflammatory disorders.

Study Approach:

- Alveolar epithelial cell line (A549) and Monocyte cell line (WT & KO cell line) will be used for disease model based in-vitro study.
- Cell lines will be treated with a known concentration of Lipopolysaccharide (priming) followed by different concentrations of N-acetyl-D sphingosine (C2 ceramide) and Bleomycin as an activator.
- Post treatments of different time points (0hr-24hrs), gene expression levels and protein levels of NLRP3 inflammasome signalling pathway will be examined.

Study Findings : Our *in-vitro* data using the human monocytes and human lung alveolar cells revealed altered expression of specific genes in the NLRP3 inflammasome pathway during the ceramide induction studies. Additionally, our cellular functional analysis with these inducers for nuclear localization signaling experiments documents nuclear translocation of protein for the NLRP3 inflammasome pathway studies. Furthermore, our preliminary data suggested the association of a binding of transcription factors for calcium mediated NLRP3 inflammasome pathways through GRA/EMSA experimental studies. Thus, it is essential to understand the molecular mechanisms of Ceramide-NEK7-NLRP3 axis and the up- / downstream factors of NLRP3 inflammasome regulation and modulating the disease pathogenesis.

Examining expression of specific genes involved in NLRP3 inflammasome pathway in ceramide treated THP1 cell line by qPCR assay

THP-1 monocytes were washed with PBS and allowed to rest 24 hours prior to stimulation. Cells were treated with ceramide and bleomycin (100 & 150 ug/ml) for 24 hours. After treatment, RNA was extracted, and mRNA levels determined using quantitative real-time RT-PCR were analyzed . To identify the impact of ceramide treatment on the different key gene expression levels of NLRP3 inflammasome signalling were examined by using quantitative real-time RT-PCR. Ceramide treatment by itself significantly induced all the various inflammasome related gene expressions (Fig. 1A. i-iv). (i) NLRP-3 (ii) ASC (iii) IL-1 β (iv) IL-6, normalized with house-keeping gene β-Actin.

Examining Apoptosis Assay in ceramide treated THP1 cell line mediated through NLRP3 inflammasome pathway by MTT assay

The cytotoxicity was measured by the MTT assay. Briefly, THP-1 monocytes cells (1×104 cells/well of 96-well plate) were treated with different ceramide concentrations (25, 50

&100 ug/ml) for 24 hours and MTT dye (100 µg/well) was added to each well for 2 h at 37°C. Thereafter, 0.1 mL of the extraction buffer (20% SDS, 50% dimethylformamide) was added in each well for 12 h at 37°C and the absorbance was red at 570 nm. To identify impact of ceramide treatment on the cell viability was assayed and absorbance was taken. The cell viability was decreased or differentially regulated in ceramide-treated cells or with or without priming with LPS (Fig. 1B. i-ii), indicating the sensitivity of ceramide to human primary cells.

FIGURE 1. Examining expression of specific genes involved in NLRP3 inflammasome pathway in ceramide treated THP1 cell line by qPCR assay and Apoptosis Assay in ceramide treated THP1 cell line by MTT assay

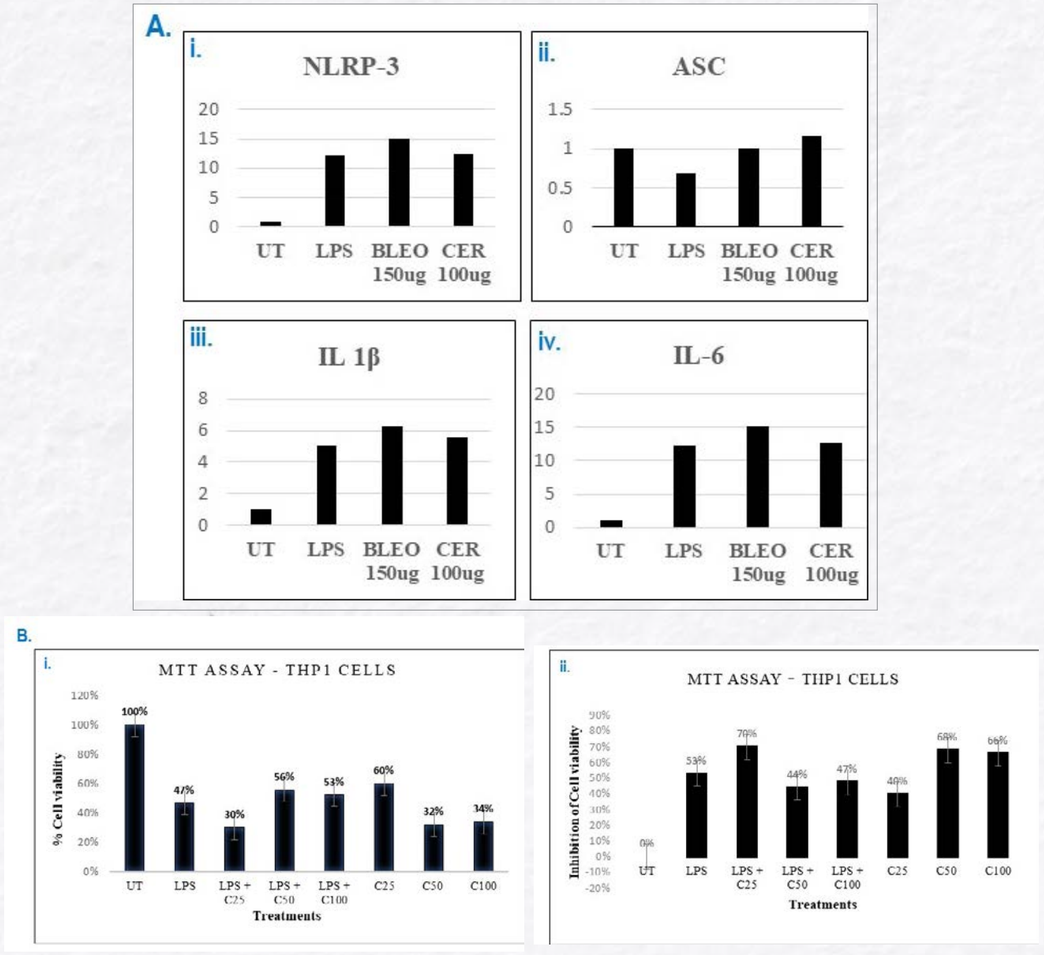

Figure 1: ***A.*** RT-qPCR analysis of the expression levels with the treatments with ceramide and bleomycin of various specific genes that are involved in NLRP3 inflammasome pathway*; (i) NLRP-3 (ii) ASC (iii) IL-1 β (iv) IL-6 in human monocytes.* ***B.*** MTT analysis for cell viability or apoptosis with treatments of ceramide of different concentrations that are involved in the NLRP3 inflammasome pathway was measured in human monocytes as shown in the *(i) & (ii)* The results represented as percentage or inhibition of cell viability.

Examining the Nuclear Translocation Assay of p65 protein in ceramide treated THP1 cell line mediated through NLRP3 inflammasome pathway by Western blot assay

To identify impact of ceramide treatment on nuclear localization signaling (NLS), nuclear translocation assay was examined in p65 protein. Briefly, cells were treated with different ceramide concentrations (25, 50 & 100 ug/ml) with or without priming with LPS for 24 hours, and cytoplasmic and nuclear extracts were prepared. Cell extract protein (50 µg) was analyzed for p65 by Western blot along with GAPDH and Histone 3 as loading controls and detected by chemiluminescence, nuclear translocation was observed as shown; **(Fig. 2A. i-ii).**

Examining the binding of transcriptional factors NF-κB /AP1 / SP1/ NFATC1 to its target in ceramide treated THP1 cell line mediated through NLRP3 inflammasome pathway by EMSA / GRA assay

To identify the impact of ceramide treatment on the NF-κB /AP1 / SP1/ NFATC1 activation was examined in the EMSA experiments. Briefly, cells were treated with different ceramide concentrations (25, 50 & 100 ug/ml) with or without priming with LPS for 24 hours. DNA–protein complex formed was separated from free oligonucleotide on 6.6% native polyacrylamide gels. The specificity of binding was examined by competition with unlabeled oligonucleotide. Visualization of radioactive bands was done in PhosphorImager (Fuji, Japan) using Image Reader software. Ceramide induced high NF-κB /SP1/ NFATC1 DNA binding activity in a dose-dependent manner, as observed shown; **(Fig. 2 B).** indicating upregulation of all the transcriptional factors in treated primary cells.

Impact of Health Relevance: We expect to discover the novel molecular mechanisms of Ceramide-NEK7-NLRP3 axis and the up-/downstream factors of NLRP3 inflammasome regulation in calcium mediated or Mitochondrial/Ros based pathways in modulating the disease pathogenesis. Furthermore, we will validate precise role of distinct metabolities (ceramide/ s1p) acutely engaged in inflammation responses and related diseases, leading to essential and possibly transformative information for development of therapies for NLRP3 inflammasome associated inflammatory disorders.

FIGURE 2. Examining the Nuclear Translocation Assay of p65 protein and in ceramide treated THP1 cell line mediated through NLRP3 inflammasome pathway by Western blot assay and the binding of transcriptional factors NF-κB /AP1 / SP1/ NFATC1 to its target in ceramide treated THP1 cell by EMSA / GRA assay.

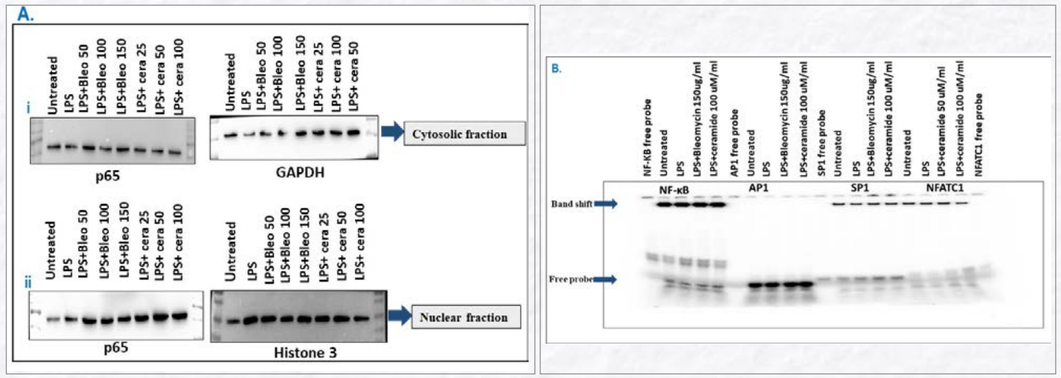

Figure 2: ***A.*** Nuclear Translocation Assay of p65 protein with the treatments with ceramide and with different concentrations (25, 50 & 100 ug/ml) with or without priming with LPS that is involved in the NLRP3 inflammasome pathway*; (i) p65 cytosolic fraction (ii) p65 nuclear fraction in human monocytes.* ***B.*** Binding of transcriptional factors with the treatments with ceramide and with different concentrations (25, 50 & 100 ug/ml) with or without priming with LPS thats involved in the NLRP3 inflammasome pathway; *NF-κB /AP1 / SP1/ NFATC1* assayed from nuclear extracts by gel retardation in human monocytes.

Project 2:

IDENTIFICATION OF GENETIC VARIATIONS AND MICROBIOME DYNAMICS IN BIOLOGICAL SAMPLES (PATIENT VERSUS CONTROL; EAST VERSUS SOUTH) AND ITS ASSOCIATION WITH DISEASES SEVERITY

- Patient sample collection process ongoing.
- Identification of genetic variants by whole exome sequencing is under process.
- Identification of sputum microbiome by 16S rRNA sequencing is under process.
- Proteomics study initiated using patient samples from South and East India.

- To identify genetic variations in patients associated with COPD in the geographical disparity of Southern and Eastern Indian population.
- To understand the impact of microbiome dynamics (causative bacterial strains) on disease severity of COPD disease progression.

Impact of Health Relevance: Our future study plan aims towards to specifically understand the genetic polymorphisms or modifications/genetic predisposition affects and functional role of in pulmonary disorder focused for COPD disorder. Our planned study findings might demonstrate unexplored heterogenic prototype study of Indian Population to decipher mechanistic events of in COPD mediated pulmonary disease progression.

Publications

Puvula J, Maddu N, Gutam N, Parimal A, Raghavendra PB. The role of pyrethroid derivatives in autophagy and apoptosis crosstalk signaling and potential risk for malignancies. Oncotarget. 2022 Dec 17;13:1323-1340. doi: 10.18632/oncotarget.28328. PMID: 36528879; PMCID: PMC9760267..

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Research Focus

Oral cancer is a huge public health burden in India. Despite improvement in the standard treatment, the 5-years survival rate has remained around 30-50% since decades for oral cancer. Intratumoral-heterogeneity has been correlated with aggressive cancer behaviour, drug tolerance and overall poor prognosis; however, responsible cellular and molecular determinants have garnered attention, recently. Our goal is to comprehend the mechanisms responsible for intratumoral-heterogeneity in oral cancer. *Specifically, we are focused on emphasizing the non-genetic heterogeneity among stem-like cancer cells and its interactions with heterogeneous tumor microenvironment as possible determinants and targets against aggressive behaviour of oral cancer.*

Research Highlights

The reversible phenotype switching of cancer cells is being reported in a variety of cancer. These cells co-exist and co-operate with each other within the tumor and facilitate tumor evolution and progression. Such observed plasticity in cancer cells may act as one of the prerequisite mechanisms facilitating adaptation to the changing environment, including anticancer treatment. Therefore, *at any given time, we anticipate to observe non-genetic heterogeneity among genetically identical clones.* Adding to the complexity, it is well known that tumors reside in a complex ecosystem comprising the tumor microenvironment (TME). Dynamic interactions among heterogeneous subtypes of cells in TME is found to be responsible for maintaining the ecosystem in solid tumor. *The collective behaviour of these group of cells determines overall tumor behaviour impacting the aggressive cancer behaviour, drug tolerance and overall poor prognosis.*

Major cellular components of TME are cancer associated fibroblasts (CAFs), which provides a fertile soil for successive cancer development and evolves along with cancer progression. During the early stage of cancer progression, fibroblasts differentiate into more active myofibroblastic state. Myofibroblasts are spindle shaped cells characterized by stress-fiber of alpha smooth muscle actin (αSMA) with contractile nature. SERPINE-1 and αSMA expression of myofibroblastic-CAF at the invading front of oral cancer is associated with extracapsular spread in cervical lymph nodes and predicted a poor disease-free survival of patients. Earlier, we have reported the inter-CAF heterogeneity among different oral cancer patients. Further, we have suggested the differential role of CAFs in maintaining stemness in gingivobuccal oral cancer and provided evidences of CAF-heterogeneity with both tumor promoting and restraining role in oral cancer depending on the extent of myofibroblastic differentiation of CAFs. Based on differential gene expression including *Tie2* and αSMA expression, we categorized CAFs into two subtypes, non-myofibroblastic or C1-type CAF and myofibroblastic or C2-type CAF. Since *Tie2 expression had not been discussed with respect to the fibroblasts functions, previously; we set to investigate the role of Tie2 in TGFb induced CAFs.* Here we have discussed our results, showing the effect of Tie2 activated CAFs over the cancer cells, using the conditioned media (CM). Results suggested that Tie2 play a key role in maintaining the aggressive CAF phenotype (data not shown). Interestingly, oral cancer cells exposed to CAF^{TGFb} CM showed significantly increased sphere forming ability

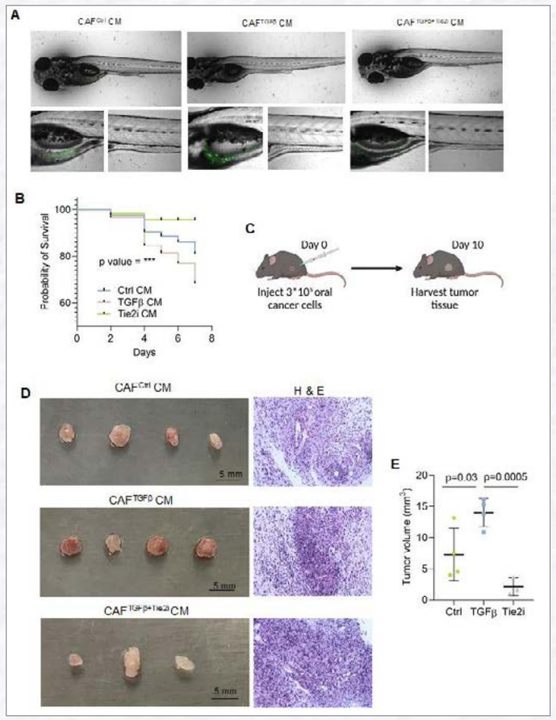

Figure 1: Reversal of pro-tumorigenic ability of CAFs after Tie2 inhibition: SCC070-pLenti-GFP cells were exposed to untreated, TGFb-treated and TGFb-treated-Tie2-inhibited conditioned media (CM). Cells were harvested, and 100 cells were injected into each zebrafish embryo (48 hrs post fertilization). (A) Representative images of zebrafish embryos after five days of cell inoculation. Green signal of GFP-positive cells suggest the oral cancer xenografts formation. (B) Survival of zebrafish embryos post inoculation was recorded and probability of survival for each condition was calculated using GraphPad prism and plotted. (C) Schematic showing the subcutaneous transplantation of C57BL/6 mice with syngeneic oral cancer cell ($3x10^5$ cells) exposed to various conditions media. (D) ten days post-transplantation, mice were sacrificed, and tumors were harvested. Representative image of H&E-stained tumor samples showing the histopathology of developed tumors. (E) Volume of the tumors were measured in ImageJ and plotted in GraphPad prism.

compared to CAF^{Ctrl} CM, which was significantly reduced with CM derived from CAF^{TGFb} cells exposed to Tie2 inhibitor ($CAF^{TGFb+Tie2i}$). Similar results were observed for all the three tested oral cancer cell lines exposed to various CM obtained from two different CAFs. Since, the sphere forming ability of cancer cells is associated with stemness property, we next confirmed the stemness related genes, *NANOG, OCT4* and *ALDH1A1* in cancer cells exposed to CAF^{TGFb} and $CAF^{TGFb+Tie2i}$ CMs. As anticipated, expression of tested set of genes were downregulated in cancer cells exposed to Tie2 inhibited CAF^{TRT} CM (Data not shown). Inhibition of Tie2 in TGFb induced CAF reverted not only the CAF phenotype, but also it inhibited the stem cell like property of cancer cells.

We next explored the tumorigenic ability of oral cancer cells exposed to these various CMs. EGFP expressing SCC070 oral cancer cells were exposed to CAF CM (CAF^{Ctrl}, CAF^{TGFb} or $CAF^{TGFb+Tie2i}$) for 48 hours. Cells were harvested and 100 cells were injected in every embryo (2dpf). Minimum 20 embryos were injected for each condition and monitored for seven days for recording survival. On 4th day post-inoculation, confocal images were taken to visualize GFP-positive cancer cell at the site of inoculation, as indicator of tumor initiation. Cancer cells exposed to CM of CAF^{TGFb} showed higher tumor initiation ability compared to CAF^{Ctrl} CM exposed cells (Figure 1A). Interestingly, no such GFP signal were observed suggesting the reduced tumor initiating ability of cancer cells upon induction by CM of $CAF^{TGFb+Tie2i}$. Correspondingly, the survival probability of zebrafish embryos was maximum for this condition as compared to other treatment conditions (Figure 1B).cNext, we injected mouse oral cancer cells (MOC2) subcutaneously into wild type C57BL/6 mice. MOC2 cells were exposed to the CM derived from CAF^{Ctrl}, CAF^{TGFb} and $CAF^{TGFb+Tie2i}$ conditions. Cells were harvested after 48hrs and $3x10^5$ cells were subcutaneously injected into the right flank of mice (Figure 1C). Parallelly, same cells were seeded for sphere formation. After 5 days of inoculation, palpable tumors were observed in mice for both CAF^{Ctrl} and CAF^{TRT} conditions but not for $CAF^{TGFb+Tie2i}$ condition (Figure 1D). Also, sphere formation assay showed the similar pattern (Data not shown). Tumors were harvested after 10 days of inoculation and tumor volume was measured (Figure 1D). MOC2 cells exposed to CAF^{TGFb} CM resulted in larger tumors volume compared to CAF^{Ctrl} CM (Figure 1D, E), however, the tumors generated from $CAF^{TGFb+Tie2i}$ CM exposed cells showed significantly reduced tumor volume (Figure 1E).

In summary, TGFb-rich oral tumor microenvironment with higher abundance of activated CAFs support stemness in cancer cells and provide aggressive behaviour. Tie2 inhibition in activated CAFs may result in reversal of its activated phenotype resulting in the loss of tumorigenic ability of oral-SLCCs.

Publications:

Ghosh S, Mitra P, Saha U, Vipparthi K, Singh S. Characterizing diversity among human oral stem-like cancer cells using flow cytometry. STAR Protoc. 2022 Dec 16;3(4):101763. doi: 10.1016/j.xpro.2022.101763. Epub 2022 Oct 12.

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Roy S, Mitra P, Acharya S, Roy S, Ghosh S, Maji M, Modak N, Ghosh N, Acharya M, Singh S, Mukherjee A. A photoactive lysosome targeting RuII complex downregulates stemness genes in oral squamous cell carcinoma. Inorg. Chem. Front.. 2022; 9(22):5840-5852. doi: 10.1039/D2QI01079H.

Group photo of the Lab

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Research Focus

Sex-difference in human is extremely prevalent and are especially common in case of complex disease traits. I am investigating the underlying multivariate genetic architecture of sex-differences in genetically correlated complex traits and diseases. Currently, I am exploring sex-difference in anthropometric, fat depositional and sex-hormonal traits using GWAS summary statistics from UK Biobank.

Research Highlights

I investigated sex-differences in genetic architecture of humans for three trait categories, anthropometric, fat depositional and sex-hormonal. The anthropometric traits included height, weight, BMI, waist circumference (WC), hip circumference (HIP), and waist-to-hip ratio (WHR). Arm fat ratio (AFR), leg fat ratio (LFR) and trunk fat ratio (TFR) are the fat depositional traits. The sex-hormone traits were total testosterone (TESTO), calculated bio-available testosterone (CBAT) and sex-hormone binding globulin (SHBG). All the summary statistics were from sex stratified GWA studies based on UK Biobank dataset, and in total there were 12 male and 12 female traits across all categories. I explored the sex-differences in the multivariate genetic architecture at 3 levels:

1) Estimating genetic correlations:

I found sex-difference both across traits and across sex genetic correlations of testosterone, CBAT, AFR, LFR and TFR (Figure 1). Variants which increase testosterone decrease anthropometric traits in males, but contrastingly increase anthropometric traits in females. Hence, men who have high testosterone have low BMI, but high testosterone women have high BMI. I have also found that the females who have high BMI also have high arm fat but low leg fat, but high BMI males also have high leg fat. LFR, AFR and TFR are highly dimorphic next to testosterone and CBAT, which are the most sexually dimorphic traits.

Cross-sex-cross-trait genetic correlations also show extensive sex-differences. I have observed that variants which increases testosterone in females also increase BMI in males, and variants increasing testosterone in males decreases BMI in females. This is sexually antagonistic pleiotropy between testosterone and the anthropometric traits. We estimated an overwhelming number of negative genetic correlations, which indicates sexual antagonism in humans. Most genetic correlations reported in non-human species are found to be positive, and this opposing direction of correlations in

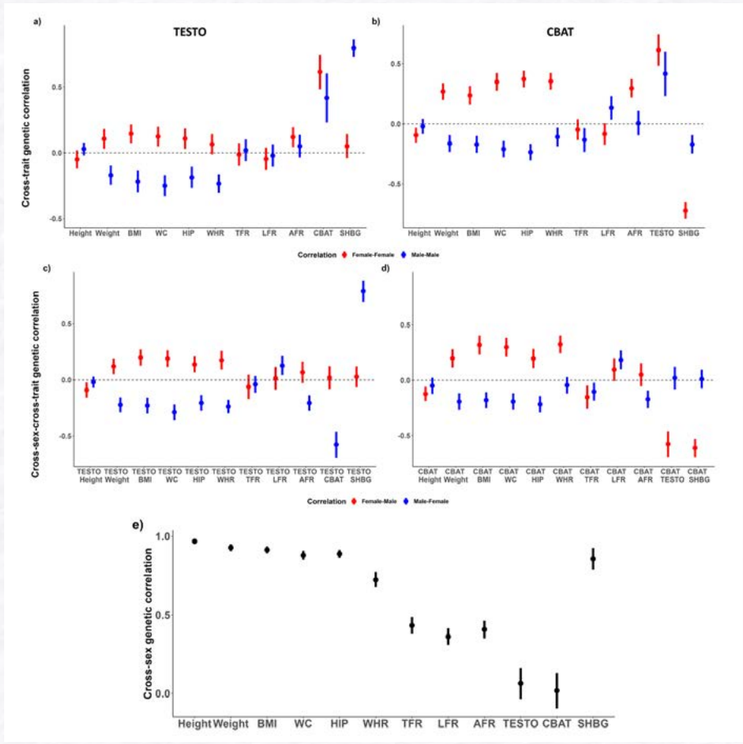

Figure 1: Genetic correlations of all 12 traits with testosterone and CBAT. a) and b) cross-trait genetic correlations of testosterone and CBAT with other traits in males and females. c) and d) cross-sex-cross-trait genetic correlations of testosterone and CBAT. e) Cross-sex genetic correlations of all 12 traits.

humans are probably the signatures of sex-specific selection which led to the sex-difference that we observe in contemporary humans.

2) Estimating polygenic overlap:

I estimated the number of variants affecting each trait independently as well as the number of variants which are influencing combinations of traits (Figure 2), by implementing bivariate causal mixture models. Among the 24 traits, we found that the most polygenic with the largest number of causal variants is male WHR (11.2K, s.d. 1.2), whereas the least polygenic is male SHBG (0.40K, s.d. 0.07). These causal

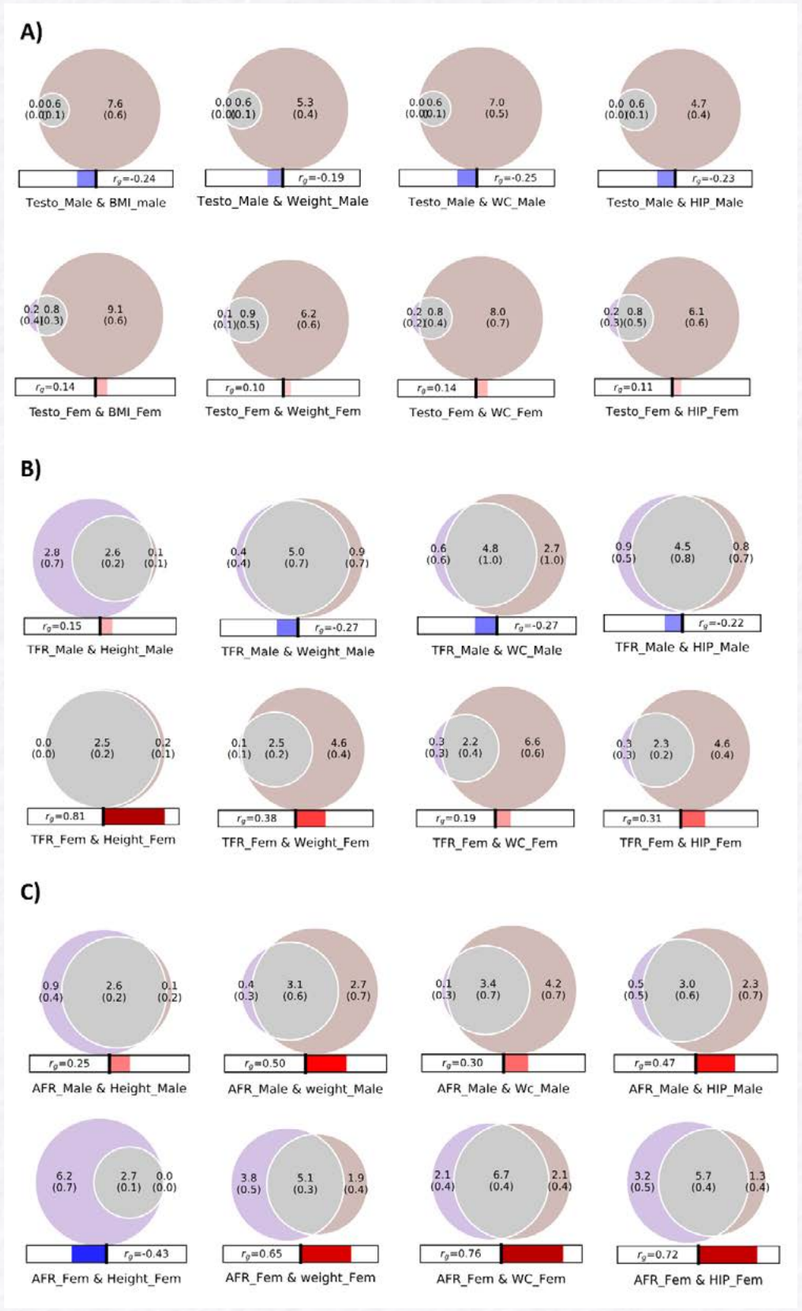

Figure 2: Venn diagram showing polygenic overlap between testosterone, TFR, AFR and other anthropometric traits (height, weight, WC and HIP) in males and females. The number of variants is shown in thousands (with standard deviation). Mauve circles depict unique variants for trait 1, and brown circle depicts that of trait 2. Shared variants between two traits are shown in grey. r_g depicts the genetic correlation between traits. Shades of red denote positive genetic correlations whereas shades of blue denote negative genetic correlations. Panels represent shared variants between anthropometric traits and A) testosterone, B) TFR, C) AFR.

variants explain 90% of SNP heritability of these specific traits. In the overlap between testosterone, and weight, BMI, WC and HIP in males, all the variants which affect TESTO also influenced all these traits (Figure 3A). But in females, TESTO has independent causal variants besides having overlapping variants with weight, BMI, WC and HIP. Moreover, the fraction of concordant variants, i.e., variants which have the same sign, between TESTO and these four traits in males are between 10%-26%, but much higher, between 60%-74%, in females.

Polygenic overlap is also estimated between the fat ratios and the anthropometric traits, which show sex-specific patterns (Figure 2B and 2C). TFR and LFR have more independent variants in male, and the size of overlap is also greater in males. The smallest overlap is between TFR and weight in females (2.5K, s.d 0.2) with only 100 independent variants for TFR female. In contrast, the shared loci between TFR and weight in males is twice that of females (5.0K, s.d. 0.7) with 400 independent variants for TFR male. All the variants causal to LFR are also causal to weight, WC and HIP in females, but not in males. On the other hand, in the overlaps between AFR and the anthropometric traits, the number of independent causal variants of AFR are much higher in females compared to males. The highest overlap is between AFR and WC in females (6.7K, s.d. 0.4) with 2.1K (s.d. 0.4) independent variants for AFR. Genetic overlap between AFR and WC in males is only for 3.4K (s.d. 0.7) variants with 100 independent variants for AFR. We have also found that the sexes are most different for AFR sharing only 36% of variants in contrast to LFR for which sexes shared 50% of the variants. The overall difference in overlap between AFR, and TFR and LFR indicates that AFR shares more loci with traits in females, and hence is a more female specific fat depot compared to TFR and LFR which are male biased fat depositional phenotypes.

3) Shared SNPs and pathway enrichment:

I identified shared SNPs between two traits based on a P-value cut off for three SNP classes – a) SNPs significant for both the traits, b) significant for trait1, c) significant for trait2. Figure 4 shows the genome-wide distribution of the overlapping SNPs belonging to the three SNP classes for AFR and BMI in males and females. The plots demonstrate that even at the level of shared significant SNPs between traits, there is genome-wide difference between males and females for every SNP classes.

Variants that were significant for both traits were further annotated, mapped, and checked for enrichment in gene-sets and pathways. I have mostly investigated the enrichment for the shared variants among adiposity and fat distribution phenotypes, and in the process, I have singled out three major pathways that are involved in fat deposition. 1) **The 16p11.2 deletion pathway**, containing SH2B1 among other genes. SH2B1 has been related to early onset obesity and insulin resistance in humans, and deletion of sh2b1 gene in mice increases food intake, obesity, and insulin resistance. 2) **Arylamine metabolism pathway** – SULT1A1 gene is the major player of this pathway, and is upregulated in differentiating adipocytes. 3) **Zinc homeostasis pathway**, containing the metallothionein class of genes, which are involved in both zinc and copper homeostasis. Metallothioneins have been shown to have a protective role against obesity in female mice but not in males.

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Research Focus

In the central nervous system, astrocytes and oligodendrocytes couple through gap junctions (GJs) over distant regions forming a "panglial syncytium", which is vital for maintaining neuronal homeostasis. Recent studies suggest that glial GJs comprising of proteins called connexins are impaired in several neurodegenerative conditions. However, molecular mechanisms that deregulate connexins in complex neurodegenerative conditions are not well understood. Further, how astroglial GJ alterations affect oligodendrocyte function through heterotypic GJ coupling remains an open question. Our goal is to understand the molecular mechanisms by which glial GJs are deregulated and how that contributes to development/progression of chronic demyelinating and neurodegenerative pathologies.

Research Highlights

Investigating the role of oligodendroglial gap junction protein Connexin 47 in axon-myelin interaction during virus-induced demyelination of the central nervous system

In different demyelinating disorders of the central nervous system (CNS) including Multiple Sclerosis (MS), it has been reported that the loss of myelin is associated with the loss of gap junction (GJ) proteins in the myelinating glial cells, the oligodendrocytes. The oligodendrocytes mainly form GJs with the astrocytes forming a "panglial syncytium", which performs a central role in maintaining ionic buffering, small molecule exchange and nutrient homeostasis that are crucial for maintaining CNS myelination and neuronal activity. However, their contribution to the development and progression of a chronic demyelinating pathology is not well understood. Oligodendrocytes mainly express GJ proteins, connexin 47 (Cx47), Cx32 and Cx29, whereas the astrocytes express Cx43, Cx30 and Cx26. In particular, Cx43/Cx47 heterotypic channels are exclusively important for astrocyte/oligodendrocyte (A/O) crosstalk in the CNS and has been suggested to be down regulated in demyelinating pathologies. However, it remains elusive whether the loss of oligodendrocyte GJs is a cause or a consequence of the chronic demyelinating pathology. Thus, the overall goal of the proposed project is to understand the involvement of altered oligodendroglial Cx47 GJ communication in chronic expansion of the demyelinating lesions in a mouse model of virus-induced demyelination. Earlier studies have established that intracerebral inoculation of a neurotropic strain of mouse hepatitis virus (MHV-A59) in 4-week-old wildtype C57BL/6 mice serves as a model of neuroinflammatory demyelination which mimics the pathological symptoms of the human neurological disease, MS. Further it has been shown that there is persistent downregulation of Cx47 levels in association with demyelination. We have done an in depth spatio-temporal analysis of the changes in Cx47 and its heterotypic coupling partner Cx43 expression in different spinal cord regions following infection. Our results highlighted an altered expression profile of the Cx47 GJs in different oligodendrocyte lineage cells affecting the Cx43-Cx47 astrocyte-oligodendrocyte axis leading to a demyelinating pathology. Using different cellular, biochemical and omics approaches we are aiming to understand the underlying pathogenic mechanisms causing impairment of Cx47 GJs as an important mechanism of chronic expansion of demyelinating lesions in virus-induced neuroinflammatory demyelination.

Understanding the role of the predominant astroglial gap junction protein Connexin 43 in amyloid-β related neurodegeneration

Reactive astrogliosis is a prominent feature of all Alzheimer's disease (AD) brains. In physiological conditions, astrocytes are connected over distant regions in the brain through gap junctions which are made up of proteins called connexins (Cx). Connexin 43 (Cx43) is a major astroglial gap junction (GJ) protein that mediates astrocyte-astrocyte GJ coupling and astrocyte-oligodendrocyte GJ coupling through heterotypic coupling *via* Cx47 in oligodendrocytes. Proper functioning of the Cx43 GJs are of paramount importance for the maintenance of the panglial network in CNS and maintaining brain homeostasis. Studies in postmortem AD brain tissue suggest increased Cx43 immunoreactivity in the reactive astrocytes surrounding the amyloid-β (Aβ) plaques, a key pathological hallmark of AD brains. Earlier studies in animal models and rodent cell culture models have shown that Aβ impairs gap junction intercellular communication between astrocytes and triggers overactivation of uncoupled Cx43 hemichannels (HCs) releasing excitotoxic ATP and glutamate eventually leading to memory impairment in AD. Based on these earlier studies, Cx43 has been suggested to be a potential therapeutic target for developing future treatment strategies for AD. Given that Cx43 forms both HCs and GJs which are differentially regulated in health and disease, we need to keep in mind that consequences of Cx43 modulation can be manifold. Therefore, an in depth understanding of the underlying molecular mechanisms by which Cx43 channels are altered in AD warrants further investigation. Towards that understanding we have used primary human astrocyte cultures and treated them with human Aβ peptide for different durations. Our results suggested that exposure to Aβ alters intracellular trafficking of Cx43 and functional gap junction communication in a time-dependent manner. The overall goal of the proposed study is first to decipher the mechanistic underpinnings of Aβ-induced deregulation of astroglial Cx43 channel functions. Further, we intend to understand how the altered astroglial Cx43 function may affect the astroglial Cx43-oligodendroglial Cx47 gap junction coupling and contribute to the development and progression of white matter pathology in AD brain tissues.

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Research Focus

How does epigenetic modification regulate disease progression is primary focus of my lab. Enhancers serve as critical regulators for transcriptional programs by directing development, homeostasis and disease states. A reliable epigenetic feature of active enhancers is the production of enhancer-directed transcripts (eRNAs). Here we will identify the differentially expressed eRNAs in chronic inflammatory condition and investigate their role in pancreatic cancer progression.

Immune response is often heterogenous and varies between individuals. We will investigate the role of differential epigenetic modifications in disease severity during viral infection. We will also explore any novel function of eRNAs in anti-viral immune response.

Research Highlights

A. To investigate the role of enhancer RNAs (eRNAs) and epigenomic modification in Pancreatic Cancer

Pancreatic cancer is one of the major aggressive and devastating cancers with the lowest five-year survival rate (<10%) among common cancers. It starts with chronic inflammation due to precancerous lesions; however, the malignant form, known as pancreatic ductal adenocarcinoma (PDAC) is most prevalent among the patients (>90%). As this cancer can't be distinguished from typical abdominal pain at the early stage, it is often detected after metastasis or malignancy, which makes it difficult to cure the patient leading to low survivability. Genomic profiling of PDAC has determined that the *MYC* gene is one of the most commonly amplified in PDAC patients. In cancer cells, *MYC* gene dysregulation is achieved due to the activation of tumor-specific super-enhancers. So, it is postulated that the enhancer-derived enhancer RNAs (eRNAs) might play a significant role in several gene expressions thus regulating important cellular functions.

Higher expression of MYC eRNAs as well as MYC mRNA were observed in chronic inflammatory condition

We have analysed the already published ChIP-seq data set for active enhancer marks (H3K27ac and H3K4me1) upstream of *MYC* gene and aligned with the GRO-seq peak for the same region to identify putative eRNAs. We have identified three *MYC* eRNAs synthesized from -490 kb, -425 kb and -510 kb upstream of *MYC* promoter. We have validated the synthesis of those eRNAs in MIA PaCa-2 cell, a pancreatic adenocarcinoma cell line. To mimic the chronic inflammatory condition, we treated the cells with TNF-α for 24 hours and measured *MYC* eRNA level by RT-PCR. We have observed significant increased expression of *MYC*-490 kb and *MYC*-425 kb eRNAs as well as *MYC* mRNA in chronic inflammatory condition (**Figure 1A**).

Next, we did pulse-chase experiment by using Click-iT chemistry to capture nascent RNA in the nuclear fraction and observed higher expression of *MYC*

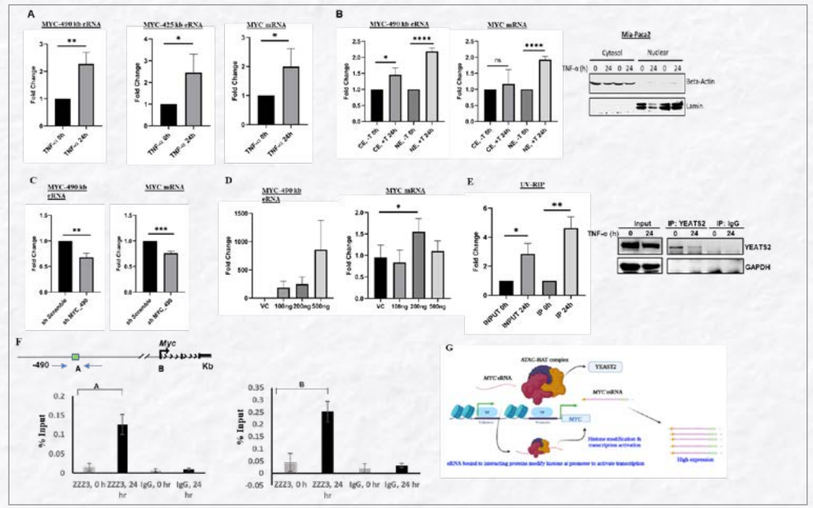


Figure 1: Identification of *MYC* eRNAs and their role in *MYC* gene expression. (**A**) *MYC* eRNA and *MYC* mRNA expression was validated by RT-PCR from TNF-α stimulated MIA PaCa2- cells. (**B**) Nascent RNAs were pulse-labelled and then chased for 1 hour to capture eRNA expression from nuclear fraction. Nascent *MYC* mRNAs were also checked in the same condition (middle panel). Western blot for Beta-actin and Lamin, showing clear fractionation of MIA PaCa-2 cells (right panel). (**C**) *MYC*-490 kb eRNA level was decreased by shRNA against the eRNA. *MYC* mRNA level was decreased due to shRNA-mediated knock-down of *MYC*-490 kb eRNA. (**D**) *MYC*-490 kb eRNA was over-expressed in MIA PaCa-2 cells in different stoichiometric ratio for 48 hours. At a particular stoichiometric ration, *MYC*-490 kb eRNA increased the MYC gene expression. (**E**) UV-RIP followed by RT-PCR showed higher association of *MYC*-490 kb eRNA to YEATS2 protein in TNF-α stimulated MIA PaCA-2 cells. (**F**) Schematic of *MYC* enhancer and promoter region (upper panel). ChIP-qPCR was performed using ZZZ3 antibody to check binding of YEATS2-containing ATAC complex to *MYC* enhancer as well as *MYC* promoter region. (**G**) The overall summary of this study has been depicted as graphical abstract.

eRNAs as well as *MYC* mRNA with chronic inflammation (**Figure 1B**). Interestingly, we have also observed higher level of *MYC* eRNAs as well as *MYC* mRNA in chronic pancreatitis (CP) and Pancreatic Cancer (PDAC) patient samples compared to normal tissue samples, thus strengthening our hypothesis.

MYC eRNA regulates MYC gene expression in pancreatic cancer cells.

Next, we checked whether *MYC*-490 kb eRNA can regulate *MYC* gene expression in MIA PaCa2 cells. When we knocked-down *MYC*-490 kb eRNA by shRNA designed against the eRNA, we found around 30% decrease in *MYC* gene expression (**Figure 1C**).

To confirm the regulatory role of *MYC* eRNA on *MYC* gene expression, we have cloned *MYC*-490 kb eRNA in mammalian expression vector and over-expressed *MYC*-490 kb eRNA by transient transfection in MIA PaCa-2 cells. We have observed significant increase in *MYC* gene expression at a particular stoichiometric ratio of *MYC*-490 kb eRNA in MIA PaCa-2 cells (**Figure 1D**).

MYC-490 kb eRNA interacts with YEATS2 protein

To determine how does *MYC*-490 kb eRNA regulate *MYC* gene expression, we investigated the *MYC*-490 kb eRNA interacting protein partners by RAP-MS. We have found that YEATS2, a histone reader protein, was one of the *MYC*-490 kb eRNA proteins. We confirmed the interaction of *MYC*-490 kb eRNA and YEATS2 by UV-RIP from TNF-α stimulated MIA PaCa-2 cells (**Figure 1E**). Further, we observed higher interaction of *MYC*-490 kb eRNA with YEATS2 protein in TNF-α stimulated cells. So, the differential binding of *MYC* eRNA and YEATS2 between pancreatic cancer indicated cancer-specific enhancer landscape and gene regulation.

Binding of MYC eRNA to YEATS domain Augments YEATS2 interaction to Crotonylated Histone

YEATS domain-containing 2 (YEATS2) is a scaffolding subunit of the Ada-two-A-containing (ATAC) complex, a conserved metazoan HAT complex. YEATS2 binds to acetylated histone H3 via its YEATS domain.

So, we first checked the binding of YEATS2-containing ATAC complex to *MYC* enhancer and promoter region by ChIP-qPCR. We have observed higher binding of ATAC complex in *MYC* enhancer region as well as in *MYC* promoter in chronic inflammatory condition in MIA PaCa-2 cells (**Figure 1F**). Moreover, it has also been reported that YEATS2 has higher affinity towards crotonylated histone compared to acetylated histone. Here, we have observed that with TNF-α stimulation, the crotonylation level didn't change significantly in *MYC* enhancer or promoter region. But, we have observed *MYC*-490 kb eRNA augmented the association of YEATS2 to crotonylated histones. So, these data prove that in chronic inflammatory condition, *MYC* eRNAs are induced from *MYC* super-enhancer regions that in turn augment the binding of YEATS2-containing ATAC complex to crotonylated Histones to specifically increase *MYC* oncogene leading to pancreatic cancer (**Figure 1G**). It would be interesting to check the effect of eRNAs on corresponding gene expression genome-wide to further strengthen our hypothesis.

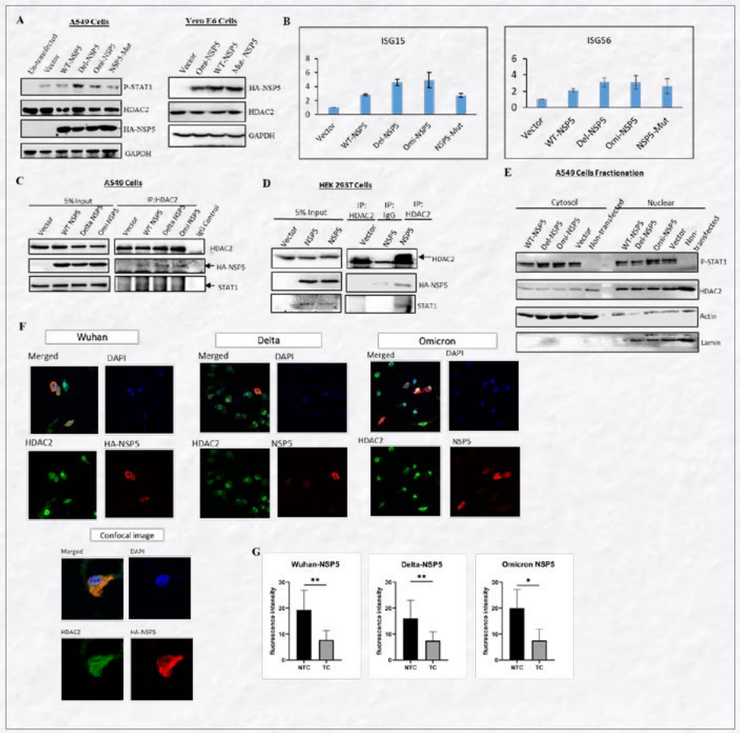


Figure 2: (**A**) Western Blot showing overexpression of HA-NSP5 of Wuhan, Delta and Omicron variants of SARS-CoV-2 virus in A549 cells as well as in Vero E6 cells. (**B**) Real-time PCR data showing upregulation of ISG15 and ISG56 genes for Wuhan, Delta or Omicron NSP5 overexpression in A549 cells. Co-immunoprecipitation followed by western blotting was performed to check association of HA-NSP5 with HDAC2 protein in A549 cells (**C**) as well as in HEK293 cells (**D**). Cellular fractionation was performed and visualized by western blotting to check the localization of HDAC2 and P-STAT1 in A549 cells (**E**). (**F**) Co-immunofluorescence microscopy picture showing colocalization of HA-NSP5 and HDAC2 in A549 cells. Confocal image was taken for a single cell to visualize the colocalization prominently. (**G**) Fluorescence intensity of nuclear regions of NSP5 transfected and un-transfected cells were measured for green channel and average intensities were plotted as bar graph.

B. Deciphering the role of epigenetic modification in anti-viral immune response.

Regulation of enhancer activity by histone acetylation/deacetylation mediated by viral proteins

As evident from previous literature, the acetylation/deacetylation cycle of histone regulates the enhancer activity, hence active transcription of the associated gene. Recent pandemic caused by SARS-CoV-2 virus also had differential effects on ISG expression in infected cells. So, we looked for whether the SARS-CoV-2 virus had any effect on histone acetylation/deacetylation.

It has been reported that NSP5 protein of SARS-CoV-2 could interact with HDAC2 protein of human host cells (Gordon *et. al.*, Nature 2020). HDAC2 is a deacetylase that can remove the acetyl group from different histone tails and thus regulates gene activation. So, we investigated the role of SARS-CoV-2 NSP5 protein in ISG expression. We have sub-cloned NSP5 gene from Wuhan, Delta and Omicron variants of SARS-CoV-2 virus into HA-tagged mammalian expression plasmid. Overexpression of NSP5 proteins were checked by Western Blot in A549 and Vero E6 cells (**Figure 2A**). We have also seen that NSP5 overexpression increased the STAT1 phosphorylation which is a mark for STAT1-mediated anti-viral immune response activation. It is already known that STAT1 activation may lead to ISG expression. So, we checked ISG15 and ISG56 expression by RT-PCR (**Figure 2B**) and found that those gene expressions were correlated with STAT1 activation *i.e.*, higher phospho-STAT1 level in Delta-NSP5 overexpressed cells showed higher ISG15 level.

SARS-CoV-2 NSP5 protein interacts with HDAC2 protein

Next, we investigated whether NSP5 interacted with HDAC2 or not by Co-immunoprecipitation (Co-IP) experiments. From the co-IP experiment, we found that NSP5 of different SARS-CoV-2 variants were interacting with HDAC2 in A549 cells (**Figure 2C**) as well as in HEK293 cells (**Figure 2D**). Then we performed cytoplasmic/nuclear fractionation study to check localization of over-expressed NSP5 as well as HDAC2 in A549 cells. From **figure 2E**, it has been observed that NSP5 proteins were mostly localized in the cytosol whereas HDAC2 protein was mostly nuclear. Being spatially separated, it was puzzling that how NSP5 was regulating HDAC2 activity.

NSP5 sequester HDAC2 protein in the cytosol, thus inhibiting the nuclear activity of HDAC2

To understand the mechanism of action, next we performed co-immunofluorescence microscopy to check co-localization of NSP5 and HDAC2 proteins in A549 cells (**Figure 2F**). The HA-NSP5 protein was labelled with Alexa-594 (Red), HDAC2 was labelled with Alexa-488 (Green) and DAPI was used to stain the nucleus (Blue). We have observed that NSP5 proteins of different variants were interacting with HDAC2 protein mostly in the cytoplasm but not in the nucleus. Interestingly, we have found that the intensity of nuclear HDAC2 was lower in NSP5 transfected cells compared to un-transfected neighbouring cells for all three variants (**Figure 2G**). Hence, it could be possible that NSP5 is sequestering HDAC2 into the cytosol, thus inhibiting HDAC2's deacetylation activity. So that the Histone tails are more acetylated in the nucleus leading to higher gene expression for ISG15. We will investigate this sequestering of HDAC2 with live SARS-CoV-2 infection and also investigate the consequence in anti-viral immunity.

Publications

Anindita Banerjee[1*], **Anup Mazumder**[1*], Jayita Roy[1], Jagyashila Das[1], Agniva Majumdar[2], Ananya Chatterjee[2], Nidhan K Biswas[1], Mamta Chawla Sarkar[2], Saumitra Das[3,1], Shanta Dutta[2], Arindam Maitra[1]. Emergence of a unique SARS-CoV-2 Delta sub-cluster harboring a constellation of co-appearing non-spike mutations. *J Med Virol* 2023 Jan; 95(1):e28413. doi: 10.1002/jmv.28413, PMID: 36541745

(*__Co-First author).__

Animesh K Singh[#1], *et. al.* Contrasting Distribution of SARS-CoV-2 Lineages across Multiple Rounds of Pandemic Waves in West Bengal, the Gateway of East and North-East States of India. *Microbiol Spectr* 2022 Aug 31;10(4):e0091422. doi: 10.1128/spectrum.00914-22. Epub 2022 Jul 19. PMID: 35852336

Group photo of the Lab

Programme Projects

Establishment of Genetics Service Unit (GSU)

PI: Professor Partha P Majumder (2016-2023 March),

Dr. Sagar Sengupta (Current)

Faculty In-Charge (Current):

Prof. Arindam Maitra

Consultant Scientist (NIBMG):

Dr. Paramita Bhattacharya

Project linked personnel:

Debjani Debnath

Purnendu Guha

Research Focus-

Establishment of Genetics Service Unit (GSU)

"Genetics Service Unit" at BioMedical Genomics Unit (BMGU) was established in October, 2016 as a project funded by Department of Biotechnology, Government of West Bengal, as an initiative to set up a much-needed genetic testing centre and for providing reliable genetic testing service to needy patients referred by Government hospitals of West Bengal for diagnosis and carrier testing of genetic disorders. The Government of West Bengal, also concurrently provided substantive funds for purchase of reagents so that reliable genetic testing services may be provided free-of-cost or at subsidized rates to needy patients and at-risk family members reporting to various public hospitals of the State.

Project Highlights

Genetics Service Unit provides testing service for about 50 genetic conditions: The Genetics Service Unit (GSU) was rapidly established on the top floor of the P.G. Polyclinic Building (Annex-3, SSKMH) within the Biomedical Genomics Centre of the National Institute of Biomedical Genomics, and the Unit started to provide genetic-testing services. Over a period of time, the number of different genetic tests increased. Currently, GSU offers 50 different genetic tests for various genetic diseases. Referrals are taken from State Government hospitals and tests and reports are provided free-of-charge to patients, per instructions from the Hon'ble Chief Minister's office. During this period, GSU has standardized and added 4 new tests to its repertoire of tests.


EMQN

Certificate of Participation

2022

this is to certify that

National Institute of Biomedical Genomics

participated in 2022 in the following EMQN CIC external quality assessment schemes:

Scheme	Genotyping	Interpretation	Clinical	Result
DNA SEQUENCING - SANGER	0.00	0.00		satisfactory

The laboratory participated in 1 scheme and passed 1 of them. The detailed performance data are given on the Individual Laboratory Report (ILR). When viewing the certificates, you should ensure that 1 scheme is listed.

Key: Scheme Passed | Scheme Failed | Poor performance

WES: No Results Submitted WES: Withdrew From Scheme

Signed by:

Prof. David Barton, Chair of EMQN

Dr. Simon Patton, Managing Director

Reliability of tests carried out by Genetics Service Unit matches global standards: About a year after GSU initiated providing services, we considered it prudent and imperative to compare our proficiency against global standards. We participated in proficiency-assessment conducted by the European Molecular Genetics Quality Network (EMQN). In each year, we have successfully passed the global proficiency assessment for genetic testing services. The picture of the certificate for the year 2022 is presented here. We are registered to participate in the next year, as well. Genetic Services Unit has also been

maintaining its registration with the National Centre for Biotechnology Information (NCBI), United States of America; Gene Test Registry (GTR) (GTR Lab ID: 506786) (https://www.ncbi.nlm.nih.gov/ gtr/labs/506786/).

Genetics Service Unit is accessed by 13 Government Hospitals: Referrals are obtained from State Government hospitals located not only in Kolkata, but in other districts as well. During this year, 1,412 test referrals were sent by 326 physicians from 49 departments of 13 hospitals. 58 of these were additional test requests (different test for the same patient) and 8 were duplicate requests (same test for same patient).

Manpower trained: Members of GSU are involved in training students in the area of clinical genetics: Dr. Paramita Bhattacharya supervised Ms. Rohini Mukherjee from Department of Biochemistry, University of Calcutta during her summer training programme for 2 months (Jun 2022 to Jul 2022).

Future Plan: We aim to sustain the much-needed genetic testing service, we are currently providing, to needy patients and try to reach out to other Government Hospitals of West Bengal, so that more patients can get benefitted from this project. We shall also expand on the types of genetic tests to cover more genetic conditions, based on frequently received requests from clinicians.

Details of publications/presentations:

Dissertation for Degree of MD (Pediatric Medicine) of The West Bengal University of Health by Dr. Saheli Roy, MBBS (IPGMER), 2022. Title: "IMPACT OF XMN1 POLYMORPHISM (rs7482144) OF HBG2 GENE ON EFFICACY OF HYDROXYUREA IN CHILDREN WITH NON TRANSFUSION DEPENDENT THALASSEMIA IN A TERTIARY CARE CENTRE OF WEST BENGAL." (Co-Guide: Dr. Paramita Bhattacharya)

Dr. Saheli Roy, MBBS (IPGMER), received '*Bhaskarmoni-Kalpana Award'* for securing *First Prize* for oral presentation in 2021, based on her Dissertation for Degree of MD (Pediatric Medicine) awarded in 2022 of The West Bengal University of Health. (Co-Guide: Dr. Paramita Bhattacharya)

Sinha, S., Dutta, A.K., Bhattacharya, P. *et al.* Spectrum of Rare and Novel Indel Mutations Responsible for β Thalassemia in Eastern India. *Ind J Clin Biochem* (2023). https://doi.org/10.1007/s-01098-022-12291w

Dr. Paramita Bhattacharya delivered an invited lecture at a Seminar on Rare Diseases held at Academic Building, IPGME&R to raise awareness for Rare Diseases on 28th Feb., 2023, organized by Centre of Excellence for Rare Diseases, IPGME&R, Kolkata.

National Genomics Core Phase-1

Name of the Project Coordinator and PI :

Professor Partha P Majumder &
Professor Arindam Maitra

Project linked personnel:

Dr. Disha Banerjee
Ms. Ridhima Mitra
Mr. Bijan B Bairagya
Mr. Sumanta Sarkar
Mr. Shekhar Ghosh
Mr. Sudip Kundu
Ms. Tithi Pal
Dr. Kuntal Ghosh

Collaborators:

Centre for DNA Fingerprinting and Diagnostics - CDFD

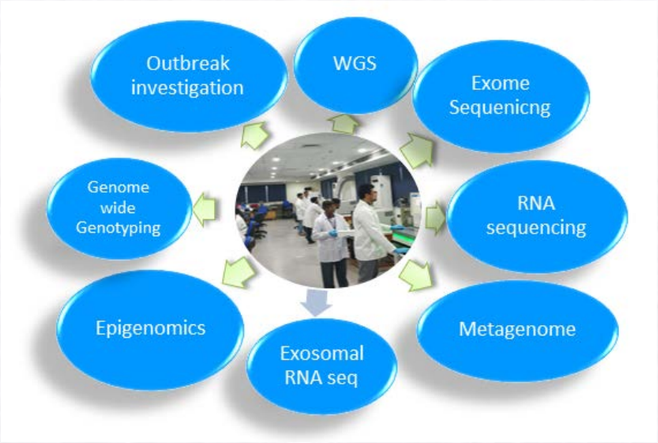
Research Focus

The National Genomics Core has been created to catalyse and facilitate genomics-driven discoveries and applications. The Core is accessible to all academic institutions and the industry for their own research and development. The Core houses and provides access to high-end genomics, computational and other relevant platforms to catalyze discoveries in all domains of biology. The ease of access to these platforms alleviates the challenges faced by researchers to generate and analyze genomics data on scale. This enabling environment attracts biologists to make insightful discoveries using the power of genomics.

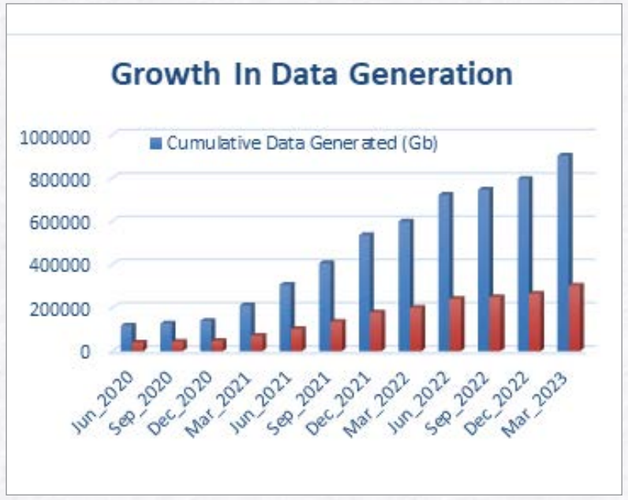
Project Highlights

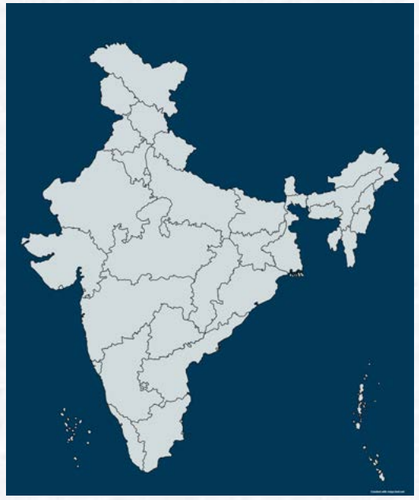
NGC-NIBMG has been able to set up an automated sequencing workflow for taking up challenges of high sample number projects of national importance to very low sample inclusive quick batches in clinical and/or urgent delivery projects. We have deployed Biomek i7 platform for automated sequencing library preparation, Novaseq 6000 for high-throughput and Miseq for low-throughput data generation and DRAGEN (Dynamic Read Analysis for GENomics) Bio-IT platform for automated data analysis. These equipment platforms are utilised for service delivery of whole genome resequencing of diverse species ranging from bacteria till human, whole exome sequencing, whole transcriptome sequencing, metagenomic sequencing, ChIP Sequencing, small RNA Sequencing, RIP Sequencing, gene-panel based assays and many others.

Last year NGC-NIBMG has also installed high-throughput long-read sequencing platforms to augment its capability of providing services pertaining to non-model



organism genomics and understanding structural variation & epigenomics modification in complex genomes. Thus, NGC-NIBMG is now uniquely poised in the country for fast and consistent delivery of high-volume data in diverse application areas. NGC-NIBMG also standardized the method of new areas of application like single cell genomics, FFPE DNA sequencing , ATAC seq ,meta-transcriptomics, etc and service of these area will be provided in near future.





During last one year, National Genomics Core has increased its national presence to 15 states catering to 51 clients based in 35 organizations across India. Service provided by NGC-NIBMG during last 1 year are tabulated as below.

Service Area	Number of samples completed
Human Whole Genome Sequencing	1280
Non-human eukaryotic whole genome sequencing	41
Exome Sequencing	885
Transcriptome Sequencing	765
Bacterial and viral Whole genome Sequencing	503
16s amplicon based metagenomics sequencing	1871
Shotgun Metagenomic Sequencing	210
Microarray based genotyping	1888
Microarray based methylation study	8
ChIP Sequencing	20

Table 1: Genomic Service provided in last one year

NGC-NIBMG is the service providing partner of the prestigious Genome India project and also has been involved in The PAN-INDIA 1000 SARS-CoV-2 RNA Genome Sequencing Consortium and the INSACOG. It has also started working with clinical entities like institutions, such as, ACTREC for their sequencing requirements.

Genomic Surveillance of SARS-CoV-2 In India: Indian SARS-CoV-2 Genomics Consortium (INSACOG)

Convenor, Co-Cordinator and PI: Prof. Arindam Maitra:

Co-PIs: Dr. Sreedhar Chinnaswamy
Dr. Nidhan K. Biswas
Dr. Souvik Mukherjee
Dr. Anup Mazumder

Project linked personnel (Current):

Dr. Shreelekha Dutta
Dr. Sabysachi Bhattacharya
Mr. Animesh Kr Singh
Ms. Madhurima Chakavarty
Mr. Satyajit Chowdhury
Ms. Ayanteeka Mondal

Collaborators:

School of Tropical Medicine, Kolkata: Dr. Bhashwati Bandhopadhyay;

College of medicine and JNM Hospital, Kalyani: Dr. Kuhu Pal;

Guwahati Medical College, Guwahati: Dr. Ajanta Sharma

Research Focus- Indian SARS-CoV-2 Genomics Consortium (INSACOG)

The Indian SARS-CoV-2 Genomics Consortium (INSACOG), jointly initiated by the Department of Biotechnology (DBT) and Union Ministry of Health and with Council for Scientific & Industrial Research (CSIR) and Indian Council of Medical Research (ICMR), is a multi-institutional consortium to monitor the genomic variations in the SARS-CoV-2.

The purpose to establish INSACOG was to:

- Help in understanding the spread and evolution of the virus, and to tackle its future spread.
- Genomic study of accumulated mutations to enable comparison of virus samples and viral lineages in order to understand if local outbreaks are caused by transmission of specific viral lineages.
- Analysis of SARS-CoV-2 genome sequences also allow to assess whether these mutations effect clinical outcomes, severity,
- Genomics data has direct application such as public health intervention measures; develop diagnostics for specific SARS-CoV-2 variant, support the development of therapies and vaccines for specific variant; evaluate and improve understanding of re-infection cases and differentiate between prolonged infection and re-infection.

The sequenced viral genomes analyzed by the respective sequencing laboratory are regularly shared with The Central Surveillance Unit (CSU) works under Integrated Disease Surveillance Programme (IDSP) at the National Centre for Disease Control (NCDC). The NCDC further correlate this data with the field data trends and establish the associations (if any) between the emerging SARS-COV-2 variants and epidemiological drifts based on COVID data generated by State and District Surveillance Units of IDSP.

Project Highlights

NIBMG played a leading role in Coordinating the Consortium during its inception, which started with 10 National laboratories. Today the consortium has expanded to 67 laboratories in a Hub and Spoke model. The consortium in its efforts to reduce turnaround time and reporting has developed Integrated Health Information Platform (IHIP) through which results uploaded by the sequencing laboratories is shared directly to NCDC as well as States for public health measures. The consortium has consolidated genome sequencing data at RCB-IBDC Faridabad. Mirrored data repositories of sequences generated by the consortium is also maintained at NIBMG.

NIBMG has been responsible for sequencing samples from the North East states: Assam, Meghalaya, Mizoram, Manipur, Nagaland, Sikkim. NIBMG has so far sequenced over 33,000 SARS-CoV-2 genomes and submitted to GISAID. NIBMG team has been actively contributing in centralized analysis for the consortium and is responsible for phylogenetic analysis of genomes.

Several studies have demonstrated that increases in SARS-CoV-2 RNA can be detected in environmental samples several

Collaborators:

Zoram Medical College, Falkawn: Dr. Swagnik Roy
Mizoram University: Dr. Senthil Kumar N.
Agartala Medical College, Agartala: Prof. Tapan Majumdar
Sir Thutob Namgyal Memorial Hospital, Gangtok: Dr. Srijana Gurung
Jawaharlal Nehru Institute of Medical Sciences, Imphal: Dr. R K Manojkumar

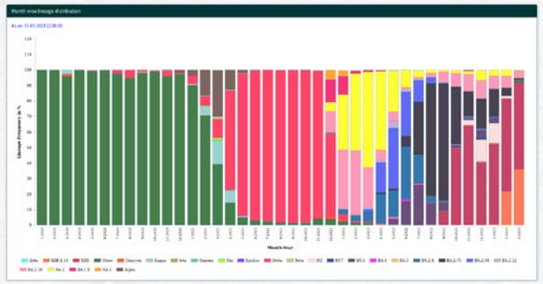


Fig: Monthwise distribution of circulating variants and sub-lineages in India

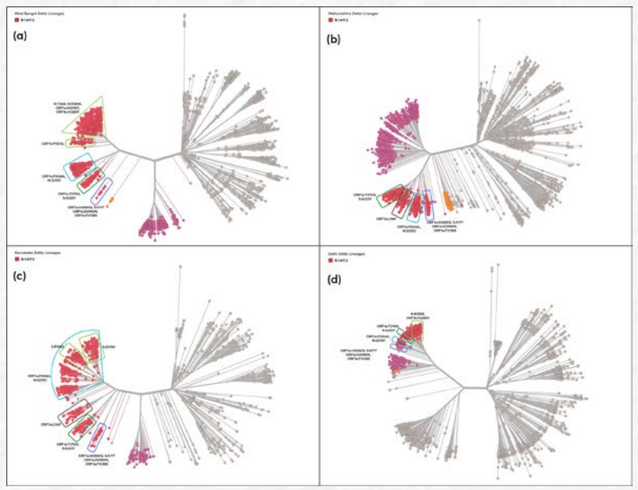


Fig 1: Unrooted phylogenetic tree of the Delta sublineages for sequences sampled from the state of West Bengal (a), Maharashtra (b), Karnataka (c), and (d) Delhi (d).

days before detection of COVID-19 through clinical surveillance. The concept is based on recent published evidence which suggest that ACE2 receptors for SARS-COV-2 are abundantly expressed in small intestine in both symptomatic and asymptomatic individuals allowing viral replication and its secretion into environment through human faeces. Consequently, there is potential to use environmental surveillance for early warning, particularly of clusters or outbreaks in countries that have already contained transmission and are easing public health and social measures, or in the event of seasonality. To establish an early warning management system that will enable the government authorities and healthcare settings to prepare themselves and take appropriate measures in combating the expected COVID-19 outbreak or wave in a locality that will stop the further spread of new genetic variants of SARS-CoV-2, INSACOG established Waster water surveillance for monitoring SARS-CoV-2 viral load, which is being coordinated by NIMBG.

Sewage samples are being collected from 5 study sites in Kalyani and regularly monitored for changes in viral load.

Publications

Singh AK, Laskar R, Banerjee A, Mondal RK, et al. Contrasting distribution of SARS-CoV-2 lineages across multiple rounds of pandemic waves in West Bengal, the gateway of east and north-east states of India. Microbiol Spectr. 2022 Aug 31;10(4):e0091422. doi: 10.1128/spectrum.00914-22. Epub 2022 Jul 19.PMID: 35852336

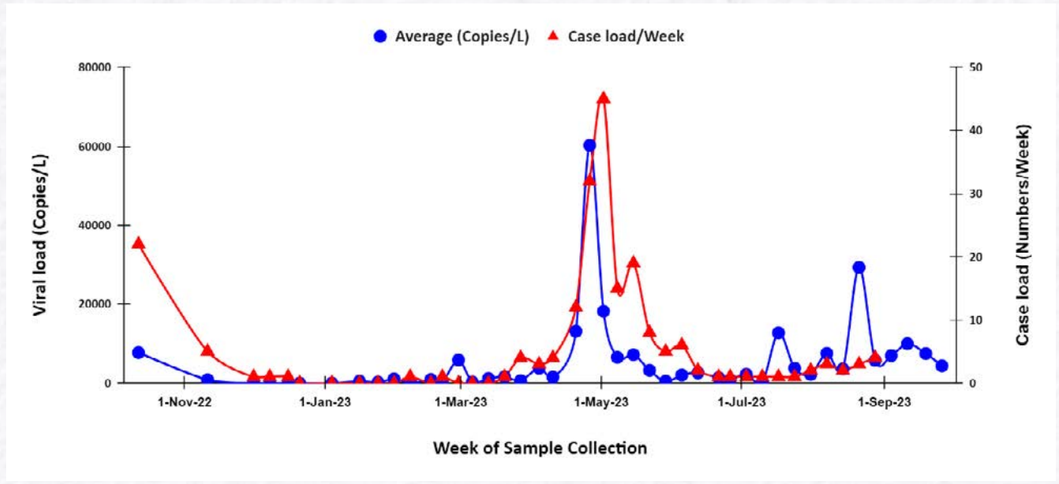


Fig 2: Week wise distribution of average Viral Load (Copies/L) of the sewage samples collected from five sites of Kalyani and SARS-CoV-2 Case Load (numbers/week) in the Nadia district

List of Participating Institutions of Insacog

A. SENTINEL SURVEILLANCE

1. National Institute of Biomedical Genomics (**NIBMG**), **Kalyani**
2. National Centre for Disease Control (**NCDC**), **Delhi**
3. Institute of Genomics and Integrative Biology (**IGIB**), **Delhi**
4. Centre for Cellular and Molecular Biology (**CCMB**), **Hyderabad**
5. Institute of Life Sciences (**ILS**), **Bhubaneshwar**
6. **InSTEM/NCBS**, **Bengaluru**
7. Centre for DNA Fingerprinting and Diagnostics (**CDFD**), **Hyderabad**
8. National Centre for Cell Science (**NCCS**), **Pune**
9. National Institute of Virology (**NIV**), **Pune**
10. National Institute of Mental Health and Neuro Sciences Hospital (**NIMHANS**), **Bengaluru**
11. North East Institute of Science and Technology (**NEIST**), **Jorhat**
12. Indian Institute of Chemical Biology **(IICB), Kolkata**
13. National Chemical Laboratory **(NCL), Pune**
14. Byramjee Jeejeebhoy Government Medical College **(BJGMC), Pune**
15. Indian Institute of Science Education and Research **(IISER), Pune**
16. Central Drug Research Institute **(CDRI), Lucknow**
17. National Botanical Research Institute **(NBRI), Lucknow**
18. Gujarat Biotechnology Research Centre **(GBRC), Gandhinagar**
19. Institute of Bioresources and Sustainable Development **(IBSD), Imphal**
20. Institute of Microbial Technology **(IMTECH), Chandigarh**
21. Institute of Liver and Biliary Sciences **(ILBS), New Delhi**
22. All India Institute of Medical Sciences, **(AIIMS), New Delhi**
23. Rajiv Gandhi Centre for Biotechnology **(RGCB), Thiruvananthapuram**
24. Regional Medical Research Centre **(RMRC), Bhubaneswar**
25. National Institute for Research in Tuberculosis **(NIRT), Chennai**
26. Regional Medical Research Centre **(RMRC), Dibrugarh**
27. Centre for Brain Research - Indian Institute of Science **(CBR-IISc), Bangalore**
28. Jawaharlal Nehru Centre for Advanced Scientific Research **(JNCASR), Bengaluru**
29. Translational Health Science and Technology Institute **(THSTI), Faridabad**
30. Indira Gandhi Institute of Medical Sciences **(IGIMS), Patna**
31. Government Doon Medical College **(GDMC), Dehradun**
32. Mahatma Gandhi Medical College **(MGMC), Jaipur**
33. All India Institute of Medical Sciences **(AIIMS), Bhopal**
34. Gandhi Medical College **(GMC), Secunderabad**
35. Sri Aurobindo Institute of Medical Sciences & Post Graduate Institute **(SAIMS & PGI), Indore**
36. Government Medical College, **Patiala**
37. Kempegowda Institute of Medical Sciences **(KIMS), Bengaluru**
38. Kasturba Hospital for Infectious Diseases, **Mumbai**
39. Sawai Man Singh Medical College, **Jaipur**
40. Lok Nayak Hospital-Maulana Azad Medical College, **New Delhi**
41. Pasteur Institute, **Shillong**
42. Maharshi Dayanand University, **Haryana**
43. State Public Health Laboratory, **Chennai**
44. Mysore Medical College & Research Institute, **Mysuru**
45. MRU, Dept of Anatomy, Institute of Medical Sciences, BHU, **Varanasi**
46. Shri Vinoba Bhave Civil Hospital & NAMO Medical Education & Research Institute, **Silvassa**
47. National Institute of Immunology **(NII), New Delhi**
48. Siddharth Medical College, **Vijayawada**
49. King George's Medical University**(KGMU), Lucknow**
50. National Environmental Engineering Research Institute **(NEERI), Nagpur**
51. Defense Research & Development Establishment **(DRDE), Gwalior**
52. Bangalore Medical College & Research Institute **(BMCRI), Bengaluru**
53. Dr. S N Medical College, **Jodhpur**
54. All India Institute of Medical Sciences **(AIIMS), Raipur**
55. All India Institute of Medical Sciences **(AIIMS), Jodhpur**
56. Zoram Medical College **(ZMC), Mizoram**
57. Agartala Government Medical College **(AGMC), Agartala**
58. Genome Sequencing Lab, Chuchot, **Ladakh**
59. Haffkine Institute for Training, Research & Training, **Mumbai**
60. North Goa Hospital, **Goa**
61. Government Medical College, **Haldwani**
62. Rajendra Institute of Medical Sciences, **(RIMS) Ranchi**
63. Veer Chander Singh Garhwali Govt. Institute of Medical Sciences and Research (**VCSGGIMSR**), Garhwal, Uttarakhand
64. All India Institute of Medical Sciences **(AIIMS), Nagpur**
65. STNM Hospital, Gangtok, **(STNM) Sikkim**
66. Shri Lal Bahadur Shastri Government Medical College, Mandi, **(SLBSGMC) Himachal Pradesh**
67. Sher-i-Kashmir Institute of Medical Sciences, Srinagar, **(SKIMS) Jammu and Kashmir**

B. SEWAGE SURVEILLANCE

1. National Institute of Biomedical Genomics **(NIBMG), Kalyani**
2. Gujarat Biotechnology Research Center **(GBRC), Gandhinagar**
3. Institute of Life Sciences **(ILS), Bhubaneswar**
4. National Centre for Cell Science **(NCCS), Pune**
5. Dr. G.M Taori Central India Institute of Medical Sciences **(CIIMS), Nagpur**
6. Veer Narmad South Gujarat University **(VNSGU), Surat**
7. Indian Institute of Sciences **(IISc), Bengaluru**
8. Indian Institute of Chemical Technology **(IICT), Hyderabad**
9. Translational Health Science and Technology Institute **(THSTI), Faridabad**
10. Institute of Bioresources and Sustainable Development **(IBSD), Imphal**

ACADEMIC CAPACITY DEVELOPMENT

Publications (Original Research papers)

1. Banerjee A, **Mazumder A**, Roy J, Das J, Majumdar A, Chatterjee A, **Biswas NK**, Chawla Sarkar M, Das S, Dutta S, **Maitra A***. Emergence of a unique SARS-CoV-2 Delta sub-cluster harboring a constellation of co-appearing non-Spike mutations. J Med Virol. 2023 Jan; 95(1):e28413. doi: 10.1002/jmv.28413. PMID: 36541745; PMCID: PMC9878222.

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EVENTS

Foundation Day, 6th August 2022

We celebrated our 13th Foundation Day on 6th August. Prof. Sanjeev Jain from NIMHANS delivered the Foundation Day lecture. He engaged with the faculty members and students. Following this, a debate competition was organized for our staff and students. Dr. G Taru Sharma, Director of National Institute of Animal Biotechnology, Hyderabad, was a Special Guest in the event.







Independence Day, 15th August 2022

NIBMG celebrated the 76th Independence Day with Flag hoisting by Prof. Arindam Maitra, Officiating Director, NIBMG. This was followed by speeches by senior members of NIBMG fraternity. The event concluded with a short cultural program by our staff and students.




Obaid Siddiqui Memorial Oration, 7th January 2023

Prof. S.C. Lakhotia delivered the 10th Obaid Siddiqi Memorial Oration at NIBMG on 7th January 2023. He was welcomed by our Director, Dr Sagar Sengupta, and felicitated by our Founder and National Science Chair, GOI, Prof. Partha P. Majumder. He also interacted with our faculty members and Students during the day.


Republic Day, 26th January, 2023

We celebrated 74th Republic Day with the unfurling of the National Flag by Dr. Sagar Sengupta, Director, NIBMG. This was followed by the National anthem and his speech. A cultural program was organized by our Students, Staff and Faculty Members.



Institute Day, 23rd February 2023

NIBMG celebrated its Institute Day on 22nd and 23rd February. As a part of the Institute Day celebrations, a sports carnival was organized at the Institute. All members of the NIBMG family enthusiastically participated in the event. Following this, on 22nd February our staff and students organized a cultural event.

On 23rd February the Institute Day lecture was delivered by Lt. Gen Madhuri Kanitkar, Vice Chancellor, Maharashtra University of Health Sciences and the Keynote address was delivered by Professor Louis J. Muglia, President and CEO Burroughs Wellcome Funds.







Women's day, 8th March 2023

We celebrated International Women's Day on 9th March 2023. Our speakers for the were Dr. Sharmila Bapat, NCCS, Pune and Dr. Manjiri Bakre, CEO, OncoStem Diagnostics. Both the speakers and the Chairperson were felicitated by Dr Sagar Sengupta, Director, NIBMG. They speakers spent time sharing their experiences with our Faculty and Students after their talk. Following this we conducted competitions for our staff and students.

DNA Day

We commemorated DNA Day with a talk by Prof. Partha P. Majumder followed by various competitions organized for our staff and students.


Swachh Sagar Surakshit Sagar Campaign

NIBMG participated in the Swachh Sagar Surakshit Sagar campaign, to clean up the 7,500 km long coastline of India, launched by the Ministry of Earth Sciences in association with the Indian Coast Guard, and Environment Ministry with three strategic goals — consume responsibly, segregate waste at home and dispose of responsibly. Our volunteers participated in the clean up drive of Diamond Harbour Beach, West Bengal.

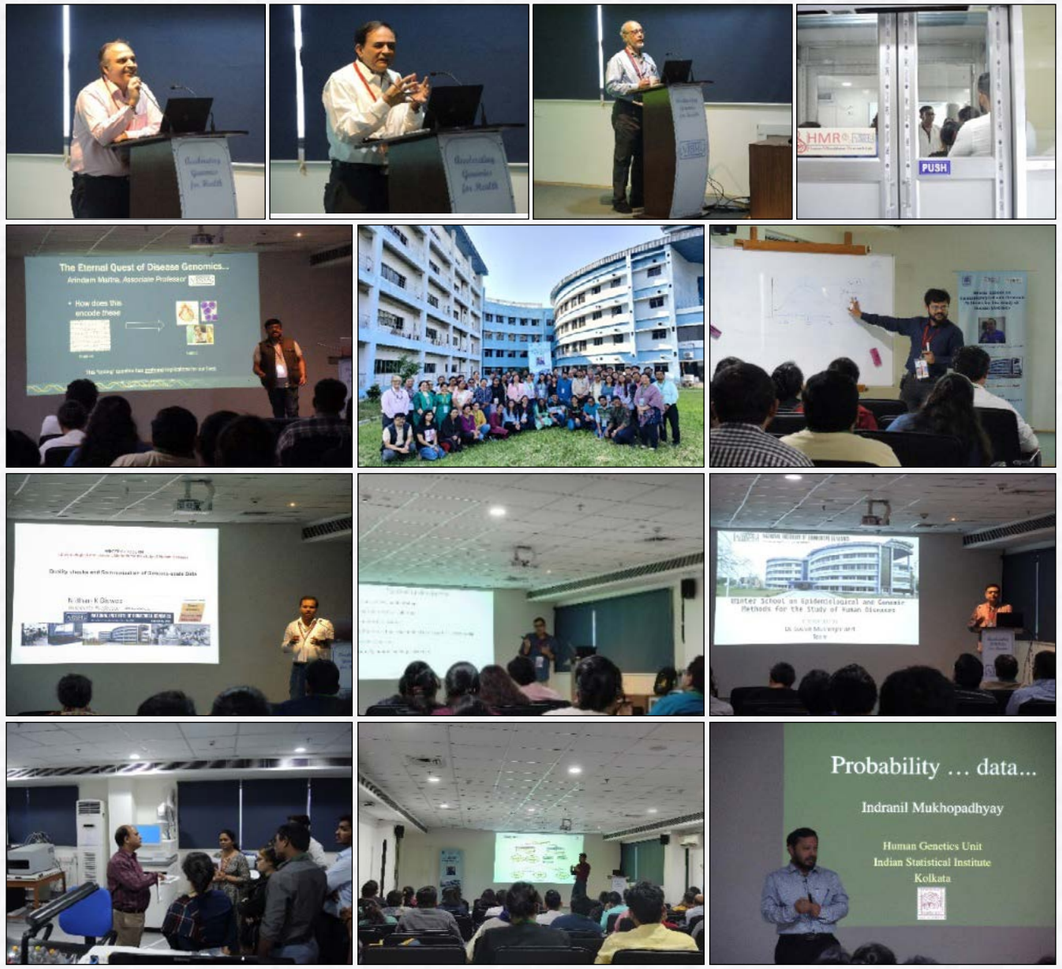



SEMINARS & WORKSHOPS

Winter School on Epidemiological and Genomic Methods for the study of human diseases

NIBMG in collaboration with ISI, Kolkata and CHRD-SAS, New Delhi, organized a Winter School on Epidemiological and Genomic Methods for the study of human diseases from 28th November to 3rd December 2022 sponsored by Sun Pharma Science Foundation. The objective of the *Winter School* was to impart training on study design and various quantitative and statistical methods required for the dissection of complex human diseases. The *Winter School* was open to young researchers working in universities and institutions in India.

In all, 116 applications were received from researchers and clinicians across the country. The number of selected applicants was 59, of whom 51 candidates actually participated in the *Winter School*.



Hands-on training workshop on SARS-CoV-2 sequencing and surveillance

NIBMG conducted a Hands-on Training Workshop and Network meeting on SARS-CoV-2 genome sequencing and surveillance from 21st- 25th November 2022 for collaborators in West Bengal and the NE states of Sikkim, Tripura and Mizoram for capacity building and discussion on initiating Sewage Surveillance. The event was attended by a total of 20 participants and collaborators from these states.

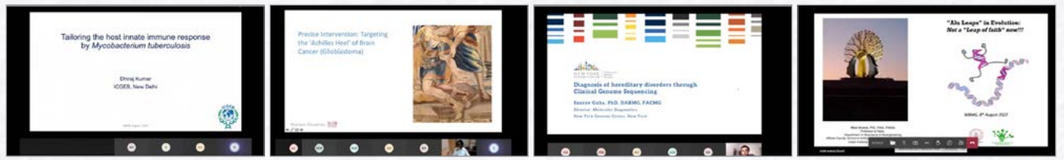
World Microbiome Day

We commemorated World Microbiome Day with a talk by Dr. Sandip Paul, Associate Professor, JIS Institute of Advanced Studies and Research. This was followed by talks by, Dr. Suman K. Paine, Project Manager, Genome India, and Ms. Mousumi Sarkar, PhD student, researchers from NIBMG working on Human Mircrobiome. The event was concluded by a Vote-of Thanks by Dr. Souvik Mukherjee.

Seminar series on Genomics of Health and Wellbeing

As a part of a seminar series on Genomics of Health and Wellbeing, we invited National and International scientists to share their research work. The seminar series was either virtual or in hybrid mode.

Dr. Dhiraj Kumar, ICGEB | Dr. Shantanu Chowdhury, IGIB | Dr. Saurav Guha, New York Genome | Dr. Mitali Mukerji, IIT Jodhpur



Dr. Sudipto Roy, Institute of Molecular and Cell Biology

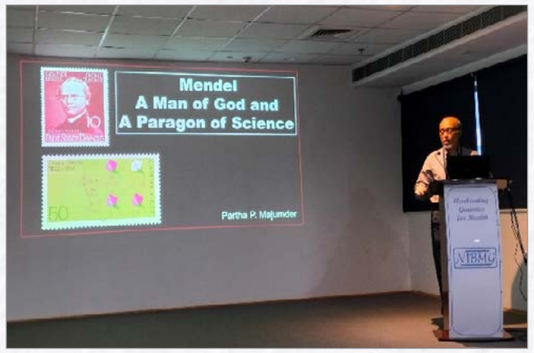
Dr. Arnab Pal, PGIMER

Dr. R. Sankaranarayanan

Dr. Sushanta Banerjee, University of Kansas Medical Centre

Gregor Mendels' 200th Centenary

NIBMG commemorated 200 years of Gregor Mendel's Birth Anniversary by organizing a seminar on 20th July. Our speaker was Prof. Partha Pratim Majumder and his talk was titled "Mendel: A Man of God and A Paragon of Science". This lecture was streamed live on NIBMG's YouTube channel.



SEMINARS AT THE INSTITUTE

Speaker	Affiliation	Date	Title
Dr Ujjwal Neogi	Karolinska Institutet, Sweden	05 July 2022	Immuno-metabolic reprogramming regulating SARS-CoV-2 replication and COVID-19 disease severity: A systems biology perspectives
Dr Sudipto Roy	Institute of Molecular and Cell Biology, Singapore	07 July 2022	Cilia, Crooked Spines and Infertility
Prof. Partha Pratim Majumder	National Institute of Biomedical Genomics	20 July 2022	Mendel: A Man of God and a Paragon of Science
Dr Kushal K. Dey	Harvard T.H. Chan School of Public Health, USA	15 July 2022	Identifying disease-critical variants, genes and cell types using genetic and genomic data
Prof. Shantanu Chowdhury	IGIB, New Delhi	25 July 2022	Precise Intervention: Targeting the 'Achilles Heel' of Brain Cancer (Glioblastoma)
Dr. Dhiraj Kumar	ICGEB, New Delhi	01 August 2022	Tailoring the host innate immune response by *Mycobacterium tuberculosis*
Dr. Mitali Mukerji	IIT Jodhpur	08 August 2022	"Alu Leaps" in Evolution - Not a "Leap of faith" now !!
Prof. Sanjeev Jain	NIMHANS, Bangalore	06 August 2022	Challaneges, Promises and Pitfalls: the hunt for the genetic basis of the diseases of the mind
Dr. Saurav Guha	New York Genome Centre, USA	26 August 2022	Diagnosis of hereditary disorders through Clinical Genome Sequencing
Dr. R. Sankarnarayan	CSIR-CCMB, Hyderabad	19 October 2022	Chiral proofreading and its evolutionary implications
Dr. Arnab Pal	PGIMER, Chandigarh	27 October 2022	Biomarker discovery for oral squamous cell carcinoma: Candidate Gene vs OMICS approach
Dr. Susanta Banerjee	University of Kansas, USA	03 November 2022	Role of CCN1 in Mutant KRAS addiction in Pancreatic Cancer Growth and Progression
Dr. Kaustubh Adhikari	The Open University, UK	05 December 2022	Prediction of pigmentation in ancient DNA samples – a survey through time and space
Dr. Arkajyoti Bhattacharya	University Medical Center Groningen, Netherlands	13 December 2022	Robust transcriptional components depicting effect of CNAs, Metabolism and immune in large set of patient-derived cancer-related samples and cell lines
Dr. Anil K. Mondal	LVPEI Hyderabad	29 December 2022	Congenital Glaucoma: Insights on Heterogeneity derived from 30 years' experience at L.V. Prasad Eye Institute
Prof. S.C. Lakhotia	BHU Distinguished Professor	07 January 2023	Gene- An Evolving Concept
Prof. Louis J. Muglia	Burroughts Wellcome Funds	23 February 2023	Preventing Prematurity: The Genomics of Birth Timing
Lt. Gen Madhuri Kanitkar	Maharashtra University of Health Science	23 February 2023	My Journey towards making my research count

Speaker	Affiliation	Date	Title
Dr. Indranil Mukhopadhyay	ISI Kolkata	06 February 2023	Pseudotime reconstruction using single cell RNA-seq data
Dr. Navonil De Sarkar	Medical College of Wisconsin, USA	13 February 2023	Beyond genomics with ctDNA: Accurately inferring epigenetically diverse phenotypic subtypes of prostate cancer
Dr. Sharmila Bapat	NCCS, Pune	09 March 2023	Knowledge enablers in targeting ovarian cancer
Dr. Manjiri Bakre	OncoStem Diagnostics	09 March 2023	Where there is will there is a way: Journey of OncoStem

OUTREACH

National Science exhibition

NIBMG participated in the 25th National Science Exhibition, Salt Lake, West Bengal from 24th to 27th August, 2022 where faculties along with administrative and technical staff students at the Institute demonstrated our research work to school & college students and members of general public.




Kalyani Book Fair

NIBMG participated in the Kalyani Book Fair held from 10th-18th December. Our stall was visited by many students, teachers and general public.



India International Science Festival

We participated in the Indian International Science Festival at Manit, Bhopal from 21st-24th January. We showcased our research using scientific posters, slides, videos and 3d models. Our stall attracted visitors from diverse backgrounds and of all age groups.




National Science Day

NIBMG celebrated the National Science Day on 28 Feb 2023 to commemorate the announcement of the discovery of the "Raman effect" by C V Raman. It was also celebrated as Open Day at NIBMG. The visitors included more than 115 students and teachers from the schools and colleges around NIBMG including Garden High School, IISER-Kolkata; Springdale High School, Kalyani; Delhi World Public School, Kalyani; Ramakrishna Mission Vivekananda Centenary College, Rahara, Kolkata; Dhruba Chand Halder College, Barasat. The guided tours within NIBMG campus included visits to the Human Genome Hall, Genome Laboratory, Genome Sequencing demonstration, science models, interactive games and quiz. There was also a debate organized on the following theme: Artificial Intelligence is a threat to human intelligence.


School college visits

We also organized Outreach visits for school and college students from in and around Kolkata. The students had a chance to interact with our Faculty members and visit our research facilities, Genome Hall and Genome Lab.




DETAILS OF VISITS

Date	No. of Students	No. of faculties	Dept/univ/coll
05-Jul-22	42	3	BB College,Asansol
06-Apr-22	16		WB State University,Dept. of Physiology
20-Aug-22	18		Calcutta University,Department of Biotechnology
22-Sep-22	22	1	Srirampore College,Dept. of Physiology
29-Oct-22	50	2	AIIMS
8/Nov/2022-12/Nov/2022	11	1	Bodoland University
26-Nov-22	25		Department of Microiology,Sister Nivedita University

Academic work performed outside of the institute by members

Name of the Member	Description of the work
Moulinath Acharya	◆ Invited talk titled "Delineating genetic heterogeneity in spectrum disorders" in the Department of Psychiatry at All India Institute of Medical Sciences, Kalyani on April 2, 2022. ◆ Invited talk titled "Investigating heterogeneity in neurodegenerative spectrum disorders of eye and muscle" in the 46th Annual Meeting of Indian Society of Human Genetics on April 9, 2022. ◆ Invited talk titled "Zebrafish model to study human diseases" in the workshop on Biomolecular techniques of waste valorization organized by the Department of Environmental Sciences at Burdwan University on June 2, 2022. ◆ Invited talk titled "Investigating heterogeneity in glaucomatous neurodegeneration using functional genomics" in the 20th All India Congress of Genetics and Genomics at CSIR-IICB Kolkata on January 30, 2023. ◆ Participated and gave an invited talk titled "Zebrafish model to study human diseases" in the workshop on Biomolecular techniques of waste valorization organized by the Department of Environmental Sciences at Burdwan University on June 2, 2022.
Anupam Basu	◆ PhD thesis evaluated i) Vinay J (NISER, Bhubaneswar): Genetic predisposition and molecular mechanistic studies of Matrix metalloproteinases in Gallbladder carcinogenesis ii) Mrinmay Dhauria (Calcutta University) : A Study Of Polymorphisms In Candidate Genes Related To Human Happiness And Subjective Well Being. iii) Shashank Shekhar (JNU): Identification and characterization of origin(s) of replication in human malaria parasite *Plasmodium falciparum* ◆ Chair the session (Disease Biology) at 91st Annual Meeting of the Society of Biological Chemists (India), held on 8-11 December 2022, Kolkata
Samsiddhi Bhattacharjee	Acted as External Expert in the seminar for defense of a research proposal by Mr. Arnab Khan, Senior Research Fellow, Human Genetics Unit, Indian Statistical Institute, Kolkata on 20th July, 2022.

Name of the Member	Description of the work
Nidhan K. Biswas	◆ Delivered multiple talks in the Mizoram University - Accelerate Vigyan - NGS workshop, October 2022 ◆ Delivered talk in the 3rd Immunology workshop on Advanced immunology concepts and techniques - CMC Vellore on 31st March, 2023. ◆ Delivered a talk in the Wastewater surveillance workshop (21-25 Nov 2022) in NIBMG, Kalyani. ◆ Delivered two talks in the NIBMG Winter School on Genetic Epidemiology. ◆ Microbiologist society of India - National seminar on "Recent Advancement in Microbial Biotechnology on 23rd April 2022. "Title of talk: Tracking temporal evolution of SARS-Cov-2 lineages" ◆ Delivered a talk in the MPAICON conference on 06 Aug 2022.- "Title of talk: Mapping molecular progression of oral precancers to frank oral cancer". ◆ Delivered a talk in the Society of Biological Chemists annual meeting on 9th Dec 2022, Kolkata - "Title of talk: Genomics insights into the mutational trajectories of oral precancerous lesions progression to frank oral cancer
Sreedhar Chinnaswamy	◆ Have been participating in regular meetings of the DBTs IDB TEC meetings and DST-SERB COVID-19 special task force for evaluation of R&D proposals ◆ Serving as the DBT nominee of Krish Biotech, Kalyani IBSC ◆ Served as a session chair in the recently held 7th Molecular Virology Meeting at IISc Bengaluru ◆ Evaluated a thesis from Genetics Dept. of UoH, Telangana ◆ Gave a science day talk as part of National Science Day celebrations in a local school in Kalyani ◆ Participated in question paper setting for MSc exams of Presidency University, Kolkata
Srikanta Goswami	◆ Invited lecture titled "Genetics of Functional Gastrointestinal Disorders: Review of Current Knowledge" at the 26th Annual Meeting of Indian Society of Gastroenterology (WB Chapter) on February 03, 2023. ◆ Participated in a Panel Discussion on "Cancer genomics could be a game changer in precision oncology" as a Panellist at the 'International Onco-Summit 2023' Organized by Netaji Subhas Chandra Bose Cancer Hospital on January 28-29, 2023, in Kolkata. ◆ Invited lecture titled "A Tale of Two Diseases: Their individuality and how do they Cross-talk" at the Fifth Regional Science and Technology Congress, held at West Bengal State University on January 19, 2023. ◆ Invited Lecture, entitled "The noncoding-RNome in Pancreatic Cancer: Exploring Function and Decoding their Global Regulation" at the 11th RNA Group Meeting, held during December 2-4, 2022 in NCCS, Pune. ◆ Co-chaired a session at the 91st Annual Conference of the Society of Biological Chemists (India) held during December 8-11, 2022 in Kolkata. ◆ Invited Lecture, entitled "SMART-COV-2: Strategies by COVID-19 virus to fool Host RNA Decay Machinery" at Department of Biotechnology, Banaras Hindu University, Varanasi July 15, 2022 ◆ Invited Lecture, entitled "Trajectories of Disease Biomarker: Translational Research Perspective" at the 'Refresher Course in Biotechnology' organized by Kalyani University, July 13, 2022 ◆ Participated as External Expert at multiple Departments of Calcutta University, Burdwan University, St. Xaviers College, Kolkata. Acted as PhD Thesis Examiner of JNU and KIIT University.

Name of the Member	Description of the work
Arindam Maitra	*Talks Given:* ◆ Longitudinal DNA Methylation Profile of Peripheral Blood in Women During Pregnancy Informs Birth Outcome" in the 2nd Subhash Mukhopadhyay Symposium organized by Adamus University on 15th January 2023. ◆ Genome-wide association study identifies maternal genotypes associated with spontaneous preterm delivery in Indian Women. Australasia Symposium 2022 Youth Forum of the Preterm Birth International Collaborative (PREBIC), on 17 December 2022. ◆ Genomics Surveillance of the Pandemic.... The Past, The Future and What We Achieved. 91st Annual Meeting of the Society of Biological Chemists (India), 08 December 2022. ◆ Detecting Genomic Variation in a Group of Individuals. Winter School on Epidemiological and Genomic Methods for the Study of Human Diseases. Sun Pharma Foundation & NIBMG, 30 November 2022. ◆ Single Cell Profiling and Identification of Hypoxia Induced Cellular Transitions in Oral Squamous Cell Carcinoma. North Zone Conference of Indian Academy of Biomedical Sciences (NZIABSCON 2022) in PGIMER, Chandigarh on 4th November 2022. ◆ Next Generation Sequencing in Disease Genomics. Symposium organized by North Bengal University, 10 September 2022. ◆ Human Genome Initiatives. Symposium on Making of Global Hub - Biologics and Biosimilars, organized by ASSOCHAM, 29 April, 2022. *External engagements* ◆ Participated as Invited Faculty in Department of Biochemistry, University of Calcutta, Ramakrishna Mission Vivekananda Educational Research Institute, NIPER, Guwahati. ◆ Member of PG Board of studies, Dept. of Genetics, University of Calcutta. ◆ Adjunct faculty of Regional Centre for Biotechnology. ◆ Co-supervised two PhD students in PGIMER Chandigarh. ◆ Acted as PhD examiner of Banaras Hindu University, University of Calcutta, North-Eastern Hill University, Kalinga Institute of Industrial Technology Deemed to be University and Jaipur National University. ◆ Reviewed grant and fellowship proposals for DBT-Welcome Trust India Alliance and participated as a member of their selection committees for Early Career fellowships and Research Management fellowships. ◆ Reviewed grant proposals in the areas of human genomics, human disease and developmental biology and cancer biology submitted to DBT, Govt. of India. ◆ In the organizing committee of the 91st Annual Meeting of the Society of Biological Chemists (India), 8 – 11 December 2022, Biswa Bangla Convention Centre, Kolkata. Chair of Scientific Program Committee & Member of Organizing Committee. Peer reviewed many manuscripts for many international journals e.g. Nature Communications, Communications Biology, Epigenomics, Journal of Medical Virology, Scientific Reports, Journal of Biosciences etc.

Name of the Member	Description of the work
Souvik Mukherjee	◆ Delivered a talk titled "Role of Human Microbiome in Health and Disease" in Science Talk on World Microbiome Day organised by Microbiologists Society of India on 28th June 2022. ◆ Delivered a talk titled "Importance of Metagenomics in Human Health and Pathogen and Detection in Diseases" in Accelerate Vigyan Karyashala (High-end Workshop) Programme, SERB, Govt. of India at JIS Institute of Advanced Studies & Research, Kolkata on 18th July 2022. ◆ Delivered a talk titled "Importance of Sequencing Microbial Pathogens: Role of Metagenomics in Pathogen Detection" in TROPACON Northeast Chapter-2022 at Zoram Medical College on 10th November 2022. ◆ Delivered a talk titled "Host-Microbiome interactions in Human health and diseases" in 47th Annual Conference of the INDIAN SOCIETY OF HUMAN GENETICS (ISHG) at Andhra University on 23rd – 25th January 2023. ◆ Delivered a talk titled "Host-Microbiome Interactions in Chronic Lifestyle Disorders in Indian Population" in the Workshop conference on "HUMAN MICROBIOME IN HEALTH AND DISEASE" (HMCW_2023) at THSTI, Faridabad on 15th February 2023.
Bhaswati Pandit	◆ Delivered talk on 'Host genomic variations and it influence on susceptibility to tuberculosis' at Haldia Institute of Technology, 2023 ◆ Presented poster on 'Replication and Validation of host genetic variants associated with Tuberculosis' in the conference 'Towards End TB: Achievements, Challenges and Future Directions' at THSTI
Sagar Sengupta	◆ Member of DBT Cancer Biology Task Force ◆ Member of SERB Program Advisory Committee (PAC) on Interdisciplinary Biological Sciences ◆ Member of the Scientific Advisory Board of Advanced Research Unit on Metabolism, Development & Aging (ARUMDA) ◆ Member of Monitoring Committee for CSIR-MLP project om T cell immune monitoring for Covid-19 ◆ Member of the screening committee for DBT Research Resources, Service Facilities and Platforms (RRSEP) ◆ Member of DBT Sectorial Expert Committee on Biomanufacturing ◆ Invited speaker in 91st Annual meeting of Society of Biological Chemists, Biswa Bangla Convention Centre, Kolkata, India, 8th – 11th December, 2022. ◆ Invited speaker in "Biological Transactions: From molecules to organisms", Indian Institute of Science, Bangalore, India, 18th–21st January, 2023. ◆ Delivered Prof. G. K. Manna memorial lecture award during 20th All India Congress of Genetics and Genomics (AICGG) organized at the Indian Institute of Chemical Biology, Kolkata, 30th–31st January, 2023. ◆ Invited speaker in International symposium "Interdisciplinary approach to Biological Sciences-2023" (IABS-2023), Indian Association for Cultivation of Science, Kolkata, India, 1st – 3rd February, 2023. ◆ Session chair and invited speaker in Royal Society Yusuf Hamied Workshop for India and the UK, 23rd-24th February, 2023. ◆ Invited speaker in Emerging Materials in Cancer Therapy (EMCT), Indian Institute of Technology & Rubber Technology Centre, Kharagpur, India, 24th March, 2023.

Name of the Member	Description of the work
Sandeep Singh	◆ Invited talk on the occasion of foundation day of Biochemistry Department; organized by AIIMS-Bhopal on 20.01.2023. Title of the talk: "Cellular plasticity: Target against aggressive cancer behaviour". ◆ Invited faculty for Christie College, London-TMC-Kolkata first FRCR preparatory course organized on 14.01.2023 ◆ Delivered Invited talk in 91st Annual Meeting of Society of Biological Chemists (India) organized by SBC(I) Kolkata Chapter, Bose Institute, CSIR-IICB, NIBMG, and Sister Nivedita University, Kolkata on 08.12.2022. Title of the talk: 'Cellular plasticity' : Non-genetic driver of aggressive cancer ◆ Appointed as a member of the Board of Examiners relating to M.Sc. Semester-II Practical Examination 2022 scheduled to be held on 27.09.2022, in Molecular Biology Paper- MSMHG 205 at Department of Zoology, The University of Burdwan ◆ Invited to deliver a Lecture in the Foundation for Head-Neck Oncology (FHNO) Annual Meeting-FHNO 2022, 4th-6th November, 2022 at Guwahati. The topic of the talk: Oral cancer stem cells: Present status and future clinical implications in Head Neck Cancer ◆ Invited Lecture on the occasion of World Cancer Research Day on September 24th, 2022 organized by IACR Mumbai Chapter at ACTREC, Mumbai. Title of the talk: Nongenetic-heterogeneity: Driver of oral cancer ◆ Chaired the session and acted as evaluator on 27th May 2022, in the AppSciCon22 conference organized by MAKAUT, West Bengal
Anup Mazumder	◆ Served as an external examiner at Department of Biotechnology, School of Life Science, Adamas University, Barasat, West Bengal on 22nd July 2022.

Academic work performed outside of the institute by NIBMG-students

Name of the Member	Description of the work
Arunima Acharya	◆ Received certified training on "*Hands-on Training on Basics of Laboratory Animal Handling (mice) & Research*" from September 19-26, 2022 at Indian Institute of Science and Research, Kolkata (IISER-K). ◆ Presented poster entitled "*Characterization of Slow Cycling Cells (SCC)" in Gingivo-Buccal Oral Squamous Cell Carcinoma.*" at the 47th Indian Society of Human Genetics (ISHG) from January 23-25, 2023 at Visakhapatnam, Andhra Pradesh.
Tahseen Ahmed	◆ Poster presentation titled "*Investigating the role of WT1 transcription factor in the pathogenesis of developmental glaucoma spectrum disorders*" at the Society of Biological Chemists, Kolkata Chapter held in Sister Nibedita University on 9th April 2022. ◆ Oral presentation titled "*Investigating the role of WT1 transcription factor in the pathogenesis of developmental glaucoma spectrum disorders*" at 47th Annual Meeting of Indian Society of Human Genetics held in Vizag, India from 21st to 24th January, 2023.
Gaurav R. Amale	◆ Acted as Volunteer for NIBMG exhibits at India International Science Festival, IISF 2022
Esha Bhattacharjee	◆ Presented poster entitled "*Genetic determinants of gestational length in Indian women: A two-stage genome wide association study.*" in 2022 Annual Meeting of American Society of Human Genetics (ASHG) from October 25-29, 2022 in Los Angeles, California.
Diptanil Biswas	◆ Acted for Volunteer for NIBMG exhibits at Kalyani Book Fair, 2022

Name of the Member	Description of the work
Sudipta Chakraborty	◆ Poster presentation titled ***"A quantitative trait GWAS on lens thickness identifies risk loci on PTPRM in the narrow-angle individuals anatomically susceptible to primary angle closure glauocoma***" in the symposium of Society of Biological Chemists which will be held at Sister Nivedita University on 9th April 2022. ◆ Poster presentation Received ARVO regsitation waiver award to present his work virtually in the annual meeting of the Association for Research in Vision and Ophthalmology (ARVO) 2022, at Denver, USA from 1th to 4th May 2022. His abstract, titled ***"A genome wide copy number variation analysis reveals NLGN1 is over represented in primary angle closure glaucoma patients,"*** was published in the Investigative Ophthalmology & Visual Science journal's supplementary section. ◆ Presented a poster entitled "***A whole-exome analysis of non-syndromic hearing loss patients from India reveals a wide spectrum of known and novel mutations***", virtually in the Rare genetic research summit-2022 (REDRESS2022) which was held in Bangaluru, from 24th -25th November 2022. ◆ Oral presentation titled "***discovery of risk gene in angle closure glaucomatous neurodegeneration***" as a shortlisted candidates for the final round in the Indian National Young Academy of Sciences (INYAS) Saransh – Thesis Competition for PhD students 2022 in the 17th December 2022. ◆ Oral presentation, titled "***A Comprehensive Genome Wide Association to Functional Study Reveals the Role of CNTNAP5 in Glaucomatous Neurodegeneration in Primary Angle Closure Glaucoma***" in the 25th Biennial Meeting International Society for Eye Research, Gold Coast, Queensland, Australia from 19th February to 23rd February 2023.
Shouvik Chakravarty	◆ Attended Society of Biological Chemists (SBC)- Kolkata chapter meeting from April 9-10, 2022, Kolkata
Pratyusha Chikkala	◆ Delivered an oral presentation at 47th Annual Conference of the Indian Society of Human Genetics (January 25, 2023) at Visakhapatnam, Andhra Pradesh ◆ Presented a poster at 11th General Assembly of the Asia Pacific Organisation for Cancer Prevention (APOCP11- Dec 8-10, 2022), Kolkata ◆ Volunteered in the Kalyani Book Fair (Boi Mela) from Dec 11th - 18th, 2022 ◆ Volunteered in the 25th National Science Exhibition, Kolkata from August 24- 28, 2022
Jagyashila Das	◆ Delivered oral presentation entitled "*Genomewide longitudinal DNA methylation profiling of pregnant women and its association with preterm birth outcome – a GARBH-Ini study.*" in 14th International Congress of Human Genetics (ICHG) 2023 from February 22-26, 2023 in Cape Town, South Africa. ◆ Presented poster entitled "*Genomewide temporal changes of DNA methylation in pregnant women delivering at term and preterm – a GARBH-Ini study.*" in 91st Annual meeting of Society of Biological Chemists (India), December 8-11, 2022.
Subrata Das	◆ "High-End Workshop on Next Generation Sequence Data Analysis in Human Diseases" – Accelerate Vigyan Karyashala Program jointly organized by Mizoram university and NIBMG from 17 - 22 Oct. 2022."
Kallol Datta	◆ Attended Winter School on Deep Learning - From Perceptrons to Diffusion Models Organized by: Electronics and Communication Sciences Unit (ECSU), Indian Statistical Institute, Kolkata from January 06 - March 04, 2023
Debjit De	◆ Delivered a talk titled "Sailing through Human Microbiome Project and Human Microbiome Data Analysis" in the "Workshop on Winter School on Epidemiological and Genomic Methods for the Study of Human Diseases" at NIBMG on 3rd December 2022

Name of the Member	Description of the work
Anuradha Gautam	◆ Had an oral presentation titled 'IL32: an unusual cytokine and its association with pulmonary tuberculosis', at the Annual conference on Indian Association of Human Genetics, 2023. ◆ Presented a poster titled 'Contribution of Myeloid Cells towards TB Specific Host Gene Signature: Revelations from Re-Analysis of Publicly Available Datasets' Tuberculosis' in the conference 'Towards End TB: Achievements, Challenges and Future Directions' at THSTI, 2023
Supratim Ghosh	◆ Received certified training in Satellite meeting cum workshop on "*Chemistry and Biology of Epigenetics.*" from September 29-30, 2022 from Indian Institute of Chemical Biology (IICB), Kolkata. ◆ Attended and participated in Human Cell Atlas (HCA) Asia 2022 meeting on behalf of Asian Immune Diversity Atlas (AIDA) – India team from November 3-5, 2022 in Bangkok, Thailand. ◆ Delivered oral presentation entitled "*Understanding the role of genomic variations in regulating cell-type-specific gene expression of peripheral blood immune cells in healthy individuals.*" at the 47th Indian Society of Human Genetics (ISHG) from January 23-25, 2023 at Visakhapatnam, Andhra Pradesh.
Arnab Ghosh	◆ "High-End Workshop on Next Generation Sequence Data Analysis in Human Diseases" – Accelerate Vigyan Karyashala Program jointly organized by Mizoram university and NIBMG from 17 - 22 Oct. 2022."
Aniket Kumar	◆ Participated in National Science fair as a member from NIBMG.
Dr. Sillarine Kurkalang	◆ Presented poster entitled "*Single-Cell Transcriptomic Analysis of Gingivo-buccal Oral Cancer Reveals Cell Types and Cell States Important in Antitumor Immune Response in OSCC-GB.*" in Human Cell Atlas (HCA) Asia 2022 meeting from November 3-5, 2022 in Bangkok, Thailand. ◆ Presented poster entitled "*Single-Cell Transcriptomic Analysis of Oral Squamous Cell Carcinoma Gingivo-buccal (OSCC-GB) Reveals Tumor Cell Populations Recapitulate Signatures of Early Development."* in Human Cell Atlas (HCA) Developmental and Paediatric meeting from November 20-22, 2022 in Paris, France. ◆ Presented poster entitled "*Characterization of Cellular Diversity in Oral Squamous Cell Carcinoma Gingivo-buccal (OSCC-GB) Reveals Two Dominant Cellular Programs.*" in 91st Annual meeting of Society of Biological Chemists (India), December 8-11, 2022.
Ankita Maddheshiya	◆ Presented a Poster titled "Characterization of Infant Gut Microbiome associated with Early Childhood Growth Failure" in in the conference on "HUMAN MICROBIOME IN HEALTH AND DISEASE" (HMCW_2023) at THSTI, Faridabad on 15th February 2023 and received the Best Poster Award. ◆ Attended a hands-on workshop on "Next Generation Sequencing and Data Analysis" in THSTI, Faridabad on 16-17th February 2023. ◆ Attended a national workshop titled "Hands-on training for Next Generation Sequencing and data analysis" supported by Accelerate Vigyan Karyashala (High-end Workshop) Programme, SERB, Govt. of India at JIS Institute of Advanced Studies & Research, Kolkata on 18-23rd July 2022.
Tuneer Ranjan Mallick	◆ Delegated for NIBMG at IISF 2023 held at MANIT, Bhopal from January 21-24, 2023 ◆ Volunteered in Kalyani Book Fair from December 11-18, 2022 ◆ Presented a poster at the 42nd Annual Conference of the Indian Association for Cancer Research (IACR) held at ACTREC, Navi Mumbai from January 12-15, 2023 ◆ Volunteered for National Science Day Celebration held at NIBMG on 28th February, 2023

Name of the Member	Description of the work
Paromita Mitra	◆ Poster presentation at Cell Symposia: Hallmarks of Cancer, San Diego, USA, October 30th to November 1st, 2022, organized by Cell Press. Title of presentation: TGFβ induced Tie2 expression in cancer associated fibroblasts remodels tumor microenvironment to support stemness in oral cancer cells
Sukanya Mitra	◆ Poster presentation titled "*A whole-exome analyses of non-syndromic hearing loss patients from India reveals a wide spectrum of mutations*" at 47th Annual Meeting of Indian Society of Human Genetics held in Vizag, India from 21st to 24th January, 2023.
Moumita Mukherjee	◆ Presented Poster on her ongoing Research work at 8th RNA Group meeting at NCCS, Pune in December, 2022. ◆ Presented Poster on her ongoing Research work at Annual meeting of Society of Biological Chemists at Kolkata in December, 2022.
Shankha Nath	◆ Presented Poster titled "Upper Respiratory Tract Microbiome Profiles in SARS-CoV-2 Delta and Omicron Infected Patients Exhibit Variant Specific Patterns and Robust Prediction of Disease Groups" in 91st Annual Meeting of Society of Biological Chemists (India), on 8-11th December 2022. ◆ Delivered a talk titled "Sailing through Human Microbiome Project and Human Microbiome Data Analysis" in the "Workshop on Winter School on Epidemiological and Genomic Methods for the Study of Human Diseases" at NIBMG on 3rd December 2022
Samadrita Ojha	◆ Had an oral presentation titled 'Association of Siglec14-Siglec5 Receptor Pair Polymorphism with Tuberculosis Disease', at the Annual conference on Indian Association of Human Genetics, 2023.
Shouvik Paul	◆ Presented a poster titled, "Characterization of Wound Microbiome and Antibiotic Resistance Patterns in Chronic Diabetic Foot Ulcer Infections" in TROPACON-2022, NE chapter, Mizoram on 10th November 2022.
Jayita Roy	◆ Participated in Kalyani Book fair as a member from NIBMG.
Barsha Saha	◆ Presented Poster on her ongoing Research work at 8th RNA Group meeting at NCCS, Pune in December, 2022. ◆ Presented Poster on her ongoing Research work at Annual meeting of Society of Biological Chemists at Kolkata in December, 2022.
Uday Saha	◆ Attended 91st Annual Meeting of Society of Biological Chemists (India) organized by SBC(I) Kolkata Chapter, Bose Institute, CSIR-IICB, NIBMG, and Sister Nivedita University, Kolkata from 08.12.2022 to 11.12.2022 and presented poster on 8.12.2022. Title of presentation: Drug induced cellular plasticity as novel therapeutic target in oral stem-like cancer cells ◆ Attended India International Science Festival (IISF), 2022, in Bhopal from 21.01.2023 to 24.01.2023, organized by Ministry of Science and Technology, Ministry of Earth Science of Government of India in association with Vijnana Bharati and displayed several scientific models engaging the public with science and technology being pursued at NBMG
Mousumi Sakar,	◆ Presented a poster titled "Characterization of Altered Vaginal Microbiome Associated with Preterm Birth in an Indian Cohort Study (GARBH-Ini) of Pregnant Women" in 91st Annual Meeting of the Society of Biological Chemists (India), 8-11th December 2022. ◆ Delivered an oral presentation on the selected abstract titled "Altered Vaginal Microbiome Associated with Preterm Birth in an Indian Cohort of Pregnant Women" in 5th Regional Science & Technology Congress, Dept of Science Technology and Biotechnology, Govt. of West Bengal, at Cooch Behar Panchanan Barma University on 17th-18th January 2023 and received the Outstanding Paper Award.

Name of the Member	Description of the work
Shamita Sanga	◆ Oral presentation titled "*Investigating genetic profile of congenital muscle disorders in Indian patients, focusing on the functional role of novel desmin and lamin mutations*" at Society of Biological Chemists (SBC) organized by Sister Nivedita University, Newtown, West Bengal, India during April 9-10, 2022. ◆ Poster presentation titled "*Functional characterization of mutations causal to rare muscle disorders in genes encoding intermediate filament proteins"* at European Society of Human Genetics Conference (ESHG), Vienna, Austria during June 11-14, 2022. ◆ Poster presentation titled "*Investigation of probable founder effect of frequently occurring mutation SGCB c.544A>C in Indian patients affected with sarcoglycanopathy*" at National Rare Diseases Research Summit (REDRESS-2022), Bengaluru during Nov 24-25, 2022.
Jyotishman Sarma	◆ Poster presentation titled as "*Functional investigation of GWAS associated SNP rs1800629 located in the promoter region of TNFa in glaucomatous neurodegeneration.*" at 47th Annual Meeting of Indian Society of Human Genetics held in Vizag, India from 21st to 24th January, 2023.
Deepshikha Shaw	Visited Dr. K. Kemparju laboratory, department of Biochemistry, University of Mysore (14 Aug – 15 Sep 2022). She performed the following experiments there. ◆ Neutrophil isolation from healthy human blood. ◆ NETosis (Neutrophil Extracellular Trap formation) induction. ◆ NET quantification. ◆ Imaging.

Awards and Honours

Name of the Member	Description
Nidhan K Biswas	Elected Fellow, 2023, West Bengal Academy of Science and Technology
Analabha Basu	Elected Fellow, 2023, Indian Academy of Sciences
Mr. Sudipta Chakraborty	◆ Received an **Outstanding Paper Award** in Medical Science at 5th Regional Science and Technology Congress 2023, West Bengal State University. The title of his presentation was "***Investigating the Genetic Architecture of Human Disease using Genome Wide Association Studies: Benefits and Challenges*** was published in the West Bengal State University, Fifth Regional Science and Technology Congress, Region Three from Scholars' Book Hub (ISBN: 978-81-959389-7-1) ◆ Received a travel award from the CSIR, Govt. of India to attend the 25th Biennial Meeting International Society for Eye Research, Gold Coast, Queensland, Australia from 19th February to 23rd February 2023.
Ms. Shamita Sanga	Received a travel award from the DBT, Govt. of India to attend the European Society of Human Genetics Conference (ESHG), Vienna, Austria during June 11-14, 2022.
Arunima Acharya	Received the second best poster award for presentation at the 47th Annual Conference of the Indian Society of Human Genetics (ISHG) and International Symposium on "New Genetics and its Contributions to Human Health and Wealth" from January 23-25,2023.
Esha Bhattacharjee	Received the Young Scientist Award for presentation at the 47th Annual Conference of the Indian Society of Human Genetics (ISHG) and International Symposium on "New Genetics and its Contributions to Human Health and Wealth" from January 23-25,2023.
Dr. Sillarine Kurkalang	Received the best poster award at 91st Annual meeting of Society of Biological Chemists (India), 8th-11th December,2022.

Name of the Member	Description
Mousumi Sarkar	Received the “Outstanding Paper Award” by 5th Regional Science & Technology Congress, Dept of Science Technology and Biotechnology, Govt. of west Bengal, held on 17th-18th January 2023 at Cooch Behar Panchanan Barma University.
Ankita Maddheshiya	Received the best poster presentation award in the conference on “HUMAN MICROBIOME IN HEALTH AND DISEASE” (HMCW_2023) on 15th February 2023 at THSTI, Faridabad.
Paromita Mitra	Awarded with DBT CTEP travel grant from DBT to attend Cell Symposia: Hallmarks of Cancer, San Diego, USA on 31st October 2022 and make poster presentation titled ‘TGF induced Tie2 expression in cancer associated fibroblasts remodels tumor microenvironment to support stemness in oral cancer cells’
Pratyusha Chikkala	Won second best poster award at the conference of Oncopathology updates- February 25-26, 2023 organized by AIIMS, Kalyani

PhD Degrees Awarded

Bishnupriya Chhatriya
PI: Srikanta Goswami

Shrayashi Biswas
PI: Samsiddhi Bhattacharjee

Debashree Tagore
PI: Analabha Basu

Kavya Vipparthi
PI: Sandeep Singh

HUMAN RESOURCE, RESEARCH AND TRAINING INFRASTRUCTURE

Brief Profiles of Academic Members

Faculty members

Sl No.	Name	Designation
1	DR. SAGAR SENGUPTA	DIRECTOR (ON DEPUTATION)
2	PROF. ARINDAM MAITRA	ASSOCIATE DIRECTOR
3	PROF. ANUPAM BASU	ASSOCIATE DIRECTOR (ON LIEN)
4	DR. SREEDHAR R. CHINNASWAMY	ASSOCIATE PROFESSOR
5	DR. BHASWATI PANDIT	ASSOCIATE PROFESSOR
6	DR. SAROJ K. MAHAPATRA	ASSOCIATE PROFESSOR
7	DR. SRIKANTA GOSWAMI	ASSOCIATE PROFESSOR
8	DR. PRIYADARSHI BASU	ASSOCIATE PROFESSOR
9	PROF. KARTIKI V. DESAI	PROFESSOR
10	DR. SAMSIDDHI BHATTACHARYA	ASSOCIATE PROFESSOR
11	DR. MOULINATH ACHARYA	ASSOCIATE PROFESSOR
12	DR. SANDEEP SINGH	ASSOCIATE PROFESSOR
12	DR. ANALABHA BASU	ASSOCIATE PROFESSOR
13	DR. NIDHAN K. BISWAS	ASSOCIATE PROFESSOR
15	DR. SOUVIK MUKHERJEE	ASSISTANT PROFESSOR
16	DR. ARVIND KORWAR	ASSISTANT PROFESSOR
17	DR. PONGALI B. RAGHAVENDRA	ASSISTANT PROFESSOR

Sl No.	Name	Designation
1.	PROF. PARTHA P. MAJUMDER	DISTINGUISHED PROFESSOR
2.	PROF. SHARMILA SENGUPTA	EMINENT SCIENTISTS

Brief Profiles of Technical Members

Sl No.	Name	Designation
1	Mr. Bikram Roy	Systems Analyst
2	Dr. Subrata Patra	Senior Technical Officer
3	Mr. Indranil Bagchi	Senior Laboratory Technician
4	Dr. Ranjan Dhar	Senior Laboratory Technician
5	Mr. Surajit Mahapatra	Senior Laboratory Technician

Brief Profiles of Administrative Members

Sl No.	Name	Designation
1	Rajeev Kumar	Senior Manager (Administration and Finance) (On lien to C-Dot)
2	Nabarun Mukherjee	Manager (Administration)
3	Debasish Kumar Mandal	Manager (Finance)
4	Sujata Das	Staff Officer to Director
5	Supriyo Chatterjee	Section Officer (Administration)
6	Ayan Majumder	Section Officer (Academic) (on deputation to CNCI)
7	Arindam Majumdar	Section Officer (Finance)
8	Sandeep B. Mukherjee	Section Officer
9	Tamoghno Chatterjee	Management Assistant (Academic)
10	Sujit Halder	Management Assistant (Administration)
11	Tapan Sikder	Junior Management Assistant (Administration)
12	Sayantony Nandi	Junior Management Assistant (Administration)

Integrated Ms-PhD (Aug 22 session)

S/N	FELLOW NAME	DOJ
1	Aniket Kumar	17-08-2022
2	Arun Kumar Mishra	17-08-2022
3	Bhagyashree Pradhan	17-08-2022
4	Dhanashree Ajay Khedekar	17-08-2022
5	Gautam Kumar	17-08-2022
6	Mourya Mondal	17-08-2022

S/N	FELLOW NAME	DOJ
7	Paresh Arvind Patil	22-08-2022
8	Pritika Kwatra	17-08-2022
9	Rishika Maji	17-08-2022
10	Sai Gayathri R	16-08-2022
11	Shreyasi Ghosh	17-08-2022
12	Sneha Saha	17-08-2022

SUMMER TRAINEES

S/N	Name	Guide	Affiliation
1	Ms. Kaushani Ghosh	Dr. Srikanta Goswami	Calcutta University
2	Ms. Dishari Banerjee	Dr. Souvik Mukherjee	Kalyani University
4	Ms. Srijita Maity	Dr. Bhaswati Pandit	Calcutta University
5	Ms. Rohini Mukherjee	Dr. Paramita Bhattacharya	Calcutta University
6	Siddharth Bhardwaj	Prof. Kartiki.V. Desai	Guru Ghasidas Vishwavidyalaya
7	Saswata Naha	Dr. Samsiddhi Bhattacharjee	ISI,Kolkata
8	Utsab Bhattacharyaa	Dr. Mahua Mallick	Kalyani University
9	Ayesa Juleka	Prof. Anupam Basu	Kalyani University
10	Prasoon Kr. Mishra	Dr. Nidhan K. Biswas	IIT,Bombay
11	Sankhadip Das	Dr. Moulinath Acharya	Kalyani University
12	Riddhi Chakraborty(INSA)	Dr. Arvind Korwar	St.Xavier's College
13	Shreya Baisakhiya (INSA)	Dr. Nidhan K. Biswas	National Institute of Technology,Rourkela
14	Rakesh Kumar Patail	Dr. Sreedhar Chinnaswamy	Guru Ghasidas Vishwavidyalaya
15	Deepashree Chanda	Dr. Priyadarshi Basu	Calcutta University
16	Mr K Akash (INSA)	Dr. Sandeep Singh	Shoolini University
17	Ajay Mallik (INSA)	Prof. Arindam Maitra	Savitribai Phule Pune University
18	Sreemoyee Saha	Dr. Moulinath Acharya	Calcutta University
19	Sagnik Dasgupta	Dr. Arvind Korwar	MAKAUT
20	Abhirup Ghosh	Dr. Arvind Korwar	VIT University
21	Anwesha Maity	Prof. Kartiki.V. Desai	VIT University
22	Arpan Narayan Roy	Dr. P.B.Raghavendra	IISER Kolkata
23	Ananya Paul	Dr. Srikanta Goswami	VIT University
24	Sudhangshu Shekhar Dash	Dr. Saroj K. Mohapatra	Siksha O Anusandhan,Bhubaneswar
25	Poulami Das	Dr.Souvik Mukherjee	MAKAUT
26	Sayani Bagchi	Dr. Moulinath Acharya	MAKAUT
27	Hriday Home Chowdhury	Dr.Nidhan Kr. Biswas	North Bengal University
28	Subhasree Sahu	Arindam Maitra	Central University South Bihar
29	Nasrina Parvin	Srikanta Goswami	MAKAUT
30	Vasudha Singh	Dr. Moulinath Acharya	St. Thomas College
31	Dilpreet Kaur Sawhney	Dr. Mahua Maulik	Navracha Azad University
32	Mansi Patel	Dr. Samsiddhi Bhattacharjee	Guzarat University

S/N	Name	Guide	Affiliation
33	Abhishek Kumar	Prof. Arindam Maitra	Central University South Bihar
34	Ankan Ghosh	Dr. Bhaswati pandit	VIT University
35	Kamal Lochan Sharma	Dr. P.B.Raghavendra	Bodoland University
36	Sunit Chakraborty	Dr. Anup Majumdar	MAKAUT
37	Sanjana Banerjee	Dr. Sandeep .Singh	St.Xavier's College
38	Payel Das	Dr. Bhaswati pandit	Techno India Univerrsity
39	Adrija Saha	Dr. Souvik Mukherjee	JIS Institute of Advanced Studies and Research
40	Misba Ahmmed	Dr. Priyadarshi Basu	MAKAUT
41	Trisha Chakraborty	Dr. Sandeep Singh	MAKAUT
42	Sreshtha Ghosh	Dr. Bhaswati Pandit	Amity University
43	Rwik Suvro Roy	Dr. Priyadarshi Basu	St.Xavier's College
44	Kirti Kashyap	Prof. Kartiki.V.Desai	Guru Ghasidas Vishwavidyalaya

हिन्दी प्रकोष्ठ, राष्ट्रीय जैव चिकित्सा जीनोमिक्स संस्थान (ऐन.आई.बी.एम.जी.), कल्याणी के कार्यक्रम

राष्ट्रीय जैव चिकित्सा जीनोमिक्स संस्थान (ऐन.आई.बी.एम.जी.), कल्याणी के हिन्दी प्रकोष्ठ की बैठक वर्ष 2021-22 के दौरान नियमित अंतराल पर आयोजित के गई थी। साथ ही साथ नगर राजभाषा कार्यान्वयन समिति, कोलकाता (कार्यालय-2) के सदस्य के रूप में, ऐन.आई.बी.एम.जी. ने इस संगठन द्वारा आयोजित सभी बैठकों में साकिर्य भाग लिया तथा सदस्यता के लिए अपना वार्षिक योगदान विधिवत प्रदान किया। पिछले सभी वर्षों की तरह इस वर्ष भी संस्था में हिंदी दिवस कार्यक्रम धूमधाम से मनाया गया। वर्ष 2022 के लिए ऐन.आई.बी.एम.जी. की हिन्दी दिवस का कार्यक्रम 14.09.2022 आयोजन किया गया था। एनआईबीएमजी के छात्रों और कर्मचारियों ने हिंदी कविता पाठ, प्रश्नोत्तरी प्रतियोगिता, अंताक्षरी, गायन और नृत्य और अन्य सांस्कृतिक कार्यक्रमों के साथ कार्यक्रम को सजाया। संस्था के अनुशंधानरत छात्रों के द्वारा हिंदी में थीसिस की लघु प्रस्तुति इस कार्यक्रम का मुख्य आकर्षण बनी | कार्यक्रम के शुरुआत माननीय निदेशक महोदय, प्रोफ. अरिंदम मित्र के स्वागत भाषण से हुआ | निदेशक महोदय ने संस्था के सभी सदस्यों को सरल एवं सुबोध हिंदी भाषा को अपने मूल कार्यों में इस्तेमाल करने के लिए प्रेरित किया |

विभिन्न प्रतियोगिताएं में विजेताओं के नाम:

3 मिनट की थीसिस	प्रश्नोत्तरी प्रतियोगिता	अंताक्षरी प्रतियोगिता	टैग- ईट	हिंदी खजाने की खोज
प्रथम स्थान: सीमा भारतीय दूसरा स्थान: पारोमिता मित्रा तीसरा स्थान: ज्योतिषमान सरमा	प्रथम स्थान टीम: ज्योतिषमान सरमा, आदित्य अग्रवाल, उदय साहा दूसरे स्थान पर रहने वाली टीम: डॉ. मौलीनाथ आचार्य, डॉ. समसिद्दी भट्टाचार्जी, सुदीप्तो चक्रवर्ती सह-दूसरे स्थान पर रहने वाली टीम: अरित्रा गुप्ता, तहसीन अहमद, रूपायन मुखर्जी	प्रथम स्थान टीम: चित्रार्पिता दास, चंद्रिका भट्टाचार्य, वत्सल पटेल दूसरे स्थान पर रहने वाली टीम: सुवामिता राउत, पियाली मंडल, रूपायन मुखर्जी	विजेता- अर्घ्य डे	हिंदी खजाने की खोज: विजेता टीम: सीमा भारतीय, अनुराधा गौतम

हिंदी प्रकोष्ठ के अध्यक्ष डॉ. संदीप सिंह ने एन.आई.बी.एम.जी सदस्यों की सक्रिय भागीदारी और इस कार्यक्रम को एक बड़ी सफलता बनाने के लिए सभी को धन्यवाद दिया। कार्यक्रम के अंत में जलपान की व्यवस्था की गई| विजेताओं को पुरष्कार आगामी इंस्टिट्यूट डे के अवसर माननीय निदेशक महोदय द्वारा प्रदान की गयी।

फेसबुक पर प्रकाशित पोस्ट से एक तस्वीर:

COMMITTEES OF THE INSTITUTE

GOVERNING BOARD MEMBERS

NAME	ROLE	AFFILIATION
Dr. Rajesh S. Gokhale	Chairperson	Secretary, DBT, Delhi
Shri Chaitanya Murti	Ex-Officio Member	Jt. Secretary (Adm), DBT,Delhi
Shri Vishvajit Sahay	Ex-Officio Member	Financial Adviser, DBT, Delhi
Prof Arindam Maitra	Ex-Officio Member	Director-In-Charge, NIBMG
Dr. Sandhya Shenoy	Ex-Officio Member	Scientist F, DBT, Delhi
Shri Rajeev Kumar	Ex-Officio Member	Sr Mgr (Adm & Fin), NIBMG
Dr. Anup Anvikar	Nominated Member	Director, National Institute of Biologicals, New Delhi
Dr. Sudeep Gupta	Nominated Member	Director, ACTREC, Mumbai
Dr. Giriraj Ratan Chandak	Nominated Member	Chief Scientist, CSIR-CCMB, Hyderabad
Dr. Debasish Bandyopadhyay	Nominated Member	Professor, University of Calcutta, Kolkata

INTERNATIONAL ADVISORY COMMITTEE

Dr. Aravinda Chakravarti	Director of the Center for Human Genetics & Genomics at New York University
Prof Takashi Gojobori	Vice-Director of the National Institute of Genetics and Distinguished Professor at Center for Information Biology and DNA Data Bank of Japan in NIG, Mishima, Japan
Prof David J Lipmanc	Former Director of the National Center for Biotechnology Information at the National Institutes of Health
Prof Edison T Liu	Former President and CEO of The Jackson Laboratory, and the former director of its NCI-designated Cancer Center
Prof G Padmanaban	Former Director of the Indian Institute of Science, and presently serves as honorary professor in the department of biochemistry at IISc and Chancellor of Central University of Tamil Nadu
Prof Terry P Speed	Senior Principal Research Scientist at the Walter and Eliza Hall Institute of Medical Research, University of California, Berkeley
Prof Michael Stratton	British Clinical Scientist and theDirector of the Wellcome Trust Sanger Institute, UK
Prof Huan-Ming Yang	Chairman and co-founder of the Beijing Genomics Institute, China

SCIENTIFIC ADVISORY COMMITTEE

NAME	ROLE	AFFILIATION
Dr. Anurag Agarwal	Chairperson	Dean, Ashoka University, Delhi
Dr. Sandhya Shenoy	Ex-Officio Member	Scientist F, DBT, Delhi
Prof. Arindam Maitra	Member Secretary	Director-In-Charge, NIBMG
Dr. Anup Anvikar	Nominated Member	Director, National Institute of Biologicals, New Delhi
Dr. Sudeep Gupta	Nominated Member	Director, ACTREC, Mumbai
Dr. Giriraj Ratan Chandak	Nominated Member	Chief Scientist, CSIRCCMB, Hyderabad
Dr. Debasish Bandyopadhyay	Nominated Member	Professor, University of Calcutta, Kolkata
Dr. Arun Bandopadhyay	Nominated Member	Director, CSIR, IICB, Kolkata
Professor Ramakrishna Ramaswamy	Nominated Member	Professor, IIT, Delhi
Professor K. Natarajan	Nominated Member	Dr. B Ambedkar Centre for Biomedical Research (ACBR), University of Delhi
Dr. Shiv Pillai	Nominated Member	Harvard Medical School, Boston

FINANCE COMMITTEE MEMBERS

NAME	ROLE	AFFILIATION
Shri Vishvajit Sahay	Chairperson	Financial Adviser, DBT, Delhi
Dr. Suchita Ninawe	Ex-Officio Member	Scientist G, DBT, Delhi
Prof Arindam Maitra	Ex-Officio Member	Director-In-Charge, NIBMG
Shri Rajeev Kumar	Ex-Officio Member	Sr Mgr (Adm & Fin), NIBMG
Dr. Sudhanshu Vrati	Nominated Member	Executive Director, RCB, Faridabad
Shri J P S Chawla	Nominated Member	Former Controller General of Accounts, Min of Finance, Delhi
Ms Neeru Abrol	Nominated Member	Chairman & Managing Director, National Fertilizers Ltd, New Delhi

SOCIETY MEMBERS

NAME	ROLE	AFFILIATION
Dr. Jitender Singh	President	Minister of Science & Technology, Delhi
Dr. Ratna De Nag	Ex-Officio Member	Min-in-charge, DBT, West Bengal
Dr. Rajesh S. Gokhale	Ex-Officio Member	Secretary, DBT, Delhi
Dr. Balram Bhargava	Ex-Officio Member	Secretary, Dept of Health Research, Delhi
Dr. Shekhar C Mande	Ex-Officio Member	Secretary, DSIR and Director General, Council of Scientific & Industrial Research, Delhi
Shri Anil Verma, IAS	Ex-Officio Member	Principal Secretary-in-charge, DBT, West Bengal
Shri S N Narain	Ex-Officio Member	Deputy Secretary (Adm), DBT, Delhi
Shri Vishvajit Sahay	Ex-Officio Member	Financial Adviser, DBT, Delhi
Dr. Sagar Sengupta	Member Secretary	Director, NIBMG
Prof S Chandrasekhar	Nominated Member	Director, CSIR-Indian Institute of Chemical Technology, Hyderabad
Prof Ramakrishna Vijayacharya Hosur	Nominated Member	Distinguished Visiting Professor, IIT, Mumbai
Dr. B Ravindran	Nominated Member	Emeritus Professor, ILS, Bhubaneswar
Dr. Sib Sankar Roy	Nominated Member	Sr Principal Scientist, CSIR, IICB, Kolkata
Professor Basuthkar Jagadeeswar Rao	Nominated Member	VC, University of Hyderabad
Shri Binish Chudgar	Nominated Member	Vice Chairman & Managing Director, Intas Pharmaceuticals, Ahmedabad

ANTI RAGGING CUM STUDENTS DISCIPLINARY COMMITTEE

NAME	ROLE	AFFILIATION
Dr. Kartiki V. Desai	Chairperson	Professor, NIBMG
Dr. Analabha Basu	Member	Associate Professor, NIBMG
Dr. Moulinath Acharya	Member	Associate Professor, NIBMG
Shri Nabarun Mukherjee	Member	Manager (Administration), NIBMG
Shri Ayan Majumder	Member - Convener	Section Officer (Academic)

INSTITUTE HOSTEL WARDENS

NAME	ROLE	AFFILIATION
Dr. Sandeep Singh	Chairperson	Associate Professor, NIBMG
Dr. Moulinath Acharya	Member	Associate Professor, NIBMG
Dr. Anup Mazumder	Member	NIBMG Fellow
Mr. Supriyo Chatterjee	Member	Convenor Section Officer (Administration

INSTITUTE HINDI CELL MEMBERS

NAME	ROLE	AFFILIATION
Dr. Sandeep Singh	Chairperson	Associate Professor, NIBMG
Dr. Kartiki V. Desai	Member	Professor, NIBMG
Ms. Sujata Das	Member	Staff Officer to Director, NIBMG
Mr. Supriyo Chatterjee	Convener	Section Officer (Administration)

NIBMG BIOSAFETY COMMITTEE

NAME	ROLE	AFFILIATION
Dr. Sreedhar Chinnaswamy	Chairperson	Associate Professor, NIBMG
Dr. Mamata Chawla-Sarkar	DBT Nominee	NICED, Kolkata
Dr. Bhaswati Pandit	Member Secretary	Associate Professor, NIBMG
Dr. Joyoti Basu	External Expert	Bose Institute, Kolkata
Dr. Provash Sadhukhan	External Expert	NICED, Kolkata
Dr. Suvendra N. Bhattacharyya	External Expert	IICB, Kolkata
Dr. K K Majumder	Biosafety Officer	AMA, Doctor, Kalyani
Dr. Moulinath Acharya	Internal Member	Associate Professor, NIBMG
Dr. Sandeep Singh	Internal Member	Associate Professor, NIBMG
Dr. Saroj Kant Mohapatra	Internal Member	Associate Professor, NIBMG

NIBMG ETHICS COMMITTEE

NAME	ROLE	AFFILIATION
Professor Saumitra Das	Chairperson	Professor, IISc, Bangalore
Professor Susanta Roychoudhury	Vice-Chairperson	Professor, IICB, Kolkata
Professor Mitali Chatterjee	Member	Professor, IPGMER, Kolkata
Dr. Mandip Paul	Member	Professor, College of Medicine, JNM Hospital, Kalyani
Mrs Subhra Sinha	Member	Headmistress (Retired), Spring Dale High School (H.S), Kalyani
Maharaj Sarveswarananda Puri	Member	Revered President Maharaj, Matri Mission O Mandir, Kalyani
Mr. Rajib Ckahkaborty	Member	Advocate, Kalyani Court
Professor Arindam Maitra	Member	Associate Director, NIBMG
Dr. P B Raghavendra	Member	Assistant Professor, NIBMG
Professor Kartiki V Desai	Member-Secretary	Professor, NIBMG

INTERNAL COMPLAINTS COMMITTEE

NAME	ROLE	AFFILIATION
Prof Kartiki Desai	Chairperson	Professor, NIBMG
Dr. Bhaswati Pandit	Member	Associate Professor, NIBMG
Shri Supriya Basu	Extrernal Expert	
Smt. Sujata Das	Member	Staff Officer to Director
Shri Ayan Majumder	Member Secretary	Section Officer (Academic)

INSTITUTE PURCHASE COMMITTEE

1. CONSUMABLE PURCHASE COMMITTEE

(a) Professor Analabha Basu – Chairperson
(b) Dr. Sreedhar Chinnaswamy – Co -Chairperson
(c) Dr. Arvind M. Korwar – Member
(d) Mr. Nabarun Mukherjee – Member
(e) Mr. Debasish Kumar Mandal - – Member
(f) Mr. Sandeep B. Mukherjee – Convenor

2. EQUIPMENT PURCHASE COMMITTEE

(a) Professor Anupam Basu – Chairperson
(b) Dr. Saroj Kant Mohapatra – Co -Chairperson
(c) Dr. Sandeep Singh – Member
(d) Mr. Nabarun Mukherjee – Member
(e) Mr. Debasish Kumar Mandal - – Member
(f) Mr. Sandeep B. Mukherjee – Convenor

3. INFRASTRUCTURE PURCHASE COMMITTEE

(a) Professor Arindam Maitra – Chairperson
(b) Dr. Srikanta Goswami – Co -Chairperson
(c) Dr. Moulinath Acharya – Member
(d) Mr. Nabarun Mukherjee – Member
(e) Mr. Debasish Kumar Mandal - – Member
(f) Mr. Sandeep B. Mukherjee – Convenor

ANNUAL REPORT RELATING TO RTI

Annual Report relating to RTI Right to Information Act 2005 mandates timely response to citizen requests for government information. It is an initiative taken by Department of Personnel and Training, Ministry of Personnel, Public Grievances and Pensions to provide a– RTI Portal Gateway to the citizens for quick search of information on the details of first Appellate Authorities, PIOs etc. amongst others, besides access to RTI related information / disclosures published on the web by various Public Authorities under the government of India as well as the State Governments.

The basic object of the Right to Information Act is to empower the citizens, promote transparency and accountability in the working of the Government, contain corruption, and make our democracy work for the people in real sense. It goes without saying that an informed citizen is better equipped to keep necessary vigil on the instruments of governance and make the government more accountable to the governed. The Act is a big step towards making the citizens informed about the activities of the Government.

RTI Act has been made by legislation of Parliament of India on 15th June 2005. The Act came into effect on 12th October 2005 and has been implemented ever since to provide information to crores of Indian citizens. All the constitutional authorities come under this Act, making it one of the most powerful laws of the country.

From 31st March 2017, RTI-MIS (Right to Information Request & Appeal Management Information System) portal has been implemented at NIBMG. From 1st April 2022 to 31st March 2023 NIBMG has received total 14 nos. All 14 nos. of RTI have been replied. This RTI-MIS portal is used by Indian Citizens for all Ministries/Departments and few other Public Authorities of Central Government to file RTI applications/first appeals online along with payment gateway. Payment is made through internet banking of SBI & its associate banks, debit/ credit cards of Master/Visa and RuPay cards.

PROGRESS OF CAMPUS CONSTRUCTION OF NIBMG

STATUS OF CONSTRUCTION ACTIVITIES FROM APRIL 2022 TO 2023

1. **Main Building:** Functional.
2. **Solar Power:** Installed and fully operational.
3. **ATM Building:** Functional
4. **Car Parking:** Construction of approx. 20,000 Sq. ft. light vehicle parking stand competed with provision for parking of 25 nos. of Car.

National Institute of Biomedical Genomics

P.O: N.S.S, District – Nadia, Kalyani, Pincode – 741251

AUDITED FINANCIAL STATEMENTS

FOR THE FINANCIAL YEAR – 2022-23

S. GUHA & ASSOCIATES
Chartered Accountants

Head Office:

16/1, Girish Vidya Ratna
Lane, Kolkata-700009
Tel.Nos.033-23506991/23609686

E_mail:sguhaassociates@gmail.com

National Institute of Biomedical Genomics

BALANCE SHEET AS AT 31st MARCH 2023

FUNDS & LIABILITIES	Schedule	Current Year	Previous Year
		₹	₹
CAPITAL FUND	1	(12,97,716.27)	2,72,30,910.38
RESERVE & SURPLUS	2	-	-
EARMARKED/ENDOWMENT FUNDS	3	56,77,46,241.29	85,41,22,073.53
SECURED LOANS AND BORROWINGS	4	-	-
UNSECURED LOANS AND BORROWINGS	5	-	-
DEFERRED CREDIT LIABILITIES	6	1,63,01,28,103.84	1,71,15,74,750.84
CURRENT LIABILITIES AND PROVISIONS	7	12,13,34,222.00	9,41,67,582.64
TOTAL		***2,31,79,10,850.86***	***2,68,70,95,317.39***

ASSETS	Schedule	Current Year	Previous Year
		₹	₹
FIXED ASSETS	8	1,63,01,28,103.84	1,71,15,74,750.84
INVESTMENTS-FROM EARMARKED/ENDOWMENT FUND	9	-	-
INVESTMENTS - OTHERS	10	-	-
CURRENT ASSETS, LOANS, ADVANCES ETC	11	68,77,82,747.02	97,55,20,566.55
MISCELLANEOUS EXPENDITURE (to the extent not written off or adjusted)		-	-
TOTAL		***2,31,79,10,850.86***	***2,68,70,95,317.39***

SIGNIFICANT ACCOUNTING POLICIES	24	
CONTINGENT LIABILITIES AND NOTES ON ACCOUNTS	25	

For Natioanl Institute of Biomedical Genomics

Director *Manager [Finance]*

डॉ. सागर सेनगुप्ता / Dr. Sagar Sengupta
निदेशक / Director
राष्ट्रीय जैवचिकित्सा जीनोमिक्स संस्थान
National Institute Of Biomedical Genomics
पो.आ.: एन.एस.एस., कल्याणी, पिन-७४१२५१, जिला-नदीया (प.बं.)
P.O.: N.S.S., Kalyani, Pin-741251, Dist. Nadia (W.B.)
भारत / India

Place : Kalyani

Date :

Signed in terms of our separate Report of even date.

FOR S. GUHA & ASSOCIATES
Chartered Accountants
Firm Registration No. 322493E

For S. GUHA & ASSOCIATES
Chartered Accountants
Sourabh Mitra
Partner

Sourabh Mitra
Partner
Membership No : 308743
UDIN No.: 23308743BGULXT4886

National Institute of Biomedical Genomics

INCOME & EXPENDITURE ACCOUNT FOR THE YEAR ENDED 31.03.2023

INCOME	Schedule	Current Year	Previous Year
		₹	₹
Income from Sales/Services	12	-	-
Grants/Subsidies	13	26,00,00,000.00	19,46,04,993.00
Fees/Subscriptions	14	5,17,000.00	89,000.00
Income from Investments	15	-	-
Income from Royalty, Publication etc.	16	-	-
Interest Earned	17	-	-
Other Income	18	19,87,88,311.00	4,16,16,145.00
Increase/(decrease) in stock of finished goods & W.I.P	19	-	-
TOTAL		***45,93,05,311.00***	***23,63,10,138.00***

EXPENDITURE	Schedule	Current Year	Previous Year
		₹	₹
Establishment Expenses	20	12,05,41,313.00	9,32,89,380.00
Other Administrative Expenses etc.	21	14,21,07,481.65	11,30,72,037.00
Expenditure on Grants, Subsidies etc.	22	-	-
Interest	23	-	-
Depreciation	8	19,57,11,676.00	4,00,21,680.00
TOTAL		***45,83,60,470.65***	***24,63,83,097.00***
Balance being excess/(shortfall) of Income over Expenditure		9,44,840.35	(1,00,72,959.00)
Prior period adjustments		(22,79,438.00)	(17,53,890.00)
Transfer to/from Capital Fund		-	-
Balance being Surplus/(Deficit) Carried to Corpus/Capital Fund		***(13,34,597.65)***	***(1,18,26,849.00)***

SIGNIFICANT ACCOUNTING POLICIES	24	
CONTINGENT LIABILITIES AND NOTES ON ACCOUNTS	25	

For Natioanl Institute of Biomedical Genomics

Director *Manager [Finance]*

डॉ. सागर सेनगुप्ता / Dr. Sagar Sengupta
निदेशक / Director
राष्ट्रीय जैवचिकित्सा जीनोमिक्स संस्थान
National Institute Of Biomedical Genomics
पो.आ.: एन.एस.एस., कल्याणी, पिन-७४१२५१, जिला-नदीया (प.बं.)
P.O.: N.S.S., Kalyani, Pin-741251, Dist. Nadia (W.B.)
भारत / India

Place : Kalyani

Date :

Signed in terms of our separate Report of even date.

FOR S. GUHA & ASSOCIATES
Chartered Accountants
Firm Registration No. 322493E

For S. GUHA & ASSOCIATES
Chartered Accountants
Sourabh Mitra
Partner

Sourabh Mitra
Partner
Membership No : 308743
UDIN No.: 23308743BGULXT4886

National Institute of Biomedical Genomics

Receipts & Payments Account for the Year ending 31st March 2023

As on 31.03.2022	RECEIPTS:	As on 31.03.2023		As on 31.03.2022	PAYMENTS:	As on 31.03.2023	
		Amount in ₹	Amount in ₹			Amount in ₹	Amount in ₹
	Opening Balance:				Expenses		
17,240.00	Cash in Hand	19,210.00		9,32,89,380.00	Establishment Expenses	12,05,41,313.00	
				11,30,36,637.00	Other Administrative Expenses	14,19,11,069.55	26,24,52,382.55
	Cash at Bank:						
66,45,578.58	In Current Accounts	6,08,76,139.98			Payment made against funds for various projects		
26,60,44,335.00	In Deposit Accounts	20,60,44,335.00		47,70,26,034.86	Extramural Projects	92,54,54,712.95	
76,81,12,154.82	In Savings Accounts	68,42,98,445.53	95,12,38,130.51	1,25,75,624.00	Fellowship	1,25,39,667.00	
				3,67,351.00	Extramural -Project (Foreign)		
				6,70,24,706.48	National Genomics Core		
				2,17,800.00	Extramural Seminar		93,79,94,379.95
	Grant Received :				Refund of Govt Grant to DBT		2,29,29,000.00
	From Govt. of India [DBT]:				Investments & deposits made		
11,25,00,000.00	Non Recurring Grant	11,00,00,000.00		-	Out of Earmarked/Endowment Funds	-	
19,46,04,993.00	Recurring Grant	26,00,00,000.00	37,00,00,000.00	-	Out of Own Funds [other Investments]		-
0.00	Extramural Seminar				Expenditure on Fixed Assets & CWIP		
32,68,37,818.08	for Extramural Projects [Net of TDS]	56,11,48,260.63		6,42,06,292.00	Purchase of Fixed Assets	10,40,24,493.00	
1,16,70,573.00	for Fellowship	1,24,66,109.00		-	Expenditure on Building	1,02,31,036.00	
25,57,989.00	Extramural -Project (Foreign)			2,13,92,680.00	Expenditure on Capital Work in Progress	-	11,42,55,529.00
5,85,06,400.68	National Genomics Core		57,36,14,369.63				
					Refund of surplus money/Loans		
			-	27,70,000.00	To the Govt. of India		
	Interest Received:			-	To the State Government	-	
5,05,214.24	Interest on Refund of Income Tax	1,71,293.00		-	To the other providers of Funds	-	
76178	On Saving account	3,41,359.00				-	-
3,89,97,113.30	On Depost Accounts [Net of TDS]	4,42,30,946.70	4,47,43,598.70		Other Payment :		
				1,81,19,312.00	Tax Deducted at Source [TDS]	1,99,94,125.00	
				62,77,047.00	Statutory Deduction from Employees	64,03,391.00	
				2,68,971.00	Statutory Deduction from Contractors	78,827.00	
98,38,971.00	Accured Interest receipt			3,53,268.00	Security Deposits [Refund & with Organisations]	37,24,165.00	
62,96,092.76	Tax Deducted from Grant		9,86,170.00	13,14,000.00	Refund of Earnest Money Deposit	29,20,800.00	
6,01,500.00	Advance amount Received			46,14,711.00	Advance to Parties	2,91,62,045.40	
	TDS on FDR		11,51,619.00	11,34,301.00	Advance to Staffs including LTC	51,07,575.00	
				36,46,359.00	GST & GST TDS	84,62,383.00	7,58,53,311.40

As on 31.03.2022	RECEIPTS:	As on 31.03.2023		As on 31.03.2022	PAYMENTS:	As on 31.03.2023	
		Amount in ₹	Amount in ₹			Amount in ₹	Amount in ₹
				1,53,795.00	Policy Premium Paid		
			-		-		
			-	14,400.00	Payment to Nabarun Mukherjee		
	Other Receipt :			20,84,189.00	Outstanding Expenses Paid	35,400.00	35,400.00
3,40,362.00	Misc Receipts		10,42,244.00	35,73,645.72	TDS on FDR	17,19,966.00	17,19,966.00
1,77,56,815.00	Tax Deducted at Source [TDS]		2,03,22,874.00		Interest Paid to Project & Overhead Researve		-
62,76,547.00	Statutory Deduction from Employees		67,25,551.00	5,05,214.24	Interest on Refund of Income Tax		-
1,66,498.00	Statutory Deduction from Contractors		91,965.00	76,178.00	On Saving account	39,777.00	
21,30,356.00	Security Deposits from Contractors		3,48,91,091.00	3,89,97,113.30	On Depost Accounts [Net of TDS]	27,29,682.00	27,69,459.00
37,74,831.00	Earnest Money Deposit from Contractors		11,23,058.00	17,53,890.00	Income Tax (Prior Priod)		
35,68,474.00	Advance recovered from Parties		2,83,82,044.82	2,30,071.00	Payment of Security Deposit (Assets)		5,80,228.00
47,24,004.00	GST & GST TDS		98,87,449.00		Advance to parties (NGC)		3,41,80,700.00
14,53,772.00	Advance recovered from Staffs including LTC		44,14,618.00		Tax Deducted from Grant		4,12,119.16
85,000.00	Fees from student [Tuition Fees]		5,17,000.00				
12,54,103.00	Hostel Charges		11,68,631.00				
96,795.00	Policy premium receiptc						
	Receipts from Security deposit assets		23,000.00		Closing Balance:		
1,14,500.00	Employees contribution to Staff Welfare Fund		1,12,400.00	19,210.00	Cash in Hand	21,377.00	
4,07,02,914.65	Hostel Development & Inst Overhead Researve		1,60,12,366.00		Cash at Bank:		
			-	6,08,76,139.98	On Current Accounts	33,70,218.48	
	Received for gratuity and leave encashment		2,72,40,470.00	20,60,44,335.00	On Deposit Accounts	23,62,28,784.00	
	Advance received from parties (NGC)		2,93,33,684.00	68,42,98,445.53	On Savings Accounts	35,95,76,134.12	
	Electric charges recovered		92,967.00		On CNA account	7,07,36,312.00	66,99,32,825.60
			-				
1,88,62,61,123.11			2,12,31,15,300.66	1,88,62,61,123.11			2,12,31,15,300.66

Signed in terms of our separate Report of even date.

For Natioanl Institute of Biomedical Genomics

डॉ. सागर सेनगुप्ता / Dr. Sagar Sengupta
निदेशक / Director
राष्ट्रीय जैवचिकित्सा जीनोमिक्स संस्थान
National Institute Of Biomedical Genomics
पो. ओ : एन.एस.एस., कल्याणी, पिन-७४१२५१, जिला-नदीया,(पश्चिम बंगाल)
P.O. : N.S.S., Kalyani, Pin-741251, Dist. Nadia,(W.B.)

Director

Sr. Mr Manager (Finance)

Place : Kalyani
Date : 26.07.2023

FOR S. GUHA & ASSOCIATES
Chartered Accountants
Firm Registration No : 322483E
Sourabh Mitra
Partner
Membership No : 308743
UDIN No.: 23308743BGULXT4886

For S. GUHA & ASSOCIATES
Chartered Accountants
Partner

National Institute of Biomedical Genomics

Schedules forming part of Balance Sheet as at 31.03.2023

SCHEDULE 1: CAPITAL FUND	Current Year		Previous Year	
	₹	₹	₹	₹
A. Non Recurring Grant in Aid				
Balance as at the beginning of the year	4,64,52,232.00		1,95,51,204.00	
Add : Contributions towards Corpus/Capital Fund	11,00,00,000.00		11,25,00,000.00	
		15,64,52,232.00		13,20,51,204.00
Less: Refund Government grant to DBT	2,29,29,000.00		-	
Less: Restated to deffered govt Grant A/C	11,42,65,029.00	13,71,94,029.00	8,55,98,972.00	8,55,98,972.00
		1,92,58,203.00		4,64,52,232.00
B. Recurring Grant in Aid				
Balance as at the beginning of the year	(1,92,21,321.62)		(73,94,472.62)	
Less: Re-stated to general reserve	(1,92,21,321.62)		(73,94,472.62)	
	-		-	
Add transfer from General Reserve	(2,05,55,919.27)		(1,92,21,321.62)	
Less surplus during the year	-	(2,05,55,919.27)		(1,92,21,321.62)
Balance at the Year End		(12,97,716.27)		2,72,30,910.38

SCHEDULE 2: RESERVES AND SURPLUS	Current Year		Previous Year	
	₹	₹	₹	₹
1.Capital Reserve:		-		-
2.Revaluation Reserve:		-		-
3.Special Reserve:		-		-
4.General Reserve:				
As per Last Account	-		-	
Restated from recurring grant in aid	(1,92,21,321.62)		(73,94,472.62)	
Add: Cumulative depriciation (opening)				
	(1,92,21,321.62)		(73,94,472.62)	
less Restated to deffered government grant	-		-	
	(1,92,21,321.62)		(73,94,472.62)	
Add: Surplus during the year	(13,34,597.65)		(1,18,26,849.00)	
	(2,05,55,919.27)		(1,92,21,321.62)	
Less: Transfer to Capital Fund	(2,05,55,919.27)	-	(1,92,21,321.62)	-
Total		-		-

National Institute of Biomedical Genomics

Schedules forming part of Balance Sheet as at 31.03.2023 Amount in ₹

Fund-wise break up

SCHEDULE 3 - EARMARKED/ENDOWMENT FUNDS	Project Fund	Fellowship Fund	Hostel Development Fund	Inst. Overhead Fund	Institute Development Fund	Institute General Fund	Staff Welfare Fund	NGC Sequencing	Total Current Year	Total Previous Year
					Annex - I					
a] Opening balance of the fund	73,39,66,039.01	43,79,332.50	12,76,538.00	2,92,51,345.61	1,95,89,350.00	3,45,08,297.21	1,41,825.00	3,10,09,346.20	85,41,22,073.53	97,06,99,195.45
b] Additions to the fund :	-	-	-	-	-	-	-	-	-	-
i] Donation/Grants/Contribution	16,05,70,625.00	1,34,13,624.00	3,51,578.00	-	-	-	1,12,400.00	11,52,93,355.29	28,57,41,582.29	37,07,97,394.68
ii] Income from investments made on account of fund	1,04,78,663.00	1,10,382.00	-	-	-	-	-	-	1,05,89,045.00	1,94,93,705.00
iii] Other (Adjustments & Outsourcing)	1,71,275.00	-	-	1,54,75,548.00	1,85,240.00	6,19,28,094.72	-	-	7,77,60,157.72	4,16,72,128.73
iv] Receivable										
Total [A+B]	90,51,86,602.01	1,79,03,338.50	16,28,116.00	4,47,26,893.61	1,97,74,590.00	9,64,36,391.93	2,54,225.00	14,63,02,701.49	1,23,22,12,858.54	1,40,26,62,523.86
c] Utilisation/Expenditure towards objectives of funds									-	-
i] Capital Expniture									15,94,83,925.00	3,57,22,605.00
Fixed Assets	15,90,48,848.00	4,35,077.00	-	-	-	-	-	-	63,717.00	-
Others	63,717.00	-	-	-	-	-	-	-	-	-
ii] Revenue Expenditure									34,25,42,007.95	42,01,30,078.48
Consumables	34,25,42,007.95	-	-	-	-	-	-	-	3,02,36,735.00	4,41,84,019.00
Manpower	3,02,36,735.00	-	-	-	-	-	-	-	1,09,32,390.00	95,75,770.00
Travel		1,09,32,390.00	-	-	-	-	-	-	18,46,613.00	8,45,332.00
Overheads	18,05,184.00	41,429.00	-	-	-	-	-	-	1,59,87,877.00	48,17,249.18
Research Grant	1,54,75,548.00	5,12,329.00	-	1,54,75,548.00	1,85,240.00	-	-	-	2,19,36,800.00	25,19,071.00
Freight,Insurance	2,14,25,389.00	5,11,411.00	-	-	-	-	-	-	-	-
Workshop/ Training	-	-	-	-	-	-	-	-	11,95,093.00	69,008.00
Contingency	11,95,093.00	-	-	-	-	-	-	-	4,59,120.00	20,68,329.00
Outsourcing	-	4,59,120.00	-	-	-	-	-	-	-	1,83,060.00
Others	1,04,49,271.38	-	-	-	-	-	-	5,34,93,903.00	6,39,43,174.38	10,56,677.00
Total [C]	58,22,41,793.33	1,26,91,756.00	-	-	-	-	-	5,34,93,903.00	64,86,27,452.33	52,11,71,398.66
d] Refund of Grant during the year	1,58,38,924.92	240.00	-	-	-	-	-	-	1,58,39,164.92	2,73,69,351.67
Net Balance as at the year end	30,71,05,883.76	50,11,342.50	16,28,116.00	4,47,26,893.61	1,97,74,590.00	9,64,36,391.93	2,54,225.00	9,28,08,798.49	56,77,46,241.29	85,41,22,073.53

National Institute of Biomedical Genomics

Schedules forming part of Balance Sheet as at 31.03.2023

	Current Year		Previous Year	
	₹	₹	₹	₹
SCHEDULE 4 :SECURED LOANS & BORROWINGS		-		-
SCHEDULE 5 :UNSECURED LOANS & BORROWINGS		-		-
SCHEDULE 6 :DEFERRED CREDIT LIABILITIES		-		-
Deffered Government Grant				
opening Balance	1,71,15,74,750.84		1,66,59,97,458.84	
Restated from Capital Fund	11,42,65,029.00		8,55,98,972.00	
Restated from General Reserve				
	1,82,58,39,779.84		1,75,15,96,430.84	
Less cumulative Depriciation (opening)	-		-	
	1,82,58,39,779.84		1,75,15,96,430.84	
Less depreciation during the year	19,57,11,676.00	1,63,01,28,103.84	4,00,21,680.00	1,71,15,74,750.84
Total		***1,63,01,28,103.84***		1,71,15,74,750.84

SCHEDULE 7 : CURRENT LIABILITIES & PROVISIONS	Current Year		Previous Year	
	₹	₹	₹	₹
A. Current Liabilities				
1. Acceptances		-		-
2. Sundry Creditors :				
i] For Capital expenditure				
ii] Others - Revenue expenditure [Annex. - iii]	3,55,400.00	3,55,400.00	35,400.00	35,400.00
3. Statutory Liabilities [Annex. - iv]		43,83,318.00		21,56,868.00
4. Deposits from Contractors [Annex. - v]		8,56,05,245.00		5,44,38,319.00
5. Earnest Money Deposits [Annex. - v]		37,49,789.00		55,47,531.00
6. Other Liabilities				
i) Liability to Govt. of India [Annex. - v]		-		3,13,87,964.64
ii) Advance amount Received		-		6,01,500.00
iii)leave salary for deputed employee				
Total [A]		***9,40,93,752.00***		***9,41,67,582.64***
B. Provisions				
1. For Taxation		-		-
2. Gratuity & Leave		2,72,40,470.00		-
3. Superannuation/Pension		-		-
4. Accumulated Leave Encashment		-		-
5. Trade Warranties/Claims		-		-
6. Others		-		-
Total [B]		2,72,40,470.00		-
Total [A+B]		***12,13,34,222.00***		***9,41,67,582.64***

National Institute of Biomedical Genomics

Schedules forming part of Balance Sheet as at 31.03.2023

Amount in ₹

SCHEDULE 8 :FIXED ASSETS

Description	GROSS BLOCK				DEPRECIATION				NET BLOCK	
	Cost/Valuation as at beginning of the year	Additions during the Year	Capitalized during the Year	Cost/Valuation at the year end	As at the beginning of the year	During the Year	On deductions during the Year	Total up to the Year end	As at the current year end	As at the previous year end
	[1]	[2]	[3]	[4=1+2+3]	[5]	[6]	[7]	[8=5+6-7]	[9=4-8]	10
A. Fixed Assets :										
Land & Building	1.00	1,02,40,536.00	1,47,75,77,107.00	1,48,78,17,644.00	-	14,84,77,739.00	-	14,84,77,739.00	1,33,93,39,905.00	1.00
Laboratory Equipment	38,58,27,747.50	7,13,69,599.00	-	45,71,97,346.50	18,38,47,198.00	3,60,15,925.00	-	21,98,63,123.00	23,73,34,223.50	20,19,80,549.50
Furniture & Fixture	2,47,45,207.50	84,44,573.00	-	3,31,89,780.50	1,07,78,706.00	19,94,049.00	-	1,27,72,755.00	2,04,17,025.50	1,39,66,501.50
Office Equipment	1,07,22,480.00	15,07,480.00	-	1,22,29,960.00	54,90,850.00	9,32,616.00	-	64,23,466.00	58,06,494.00	52,31,630.00
Computer, Software & Networking	8,67,83,718.15	1,47,41,759.00	-	10,15,25,477.15	7,59,40,411.31	73,73,867.00	-	8,33,14,278.31	1,82,11,198.84	1,08,43,306.84
Electrical Equipment	31,52,853.00	79,61,082.00	-	1,11,13,935.00	11,77,198.00	9,17,480.00	-	20,94,678.00	90,19,257.00	19,75,655.00
Books & Journals	4,62,554.69	-	-	4,62,554.69	4,62,554.69	-	-	4,62,554.69	-	-
Misc. Assets	4,470.00	-	-	4,470.00	4,470.00	-	-	4,470.00	-	-
Total of Current Year[A]	***51,16,99,031.84***	***11,42,65,029.00***	***1,47,75,77,107.00***	***2,10,35,41,167.84***	***27,77,01,388.00***	***19,57,11,676.00***	-	***47,34,13,064.00***	***1,63,01,28,103.84***	***23,39,97,643.84***
B. Capital Work in Progress :										
New Campus	1,47,75,77,107.00	-	1,47,75,77,107.00	-	-	-	-	-	-	1,47,75,77,107.00
Total of Current Year [B]	***1,47,75,77,107.00***	-	***1,47,75,77,107.00***	-	-	-	-	-	-	***1,47,75,77,107.00***
Total [A+B]	***1,98,92,76,138.84***	***11,42,65,029.00***	***2,95,51,54,214.00***	***2,10,35,41,167.84***	***27,77,01,388.00***	***19,57,11,676.00***	-	***47,34,13,064.00***	***1,63,01,28,103.84***	***1,71,15,74,750.84***

National Institute of Biomedical Genomics

Schedules forming part of Balance Sheet as at 31.03.2023

	Current Year		Previous Year	
	₹	₹	₹	₹
SCHEDULE 9 :INVESTMENTS FROM EARMARKED/ ENDOWMENT FUNDS		-		-
SCHEDULE 10 :INVESTMENTS - OTHERS		-		-

SCHEDULE 11 : CURRENT ASSETS, LOANS , ADVANCES ETC	Current Year		Previous Year	
	₹	₹	₹	₹
A. Current Assets				
1. Inventories		-		-
2. Sundry Debtors		-		-
3. Cash balances in hand				
NIBMG	21,377.00		19,210.00	
BMGC	-	21,377.00		19,210.00
4. Bank Balances :				
a] On Current Accounts :				
PNB, Kalyani Branch	28,64,100.58		5,86,72,851.58	
With PNB, Kalyani Branch [FCRA]	5,06,117.90		22,03,288.40	
With State Bank of India, Gokhale Road Branch [BMGC]	-	33,70,218.48	-	6,08,76,139.98
b] On Savings Accounts :				
Indian Bank SB ACCOUNT	2,72,40,470.00		-	
Punjab National Bank [CoTeRI]	3,67,05,629.24		2,36,64,242.83	
Punjab National Bank [ICGC]	2,81,35,013.72		18,85,46,381.42	
Punjab National Bank [Main]	7,41,00,795.78		71,01,212.78	
Punjab National Bank [Population Science Study]	39,62,408.15		40,01,797.85	
Punjab National Bank [Projects]	8,99,02,251.64		38,69,94,997.34	
Punjab National Bank [Seminar]	10,92,255.83		10,51,866.53	
Punjab National Bank [NGC]	5,45,54,952.68			
Punjab National Bank [SyMeCs]	4,38,82,357.08	35,95,76,134.12	7,29,37,946.78	68,42,98,445.53
b] On Deposit Accounts : [Annexure - VI]				
With Punjab National Bank, Kalyani Branch	5,73,50,000.00		10,60,44,335.00	
With State Bank of India, NRI Branch Kalyani	6,00,00,000.00		10,00,00,000.00	
With Indian Bank Kalyani Branch [COTERI]	11,88,78,784.00	23,62,28,784.00	-	
With CICI Bank CAN A/C Kalyani Branch		7,07,36,312.00	-	20,60,44,335.00
c. Post Office Savings Accounts	-	-	-	-
Total [A]		*66,99,32,825.60*		*95,12,38,130.51*
B. Loans, Advances & other Assets				
1. Loans :				
a] Staffs [Annexure - viii]	6,89,516.00		-54,904.00	
b] Other Entities engaged in similar activities	-		-	
c] Others	-	6,89,516.00	-	-54,904.00
2. Advances and other amounts recoverable in cash or in kind or for value to be received :				
a] On Capital Account	-		-	
b] Prepayments				
c] Contractors & Suppliers [Annexure - vii]	(15,08,694.10)		13,73,445.52	
d] Security Deposits [Annexure - ix]	8,45,194.00		2,87,966.00	
e] With Dept. of Income Tax [Annexure - xi]	97,64,215.98	91,00,715.88	1,14,11,308.98	1,30,72,720.50
3. Income Accrued				
a] On Investments from Earmarked/Endownment Fund	-		-	
b] On Investments - Others				
i] NIBMG [Annexure - x]	80,59,689.54		1,12,50,219.54	
ii] ICGC				
iii] BMGC				
c] On Loans and Advances			-	
d] Others (Payment to N. Mukherjee)	-	80,59,689.54	14,400.00	1,12,64,619.54
4. Claims Receivable		-		-
Total [B]		*1,78,49,921.42*		*2,42,82,436.04*
Total [A+B]		*68,77,82,747.02*		*97,55,20,566.55*

National Institute of Biomedical Genomics

Schedules forming part of Income & Expenditure Account for the year ended 31.03.2023

SCHEDULE 12 :INCOME FROM SALES/SERVICES	Current Year		Previous Year	
	₹	₹	₹	₹
1. Income from Sales		-		-
2. Income from Services		-		-
Total		-		-

SCHEDULE 13 : GRANTS/SUBSIDIES	Current Year		Previous Year	
	₹	₹	₹	₹
(Irrevocable Grants & Subsidies Received)				
1. Central Government		26,00,00,000.00		19,46,04,993.00
2. State Governments (Specify)		-		-
3. Government Agencies		-		-
4. Institutions/Welfare Bodies		-		-
5. International Organsations		-		-
6. Others		-		-
Total		*26,00,00,000.00*		*19,46,04,993.00*

SCHEDULE 14 :FEES/SUBSCRIPTIONS	Current Year		Previous Year	
	₹	₹	₹	₹
1. Entrace Fees		-		-
2. Annual Fees/Subscriptions		-		-
3. Seminar/Program Fees/ SUSPENCE		-		-
4. Consultancy Fees		-		-
5. Others (Tuition Fees)		5,17,000.00		89,000.00
Total		*5,17,000.00*		*89,000.00*

SCHEDULE 15 :INCOME FROM INVESTMENTS	Current Year		Previous Year	
	₹	₹	₹	₹
(Income on Investment from Earmarked/Endowment Funds transferred to Funds)		-		-
1. Interest		-		-
2. Dividend		-		-
3. Rents		-		-
4. Others		-		-
Total		-		-
Transferred to Earmarked / Endowment Funds				-

National Institute of Biomedical Genomics

Schedules forming part of Income & Expenditure Account for the year ended 31.03.2023

SCHEDULE 16 :INCOME FROM ROYALTY, PUBLICATION ETC	Current Year		Previous Year	
	₹	₹	₹	₹
1. Income from Royalty	-	-	-	-
2. Income from Publications	-	-	-	-
3. Others	-	-	-	-
Total	-	-	-	-

SCHEDULE 17 : INTEREST EARNED	Current Year		Previous Year	
	₹	₹	₹	₹
1. On Term Deposits:	-	-	-	-
2. On Savings Accounts:	-	-	-	-
3. On Loans	-	-	-	-
4. Interest on Debtors and Other Receivables	-	-	-	-
Total	-	-	-	-

SCHEDULE 18 :OTHER INCOME	Current Year		Previous Year	
	₹	₹	₹	₹
1. Profit on Sale/disposal of Assets	-	-	-	-
2. Export Incentives realized	-	-	-	-
3. Fees for Miscellaneous Services	-	-	-	-
4. Deffered Income on deffered govt grant	-	19,57,11,676.00	-	4,00,21,680.00
5. Miscellaneous Income	-	24,69,335.00	-	14,06,665.00
6. User charges	-	6,07,300.00	-	1,87,800.00
Total	-	*19,87,88,311.00*	-	*4,16,16,145.00*

SCHEDULE 19 : INCREASE/(DECREASE) IN STOCK OF FINISHED GOODS	Current Year		Previous Year	
	₹	₹	₹	₹
1.Closing stock	-	-	-	-
Finished Goods	-	-	-	-
Work-in-progress	-	-	-	-
2.Less: Opening Stock	-	-	-	-
Finished Goods	-	-	-	-
Work-in-progress	-	-	-	-
Total	-	-	-	-

National Institute of Biomedical Genomics

Schedules forming part of Income & Expenditure Account for the year ended 31.03.2023

SCHEDULE 20 : ESTABLISHMENT EXPENSES	Current Year		Previous Year	
	₹	₹	₹	₹
1. Salary & Allowances	7,95,12,331.00		7,31,63,942.00	
2. Fellowship	33,64,516.00		37,50,806.00	
3. Employer's Contribution to CPF	-		0.00	
4. Employer's Contribution to NPS	89,24,171.00		1,02,79,251.00	
5. Staff Medical Expenses	13,06,834.00		11,15,385.00	
6. Leave encashment	1,61,782.00		18,62,043.00	
7. Leave Salary & Pension Contribution	-		28,54,387.00	
8. Gratuity	2,72,40,470.00		-	
9. LTC	31,209.00	12,05,41,313.00	2,63,566.00	9,32,89,380.00
Total		*12,05,41,313.00*		*9,32,89,380.00*

SCHEDULE 21 :OTHER ADMINISTRATIVE EXPENSES ETC	Current Year		Previous Year	
	₹	₹	₹	₹
1. Consumables & Supplies :				
Laboratory Consumables	4,10,78,366.00		2,74,56,459.00	
Freight, Duty & Clearing Charges	-		-	
Entry Tax	-		-	
Computer Peripherals	67,712.00	4,11,46,078.00	4,55,706.00	2,79,12,165.00
2. Administrattive Expenditure :				
Advertisement	3,10,473.00		11,02,471.00	
Audit Fees	35,400.00		35,400.00	
Bank Charges	1,05,478.13		26,559.00	
Bio Medical Waste Management	58,155.00		55,738.00	
Electricity Charges	2,06,32,534.00		1,77,34,244.00	
Foundation Day Celebration Exp	4,71,616.00		3,48,026.00	
Internet & Telephone Charges	17,99,448.00		4,62,437.00	
Office Supplies	31,66,057.00		27,49,742.00	
Postage & Courier	77,075.00		90,192.00	
Power & Fuel	4,84,114.00		4,03,273.00	
Printing & Stationery	16,22,188.00		15,75,716.00	
Stipend to Integrated MS- phd Student	48,03,323.00		43,48,000.00	
Membership fees	6,10,000.00		8,36,000.00	
Security & Housekeeping Charges	3,79,90,624.00		3,82,73,013.00	
Salary Academic Non Permanent	1,18,890.00		-	
Salary Aadministrative Non Permanent	-		-	
Shifting Charges	-	7,22,85,375.13	-	6,80,40,811.00

3. Repair & Maintenance :				
Repairs & maintenance- Electrical	27,68,017.00		32,93,200.00	
Repairs & maintenance- Lab Equip.	36,22,327.00		17,13,086.00	
Repairs & maintenance- Office Equip.	7,79,602.00		8,39,214.00	
Repairs & maintenance- Computer	20,23,609.00		33,11,896.00	
Repairs & maintenance- others	69,41,787.00	**1,61,35,342.00**	13,43,807.00	**1,05,01,203.00**
4. Seminer ,Workshop :				
Honorarium	14,33,500.00		11,19,817.00	
Travel	5,22,428.00		37,661.00	
Publication Expenses	4,01,431.00		-	
Other Expenses	32,35,528.00	**55,92,887.00**	21,25,214.00	**32,82,692.00**
5. Seminer ,Workshop [Extramural] :				
Summer School	-		-	
Winter School	-		-	
Uchiago Workshop 2016	-		-	-
6. Travel :				
Car Hire Charges	27,77,270.00		18,88,001.00	
Travel for Academic Staff	15,81,253.00		5,94,188.00	
Travel for Administrative Staff	3,09,454.00		2,82,058.00	
Others	18,29,741.00	**64,97,718.00**	4,20,040.00	**31,84,287.00**
7. Other Expenditure :				
Legal expenses	16,520.00		-	
Processing & Filling Fees	1,40,953.00		1,24,760.00	
Fine, Interest & Penalty	-		2,500.00	
Income Tax	-		-	
Misc. Expenses	2,92,608.52	**4,50,081.52**	23,619.00	**1,50,879.00**
Total		***14,21,07,481.65***		***11,30,72,037.00***

SCHEDULE 22 :EXPENDITURE ON GRANTS, SUBSIDIES ETC.	*Current Year*		*Previous Year*	
	₹	₹	₹	₹
1. Grants given to Institutions/Organisations	-			
2. Subsidies given to Institutions/Organisations	-	-	-	-
Total		-		-

SCHEDULE 23 :INTEREST	*Current Year*		*Previous Year*	*Previous Year*
	₹	₹	₹	₹
1. On Fixed Loans	-	-	-	-
2. On Other Loans	-	-	-	-
3. Others	-	-	-	-
Total			-	-

National Institute of Biomedical Genomics

DETAILS OF ACCOUNTS AS ON 31ST MARCH, 2023

Annexure : I

	As on 31.03.2023	As on 31.03.2022
A. INSTITUTIONAL OVERHEAD RESERVE		
Name of the project & Others		
TB E Nose	21,089.00	21,089.00
Wellcome Trust IA Grant [Dr. Sandeep Singh]	29,58,023.92	29,58,023.92
Wellcome Trust IA Grant [Dr. Sreedhar Chinnaswamy]	27,77,422.00	23,80,260.00
Wellcome Trust IA Grant [Dr. Samsiddhi Bhattacharjee]	14,81,456.00	14,81,456.00
Hyperthropic Cardiomopathy	79,209.00	79,209.00
Host Virus Interaction	32,395.00	32,395.00
Indo Spanish Immune Project	1,14,212.50	1,14,212.50
PACG-GWAS	1,20,000.00	1,20,000.00
MDSCS	54,750.00	54,750.00
System Medicine cluster	15,00,000.00	15,00,000.00
Hormon Resistance Breast cancer	50,000.00	50,000.00
Cholera Vaccine	1,79,522.00	1,79,522.00
VNCI oral cancer	2,00,000.00	2,00,000.00
MPS project	1,00,000.00	1,00,000.00
IL28B Expression & Activity	76.00	76.00
Pancreatic Cancer Epigenetics	1,20,930.00	1,20,930.00
Pharma Genomics WB DBT	4,86,530.00	4,86,530.00
DFU Microbiome	1,00,000.00	1,00,000.00
Early Onset Sepsis	2,61,241.00	2,61,241.00
NER Tuberculasis	8,00,000.00	8,00,000.00
Brexo	82,500.00	82,500.00
NSM Project	17,92,000.00	11,00,000.00
Genome India project	2,87,00,000.00	1,50,00,000.00
INSACOG	19,95,000.00	15,75,000.00
Anti viral Epigenetics (Anup Majumder)	98,500.00	98,500.00
SERB- NOTCH Dr sandeep Singh	1,45,578.00	1,45,578.00
Garbh-Ini- Phase II	2,00,000.00	2,00,000.00
Oropharyngeal Cancer Genomics	9,481.00	9,481.00
Chronic Pancreatitis -Nort East	0.01	0.01
IL28B & HCV in India	592.18	592.18
Bry Box	1,12,274.00	-
Maligancy Genes in Pancreaic Cancer	1.38.886.00	-
Wellcome Trust IA Grant [Dr. Mahua Maulik]	15,226.00	-
Total	***4,47,61,893.61***	***2,92,51,345.61***

S. GUHA & ASSOCIATES KOLKATA Chartered Accountants

B. INSTITUTE DEVELOPMENT FUND	As on 31.03.2023	As on 31.03.2022
Name of the project & Others		
GCI ACT	1,96,000.00	1,96,000.00
Ramanujan Fellowship	2,99,000.00	2,99,000.00
JC Bose Fellowship	3,00,000.00	3,00,000.00
Departmental grant -UGC	9,000.00	9,000.00
EXOCARE	17,350.00	17,350.00
SERB-NPDF Vinoth kumar	1,50,000.00	1,50,000.00
SERB-NPDF Anindita Banerjee	1,50,000.00	1,00,000.00
SERB-NPDF Dipta Rup Nandi	1,50,240.00	1,50,000.00
ISR Fellowship	20,000.00	20,000.00
National Science Chair	3,00,000.00	2,00,000.00
COTERI	1,78,14,349.00	1,78,14,349.00
AIDA	3,33,651.00	3,33,651.00
DST Faculty Fellow Anusuya	35,000.00	
Total	***1,97,74,590.00***	***1,95,89,350.00***

C. INSTITUTE GENERAL FUND		
Name of the project & Others		
DBT's 30 year clebration	77,886.50	77,886.50
Interest Received	9,63,58,505.43	3,44,30,410.71
Total	***9,64,36,391.93***	***3,45,08,297.21***

Annexure : II	As on 31.03.2023	As on 31.03.2022
LIABILITIES FOR CAPITAL EXPENDITURE:	-	-
	-	-
	-	-

Annexure : III		
OUTSTANDING EXPENSES :		
Audit Fees	35,400.00	35,400.00
JC Bose Fellowship-Sagar Sengupta	3,20,000.00	-
	3,55,400.00	***35,400.00***

Annexure : IV		
STATUTORY LIABILITIES :		
Nature of Liabilities :		
Goods & Service Tax	8,565.00	1,75,528.00
TDS ON Goods & Service Tax	37,02,473.00	16,51,887.00
Income Tax deducted at source	6,06,961.00	2,78,212.00
Professional Tax	940.00	-
West Bengal Labour Welfare Cess	64,379.00	51,241.00
	43,83,318.00	***21,56,868.00***

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राष्ट्रीय जैवचिकित्सीय जीनोमिकी संस्थान NATIONAL INSTITUTE OF BIOMEDICAL GENOMICS P.O.: N.S.S., Kalyani-741251, Dist.-Nadia, West Bengal

Annexure : V

OTHER LIABILITIES

Security Deposit from :	As on 31.03.2023	As on 31.03.2022
De Bono Flexcom (I) Ltd	2,58,499.00	2,58,499.00
Ankush Furniture	9,085.00	4,697.00
IVRCL	4,21,33,820.00	4,19,33,820.00
IVRCL [Electrical Wirng-Lift shed]	46,410.00	46,410.00
Hue Services Pvt. Ltd.	4,56,952.00	4,56,952.00
Vogue furniture	3,56,069.00	1,06,820.00
Swaraj Tours & Travels	15,000.00	15,000.00
Arka Electric	92,420.00	98,202.00
AGC Networks Limited	15,95,540.00	15,95,540.00
Freezaid Sales & service	-	3,730.00
Electromech Engineers Co-Operative Society Ltd.	-	2,50,912.00
Jetmobility Pvt Ltd	15,000.00	15,000.00
Ghosh, Bose & Associates Pvt Ltd	-	2,43,167.00
M/s Vouge False Ceilling	-	3,95,685.00
Aircon India Incorporated	75,365.00	49,888.00
Aircon India Incorporated (2nd Phase)	14,73,051.00	14,73,051.00
Hindustan Construction Corporation	1,08,237.00	8,64,144.00
Bozon Technologies Pvt Ltd	-	62,498.00
Tara Aluminium Works	-	31,956.00
Charu Furniture	1,45,077.00	4,48,472.00
Monmotha Furniture	2,273.00	2,273.00
Decadon Engineering	-	1,44,735.00
M/S Vogue Vinyline flowring	1,41,036.00	1,41,036.00
D N Construction	21,932.00	18,932.00
Abdul Electricals & Infrastructure Pvt. Ltd.	-	1,78,143.00
Adhikary Enterprise	-	10,000.00
Microprocessor Unit -1	-	7,410.00
I win advisory services Ltd	1,77,159.00	1,72,130.00
Dutta Eletrical & Engineering Works	-	21,100.00
Sunshine power product pvt ltd.	2,03,633.00	8,98,867.00
Sarkar Enterorise	-	18,770.00
Biva Catering & Food Supply	5,000.00	5,000.00
Consulting Engineering Services	4,93,210.00	4,93,210.00
Dinesh Enterprise	-	1,68,897.00
Kalyani Tip Top Construction	-	1,56,168.00
K R Enterprise	-	19,684.00
Lokenath Enterprise	-	32,206.00
Mondal Electric	28,710.00	8,027.00
Smart Planet IT Solutions Pvt. Ltd.	35,87,288.00	35,87,288.00
Macro Mission	34,100.00	-

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Partha Pratim Dutta	19,680.00	-
Circuit World India Pvt. Ltd.	10,000.00	-
Micropoint Computer Pvt. Ltd	58,96,967.00	-
Nundy's	31,140.00	-
AKG Engineering Pvt. Ltd.	4,000.00	-
Omkar Engineering	42,449.00	-
Roy Contactor	28,659.00	-
Annapurna Enterprise	22,00,000.00	-
Surajit Enterprise	6,19,841.00	-
K R Instruments & Chemicals	59,614.00	-
Thermo Fisher Scientific India Pvt. Ltd.	2,51,73,607.00	-
Climaveneta	19,333.00	-
Gen work health Pvt Ltd	18,900.00	
Neel Enterprise	6,189.00	-
Total [A]	***8,56,05,245.00***	***5,44,38,319.00***

	As on 31.03.2023	As on 31.03.2022
Earnest Money Deposit from:		
Embee Software Pvt Ltd.	-	70,000.00
D N Construction	12,000.00	12,000.00
Bio Rad Pacific	36,000.00	36,000.00
Redical Scientific Equipments	8,900.00	8,900.00
Jetfleet	17,000.00	17,000.00
Omkar Travels	2,000.00	2,000.00
Lemon Car Service	2,000.00	2,000.00
Swaraj Tours & Travels	2,000.00	2,000.00
Eppendorf India Pvt Ltd	96,400.00	96,400.00
Kalyani Tip Top Construction	75,432.00	75,432.00
Bioapps	68,000.00	18,000.00
Bio Lab Equipments Pvt Ltd	18,000.00	18,000.00
AGC Networks Limited	9,00,000.00	9,00,000.00
Insprisys Solution Ltd	-	70,000.00
Innovation Pvt Ltd	-	70,000.00
Unigenetic InStruments	-	18,000.00
Bajrang BaliTravel	15,000.00	15,000.00
Nundy's	4,600.00	(12,000.00)
I - Win Advisory Services Ltd	1,00,000.00	1,00,000.00
De Bono Flexcom (I) Ltd	1,00,000.00	1,00,000.00
Invitrogen Bioservices India Pvt Ltd	20,000.00	20,000.00

Mobel India Pvt Ltd	-	2,29,000.00
Star Security & Detective Agency	7,20,000.00	7,20,000.00
Acer Pvt Ltd	70,000.00	70,000.00
Pionear scientific	18,000.00	18,000.00
Biva Catering & Food supply	-	50,000.00
Patel Chem De drugs	3,58,380.00	3,58,380.00
S V Scientific Pvt Ltd	37,573.00	37,573.00
Sunshine power product pvt ltd.	1,27,846.00	1,27,846.00
Gen work health Pvt Ltd	-	16,000.00
Converge System	-	5,00,000.00
Numal Buragohin	-	2,80,000.00
Digital Track Solution Pvt Ltd	-	6,52,000.00
Bioprojects Pvt Ltd	-	8,50,000.00
Hue Service Pvt. Ltd.	19,800.00	-
Labest Instruments	37,000.00	-
Vijay Power Generator Pvt. Ltd.	1,31,159.00	-
DN Construction	16,000.00	-
Arka Electric	16,000.00	-
Wizertech Informatics Pvt. Ltd.	36,800.00	-
Surajit Enterprise	54,135.00	-
Ikon Instrument	1,12,000.00	-
Biorad Laboratories India Pvt. Ltd.	1,20,000.00	-
Cidermatics India Pvt. Ltd.	37,350.00	-
Orbit Infosolutions	36,800.00	-
Bio Lab Equipments Pvt. Ltd.	50,000.00	-
Rajdoot	50,000.00	-
Rahul Traders	1,64,000.00	-
K R Instrumennts Pvt. Ltd.	59,614.00	-
Total [B]	***37,49,789.00***	***55,47,531.00***
Grand Total [A+B]	***8,93,55,034.00***	***5,99,85,850.00***

Liabilities to Govt Of India		
Interest on Fixed Deposits	-	*2,73,39,313.15*
Interest on Advances	-	*35,88,602.00*
Interest on Savings Bank Accounts	-	*2,09,14,049.49*
Less : Refund to Govt. of India	-	*(2,04,54,000.00)*
	-	***3,13,87,964.64***

Annexure : VI	As on 31.03.2023	As on 31.03.2022
FIXED DEPOSIT :		
Punjab Natinal Bank, Kalyani		
057920PU00000226	57350000.00	-
- 0579109280399	-	72,00,000.00
- 0579109280380	-	2.22.00.000.00
- 0579109280371	-	33,00,000.00
- 0579109280362	-	43,00,000.00
- 0579109280353	-	14,00,000.00
- 0579109280399	-	38,00,000.00
- 0579109283855	-	2,43,53,670.00
- 0579109283864	-	1,03,04,670.00
- 0579109283882	-	2,91,85,995.00
Total	***5,73,50,000.00***	***10,60,44,335.00***
Indian Bank, Kalyani		
7282975098	5,73,50,000.00	
7386783795	1,00,00,000.00	
7467738901	5,15,28,784.00	
Total	***11,88,78,784.00***	-
State Bank of India, NRI		
37673407063	1,20,00,000.00	1,20,00,000.00
37673438547	1,20,00,000.00	1,20,00,000.00
37673439595	1,20,00,000.00	1,20,00,000.00
37673440283	1,20,00,000.00	1,20,00,000.00
37673440862	1,20,00,000.00	1,20,00,000.00
37906647366	-	1,00,00,000.00
37906690683	-	1,00,00,000.00
37906691097	-	1,00,00,000.00
37906691520	-	1,00,00,000.00
Total	***6,00,00,000.00***	***10,00,00,000.00***
Grand Total	***23,62,28,784.00***	***20,60,44,335.00***

S. GUHA & ASSOCIATES Chartered Accountants KOLKATA

Annexure : VII	As on 31.03.2023	As on 31.03.2022
ADVANCE TO PARTIES :		
Kothari Medical Centre	1,19,719.00	
Genex India BioSciences	4,99,825.00	
Climaveta	30,000.00	
Balmer Lawrie & Co Ltd	2,119.00	
Bapna Enterprise	-	9,005.00
Prakash Freight movers ltd	-	2,80,974.00
CCHuGe	-	(649.40)
West Bengal Green Energy Development Corporation	-	6,000.00
Kolkata Metropolitan Water & Sanitation Authority	-	4,806.00
United Chemical Company	-	24,308.00
B C Roy Krishi Viswavidyalaya	-	67,000.00
Swissotel-BhadiHospitality	-	20,031.50
Jet Mobility Pvt. Ltd.	-	37,803.00
Holiday in Kolkata	-	17,046.00
ICICI bank Credit card	-	92,380.42
Ladhuram Toshniwal & Sons	-	14,200.00
Tata Steel Ltd	-	11,156.00
NIAB	-	(1.861.00)
World Courier	-	1,81,102.00
Patel Chem-De-Drugs	-	(4,13,661.00)
Total [i]	***6,51,663.00***	***3,49,640.52***

ADVANCE TO PARTIES (NGC):	As on 31.03.2023	As on 31.03.2022
Agartala Medical College	2,23,550.00	-
CNCI Kolkata	(2,40,181.00)	-
AIIMS Kalyani	856.00	-
Manipal Academy of Higher Education	1,22,571.00	-
CSIR-IMT	1,314.00	-
NBRC Haryana	1,11,366.00	-
ICMR RMRC	(3,01,898.00)	-
NIBMG Core & Project	18,32,250.00	-
SNIP	1,56,853.00	-
TMC	1,38,897.00	-
University of Calcutta	32,665.00	-
ISI, Kolkata	1,30,993.00	(1,55,484.00)
Bose Institute	1,97,452.00	5,458.00
IVRI Bareilly	93,478.00	(36,885.00)
NICED	56,099.90	2,35,268.00
Midnapore City College	21,920.00	2,065.00
ICAR NEH	-	58,410.00
IICB	-	1,21,977.00
IISER-Kolkata	-	29,016.00
IMTECH	-	1,78,228.00
NIO	-	2,96,466.00
RCB	-	19,824.00
RGCB	-	2,52,116.00
Unilever	9,97,389.00	
NIMHANS	(57,35,932.00)	
THSTI	-	17,346.00
	(21,60,357.10)	***10,23,805.00***
Total [A] [i+ii]	***(15,08,694.10)***	***13,73,445.52***

S. GUHA & ASSOCIATES Chartered Accountants KOLKATA

	As on 31.03.2023	As on 31.03.2022
Annexure : VIII		
Advance to Staff		
Samsiddhi Bhattacharjee	89,724.00	-
Tuneer Ranjan Maulick	1,000.00	-
Paramita Mitra	1,59,355.00	-
Neelutpal Das	3,000.00	-
Jagyashila Das	87,260.00	-
Esha Bhattacharjee	1,16,943.00	-
Debosmita Bannerjee	10,000.00	-
Anindita Bannerjee	1,01,385.00	-
Tamogno Chatterjee	65,000.00	-
Sagar Sengupta	(46,196.00)	-
Nidhan Kr Biswas	5,000.00	-
Analabha Basu	89,502.00	-
Amal kumar Halder (MF)	-	(1,50,000.00)
Bishnupriya Chhatriya	-	(32,335.00)
Priyodarshi Basu	-	6,073.00
Supriyo Chatterjee	7,463.00	20,000.00
Ankita Chatterjee	-	(4,728.00)
Arvind M korwar	-	23,400.00
Arindam Maitra	-	6,139.00
Sandeep B Mukherjee	-	42,459.00
Subrata Patra	-	6,000.00
Arindam Maitra (LTC)	80.00	28,088.00
	6,89,516.00	***(54,904.00)***

	As on 31.03.2023	As on 31.03.2022
Annexure :IX		
Security Deposits (Assets)		
Name of Party		
Semb Rambky	7,500.00	7,500.00
ELLenbarrie Industrial Gas Ltd	34,000.00	34,000.00
Genesis Indane Gas Service, Gayeshpur	13,600.00	10,200.00
Science City	6,195.00	6,195.00
West Bengal State Electricity Distribution Co Ltd	7,83,899.00	2,30,071.00
	8,45,194.00	***2,87,966.00***

Annexure : X		
Interest accrued		
D. State Bank of India NRI [F.V : ₹ 6,00,00,000/-]	69,54,554.00	1,04,31,830.00
b. Advance		
Interest accrued on Investment of WBSEDCL	11,05,135.54	8,18,389.54
	80,59,689.54	***1,12,50,219.54***

S.GUHA & ASSOCIATES KOLKATA Chartered Accountants

राष्ट्रीय जैवचिकित्सकीय जीनोमिकी संस्थान * (एनआईबीएमजी) * NATIONAL INSTITUTE OF BIOMEDICAL GENOMICS * P.O.: N.S.S., Kalyani: Dist.-Nadia (W.B.) PIN-741251 * दूरभाष: 033-2582-8012 * वेबसाईट: www.nibmg.ac.in

	As on 31.03.2023	As on 31.03.2022
Annexure : XI		
Other Receivables :		
Advance Income Tax		
Financial Year 2013-14		
United Bank of India [TDS on Interest on FDR]	-	10,41,867.00
ICICI Bank [TDS on Interest on FDR]	-	5,488.00
Financial Year 2014-15		
United Bank of India [TDS on Interest on FDR]	-	5,48,443.00
ICICI Bank [TDS on Interest on FDR]	-	5,947.00
Unilever [TDS on Grant]	-	1,00,000.00
Financial Year 2016-17		
ICGC	-	6,20,826.00
BMGC	-	2,94,597.00
Financial Year 2017-18		
TDS on Interest on FD		
NIBMG	-	11,51,619.00
BIRAC [TDS on Grant]	-	53,680.00
Unilever [TDS on Grant]	-	2,00,000.00
CoTeRI - ISI [TDS on Grant]	-	24,000.00
Financial Year 2018-19		
BIRAC [TDS on Grant]	-	53,680.00
Unilever [TDS on Grant]	-	2,00,000.00
CoTeRI - ISI [TDS on Grant]	-	22,060.00
Financial Year 2020-21		
Bose Institute - NGC Sequencing	19,590.00	1,14,610.00
WBUAFS - NGC Sequencing	5,841.00	5,841.00
EXSEGEN - NGC Sequencing	26,270.00	26,270.00
TDS on Interest on FD [NIBMG]	33,51,320.26	33,51,320.26
Financial Year 2021-22		
NGC Sequencing [TDS on Grant]	3,70,125.72	3,70,125.72
TDS on Interest on FD [NIBMG]	31,84,339.00	31,84,339.00
Financial Year 2022-23		
TDS on Interest on FD [NIBMG]	17,19,966.00	
TDS on Grant [Sunpharma]	20,147.00	
TDS on Grant	10,30,021.00	
Total [A]	***97,27,619.98***	***1,13,74,712.98***
Excess TDS deposited with Income Tax Department		
Relating to 1st Qtr of FY 2013-14	**36,596.00**	**36,596.00**
Total [B]	***36,596.00***	***36,596.00***
Total [A+B]	***97,64,215.98***	***1,14,11,308.98***

S.GUHA & ASSOCIATES KOLKATA Chartered Accountants

राष्ट्रीय जैवचिकित्सकीय जीनोमिकी संस्थान * (एनआईबीएमजी) * NATIONAL INSTITUTE OF BIOMEDICAL GENOMICS * P.O.: N.S.S., Kalyani: Dist.-Nadia (W.B.) PIN-741251 * दूरभाष: 033-2582-8012 * वेबसाईट: www.nibmg.ac.in

Statement showing receipt, utilisation & unutilised balance of projects during 2022-23

Extramural Project

Name of the Project	Avesthagen (20300)	Antiviral Epigenetics (20311)	Anti Viral Drug Designing (20320)	Big Data (20275)	BMGC	Biotech Rise	BREXO (20287)	BRY BOX (20317)	CDFR (20316)	Cholera Vaccine Response (20273)	COVID - BRICS (20308)	Development of SARS-CoV-2 Virus (20301)	Early Onset Sepsis (20284)	EXOCARE (20294)
a] Balance as on 01.04.2022	53,206.00	14,82,700.00	-	2,84,208.00	2,14,25,389.38	1,71,231.00	15,869.00	-	-	11,78,307.00	1,39,96,808.00	6,20,870.00	96,634.00	1,29,572.00
b] Grant received during the year			11,13,700.00					15,15,700.00	9,37,000.00		-	-		
c] Interest received during the year		16,805.00	7,025.00	6,320.00	-	-		15,268.00	16,034.00	11,812.00	13,917.00	12,556.00	2,028.00	3,504.00
d] Misc. Adjustment during the year														
e] Outsourcing														
Total [a+b+c+d+e]	53,206.00	14,99,505.00	11,20,725.00	2,92,528.00	2,14,25,389.38	2,71,231.00	15,869.00	15,30,968.00	9,53,034.00	12,10,119.00	1,40,10,725.00	6,33,426.00	98,662.00	1,33,076.00
e] Utilisation/Expenditure during the year														
i] Capital Expenditure :														
Equipment		4,65,350.00	-					-						
Non Recurring														
Others			-											
ii] Revenue Expenditure														
Consumables		7,48,007.00	1,96,517.00				-	7,61,812.00	29,449.00		6,98,938.00	2,02,697.00		
Manpower		1,93,242.00	22,320.00				-	1,00,424.00	80,129.00		6,78,273.00		21,420.00	
Stipend & HRA														
Travel							-	1,12,274.00	25,000.00		25,702.00	29,568.00		
Overhead									-					
Freight,Insurance														
Workshop,training														
Fellowship														
Research Grant											26,339.00	29,739.00		
Accessories for Data Analysis														
Data Analysis														
Library Preparation & Sequencing														
Contingency														
Outsourcing														
Recurring														
Others							15,869.00							
Scientific Social Responsibility														
Total Expenditure [e]	-	14,14,960.00	2,18,837.00	-	2,14,25,389.38	0.38	15,869.00	9,74,516.00	1,34,578.00	-	14,29,292.00	2,62,004.00	21,420.00	-
f] Grant Refunded during the year				78,428.00	-						1,24,848.00	28,425.30		-
Balance as on 31.03.2023 [a+b+c+d+e-f]	53,206.00	84,545.00	9,01,888.00	2,14,100.00	-	2,71,231.00	-	5,56,452.00	8,18,456.00	12,10,119.00	1,24,56,625.00	3,42,997.00	77,242.00	1,33,076.00

Statement showing receipt, utilisation & unutilised balance of projects during 2022-23

Extramural Project

Name of the Project	FG-RED (20292)	Fund for DNB Program (20243)	Fund for Fetal Programming Research (20247)	GARBH-Ini Phase - II (20306)	Gastric Cancer Mizoram (20262)	GBSSC Chemoresistance (20226)	GCI ACT (20263)	Genome India (20299)	Hereditary Hemolytic Anemias (20272)	HPV 16-E7 (20276)	HPV Persistance WB-DBT (20280)	IAS [Training] (20285)	ICGC-India Project (20277)	IMPACT (20279)
a] Balance as on 01.04.2022	4,12,459.00	7,82,229.95	25,271.00	42,62,905.00	1,80,396.00	-1,59,181.00	6,02,833.00	7,47,60,503.00	61,673.00	6,67,346.00	3,43,833.00	2,04,297.00	18,87,39,734.22	1,00,20,932.00
b] Grant received during the year	9,096.00	-	-	-	-	-	5,88,852.00	11,77,11,482.00	-	-	15,04,716.00	-	-	19,48,17,585.00
c] Interest received during the year	-	-	-	2,280.00	-	-	-	23,45,603.00	-	16,635.30	7,003.00	-	35,47,421.00	-
d] Misc. Adjustment during the year	-	-	-	-	-	1,59,181.00	-	-	-	-	-	-	-	-
e] Outsourcing	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total [a+b+c+d+e]	4,21,555.00	7,82,229.95	25,271.00	42,65,185.00	1,80,396.00	-	11,91,685.00	1,00,20,932.00	61,673.00	6,83,981.00	18,55,552.00	2,04,297.00	19,22,87,156.22	19,48,17,585.00
e] Utilisation/Expenditure during the year	-	-	-	-	-	-	-	-	-	-	-	-	-	-
i] Capital Expenditure :	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Equipment	-	-	-	5,15,841.00	-	-	-	4,139.00	-	-	-	-	-	-
Non Recurring	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Others	-	-	-	-	-	-	-	-	-	-	-	-	-	-
ii] Revenue Expenditure	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Consumables	3,11,394.00	1,29,409.95	21,340.00	36,91,612.00	-	-	-	13,98,604.00	-	39,743.30	3,11,807.00	-	16,25,59,173.00	7,69,19,427.00
Manpower	-	3,07,503.00	-	2,97,600.00	80,008.00	-	-	4,34,827.00	-	-	-	-	33,24,975.00	27,44,490.00
Stipend & HRA	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Travel	-	37,310.00	-	3,416.00	2,135.00	-	-	2,56,421.00	-	2,972.00	-	-	26,723.00	-
Overhead	-	-	-	-	-	-	-	1,37,08,000.00	-	-	-	-	-	-
Freight,Insurance	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Workshop,training	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Fellowship	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Research Grant	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Accessories for Data Analysis	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Data Analysis	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Library Preparation & Sequencing	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Contingency	-	6,162.00	-	2,077.00	47,601.00	-	-	64,074.00	1,621.00	-	-	-	4,54,499.00	-
Outsourcing	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Recurring	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Others	-	50,000.00	-	-	-	-	-	-	-	-	-	-	-	-
Scientific Social Responsibility	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Expenditure [e]	3,11,394.00	5,30,384.95	21,340.00	45,10,546.00	1,29,746.00	-	-	19,01,844.00	14,07,77,812.00	1,621.00	42,715.00	3,11,807.00	2,04,297.00	16,63,65,770.00
f] Grant Refunded during the year	-	2,51,845.00	3,931.00	-	50,650.00	-	-	19,06,100.00	60,052.00	18,677.00	-	-	63,08,260.00	
Balance as on 31.03.2023 [a+b+c+d+e-f]	1,10,161.00	-	-	-2,45,361.00	-	-	11,91,685.00	81,19,088.00	5,21,33,673.00	-	6,22,589.00	15,43,745.00	-	1,96,13,126.22

Statement showing receipt, utilisation & unutilised balance of projects during 2022-23

Extramural Project

Name of the Project	INSACOG [COVID-19] (20305)	NIBMG Flagship (20298)	NSHL (20293)	NSM (20297)	Pancreatic Cancer Epigenetics (20286)	PGI-Oral Cancer (20302)	Pharmacogenomics WB-DBT (20288)	Metformin Resistance (20236)
a] Balance as on 01.04.2022	4,42,06,307.00	-	1,19,641.00	10,59,498.00	23,836.00	12,58,494.00	5,22,463.00	86,369.00
b] Grant received during the year	38,86,449.00	16,95,600.00	-	7,00,000.00	-	-	-	-
c] Interest received during the year	4,70,129.00	2,224.00	-	15,604.00	-	29,353.00	14,112.00	-
d] Misc. Adjustment during the year	-	-	-	-	-	-	-	-
e] Outsourcing	-	-	-	-	-	-	-	-
Total [a+b+c+d+e]	*4,85,42,885.00*	*16,97,824.00*	*1,19,641.00*	*17,74,902.00*	*23,836.00*	*12,87,847.00*	*5,36,575.00*	*86,369.00*
e] Utilisation/Expenditure during the year								
[] Capital Expenditure :								
Equipment	-	-	-	-	-	-	-	-
Non-Recurring								
Others	-	-	-	-	-	-	-	-
[i] Revenue Expenditure	-	-	-	-	-	-	-	-
Consumables	2,39,65,930.00	-	75.00	9,83,946.00	-	1,02,287.00	10,249.00	10,249.00
Manpower	25,77,881.00	7,07,257.00	60,000.00	2,79,884.00	-	1,92,896.00	41,333.00	41,333.00
Stipend & HRA	-	-	-	-	-	-	-	-
Travel	74,709.00	-	-	1,38,886.00	-	-	-	-
Overhead	4,20,000.00	-	-	-	-	-	-	-
Freight, Insurance	-	-	-	-	-	-	-	-
Workshop, training	-	-	-	-	-	-	-	-
Fellowship	-	-	-	-	-	-	-	-
Research Grant	-	-	-	-	-	-	-	-
Accessories for Data Analysis	-	-	-	-	-	-	-	-
Data Analysis	-	-	-	-	-	-	-	-
Library Preparation & Sequencing	-	-	-	-	-	-	-	-
Contingency	9,10,776.00	-	13,089.00	-	-	-	-	-
Outsourcing	-	-	-	-	-	-	-	-
Recurring	-	-	-	-	-	-	-	-
Others	-	-	-	-	-	-	-	-
Scientific Social Responsibility	-	-	-	-	-	-	-	-
Total Expenditure [e]	2,79,49,296.00	7,07,257.00	73,164.00	14,00,646.00	-	2,95,183.00	51,582.00	51,582.00
f] Grant Refunded during the year	3,18,447.00	-	46,477.00	-	-	-	34,787.00	34,787.00
Balance as on 31.03.2023 [a+b+c+d-e-f]	2,02,65,142.00	9,90,567.00	-	3,72,256.00	23,836.00	9,92,664.00	-	-
	17,58,863.00	2,21,14,743.00	6,04,086.06	-2,64,472.00	2,11,443.00	18,17,482.00	5,36,575.00	81,961.00

Name of the Project	Nonoding RNA in Pancreatic Cancer (20318)	Populationa Science study (20269)	Pre Term Birth (20238)	RNA Seq on Breast Cancer (20214)	Selenium Project (20291)	SERB- NOTCH (20309)	Sun Pharma Winter School 28 Nov to 03 Dec 22	System Medicine Cluster (20268)	Multi-Omics Signatures of Human Placenta (20303)	National Geno::cs Core (20295)	NER Training (20244)	NER Tuberculjs (20290)	Multi-Omics Signatures of Human Placenta (20303)	National Geno::cs Core (20295)	NER Training (20244)	NER Tuberculjs (20290)
a] Balance as on 01.04.2022	-	39,59,471.65	2,31,59,016.00	6,55,143.20	29,229.00	14,14,551.00		7,18,88,430.04	28,98,442.00	16,62,46,659.00	10,86,382.94	59,53,655.00	28,98,442.00	16,62,46,659.00	10,86,382.94	59,53,655.00
b] Grant received during the year	19,91,531.00	-	-	-	20,96,275.00	7,00,000.00	16,10,941.00	-	47,34,903.00	30,87,000.00	-	-	47,34,903.00	30,87,000.00	-	-
c] Interest received during the year	21,669.00	3,18,731.00	-	17,688.00	23,096.00	22,993.00	-	13,34,559.00	10,113.80	13,42,466.00	29,328.00	63,537.00	10,113.80	13,42,466.00	29,328.00	63,537.00
d] Misc. Adjustment during the year	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
e] Outsourcing	-	-	12,094.80	-	-	-	-	-	-	-	-	-	-	-	-	-
Total [a+b+c+d+e]	*20,13,202.00*	*2,54,00,268.00*	*29,40,930.00*	*78,98,715.00*	*6,51,655.00*	*18,85,186.00*	*5,36,575.00*	*4,74,100.00*	*17,58,863.00*	*2,21,14,743.00*	*6,04,086.06*	*-2,64,472.00*	*2,11,443.00*	*18,17,482.00*	*5,36,575.00*	*81,961.00*
e] Utilisation/Expenditure during the year																
[] Capital Expenditure :	-	-	-	-	-	-		-								
Equipment	-	-	-	-	-	-		-								
Non Recurring	60,000.00	-	-	-	-	-		-								
Others	-	-	-	-	-	-		-								
[i] Revenue Expenditure	-	-	-	-	-	-		-								
Consumables	-	15,89,844.00	20,34,214.00	9,00,000.00	4,19,167.00	67,704.00		1,61,01,428.00								
Manpower	73,600.00	6,09,649.00	3,02,500.00	51,74,469.00	-			7,81,934.00								
Stipend & HRA	-	-	-	-	-			4,741.00								
Travel	-	1,02,031.00	-	6,59,651.00	7,666.00			-								
Overhead	-	-	-	6,92,606.00	-											
Freight, Insurance	-	-	-	-	-											
Workshop, training	-	-	-	-	-											
Fellowship	-	-	-	-	-											
Research Grant	-	-	-	-	-											
Accessories for Data Analysis	-	-	-	-	-											
Data Analysis	-	-	-	-	-											
Library Preparation & Sequencing	-	-	-	-	-											
Contingency	-	760.00	130.00	2,24,619.00	3,379.00											
Outsourcing	-	-	-	3,00,000.00	-											
Recurring	1,18,739.00	-	-	-	-											
Others	-	-	-	-	-											
Scientific Social Responsibility	-	-	-	-	-											
Total Expenditure [e]	2,54,339.00	13,02,284.00	23,36,844.00	79,50,779.00	4,30,212.00	67,704.00										
f] Grant Refunded during the year	-	7,83,241.00	-	2,12,448.00	-	-										
Balance as on 31.03.2023 [a+b+c+d-e-f]	*17,58,863.00*	*2,21,14,743.00*	*6,04,086.06*	*-2,64,472.00*	*2,11,443.00*	*18,17,482.00*										

Statement showing receipt, utilisation & unutilised balance of projects during 2022-23

Name of the Project	Extramural Project										Extramural Seminar		Foreign	
	TB : WB DBT (20296)	TB Granuloma Project (20213)	Unilever (20239)	VNCI Breast Cancer (20274)	VNCI - Oral Cancer (20283)	Wellcome Trust - Alpana Dave (20307)	Wellcome Trust - Mahua Malik (20312)	Wellcome Trust- Sreedhar Chinnaswamy (20279)	NIBMG SAS Project (CIKIM Study) (20315)	NIBMG SAS Project (WINGS Study) (20314)	Analysis of NGS Data	EMBO Symposium (50104)	Ichigan-NIBMG Winter School	Asian Immune diversity Atlas (AIDA) (70101)
a] Balance as on 01.04.2022	64,404.00	2,12,426.92	41,90,494.66	90,54,214.00	60,97,860.00	11,199.00	17,69,260.00	22,82,783.00	2,63,091.00	9,82,205.00	-	1,88,221.00	13,004.00	21,90,638.00
b] Grant received during the year	9,35,372.00	-			-	6,33,333.00	-	38,73,096.00	15,20,000.00	11,27,500.00				
c] Interest received during the year	22,213.00	-	99,079.00	1,01,718.00	-	1,298.00	36,608.00	39,777.00	26,651.00	27,999.00				-
d] Misc. Adjustment during the year	-	-	-	-	-			-			-	-	-	
e] Outsourcing	-	-	-	-	-			-			-	-	-	
Total [a+b+c+d+e]	***10,21,989.00***	***2,12,426.92***	***42,89,573.66***	***91,55,932.90***	***60,97,860.00***	***6,45,830.00***	***18,05,868.00***	***61,95,656.00***	***18,09,742.00***	***22,37,704.00***	-	***1,88,221.00***	***13,004.00***	***21,90,638.00***
e] Utilisation/Expenditure during the year				-										
i] Capital Expenditure :	-	-	-	-	-			-			-	-	-	
Equipment	-	-	-	-	-			-			-	-	-	
Non-Recurring														
Others	-	-	-	-	-			-			-	-	-	
ii] Revenue Expenditure	-	-	-	-	-			-			-	-	-	
Consumables	1,20,221.00	-	9,35,555.00	78,52,612.00		15,980.00	9,93,863.00	49,37,564.00	69,295.00	37,959.00				17,59,882.00
Manpower	-			-		6,00,000.00		3,60,000.00						
Stipend & HRA				-										
Travel				-						27,516.00				
Overhead				-	-		15,226.00	3,97,162.00						
Freight,insurance				-										
Workshop,training		-		-										
Fellowship														
Research Grant														
Accessiories for Data Analysis									10,135.00					
Data Analysis									36,750.00	82,320.00				
Library Preparation & Sequencing									6,65,670.00	18,00,000.00				
Contingency		-		3,045.00	-					1,764.00		-		
Outsourcing				-										
Recurring														
Others				-		-	-							
Scientific Social Responsibility														
Total Expenditure [e]	1,20,221.00	-	9,35,555.00	78,55,657.00	-	6,15,980.00	10,09,089.00	56,94,726.00	7,82,250.00	19,49,559.00	-	-	-	17,59,882.00
f] Grant Refunded during the year		2,12,426.92		63,085.00	2,83,990.00	1,215.00		80,683.00					-	
Balance as on 31.03.2023 [a+b+c+d-e-f]	**9,01,768.00**	-	**33,54,018.66**	**12,37,190.00**	**58,13,870.00**	**27,635.00**	**7,96,779.00**	**4,20,247.00**	**10,27,492.00**	**2,38,145.00**	-	**1,88,221.00**	**13,004.00**	**4,30,756.00**

Statement showing receipt, utilisation & unutilised balance of projects during 2022-23

Name of the Project	ICMR-Driver Gene (20322)	CoTeRI	Total
a] Balance as on 01.04.2022	-	9,00,65,360.05	73,39,66,039.01
b] Grant received during the year	8,44,648.00	29,500.00	16,05,70,625.00
c] Interest received during the year	-		1,04,78,663.00
d] Misc. Adjustment during the year			1,71,275.00
e] Outsourcing			-
Total [a+b+c+d-e]	***8,44,648.00***	***9,00,94,860.05***	***90,51,86,602.01***
e] Utilisation/Expenditure during the year			-
i] Capital Expenditure :			-
Equipment			15,90,48,848.00
Non-Recurring			63,717.00
Others			-
ii] Revenue Expenditure			-
Consumables		9,74,850.00	34,25,42,007.95
Manpower		-	3,02,36,735.00
Stipend & HRA			-
Travel			18,05,184.00
Overhead			1,54,75,548.00
Freight,insurance			-
Workshop,training			11,95,093.00
Fellowship			-
Research Grant			2,14,25,389.00
Accessiories for Data Analysis			10,535.00
Data Analysis			1,19,070.00
Library Preparation & Sequencing			24,65,670.00
Contingency			26,20,392.00
Outsourcing			3,00,000.00
Recurring			1,18,739.00
Others			48,06,704.38
Scientific Social Responsibility			8,161.00
Total Expenditure [e]	-	9,74,850.00	58,22,41,793.33
f] Grant Refunded during the year			1,58,38,924.92
Balance as on 31.03.2023 [a+b+c+d-e-f]	**8,44,648.00**	**8,91,20,010.05**	**30,71,05,883.76**

Statement showing receipt, utilisation & unutilised balance of fellowship during 2022-23

Name of the Project	CSIR Fellowship	DBT -JRF Fellowship	DBT -RA Fellowship	DST Inspire Faculty Fellowship	DST Inspire Fellowship	ICMR Fellowship	National Science Chair - PPM	SERB - NPDF	J. C. Bose Fellowship-Dr. Sagar Sengupta (40263)	UGC Fellowship	Total
A	B	C	D	E	F	G	H	I	J	K	B to K
a] Balance as on 01.04.2022	*1,75,751.00*	*1,72,636.00*	*62,781.50*	*16,07,456.00*	*16,74,880.00*	*1,28,413.00*	*83,470.00*	*4,39,637.00*	-	*34,308.00*	*43,79,332.50*
b] Grant received during the year	14,631.00	39,31,365.00	-	-	13,54,880.00	16,96,833.00	44,00,000.00	10,68,400.00	9,47,515.00	-	*1,34,13,624.00*
c] Interest received during the year	-	-	-	22,328.00	-	-	73,447.00	13,815.00	792.00	-	*1,10,382.00*
d] Misc. Adjustment during the year	-	-	-	-	-	-	-	-	-	-	-
Total [a+b+c+d]	*1,90,382.00*	*41,04,001.00*	*62,781.50*	*16,29,784.00*	*30,29,760.00*	*18,25,246.00*	*45,56,917.00*	*15,21,852.00*	*9,48,307.00*	*34,308.00*	*1,79,03,338.50*
e] Utilisation/Expenditure during the year											-
i] Capital Expenditure :	-	-	-	-	-	-	-	-	-	-	-
Equipment	-	-	-	4,35,077.00	-	-	-	-	-	-	*4,35,077.00*
Others	-	-	-	-	-	-	-	-	-	-	-
ii] Revenue Expenditure	-	-	-	-	-	-	-	-	-	-	-
Consumables	-	-	-	-	-	-	-	-	-	-	-
Manpower	-	-	-	-	-	-	-	-	-	-	-
Fellowship,Stipend & HRA	-	35,25,956.00	60,760.00	8,75,000.00	25,98,908.00	11,77,774.00	18,00,000.00	8,69,000.00	25,000.00	-	*1,09,32,390.00*
Travel	-	-	-	41,429.00	-	-	-			-	*41,429.00*
Overhead	-	-	-	35,000.00	-	-	1,00,000.00	50,000.00	3,27,329.00	-	*5,12,329.00*
Freight,insurance	-	-	-	-	-	-				-	-
Research Grant	-	-	-	-	-	-	2,14,654.00	2,96,757.00		-	*5,11,411.00*
Contingency	1,09,029.00	1,78,121.00	-	50,906.00	99,232.00	21,830.00	-	-	-	-	*4,59,120.00*
Others	-	-	-	-	-	-	-	-	-	-	-
Total Expenditure [e]	*1,09,029.00*	*37,04,079.00*	*60,760.00*	*14,37,412.00*	*26,98,132.00*	*11,99,604.00*	*21,14,654.00*	*12,15,757.00*	*3,52,329.00*	-	*1,28,91,756.00*
f] Grant Refunded during the year	-	-	-	-	-	-	-	240.00	-	-	240.00
Balance as on 31.03.2023 [a+b+c+d-e-f]	*81,353.00*	*3,99,922.00*	*2,021.50*	*1,92,372.00*	*3,31,628.00*	*6,25,642.00*	*24,42,263.00*	*3,05,855.00*	*5,95,978.00*	*34,308.00*	*50,11,342.50*

National Genomics Core (Sequencing)

Receipts & Payments Account for the period 01.04.2022 to 31.03.2023

RECEIPTS:	*As on 31.03.2023*		*PAYMENTS:*	*As on 31.03.2023*	
	Amount	*Amount*		*Amount*	*Amount*
Opening Balance		₹ 3,10,09,346.20			
			Payment for Consumables		₹ 4,78,88,795.89
			Bank Charges	₹ 1,928.16	
Receipts for Sequencing			Printing & Stationary	₹ 9,169.00	
Research and Development Services [Including Taxes]	₹ 97,92,413.35		Repair & Maintenance	₹ 23,947.00	
Reimbursement of Consumables	₹ 8,75,69,609.89		Carriage Outward	₹ 9,450.00	
Data Generation Charges	₹ 74,76,584.82	₹ 10,48,38,608.06	Misc Payment	₹ 0.00	₹ 44,494.16
			Goods & Service Tax Paid (Deposited)		₹ 25,50,850.71
Advance Received		₹ 71,29,778.00			
Interest received		₹ 3,15,207.00			
			Closng Balance		₹ 9,28,08,798.49
		₹ 14,32,92,939.26			*₹ 14,32,92,939.26*

For Natioanl Institute of Biomedical Genomics

Director
डॉ. सागर सेनगुप्ता / Dr. Sagar Sengupta
निदेशक / Director
राष्ट्रीय जैवचिकित्सा जीनोमिक्स संस्थान
National Institute Of Biomedical Genomics
Place : Kalyani
Date : 28.07.2023

Manager [Finance]

Signed in terms of our separate Report of even date.

FOR S. GUHA & ASSOCIATES
Chartered Accountants
Firm Registration No : 322498E
Sourabh Mitra
Partner
Membership No : 308743
UDIN No.: 23308743BGULXT4886

For S. GUHA & ASSOCIATES
Chartered Accountants
Partner

National Genomics Core (Secuencing)

Income & Expenditure Account for the period 01.04.2022 to 31.03.2023

Expenditure	As on 31.03.2023		Income	As on 31.03.2023	
	Amount	Amount		Amount	Amount
To Purchase of Consumable	₹ 10,18,30,028.58		By, Research & Development		₹ 1,19,08,793.60
To, Carriage Inward	₹ 9,450.00	₹ 10,18,39,478.58	By, Reimbursement of Consumables	₹ 8,75,69,609.89	
			By, Data Generation Charges (Inter-departmental)	₹ 74,92,234.82	₹ 9,50,61,844.71
To, Bad Debt	₹ 17,40,062.58				
To, Bank charges	₹ 1,835.32		By, Interest received		₹ 3,15,207.00
To, Printing & Stationary	₹ 9,169.00				
To, Repair & Maintenance	₹ 23,947.00	₹ 17,75,013.90			
To, Excess Income over Expenditure		₹ 36,71,352.83			
		₹ 10,72,85,845.31			₹ 10,72,85,845.31

Signed in terms of our separate Report of even date.

FOR S. GUHA & ASSOCIATES
Chartered Accountants
Firm Registration No.: 322498E
Saurabh Mitra
Partner
Membership No : 308743
UDIN No.: 23308743BGULXT4886

Natioanl Institute of Biomedical Genomics
Manager [Finance]

Director
Dr. Sagar Sengupta
निदेशक / Director
राष्ट्रीय जैवचिकित्सा जीनोमिक्स संस्थान
National Institute Of Biomedical Genomics
Place:- Kalyani
Date:-

NATIONAL INSTITUTE OF BIOMEDICAL GENOMICS

ACCOUNTING POLICIES & NOTES ON ACCOUNTS FORMING PART OF ACCOUNTS FOR THE YEAR ENDED 31ST MARCH 2023

SCHEDULE - 24

Significant Accounting Policies:

1. The financial statements are prepared on the basis of historical cost convention, in accordance with the applicable Accounting Standards in India.
2. Income and expenditure are accounted for on accrual basis of accounting,
3. Fixed Assets are accounted for a cost of acquisition inclusive inward freight, duties and taxes & incidental & direct expenses related to acquisition.
4. Fixed assets are stated at written down value. In addition to WDV of fixed assets, depreciation is charged on the fixed assets purchased and put to use during the year.
5. Fixed assets acquired on Government Grant which has been capitalized by the Institute as per AS 12 (Issued 1991) para 8.4, "Accounting for Government Grants".
6. Depreciation is calculated & provided in the accounts on the basis of written down value of the fixed assets at the rates specified under the Income Tax Act. 1961 and also followed accounting policies as stated in AS 12 (Issued 1991) para 8.4, "Accounting for Government Grants".
7. Deposits with Bank stated at cost under the head 'Current Assets '& interest accrued thereon is shown separately in Balance Sheet under Loans, Advances & other current Assets.
8. Grants in aid are recognized in accounts as & when the same is received. Non-recurring grants are funded & shown under Capital Fund whereas Recurring grants are recognized as income in the Income & Expenditure Accounts. A year's unutilized grant or deficit due to over -utilization of grant is carried forward & adjusted with next year's grant in aid.
9. Stocks of reagents & consumables are fully charged as expenditure as and when the same is allotted to laboratory for research purpose.

SCHEDULE -25

Contingents Liabilities & Notes on Accounts

A. **Contingent Liabilities**

1. Claims against the Institute not acknowledged as debts -Nil(previous year Nil)

Notes on Accounts:

1. The Institute has been registered on 6th August, 2009 under Societies Registration Act, XXI of 1860 under Registrar of Societies, Govt. of NCT of Delhi, having registered office at Block-2, 7th Floor, CGO Complex, Lodhi Road, New Delhi - 3 and functioning from P. O. -NSS, Kalyani, Nadia, West Bengal.

2. The Institute has been set up as an autonomous body under the administrative control of the Department of Biotechnology, Ministry of Science & Technology, and Government of India.

3. The Institute is wholly financed by the Government of India & its income is exempted from Income tax under section 10(23C) (iiiab) of the Income Tax Act, 1961.

4. Interest earned on deposit has been apportioned between extramural projects on monthly average basis.

5. Capital Work- In- progress as on 1st April 2022 was Rs. 147,75,77,107/- and addition during the year is Rs. 1,02,40,536/-totaling to Rs. 148,78,17,643/-, which has capitalized for completion of building.

7. Endowment fund consists of project fund, fellowship fund, hostel development fund, Institute overhead fund, NGC Sequencings, and staff welfare fund.

8. Annual Return of the society has been submitted up to financial year 2021-2022 & Return for the Financial Year 2021-22 has been submitted on 31.03.2023.

9. Fixed assets amounting to Rs. 15,95,47,642.00, have been purchased during this financial year out of grant received for extramural projects. The cost of the said fixed assets has been charged against extramural projects & fellowship Account. These Fixed assets have not been shown in the Fixed Assets Schedule, as these assets have been purchased for carrying out the projects for which grants are received from the external agencies and the project is in the ongoing state. Ownership of these assets does not lie with NIBMG during the pendency of the project, but NIBMG merely stands custodian of these assets.

10. Fixed Assets register is being maintained by the institute.

11. No insurance policy has been taken by the Institute for its Fixed Assets as well as Project's Fixed Assets.

12. Previous year's figure have been rearranged & regrouped whenever necessary.

13. Fellowship received and disbursed both through institute as well as Direct Benefit Transfer mode in the current Financial Year.

For National Institute of Biomedical Genomics.

[Debashish Kumar Mandal]
Manager [Finance]

[Prof. Sagar Sengupta]
Director

Dr. Sagar Sengupta
निदेशक / Director
राष्ट्रीय जैवचिकित्सा जीनोमिक्स संस्थान
National Institute Of Biomedical Genomics
पो.ओ.: एन.एस.एस., कल्याणी, पिन-७४१२५१, जिला-नदीया,(प.ब.)
P.O.: N.S.S., Kalyani, Pin-741251, Dist. Nadia,(W.B.)
भारत / India

S. GUHA & ASSOCIATES, KOLKATA, Chartered Accountants

Notes