GDDB 2025

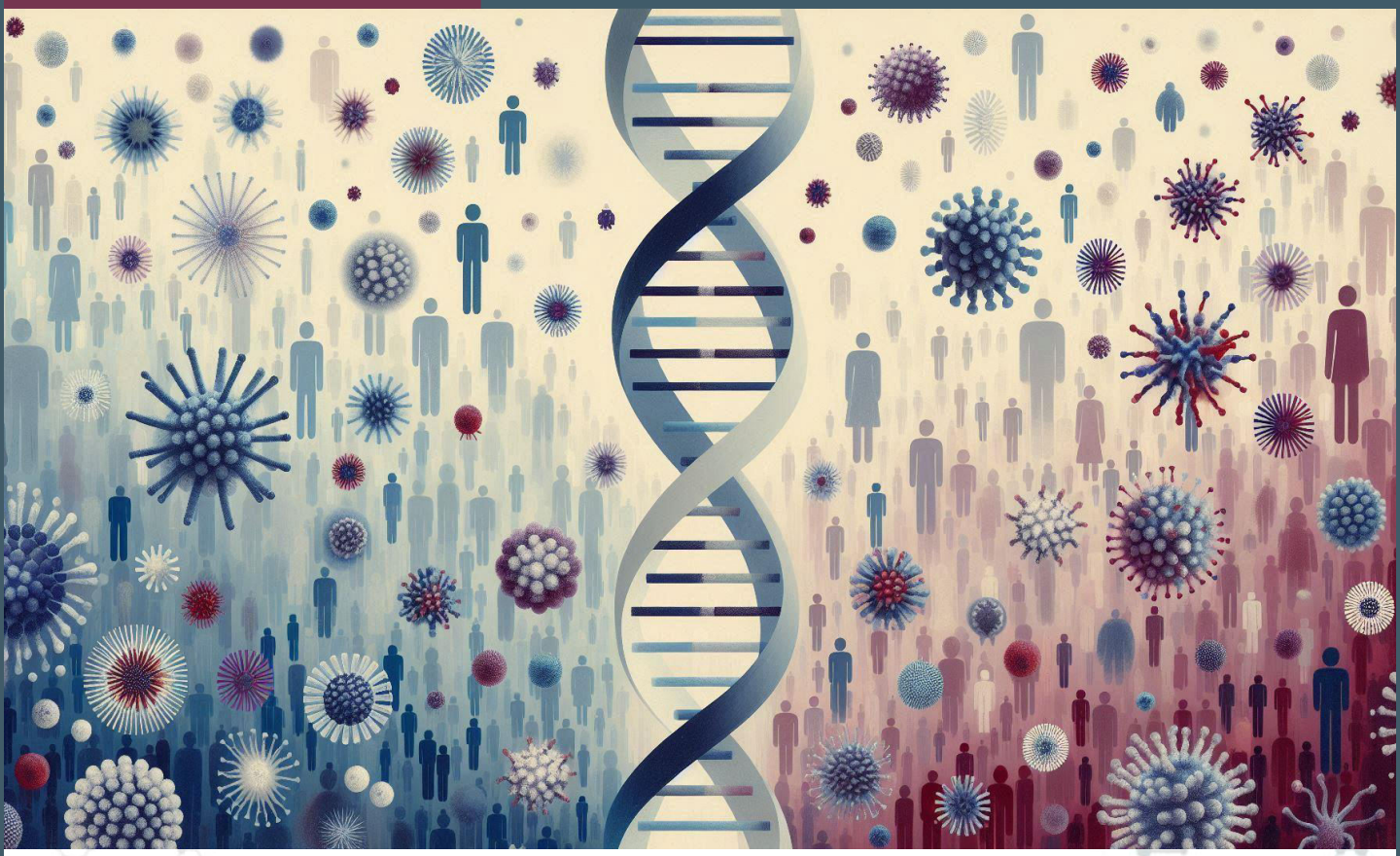
International Symposium on GENETIC DIVERSITY AND DISEASE BIOLOGY

May 23, 2025

Organized by

JIS Institute of Advanced Studies and Research (JISIASR),
JIS University

Book of ABSTRACTS



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Editors:
Kausik Basak, *PhD;* Sandip Paul, *PhD*
JISIASR, JIS University

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- Understanding mutational impact on structure and function of protein
- AI-driven computational pipeline for characterization of antibiotic resistance drug classes
- Web-based resource development for microbiome and metabolome data integration and feature identification
- Anti-tuberculosis compound development targeting c-di-AMP signalling

... and many more

Dedicated to the Pursuit of Knowledge

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GDDB 2025

International Symposium on
GENETIC DIVERSITY & DISEASE BIOLOGY (GDDB) 2025
May 23, 2025

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GDDB 2025

PROGRAM SCHEDULE

May 23, 2025

TIME	SESSIONS
09:00 am – 09:45 am	Registration and Welcome Tea
09:45 am – 10:30 am	Inauguration
	SESSION 1
10:30 am – 12:30 pm	Session Chair: Prof. Ajoy Kumar Ray, *Director, JISIASR* Keynote Speech: Genomics of Oral Cancer: Lessons Learnt, Learning, to Learn Prof. Partha Pratim Majumder *National Science Chair, Govt. of India* *Distinguished Professor, John C. Martin Center for Liver Research & Innovations* Invited Talk: The promise of precision medicine: concepts, complexities and challenges Prof. Ananyo Choudhury *Sydney Brenner Institute for Molecular Bioscience, University of Witwatersrand, South Africa*
12:30 pm – 01:30 pm	POSTER PRESENTATION
01:30 pm – 02:30 pm	NETWORK LUNCH
	SESSION 2
02:30 pm – 04:30 pm	Session Chair: Dr. Rukhsana Chowdhury, *Adjunct Professor, JISIASR* *Former Chief Scientist, Infectious Diseases and Immunology Division, CSIR-Indian Institute of Chemical Biology, Kolkata* Keynote Speech: *Helicobacter pylori* – role of type IV secretion and bacterial SNPs in gastric cancer development Prof. Steffen Backert *Chair of Microbiology, Friedrich-Alexander University, Erlangen, Germany* Invited Talk: Genomic diversity and pathogen virulence: implications for infectious disease Prof. Santasabuj Das *Director, ICMR-National Institute for Research in Bacterial Infections, Kolkata, India*
04:30 pm – 05:00 pm	Award / Certificate Distribution and Vote of Thanks

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Sardar Taranjit Singh
Chancellor, JIS University
Managing Director, JIS Group

It gives me immense pleasure to extend my heartfelt greetings to all the distinguished participants, speakers, and organizers of the International Symposium on Genetic Diversity and Disease Biology (GDDB 2025).

This symposium represents a vital confluence of scientific intellect and collaborative spirit, focused on the exploration of genetic diversity and its role in disease mechanisms. In a world increasingly shaped by the promise of personalized medicine and genomic science, such discussions are not only timely but critical. The knowledge shared here will undoubtedly contribute to the development of more inclusive, effective, and equitable health solutions.

At JIS Group, we believe in empowering innovation through education and research. We are proud to support initiatives like GDDB 2025 that bring together global perspectives, cutting-edge research, and young scientific minds with a shared goal of addressing pressing challenges in human health and disease biology.

I congratulate the organizing committee for their dedication and foresight in curating this symposium. I also wish to thank all the participants whose contributions continue to push the boundaries of scientific understanding. May GDDB 2025 serve as a beacon of inspiration, collaboration, and advancement in biomedical science.

Warm regards.

Sardar Taranjit Singh

SIMARPREET SINGH

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Sardar Simarpreet Singh
Director, JIS Group, India

It is a privilege to welcome all participants to the International Symposium on Genetic Diversity and Disease Biology (GDDB 2025), organized by JISIASR, JIS University. This significant event brings together eminent minds to delve into the complexities of genetic diversity and its profound implications for disease understanding and treatment.

We are in an era of rapid scientific advancement where interdisciplinary research is essential for meaningful innovation. This symposium provides a dynamic platform for researchers, scholars, and professionals from diverse backgrounds to share groundbreaking insights, explore emerging challenges, and foster impactful collaborations in the fields of genomics, molecular biology, and disease biology.

At JIS University, we remain steadfast in our commitment to academic excellence and research-driven progress. Events like GDDB 2025 reflect our vision of nurturing a vibrant scientific community that drives solutions to some of today's most pressing health and biological challenges.

I extend my heartfelt gratitude to all invited speakers, presenters, and delegates for contributing to the success of this symposium. Special thanks to the organizing committee for their diligent efforts in curating this meaningful event.

Warm regards,
Sardar Simarpreet Singh

Prof. Bhaskar Gupta
Vice-Chancellor, JIS University

It gives me immense pleasure to note that the **Centre for Health Science and Technology, JISIASR** under the aegis of JIS University, Kolkata organizes the one-day International Symposium on **"Genetic Diversity and Disease Biology (GDDB 2025)"** on **23rd May, 2025**. This symposium serves as a collaborative platform for leading scientists in human and microbial genomics from across the globe.

Truly it refers as a testament to the dedication, intellectual curiosity, and collaborative spirit that define our academic community. Each abstract within these pages is a snapshot of countless hours of rigorous inquiry, critical thinking, and a commitment to pushing the boundaries of knowledge in diverse fields.

In an era defined by rapid change and complex global challenges, the role of research is more critical than ever. It is through platforms like this that we foster interdisciplinary dialogue, spark new collaborations, and ultimately contribute to solutions that benefit our society as well. The insights shared here, even in their condensed form, have the potential to inspire further exploration and drive meaningful progress.

I extend my heartfelt congratulations to all the contributors for their outstanding work. Your research enriches the institute (JISIASR), strengthens its academic standards, and plays a pivotal role in shaping the future research arena. I also commend the organizing committee for their tireless efforts in bringing that event to fruition and compiling this valuable resource.

I encourage all attendees to delve into these abstracts, engage in stimulating discussions, and forge new connections. May this event be a truly enriching experience for everyone involved.

I wish all the best for the event.

Prof. Bhaskar Gupta

From the Desk of the Director, JISIASR

Prof. Ajoy Kumar Ray (*Padma Shri*)
Director, JISIASR Kolkata

It is with immense pleasure that I welcome all participants, speakers, and delegates to the International Symposium on Genetic Diversity and Disease Biology (GDDB 2025), being held on May 23, 2025, at the JIS Institute of Advanced Studies and Research (JISIASR), Kolkata.

This symposium arrives at a time when biomedical sciences are undergoing a rapid transformation, fuelled by technological innovation and deepening biological insight. The themes of this event—ranging from molecular microbiology to machine learning in healthcare—reflect the interdisciplinary nature of modern life science research. GDDB 2025 brings together a rich tapestry of experts and young researchers to engage with topics that lie at the very heart of global health challenges and solutions.

JISIASR – A mission to address modern challenges through translational research:

Founded in 2019, JISIASR was conceived not as a conventional academic institution, but as a centre of excellence committed to high-impact, translational research. Our aim is to foster cutting-edge inquiry in areas that directly address societal needs—healthcare, environmental sustainability, energy, materials, and data science.

We currently house four specialized centres:

- Centre for Health Science and Technology
- Centre for Interdisciplinary Sciences
- Centre for Data Science
- Centre for Renewable and Sustainable Energy and Green Automotive

Our faculty, many of whom come with global training and research experience, are leading over 30 sponsored research projects, worth of more than 9 crores, funded by ANRF, CCRH, DBT, ICMR, Wellcome Trust, WBDSTBT and other national agencies. These projects cut across thematic domains such as infectious disease biology, computational health informatics, drug design, and microbial genomics—forming a natural backdrop to the focus of GDDB 2025.

Our work at JISIASR in line with GDDB 2025:

The core areas reflect urgent biomedical research frontiers and embody JISIASR's strengths. Some of the key research themes being showcased and discussed include:

1. Evolution of virulence and antibiotic resistance:
The accelerating emergence of multi-drug resistant pathogens present one of the greatest threats to global health. At JISIASR, researchers are working on molecular and genomic studies that unravel the evolutionary dynamics of bacterial virulence and resistance mechanisms. The goal is to inform novel strategies for antimicrobial development and stewardship.

2. Host–pathogen interactions and the human microbiome:
Understanding the molecular crosstalk between host and pathogen is fundamental to decoding disease outcomes. Our faculty are investigating these interactions in infectious diseases such as leishmaniasis, tuberculosis, and sepsis. We are also exploring how shifts in the human microbiome contribute to chronic inflammatory conditions, metabolic disorders, and even neurodegeneration.

3. NGS data mining and methodological development:
With the explosion of next-generation sequencing (NGS) technologies, there is a growing need for robust computational frameworks. JISIASR researchers are developing algorithms and pipelines to interpret multi-omics data, identify disease-associated variants, and integrate transcriptomic and metagenomic datasets—an area that aligns deeply with the data-intensive sessions at GDDB 2025.

4. Machine learning and medical image analysis:
Artificial intelligence is reshaping the diagnostics and prognostics landscape. Our data science team is applying deep learning models to analyze medical images—such as histopathological slides and radiographs—for disease classification and risk prediction. Similarly, predictive models trained on clinical and genomic data are being explored for personalized healthcare solutions.

5. Medical microbiology and drug design:
We are engaged in designing molecular probes, inhibitors, and small molecule drugs targeting key microbial and oncogenic pathways. Through molecular modeling, structure-activity relationships, and in silico screening, our teams are accelerating the early stages of drug discovery. This complements parallel wet-lab work on bioassays and therapeutic validation.

Global collaboration and academic exchange:

The GDDB 2025 symposium exemplifies our belief that science thrives through collaboration and cross-pollination of ideas. This event features renowned speakers and participants from leading institutions in South Africa, France, Australia, Poland, and across India, including the Sydney Brenner Institute for Molecular Bioscience and several CSIR and DBT-supported labs. The thematic sessions of the symposium—hosted by the Centre for Health Science and Technology in collaboration with the Centre for Data Science—are designed to spark innovative discussions on: human genetic diversity and disease susceptibility, computational pipelines for health data; systems biology and pathway modeling; microbial ecosystems in health and disease; therapeutic target discovery and rational drug design, etc.

A message of optimism and gratitude:

It is my sincere belief that the International Symposium on Genetic Diversity and Disease Biology will provide a fertile platform for intellectual exchange, inspire young researchers, and lay the groundwork for future collaborations that transcend institutional and national boundaries. I extend my deepest appreciation to the organizing committee, our faculty colleagues, student volunteers, and every contributor who has made this event a reality. I also thank our speakers and delegates for enriching this symposium with their time, knowledge, and presence. In the spirit of discovery, dialogue, and inclusion, I welcome you all to GDDB 2025 and hope this experience is intellectually stimulating and deeply rewarding.

Best wishes,
Prof. Ajoy Kumar Ray

Prof. (Dr.) Goutam Ghosh
Pro Vice-Chancellor, JIS University

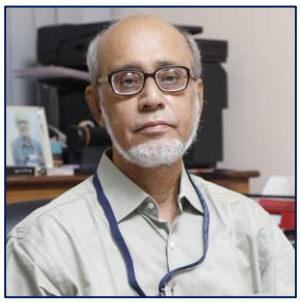
It is with great pleasure that I extend my warmest greetings to all participants of the International Symposium on Genetic Diversity and Disease Biology (GDDB 2025).

This symposium serves as a dynamic platform for the exchange of cutting-edge knowledge in the rapidly evolving fields of genomics, molecular biology, and disease research. The focus on genetic diversity as a central theme is particularly relevant in today's scientific and healthcare landscape, where personalized and precision medicine is becoming increasingly significant.

At JIS University, we are committed to fostering academic excellence, interdisciplinary research, and global collaboration. GDDB 2025 exemplifies this commitment by bringing together eminent scholars, researchers, and young scientists to address complex biological challenges and explore novel solutions that can shape the future of healthcare and biomedical sciences.

I sincerely commend the efforts of the organizing team for their vision and dedication in making this symposium a reality. I also extend my appreciation to the contributors whose scholarly work has enriched this event and will continue to inspire innovation and inquiry.

With regards,
Prof. (Dr.) Goutam Ghosh



Prof. Partha Pratim Majumder
National Science Chair, Government of India
Distinguished Professor, John C. Martin Centre for Liver Research and Innovations
Emeritus Professor, Indian Statistical Institute

On May 23, 2025, the JIS Institute of Advanced Studies & Research, Kolkata, organized an international symposium on "Genetic Diversity and Disease Biology." I received an invitation to participate in the symposium. Without any hesitation, I accepted the invitation especially because the theme of the symposium is close to my heart.

The connection between genetic diversity and disease biology is not an obvious one. Genetic diversity is about heritable variation among individuals in populations. While disease biology is about the biological, including genetic, aspects of diseases. Genetic diversity arises by the action of various evolutionary forces. New genetic variation arises by mutation. The frequency of a new variant in a population is determined primarily by natural selection, and in small populations additionally by a stochastic force termed as random genetic drift. Some of these new variants cause diseases. The genes in which these variants arise either lose their normal function or gain altered functions that cause diseases. That's the connection between genetic diversity and disease biology. A variant often does not cause disease, but raises the susceptibility of disease. The disease manifests when the individual possesses multiple genetic variants, each of which increases the susceptibility to the disease by a small amount. The joint action of the multiple genetic variants is sufficiently large to cause disease. Most late-age onset chronic diseases are due to the joint action of multiple variants. In an individual with such a disease, the genetic variants do not arise de novo, but are obtained by genetic transmission from parents. Because each of these variants only raises disease susceptibility by a small amount, these variants are not strongly influenced by natural selection after they arise. Hence, their frequencies are often seen to increase in a population over generations. Because such newly-arising variants are not quickly lost from the population, genetic diversity in the population increases. Therefore, genetic susceptibility to a common chronic disease is a significant part of this genetic diversity.

Modern genomic methods can routinely lead to the identification of variants in genes that are involved in disease susceptibility. The normal functions of these genes are usually known from past scientific research. The impact of a newly-arising variant on the function of a gene is deciphered by conducting a variety of experiments. A gene is a part of a biological pathway. When a new variant alters the function of a gene, the biological pathway is usually perturbed. The nature of perturbation also needs to be determined, since perturbed biological pathways ultimately precipitate disease. Thus, from exploring genetic diversity and the nature of variants present in a population, to identifying perturbations in biological pathways caused by gene-variants provides the understanding of the biological bases of diseases.

This compilation of scientific presentations in the symposium is a testimony to the relationship between genetic diversity and biology of diseases.

Prof. Partha Pratim Majumder

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

SDIMB
SYDNEY BRENNER INSTITUTE FOR MOLECULAR BIOSCIENCE

Prof. Ananyo Choudhury
Reader | Sydney Brenner Institute for Molecular Bioscience (SBIMB)
SBIMB, University of the Witwatersrand Johannesburg, South Africa
Co-Chair | H3Africa Genome Analysis working group

It is a great honour to extend my warm greetings to all participants of the International Symposium on Genetic Diversity and Disease Biology (GDDB 2025).

The theme of this symposium resonates deeply with the urgent global need to understand how genetic variation influences human health and disease. As researchers and scholars, we are collectively tasked with uncovering the biological mechanisms that underlie disease disparities, particularly within underrepresented populations. In this context, the integration of genetic diversity into biomedical research is not just a scientific imperative—it is an ethical one.

Events like GDDB 2025 provide invaluable opportunities for dialogue, collaboration, and innovation. They create a platform to bridge regional research efforts with global scientific advances, fostering inclusive and contextually relevant solutions to complex health challenges. It is especially heartening to witness such initiatives growing in scope and ambition, drawing contributions from across disciplines and geographies.

I commend the organizers for their vision in bringing this symposium to fruition and for cultivating a space where diverse voices in science are both represented and celebrated. To the participants—may your discussions be thought-provoking, your collaborations fruitful, and your discoveries impactful.

With best wishes for a successful and inspiring symposium.

Warm regards,
Prof. Ananyo Choudhury

Dr. Santasabuj Das
MBBS, MD (General Medicine)
Director and Scientist-G,
ICMR-National Institute for Research in Bacterial Infections, Kolkata, India

It is great to know that the organizers have decided to publish a Book of Abstract on the proceedings of the International symposium on Genetic Diversity and Disease Biology (GDDB) 2025, held on May 23, 2025 at the JIS Institute of Advanced Studies and Research, Santragachi, West Bengal. I had the privilege to attend the symposium physically and was immensely impressed by the quality of research work presented as posters and oral presentations.

The study of genetic diversity is not merely an academic exercise, but fundamental to our understanding of human health and diseases. Genetic variations can influence disease susceptibility, manifestations, progression, and response to treatment. This goes beyond inherited diseases and encompasses most infections and non-communicable diseases as well. Better understanding of genetic variations will help to prevent disease development and pave the way for more precise and targeted therapy, leading to improved disease management and more favorable outcome.

The symposium brought together national and international experts from diverse fields as well as students and faculty members of different scientific disciplines. It created an ideal platform for the exchange of cutting-edge research findings, fostering collaborations for interdisciplinary research and developing network to translate research into action and generated great excitement and enthusiasm among the young researchers.

I strongly believe that the symposium was a great success.

Best wishes,
Dr. Santasabuj Das

Dr. Rukhsana Chowdhury
Adjunct Professor, JISIASR
Former Chief Scientist, Infectious Diseases and Immunology Division,
CSIR-Indian Institute of Chemical Biology, Kolkata

I have recently participated in the International Symposium on Genetic Diversity and Disease Biology (GDDB 2025) organized by JIS Institute for Advanced Studies and Research on May 23, 2025. The conference was extremely well organized and I am sure involved a lot of meticulous planning and hard work for its execution. Congratulation to the entire team!

The topic of the conference was of particular relevance in view of recent developments in our understanding of how genetic diversity between individuals and populations affects susceptibility to disease, both systemic and infectious and also regulates response to pharmaceutical interventions. All these areas were addressed by distinguished speakers.

Professor Partha Partha Pratim Majumder's talk highlighted his team's pathbreaking work on genome analysis of oral cancer patients as well as those with precancerous lesions. Professor Steffen Backert presented interesting data on SNPs in the genome of *Helicobacter pylori* that may cause malignant alterations in the host stomach. Along similar lines, but in more generalized systems, Professor Santa Sabuj Das described the effects of bacterial as well as host genomic diversity in host pathogen interactions. The very promising area of precision medicine was addressed by Professor Ananyo Choudhury - starting with extensive population genetics data and culminating in early disease predictions.

In addition to presenting their research results, all speakers discussed the future of research on genomic diversity as reflected in the title of their talks. These included the areas where these studies could be extended, the potential benefits as well as major challenges in implementing these studies.

I once again thank the organizers for this very relevant biomedical conference and look forward to attending conferences extending the theme further in the future.

Warm regards,
Dr. Rukhsana Chowdhury

KEYNOTE & INVITED Lectures

Genomics of Oral Cancer: Lessons Learnt, Learning, to Learn

Prof. Partha Pratim Majumder
National Science Chair, Government of India
Distinguished Professor, John C. Martin Centre for Liver Research and Innovations
Emeritus Professor, Indian Statistical Institute

Abstract:

Based on analysis of genome sequences of 500 patients of oral cancer, we have identified and characterized driver mutations of oral squamous cell carcinoma of the gingivobuccal region. We have also identified genomic and transcriptomic alterations that result in metastasis and other complications in a subset of patients. We have recently carried out genomic analyses of pre-cancerous lesions from which we are learning about the sequence of genomic alterations in the progression of oral cancer. In an effort to characterize the nature and extent of heterogeneity within tumours of the oral cavity, we have performed single-cell transcriptomic analysis. Our first set of results have revealed two dominant cellular programs. I shall present the results of these studies.

Helicobacter pylori – role of type IV secretion and bacterial SNPs in gastric cancer development

Prof. Steffen Backert
Chair of Microbiology, Friedrich-Alexander University, Erlangen, Germany

Abstract:

Many Gram-negative pathogens harbor type IV secretion systems (T4SSs) that translocate bacterial virulence factors into host cells to hijack cellular processes for multiple purposes. *Helicobacter pylori* is a human-specific bacterium that causes persistent infections in the stomach associated with pathologies ranging from chronic gastritis, peptic ulceration to gastric cancer. The virulence of *H. pylori* strongly depends on the serine protease HtrA, the T4SS encoded by the cytotoxin-associated genes (cag) pathogenicity island and the injected effector protein CagA. This T4SS forms a needle-like pilus, which is induced upon host cell contact, and its assembly is accomplished by multiple protein-protein interactions. We discovered two gastric cancer-associated single nucleotide polymorphisms (SNPs) in the bacteria, which control effective T4SS functions upon infection of polarized gastric epithelial cells. Those include an alanine-to-threonine polymorphism in the tyrosine phosphorylation motif EPIYA-B of CagA, which affects gastric cancer risk by modifying intracellular signal transduction events involved in inflammation and cell division. The second SNP causes a serine-to-leucine change in HtrA which is found to be associated with – (i) elevated trimer formation and proteolytic activity, (ii) boosted cleavage of epithelial junction proteins occludin, claudin-8 and E-cadherin, (iii) induction of paracellular *H. pylori* transmigration, (iv) pronounced T4SS-pilus formation and injection of CagA, (v) promoted gastric inflammation and β-catenin-mediated cell proliferation, as well as (vi) enhanced introduction of DNA double-strand breaks (DSBs) in the host chromosome and micronuclei formation upon infection. These activities highlight the importance of the cag T4SS and SNPs in bacterial virulence, which co-operatively elicit malignant alterations in the gastric epithelium that are discussed.

The promise of precision medicine: concepts, complexities and challenges

Prof. Ananyo Choudhury
Sydney Brenner Institute for Molecular Bioscience, University of Witwatersrand, South Africa

Abstract:

I plan to begin my presentation with a brief primer on genetic variants and trends in their distribution across populations and geographies. We will then examine how the connection between genetic variants and diseases enables us to harness genomics in diagnosis, drug development, and early disease risk predictions. Next, we will discuss how genes modulate the response to common medicines and the ways in which these relationships provide an opportunity for improving treatment effectiveness by tailoring medication to an individual's genetic make-up. In the final segment, I will summarize the major challenges in implementing precision medicine and highlight the key acumen and skills that could prepare future researchers for a career in this exciting field of research.

Genomic diversity and pathogen virulence: implications for infectious disease

Prof. Santasabuj Das
Director, ICMR-National Institute for Research in Bacterial Infections, Kolkata, India

Abstract:

Pathogen virulence is genetically encoded and may result as a gain-of-function or loss-of-function trait. The former mechanism is more common and predominantly involves horizontal transfer of mobile genetic elements from a pathogenic strain to another pathogenic or non-pathogenic bacteria. Additionally, genetic recombination may also add to genomic diversity and alter the virulence by changing pathogenicity, antimicrobial resistance and biofilm formation. However, optimal virulence often requires 'pathoadaptive mutations' – a loss-of-function trait due to deletion, insertion or point mutation of antivirulence genes that limit the expression or function of ancestral or newly acquired genes required for optimal pathogenicity. Parallel adaptive mutations over time in the same pathogenic strain from multiple patients of a disease have helped to identify candidate virulence genes associated with antibiotic resistance and redox regulation. Literature suggests that more genetic diversity within a pathogen population implies a wider range of virulence factors and better adaptation to new environmental stress and host defense mechanisms. At times, genetically diverse strains complement each other, thereby increasing their overall pathogenicity. Further, host diversity also increases the selection pressure on pathogens to diversify and evolve to a more virulent strain that can overcome wide range of host defenses. Traditionally, virulence factors were identified by biochemical or forward genetic approaches, followed by the introduction of genome-wide mutagenesis techniques. However, these approaches suffer from the lack of representativeness of the genomic diversity of the entire species. Recent spurt of bacterial whole genome sequencing and comparative genomics have attempted to overcome this through identification of species-specific virulence traits from the core genome and strain specific factors by analyzing the accessory genes. Genotypic-phenotypic association is performed through Genome Wide Association Studies (GWAS) for single locus or by a more holistic machine learning approach for multi-locus analysis. Finally, epigenetic changes, especially DNA methylation also plays an important role in rapid, phase variable expression of virulence genes.

ABSTRACTS for presented posters

GDDB-PP-01

Dengue virus protein sequence diversity: Serotype prediction from individual protein sequences

Dwaipayan Chaudhuri, Kalyan Giri*

Department of Life Sciences, Presidency University, Kolkata

**Corresponding Author:*
Dr. Kalyan Giri, kalyan.dbs@presiuniv.ac.in

Abstract:
Dengue virus (DENV) is a serious worldwide health issue with four recognized serotypes (DENV-1, DENV-2, DENV-3, and DENV-4) responsible for the degree of disease severity. Correct classification of DENV proteins into serotypes is essential for diagnostics, vaccine development, and epidemiological research. In this study we examine the variability of the dengue virus protein sequences in a bid to elucidate on the viral serotype evolution and fitness of the viral protein sequences with respect to their amino acid conservancy. Using this protein sequence variability information, we aim to construct a classifier model that can precisely classify specific unknown dengue protein sequences into their corresponding serotypes. Previous studies have demonstrated that DENV shows considerable sequence variation across different geographic regions and different serotypes, which can impact viral transmission dynamics and disease outcomes. Sequence analysis of the DENV genomic and proteomic sequences has revealed distinct patterns of evolution both at proteomic level as well as serotypic level, highlighting the complex interplay between viral adaptation and host immune pressure. This study aims to provide an in-depth analysis of the sequence diversity of dengue virus proteins, at both the intra-serotypic and inter-serotypic level, focusing on the structural and non-structural proteins, and their implications for viral evolution, disease pathogenesis, and vaccine design. This study also aims at selecting proteins whose sequence patterns serves as a serotypic signature of each of the Dengue virus serotypes. A machine learning strategy making use of Support vector machine, Random forest algorithm and Artificial neural network is subsequently used to train a classifier model based on features extracted from the protein sequences utilizing the one-hot encoder to transform each protein sequence into vector form. The model is then tested against a set of known DENV serotype proteins and its performance is evaluated in terms of accuracy, precision, recall value and F1-scores making use of multiple iterative predictions. The outcomes indicate various classifier models are capable of predicting the serotype of various new dengue protein sequences, providing a useful tool for enhancing diagnostic approaches and surveillance strategies. The aim is thus, preparing a sequence classification model such that if an unknown serotype Dengue virus protein sequence is provided, the model can predict with high accuracy which serotype the protein belongs to making use of the serotype defining features embedded in the protein sequences itself. This study may serve as a stepping stone in the knowledge of the sequence variability of DENV proteins and may lead to the development of more accurate tools for controlling dengue virus outbreaks.

GDDB-PP-02

Probing the potential of the immune checkpoint molecule Cytotoxic T lymphocyte antigen-4 (CTLA-4) as a prognostic and therapeutic target in breast cancer

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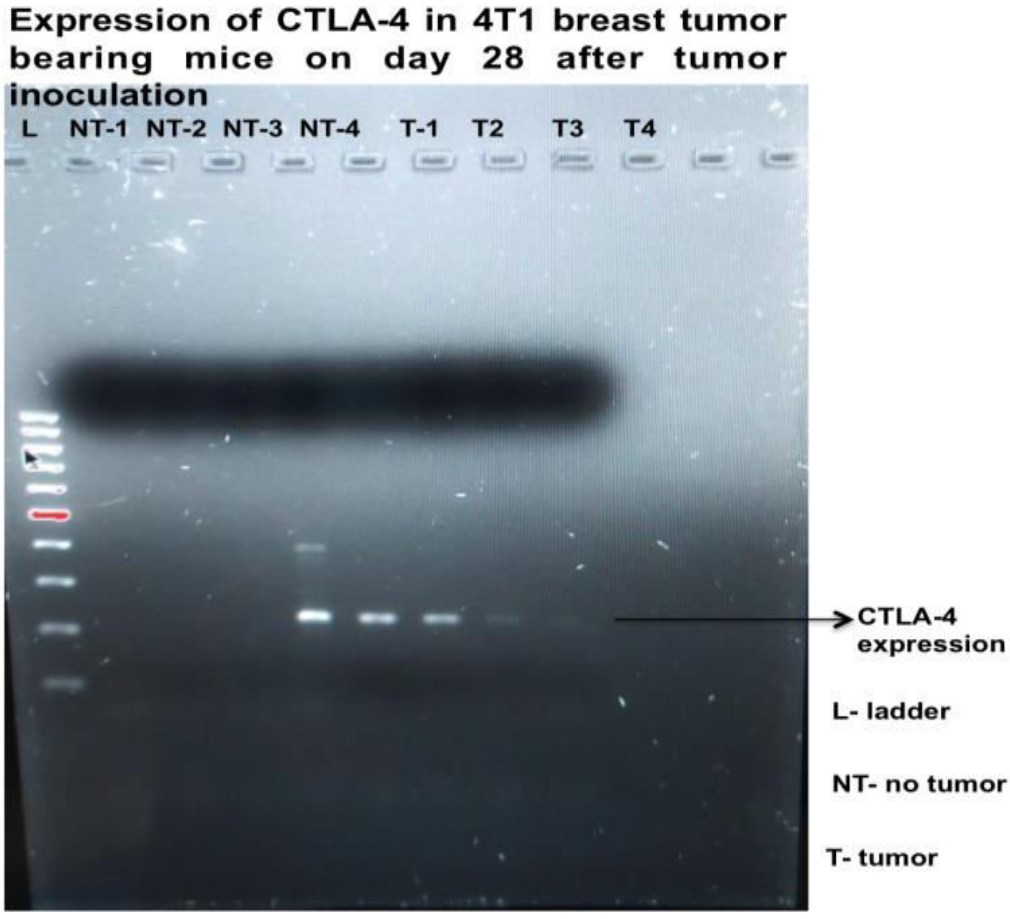
Introduction:
Exploiting the immune landscape of cancer as a prognostic and therapeutic avenue is an emerging area of cancer control. Heightened expression of Immune checkpoint molecules in cancers is correlated with poor prognosis and blocking their expression in antitumor T cells is often accompanied by palpable tumor regression. A lot remains to be explored on the potential of immune checkpoints, often called exhaustion markers in cancer prognosis and cure. Here we explored the expression of the immune checkpoint molecule, CTLA-4 in breast cancer to gain an insight into it's potential as a robust prognostic and therapeutic target in one of the most devastating cancer afflicting our society at present.

Materials and Methods:
10 BALB/c mice (6 weeks age) were inoculated with mice breast cancer cell line 4T1 keeping age matched control. Following tumor inoculation, all the mice were monitored for tumor growth for a period of two months. Once every week following tumor inoculation, blood was collected by retro-orbital puncture and peripheral blood mononuclear cells (PBMC's) were harvested. The PBMCs' were immediately processed for RNA extraction by TRIzol method. The RNA was later used for cDNA synthesis, followed by PCR with to detect the expression of CTLA-4 at various time points.

Results:
We found a striking difference in the expression of CTLA-4 on day 28 following tumor inoculations in tumor bearing mice compared to control. All the tumor bearing mice tested so far showed distinct expression of CTLA-4 while only one of the normal non-tumor bearing mice showed any expression.

Conclusion:
Our data gathered so far clearly points towards the potential of CTLA4 to be used for the dual purpose of breast cancer prognosis and a therapeutic target for cancer cure. Additional exploration will strengthen our preliminary findings.



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GDDB-PP-03

Platelet-poor plasma as a novel autologous approach to target breast cancer stem cells

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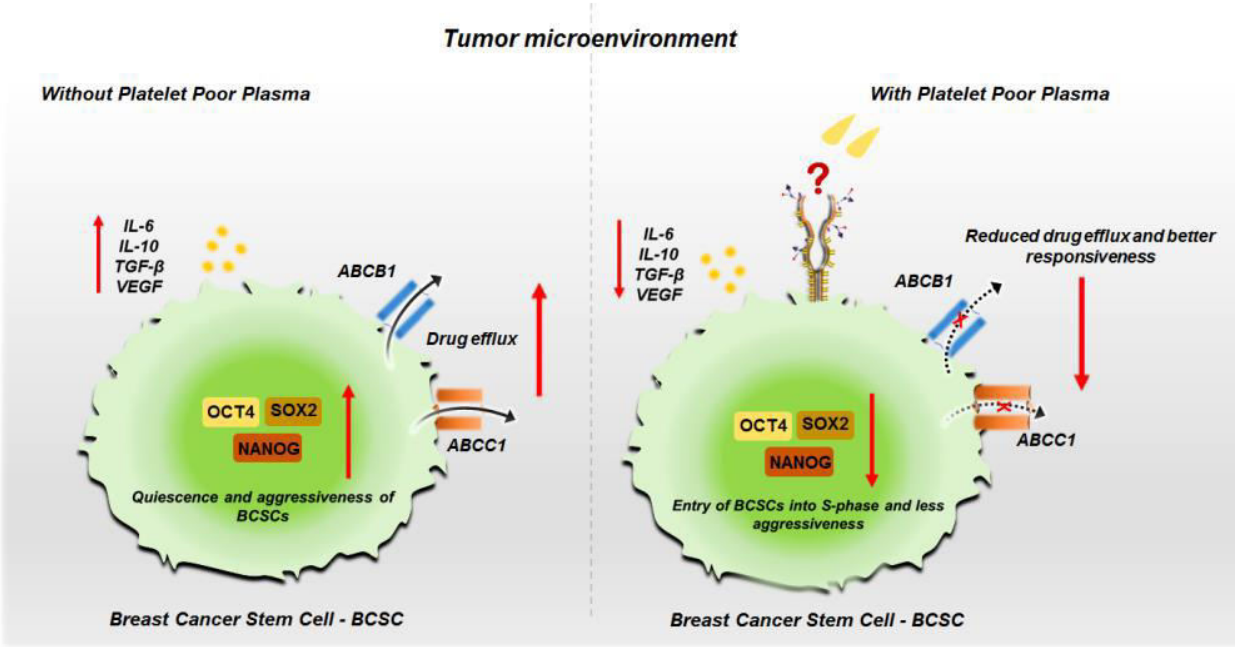
Background:
Breast cancer stem cells (BCSCs) are implicated in tumor recurrence, metastasis and multidrug resistance; posing a major challenge in effective treatment [1]. Targeting BCSCs may significantly improve therapeutic outcomes, especially in both luminal A and triple-negative breast cancer (TNBC) subtypes[1]. This study investigates, for the first time, the effect of platelet-poor plasma (PPP) on stemness, migration, tumorigenicity and drug resistance profiles of BCSCs derived from both luminal A (MCF-7) and TNBC (MDA-MB-231) cell lines, aiming to explore its potential as a clinically feasible therapeutic approach to target the root of breast cancer persistence and progression.

Methods:
$CD44^+/CD24^-$ BCSCs were magnetically sorted and cultured in stem cell-enrichment media to assess spheroid formation over 5 days. Functional assays included mammosphere assay, soft agar colony formation and scratch wound healing assay to evaluate self-renewal, anchorage-independent growth and migration, respectively. Cell proliferations via Ki67 staining and cell cycle progression were assessed by flow cytometry. Gene expression of stemness (*oct4, sox2, nanog*) and drug resistance markers (*abcb1, abcc1*) was quantified using RT-PCR. ELISA was used to analyze cytokine profiles (TGF-β, IL-6, IL-10, VEGF) in culture supernatants.

Results:
PPP treatment markedly reduced mammosphere and soft agar colony formation, indicating impaired self-renewal and tumorigenicity of BCSCs. PPP treated BCSCs exhibited adherent morphology even on non-adherent plates, resembling differentiated parental cancer cells. Further, migration was inhibited as evidenced by reduced wound healing. PPP exposed BCSCs showed a shift from G0/G1 to S-phase with increased Ki67 expression, suggesting loss of quiescence. Stemness genes *oct4* and *sox2* were also significantly downregulated, with a marginal reduction in *nanog*. Notably, PPP also hindered multidrug resistance genes *abcc1* and *abcb1*. Cytokine analysis revealed decreased TGF-β and IL-6, with no significant changes in IL-10 and VEGF levels.



Conclusion:

This study presents compelling evidence that PPP, a blood-derived autologous component [2], effectively targets multiple hallmarks of BCSCs in both luminal A and TNBC breast cancer. Of particular clinical relevance is the observation that PPP demonstrated consistent efficacy in hindering stemness and drug resistance in both hormone receptor-positive (MCF-7) and TNBC (MDA-MB-231) breast cancer models. TNBC, which lacks targeted therapies and is typically more aggressive, stands to benefit significantly from such adjunctive interventions. Translationally, these findings position PPP as a promising, biocompatible and low-cost adjunctive therapy capable of targeting the BCSC population that is often left unaffected by conventional treatments, thereby reducing recurrence risk and enhancing long-term treatment efficacy. Because PPP can be derived from the patient's own blood, it offers an accessible, autologous option with minimal immunogenic risk especially valuable for high-risk or drug-resistant breast cancer patients, including those with TNBC who currently have limited targeted therapies. This paves the way for translational studies and clinical trials exploring PPP as a supportive strategy to sensitize tumors to chemotherap, minimize relapse and improve overall survival outcomes in breast cancer care.

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GDDB-PP-04

Identifying infectious bacterial isolates in food industry wastewater: A step towards restricting the development of infectious diseases in the community health system

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Abstract:
The food industry is one of the fastest expanding manufacturing sectors, a large user of natural resources, and a considerable generator of environmental pollutants and bacterial contaminants, particularly food industry effluents. In many poor countries, the bulk of industrial wastewater is discharged untreated, acting as reservoirs and transmission vectors for pathogenic bacterial regimes, posing a significant threat to community health systems. This study looks at the morphological and biochemical properties of bacterial isolates from food industry effluents and assesses their pathogenic potential using a hemolysin assay. In addition to human health problems, large organic loads and microbial contamination help to degrade aquatic ecosystems and reintroduce deadly bacterial infections. Furthermore, in this digital era, AI-driven models implies to detect important bacterial contamination, significantly improving real-time pathogen identification and their re-emergence to avert the recurrence of bacterial infections. This project seeks to isolate, identify, and describe bacterial strains found in food industry effluents in order to better understand their variety and potential risks considering the renaissance. Therefore, the current findings provides potential insights for identifying infectious bacterial isolates in food sector wastewater can help restrict the development of infectious diseases in the community health system.

Keywords:
Food industry effluent, emerging infectious diseases, community health, AI in wastewater management.

GDDB-PP-05

Impact of flower waste as pivotal bioconstituents in the field of precision medicine and its sustainable utilization

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Abstract:
Marigold (Tagetes spp.), which is often employed in religious activities, contributes significantly to flower waste because it is often discarded untreated, polluting the environment. Historically, early civilizations such as those documented in the Rigveda relied solely on natural plant-based remedies to treat their health concerns. The marigold flower has been used in ayurveda and folk medicine since ancient times as a rich source of bioactive compounds (flavonoids, quercetin, patuletin, carotenoids, lutein, zeaxanthin, and phenolic acids), which have been scientifically proven to have antibacterial, antiinflammatory, and antioxidant properties. This study looks into the conversion of marigold flower waste into high-value drugs that are consistent with ancient wisdom and modern precision medicine practices. Flavonoids and carotenoids has been identified upon phytochemical investigations. Agar well diffusion tests for antimicrobial activities of flower waste extracts have revealed inhibition zones against *Pseudomonas sp*. Molecular docking revealed higher binding affinities of Patuletin and Quercetin (bioactive compounds) with microbial target proteins like LasR and DNA gyrase, indicating quorum sensing inhibition and bacterial DNA replication disruption. The major outcome of this study has shown that marigold flower waste could be used to make valuable bio-products (herbal ointments, antibacterial incense, mosquito repellents, and nutraceutical supplements) in the field of precision medicine. Current study not only honors ancient medicinal techniques, but it also offers an environmentally appropriate solution for floral waste disposal. This work aligns with a circular bioeconomy strategy and eco-friendly health breakthroughs by connecting indigenous knowledge systems to current biomedical sciences and precision medicinal aspects.

Keywords:
Phytochemicals, patuletin, quercetin, circular bio-economy, molecular docking, precision medicines.

GDDB-PP-06

Crosstalk between ciliopathy and cancer: An insight into Plk4 and CEP164 functionalities

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Abstract:
Primary ciliopathies and malignant cancers share overlapping molecular etiologies, implicating defective centrosome and ciliary assembly in certain cancers. The vertebrate centrosome and cilium axis comprises centrosomes, cilia, and centriolar satellites, with the centrioles serving as scaffolds for pericentriolar material recruitment and centrosome assembly. The mature mother centriole is repurposed as a basal body to nucleate primary cilia, which project from the apical surface and have sensory functions. Centrosome duplication initiates in S phase, yielding two centrosomes that form the bipolar mitotic spindle for accurate chromosome segregation. In terminally differentiated cells, centrioles dock at the plasma membrane and undergo ciliogenesis, establishing primary cilia as signalling hubs for Hedgehog and Wnt pathways. Centriole maturation spans nearly two cell cycles, producing three generations of centrioles per cell, although only the oldest gains competence for ciliogenesis. Studies have shown that Polo-like kinase 4 (Plk4) activity is important for progressive biochemical maturation and appendage assembly on nascent centrioles; Plk4 inhibition affects appendage protein accumulation, whereas unscheduled Plk4 activation accelerates centriole maturation and ciliary assembly, effectively abolishing centriole age asymmetry. CEP164, a distal appendage marker recruited during late G2/early M phases, is essential for appendage assembly and ciliogenesis as its presence distinguishes centrosomes formed by cytokinesis failure from those generated by centriole overduplication; the latter lack CEP164. Autosomal recessive mutations in CEP164 underlie nephronophthisis type 15 (NPHP15), causing defective ciliogenesis in renal epithelial cells. It has been seen that Plk4-overexpressing cells showed reduced CEP164-positive centrosomes, whereas cytokinesis-failure cells displayed increased CEP164 levels. We hypothesize that Plk4 mutations can perturb CEP164 recruitment, thereby coupling ciliopathic kidney dysfunction with oncogenic centrosome amplification. These findings support a mechanistic link to our hypothesis and suggests that there could be a functional link between Plk4 and CEP164. Thus, it is implied that there is an underlying connection between ciliopathy and cancer.

Keywords:
Ciliopathy, cancer, centriole duplication, ciliogenesis.

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GDDB-PP-07

Identification of estradiol-based pharmacophores: Potent alternatives for impaired estrogen receptor binding due to endrocrine disruptors

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Abstract:
The endocrine system of the body bears the responsibility of the producing, storing and secretion of hormones which are required for maintaining the metabolism, fertility, growth, sleep, and emotion of the body. Endocrine disruptors (EDs) are a class of non – natural chemical compounds, present in all kinds of cosmetics, pesticides, industrial solvents and lubricants, plastics and even in clothing items, which interrupts the normal functioning of the hormones inside the body leading to infertility, early puberty, less immunity, metabolic issues, obesity, heart disease, growth and learning disorder and even cancer. Therefore, it is a matter of concern to predict the effect of EDs [1,2]. In this study, Estrogen alpha (ESRα) (PDB ID: 1A52) receptor was docked against natural hormone Estradiol and non-natural chemical compounds Bisphenol A, and Bisphenol B. The non – natural chemical compounds were having binding affinities for ESRα comparable to that of Estradiol for ESRα (Table 1). Therefore, this preliminary study confers the fact that the non – natural chemical compounds compete with natural hormone to interact with the ESRα receptor and interfere with the normal functioning of the hormones. Therefore, we have aimed to predict some Estradiol-like pharmacophores which can fulfil the function of Estradiol when its activity is impaired by EDs. We have found four pharmacophores using ZINCPharmar database which were docked against ESRα and they were having binding affinities for ESRα comparable to that of Estradiol for ESRα (Table 1). Therefore, this in – silico structure-based drug discovery approach can be valuable in sketching the in - vitro assessments for predicting the potential Estradiol hormone like drugs that can be used when Estrogen receptor binding activity is impaired by EDs.

Table 1. Binding Affinities of different types of compounds for ESRα

Type of Compound	Name of Compound	Binding Affinity for ESRα (kcal/mol)
Natural Hormone	Estradiol	-7.7
Endrocrine Disruptor	Bisphenol A	-8.1
	Bisphenol B	-8.2
Pharmacophores	Compound No.1	-7.6
	Compound No.2	-7.8
	Compound No.3	-7.9
	Compound No.4	-7.6

Keywords:
Endocrine disruption, hormonal disorder, estrogen receptors, molecular docking, binding energy.

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GDDB-PP-08

In silico analysis of global genetic diversity linked to antimicrobial resistance and virulence in *Klebsiella pneumoniae*

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Background:
Klebsiella pneumoniae is a clinically significant gram-negative pathogen responsible for severe hospital-acquired infections. The emergence of multi-drug resistant (MDR) and hypervirulent strains poses a global public health threat, necessitating genomic surveillance to unravel the molecular determinants of antibiotic resistance and virulence. This study aimed to perform an *in-silico* analysis of the global genomic diversity of *K. pneumoniae*, focusing on sequence types (STs) and their association with antibiotic resistance genes (ARGs) and virulence factors (VFs).

Methods:
A total of 1,665 *K. pneumoniae* genomes were retrieved from BV-BRC database and coding sequences were downloaded from NCBI. Multi-Locus Sequence Typing (MLST) was conducted using seven housekeeping genes following the Institut Pasteur MLST scheme. ARGs and VFs were identified using the CARD Resistance Gene Identifier tool and VFDB's VFanalyzer, respectively.

Results:
MLST was accomplished for 1,585 genomes, revealing 279 distinct STs. Globally, ST11 (23%), ST15 (5%), and ST258 (4%) were found to be the most prevalent, with ST231 emerging as the most common in India. Association analysis of STs with ARG and VF profiles highlighted 10 STs (ST11, ST14, ST15, ST23, ST147, ST258, ST268, ST307, ST392 and ST1) harboring the highest numbers of resistance and virulence determinants. Notably, ST11—being identified as an emerging carbapenem-resistant clone—was found to be linked with higher mortality rates, especially in China. Furthermore, high-risk clones like MDR ST14 and ST15, extensively drug-resistant (XDR) ST147, and hypervirulent ST23 were discovered to be widely spread all over the world, highlighting their significant threat to public health. ST147 is a Multidrug-resistant clone while ST258 has Carbapenemase, ST268 is hypervirulent, ST307 is a high-risk clone, ST392 has OXA-481 gene and ST1 is Colistin-resistant.ST231, mostly predominant in India, is a carbapenem-resistant clone, with the presence of blaOXA-48-like and blaNDM-1/5 genes in a significant proportion of isolates highlighting the challenge of treating infections caused by this ST.

Conclusion:
This study provides valuable insights into the global sequence diversity of *K. pneumoniae* and its linkage to antimicrobial resistance and virulence. Further research involving clonal complex identification, pan-genome analysis, and evolutionary dynamics is essential to guide predictive modeling and improve MDR outbreak preparedness.

Keywords:
Klebsiella pneumoniae, Multi-Drug Resistance, Virulence Factors, Multi-Locus Sequence Typing, *In Silico* Genomics, Antibiotic Resistance Genes, Nosocomial infections.

GDDB-PP-09

Loss-of-function mutations and *Helicobacter pylori* pathoadaptation

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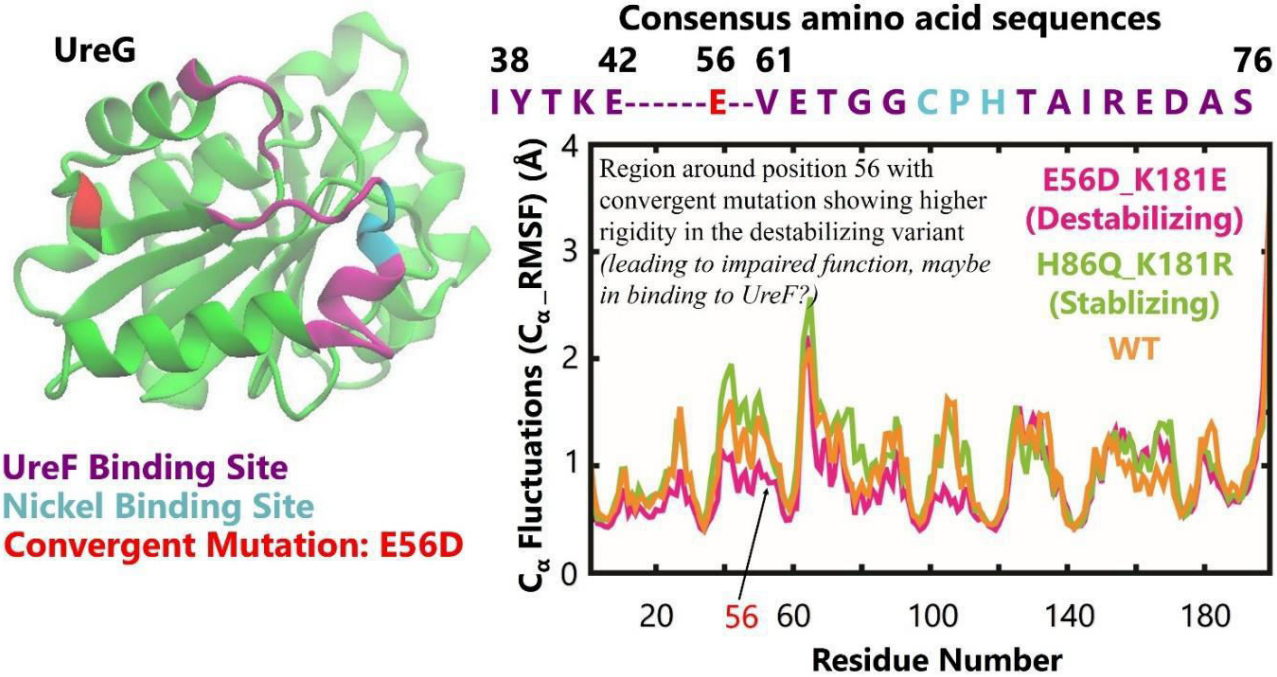
Abstract:
One common mode of bacterial adaptive evolution is the inactivation of specific genes, functional presence of which may appear detrimental to the organism in some altered environmental conditions, following 'die-or-lose' dynamics, via accumulation of either non-synonymous mutations or truncation mutations (TnM) leading to a premature stop of the translation process. We aimed to assess the potential pathoadaptive contribution of loss-of-function mutations in *Helicobacter pylori*, a rapidly evolving human-adapted organism known for its colonization in harsh acidic environments and association with gastritis, peptic ulcers, and gastric cancer. Our in-silico study on 346 complete *H. pylori* genomes detected both TnM and convergent non-synonymous mutations (CM) in a set of genes. While CM occurrence is thought to be a powerful adaptive marker, we hypothesized the overlap of CM and TnM to be an accumulation of potentially adaptive loss-of-function mutations in specific isolates.[1-3] Interestingly, one of these genes encodes UreG, essential for functional urease that helps *H. pylori* colonize and survive in the acidic gastric environment. Importantly, the CMs at position 56, flanked by two UreF-binding domains, were found to considerably destabilize the protein structure, showing significantly higher rigidity than the wild type in the molecular dynamics simulation analysis, similar to what detected for truncated variant. If this lack of conformational stability and flexibility indicate non-functionality of the corresponding variants, we find that ~7% of our analysed isolates possibly harbour inactivated *ureG* gene, majority (~65%) representing peptic ulcer patients. Future experimental studies are warranted to understand potential fitness impact of such inactivation events.



Keywords:
ureG, *Helicobacter pylori*, molecular dynamics simulation, truncation mutations, convergent mutations.

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GDDB-PP-10

ST152: An overwhelmingly predominant *Shigella sonnei* clone circulating worldwide with distinct antibiogram profiles across countries

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Abstract:
Shigella sonnei, one of the *Shigella* spp. causing diarrheal disease termed shigellosis, is tremendously expanding in industrializing regions globally, showing extensive drug resistance (XDR). We extracted 1269 publicly available *S. sonnei* having known antibiogram profile information. Of these, >85% strains were isolated from 5 countries – Australia (358), UK (270), China (255), India (118) and Vietnam (110) – showing isolation of >100 strains for each country. Interestingly, majority (>96%) of these isolates represented a single clone, i.e., ST152. However, the isolates of this single clone across countries demonstrated distinct resistance patterns to any specific set of antibiotics. According to WHO and ICMR guidelines, ciprofloxacin (quinolone) is the first-line treatment, followed by ceftriaxone (cephalosporin) and azithromycin (macrolide) as second-line options. Indian and Vietnamese isolates showed high resistance to ciprofloxacin and nalidixic acid, indicating the inefficacy of first-line treatment. UK isolates also demonstrated rising resistance to quinolones, while Australian strains showed a gradual increase. For macrolides, high azithromycin resistance was observed in Chinese and Australian isolates, whereas it was found effective against UK strains. Regarding quinolones, Chinese and Vietnamese isolates were highly resistant to ceftriaxone, with some alternatives still effective in China. Conversely, Indian and Australian isolates showed lower resistance to cefotaxime and ceftriaxone, respectively, while UK isolates exhibited increasing resistance to multiple cephalosporins. The antibiogram profile of different isolates varies greatly, suggesting region-specific antibiotic profiles of circulating *S. sonnei*. This clearly indicates the need for a country-specific regular assessment and dynamic policymaking for antibiotic selection against shigellosis, in contrast to any generalized choice as currently directed by the World Health Organization (WHO).

Keywords:
Shigella sonnei, shigellosis, ST152, antibiotic resistance/susceptibility profiling, Country-specific policy.

GDDB-PP-11

Clonal co-evolution of *E. coli* core genes potentially linked to uropathogenicity

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Abstract:
Urinary tract infections (UTIs), occurring in any part of the urinary tract (urethra, bladder, ureters, kidneys), represent one of the most prevalent diseases in patients ranging from neonates to geriatric age groups, with a lifetime occurrence risk of 60% in every woman and 13% in every man. The high incidence of virulence complications is severely associated with increasing antibiotic resistance, especially in *Escherichia coli* causing >60% of the cases worldwide. To combat the disease, via antibiotic treatment optimization in particular, one key aspect would be to decipher the positive selection footprints of urovirulence and/or antibiotic resistance. Our study involves tracking of such signatures through mutational convergence where adaptive pressures lead to the repeated, independent amino acid changes in the same structurally/functionally crucial positions of specific proteins important for increased uropathogenesis and/or antibiotic resistance. Using publicly available completely sequenced genomes from 525 fecal and urine isolates across the globe, we performed core genome profiling and clonal distribution via multilocus sequence typing (MLST). Next, we searched for core genes co-evolving via potential adaptive mutational convergence exclusively in uropathogenic isolates across a set of specific clones. We found 28 genes that exhibited convergent mutations solely among the urine isolates. Out of these, 2 sets of isolates were noted where a total of 6 genes were identified as being consistently associated with specific combinations of clones based on MLST. Of these, 4 genes, i.e., *gcvP, hypF, mdtK* and *sltY* showed convergent mutations within the set of same isolates belonging to ST219 and ST2847, while 2 other genes, i.e., *alaS* and *flK* expressed convergent mutations in another set of same isolates belonging to ST538 and ST429. As we are in the process of detailed in-silico analysis of the proteins encoded by these 6 genes with an emphasis on the naturally occurring convergent mutations to assess any contribution of such mutational convergence on urovirulence in the corresponding isolates, functional assessment of these uropathogenic *E. coli* (UPEC) specific mutations along with ongoing findings of other co-evolving genes genome-wide can offer important insights on adaptive evolution of *E. coli* as uropathogens.

GDDB-PP-12

Serum triglycerides estimation and its association as marker in diabetic nephropathy – Prospect for genetic insight

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Introduction:
Type 2 Diabetes Melitus (DM) is a heterogeneous and polygenic disease. With duration and severity Diabetes causes a variety of minor and major complications including nephropathy. Till date while glycated hemoglobin demarcates severity, urinary microalbumin indicates renal impairment. Potential predictor of diabetic nephropathy was explored in this prospective observational study. Although the understanding of the genetic architecture of Type 2 DM has exhibited considerable progress in the past few years, there may be still many additional genetic variants that might be associated with the pathogenesis of Type 2 DM and further studies are warranted to unveil the underlying pathophysiological mechanisms associated with dyslipidemia in these individuals.

Methodology:
Following WHO criteria, 100 diabetic patients were recruited and categorised as moderate (n=43) and severe (n=57) based on glycated haemoglobin (8%) level. Duration of disease, BMI, systolic and diastolic BP, Fasting and Post Prandial blood glucose, glycated haemoglobin, serum lipid profile and urinary micro albumin were recorded. The groups were compared for these parameters. Correlation was estimated. With most significant parameter, ROC curves were constructed for finding suitable cut-off value for demarcating the severity and detecting micro-albuminuria.

Results:
Significant differences were recorded for blood glucose, glycated haemoglobin, triglyceride and microalbuminuria but not for other parameters. Significant association of glycated haemoglobin was displayed with triglyceride, fasting and post prandial glucose and urine microalbumin levels. ROC curve of triglyceride level showed better performance (AUC= 0.97) than microalbuminuria (AUC= 0.88) to demarcate severity of diabetes; at a cut-off of 285mg/dl, triglyceride showed 93% sensitivity and specificity. At 252 mg/dl, 90% sensitivity and 94% specificity were displayed for detection of microalbuminuria.

Conclusions:
Triglyceride level corresponds to severity of the disease pathology and might be considered as potential predictor of diabetic nephropathy which in turn may give further scope to study the genetic susceptibility of dyslipidemia in diverse diabetic individuals corresponding with deranged glycemic status. In type V familial hyperlipidemia there is an increase in both chylomicrons and very low-density lipoprotein (VLDL). Both of the lipoproteins are hydrolyzed by lipoprotein lipase(LPL). However, as per studies, this is seen to be mainly caused by mutations in APOA5. APOA5 plays a role in stablizing the APOC2-LPL complex, which is needed to hydrolyze VLDL and chylomicrons. In this context to explore Apo A5 mutation in hypertriglyceridemic patients with diabetes mellitus will give a novel prospect in future.

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GDDB-PP-13

Comparative analysis of clonal diversity and associated antimicrobial resistance and virulence of *Staphylococcus aureus* strains from human and non-Human hosts

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Background:
Staphylococcus aureus is a major opportunistic pathogen capable of infecting both humans and animals. The acquisition of resistance genes and diverse virulence factors enables it to adapt across host species and cause a wide range of infections. The global rise of antimicrobial resistance (AMR) in *S. aureus* threatens treatment efficacy, while its virulence factors contribute to persistent infections. Though methods like Multi Locus Sequence Typing (MLST) have enhanced our understanding of classification and differentiation of *S. aureus* strains based on their genetic diversity, but the interplay between resistance, virulence, and sequence types (STs) remains underexplored. A comparative analysis of these elements is essential to uncover host-specific adaptations, track the evolution of high-risk clones, and inform strategies for surveillance, infection control, and therapeutic development.

Methods:
Complete genomes of *S. aureus* strains from human (n=666) and non-human (n=244) hosts were retrieved from BV-BRC and filtered for quality and annotation. MLST analysis (Oxford scheme) was done via PubMLST with 90% identity and coverage. ARGs were identified using RGI (CARD database) and virulence factors via BLASTn against VFDB (85% identity and coverage). Custom Python scripts generated abundance matrices for both ARGs and virulence genes.

Results:
MLST analysis identified 92 distinct STs in 564 out of 666 *S. aureus strains* with human-host and 81 distinct STs in 236 of 244 *S. aureus* strains with non-human-host. ST5 (17%), ST8 (16%), and ST30(6.5%) dominated strains with human host, while ST1292 (15.6%), ST8 (15.25%), and ST474(5.5%) were most common in strains with non-human host. *S. aureus* strains isolated from human hosts had a higher prevalence of ARGs against the drug classes Fluoroquinolones (99.8%), beta-lactams (99.3%), and tetracyclines (99.1%). The majority of these strains with human-host were isolated from Asia and Africa. Conversely, strains from non-human hosts, majorly isolated from Europe, showed the predominant presence of ARGs against the drug class fluoroquinolones. The majority of the human-associated strains, isolated from Asia, also exhibited the prevalence of VFs linked to immune modulation, exotoxin production, and biofilm formation. Correlation analysis of STs with ARGs and VFs revealed, ST5 and ST8 from human-associated strains have a high abundance of both ARGs and VFs indicating elevated pathogenic potential in humans, whereas, in non-human hosts, ST1292 and ST8 showed relatively higher presence of ARGs and VFs, indicating potential zoonotic threat.

Conclusion:
This study highlights the correlation of ST-, ARG- and VF- profiles of *S. aureus* across human and non-human hosts as well as their geographical disposition. Human-associated strains exhibit greater antibiotic resistance and virulence potential, particularly for ST5 and ST8, underscoring clinical concern, while the antibiotic resistance and virulence pattern in non-human strains reveal the importance of environmental reservoirs in the spread of ARGs and aid in the development of improved infection management strategies.

Keywords:
Staphylococcus aureus, Antibiotic resistance genes, Virulence factor genes, Sequence Types, Host-specificity

GDDB-PP-14

Diversity and evolution of c-di-AMP signaling genes and their role in mycobacterial stress adaptation

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Background:
The Mycobacterium genus comprises a diverse array of bacterial species occupying a wide range of ecological niches, including human and animal hosts as well as environmental reservoirs. While it includes well-known pathogens such as *Mycobacterium tuberculosis* and *M. leprae*, a growing number of nontuberculous mycobacteria (NTM), notably the *Mycobacterium avium* complex (MAC) and *Mycobacterium abscessus* complex (MABC), have emerged as significant opportunistic pathogens. This ecological and pathogenic diversity reflects the evolution of distinct physiological traits and molecular strategies, particularly in the realm of environmental sensing and stress adaptation. Central to these processes are small molecule secondary messengers, such as cyclic di-adenosine monophosphate (c-di-AMP), which regulate vital bacterial functions including cell wall integrity, stress response, biofilm formation, and virulence. Although c-di-AMP signaling has been partially explored in *M. tuberculosis*, its distribution and functional role across other mycobacterial species remain largely under-characterized. In this study, we conducted a comprehensive bioinformatic analysis of c-di-AMP signaling components across the Mycobacterium genus, revealing both conserved and divergent features. Additionally, we investigated the functional role of c-di-AMP in stress adaptation in *M. smegmatis* through experimental approaches, providing new insights into the broader regulatory significance of this signaling molecule in mycobacterial biology.

Methods:
A total of 214 genomes representing five Mycobacterium-related genera—Mycobacterium, Mycobacteroides, Mycolicibacter, Mycolicibacillus, and Mycolicibacterium, were analyzed to construct a core gene-based phylogenetic tree and identify genes involved in the c-di-AMP signaling pathway (DisA, PDE and AtaC) using BLASTP with custom scripts. Conserved protein domains were identified using the SMART web tool. Genetic diversity was assessed by calculating average nucleotide diversity (π) and substitution rates (dN/dS). For experimental validation, knockout mutants of *M. smegmatis* lacking DisA and PDE were generated via homologous recombination using a pPR27-based recombinant cassette carrying a kanamycin resistance marker. These mutants were subjected to various stress conditions (SDS, H_2O_2, sodium citrate) and assessed through spot dilution assays on agar plates.

Results:
The genes DisA, PDE, and AtaC were identified in 90.64%, 96.73%, and 90.19% of the analyzed Mycobacterium genomes, respectively. The MTBC and MABC groups lacked the AtaC gene, indicating its possible specificity to the Mycobacteroides genus. In contrast, DisA was absent across all Mycolicibacter genomes, including *Mycobacterium novum*. Truncated versions of DisA and PDE were observed in the reduced genomes of *M. leprae*, *M. lepromatosis*, and *M. uberis*, likely due to genome erosion associated with their obligate intracellular lifestyle.

Genetic diversity analysis showed that DisA had a nucleotide diversity (π) within the range observed for core genes, while PDE displayed moderately higher diversity. AtaC exhibited the highest variability among the three. Synonymous and non-synonymous substitution rate patterns mirrored these findings, with AtaC showing significantly elevated rates compared to DisA and PDE.
In functional assays, MSPDE knockout mutant demonstrated a significant growth defect compared to the wild type, underscoring the importance of this signaling pathway. Under various stress conditions, the Δpde strain showed an approximately one-log reduction in survival under oxidative stress (H_2O_2), surface stress (SDS), and osmotic stress (sodium citrate), with the most pronounced defect observed in the presence of sodium citrate.

Conclusion:
The phylogenetic and evolutionary analyses revealed widespread retention of functional DisA and PDE genes like other core genes and loss in course of genome reduction in several genome. Many genomes also harbored hybrid diguanylate cyclase-phosphodiesterase genes. Stress survival assays showed MSPDE mutants were sensitive to oxidative, detergent and osmotic stress highlighting crucial role of the c-di-AMP signaling pathway in maintaining mycobacterial physiology, particularly under stress conditions.

Keywords:
Small molecule signaling, cyclic di-AMP, Phylogenetic analysis, *Mycobacterium tuberculosis*, stress adaptation

GDDB-PP-15

Dynamic visualization of c-di-AMP signaling in Mycobacteria using a FRET-based biosensor

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Background:
Cyclic di-AMP has recently emerged as a broadly conserved second messenger critical for microbial growth and physiology [1]. It influences stress responses, antibiotic resistance, cell morphology, bacterial proliferation, and virulence [2]. Synthesized from ATP by di-adenylate cyclase (DAC) domain-containing proteins and degraded into pApA or AMP by phosphodiesterases (PDEs), c-di-AMP also plays an important role during infection. Pathogens such as *Listeria monocytogenes*, *Mycobacterium tuberculosis* (Mtb), and *Chlamydia trachomatis* secrete c-di-AMP via multidrug resistance (MDR) transporters, eliciting an IFN-β-mediated immune response in the host [3]. In Mtb, c-di-AMP has been implicated in virulence, although the underlying mechanisms remain unclear. Accurate and real-time detection of c-di-AMP is essential to uncover its role in key cellular processes such as cell wall homeostasis, osmoregulation, and host-pathogen interactions. A reliable biosensor will provide sensitive, specific, and temporally resolved insights into c-di-AMP dynamics, particularly during bacterial cell division at the single-cell level. This biosensor will also aid in identifying environmental stimuli that activate c-di-AMP signaling in mycobacteria, advancing our understanding of how this pathway is regulated. Such knowledge could ultimately inform strategies to modulate the outcome of Mtb infection. Additionally, mapping host cells that sense c-di-AMP will help elucidate how bacterial secretion of this molecule contributes to innate immune activation.

Method:
To develop the FRET-based c-di-AMP biosensor, a truncated version of the c-di-AMP binding protein Lmo0553 from *Listeria monocytogenes* was fused between two fluorophores, eCFP and eYFP. This biosensor was expressed in *Mycobacterium smegmatis* under various promoters and studied by FACS and fluorimeter analysis. Mutant strains of *M. smegmatis* lacking the *disA* (ΔdisA) or *pde* (Δpde) genes were generated via homologous recombination. FRET signal changes were measured in these mutants to assess variations in intracellular c-di-AMP levels. Additionally, various environmental conditions were applied to evaluate the biosensor's response to fluctuations in c-di-AMP concentration. Bacterial survivability under stress was assessed by counting colony-forming units (CFUs) in treated and untreated samples.

Results:
Optimum expression of the biosensor was observed under tetracycline inducible promoter. The highest percentage of cells showing FRET activity was observed in *pde* deletion mutant strains because in the absence of phosphodiesterase amount of cyclic-di-AMP inside the bacterial cell is high. On the other hand, the lowest percentage of cells showing FRET activity was observed in *disA* deletion mutant strains because in the absence of DisA amount of cyclic-di-AMP inside the bacterial cell is low.

To identify environmental conditions that influence intracellular c-di-AMP levels, *Mycobacterium smegmatis* cells expressing the c-di-AMP biosensor were treated with various osmolytes, antibiotics, DNA-damaging agents, and membrane-disrupting reagents. Among these treatments, significant changes in the FRET ratio were observed under phosphate-depleted conditions, in the presence of 1 M NaCl, and at pH 4. These results indicate that the intracellular concentration of c-di-AMP is altered in response to these specific stress conditions.

Conclusion:
This study demonstrates that the FRET-based biosensor reliably detects changes in intracellular c-di-AMP levels in *Mycobacterium smegmatis*. Mutant analysis revealed elevated c-di-AMP levels in the Δpde strain and reduced levels in the ΔdisA strain. Significant FRET signal changes were also observed under phosphate starvation, high salt (1 M NaCl), and acidic pH (pH 4), indicating that these stress conditions modulate c-di-AMP levels. CFU analysis confirmed that these changes were not due to cell death. Using this biosensor, we have identified—for the first time—the environmental signals that modulate c-di-AMP levels in mycobacteria. Overall, the biosensor serves as a valuable tool for studying c-di-AMP dynamics under various stress conditions.

Keywords:
Mycobacterium smegmatis, second messenger signaling, Cyclic-di-AMP, Biosensor, Stress response, FRET

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GDDB-PP-16

Understanding the role of cyclic-di-AMP in potassium transport of *Mycobacterium tuberculosis*

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Background:
Cyclic di-AMP (c-di-AMP) is an essential second messenger involved in regulating potassium homeostasis, virulence and other physiological processes in many bacterial species. While its role in potassium regulation is well established in organisms such as *Bacillus subtilis* and *Listeria monocytogenes*, its function in *Mycobacterium tuberculosis* (Mtb) is not yet fully understood. In Mtb, c-di-AMP is synthesized by diadenylate cyclase (DAC) and degraded by phosphodiesterase (PDE), and it plays a broader role in bacterial survival, growth, and virulence. Potassium is a critical ion for maintaining cellular functions such as osmoregulation, membrane potential, and enzymatic activity. Mtb possesses two key potassium transport systems—TrkA and KdpD—that are believed to be regulated by c-di-AMP. Our study focuses on investigating the interaction between c-di-AMP and these transporters, including how c-di-AMP levels influence transporter activity and the expression of the *kdpFABC* operon. By studying these mechanisms in both Mtb and *Mycobacterium smegmatis* (*Msmeg*), we aim to elucidate the role of c-di-AMP in mycobacterial potassium homeostasis. A better understanding of this signaling pathway could provide new insights into Mtb physiology and identify potential targets for novel anti-tuberculosis therapies.

Methods:
Overexpression and purification of *kdpD* and *trkA* from *M. smegmatis*, and *kdpD*, *ceoB*, and *ceoC* from *M. tuberculosis* were carried out. To assess the interaction between c-di-AMP and potassium transporters, pull-down assays were performed using 2′-AHC-c-di-AMP agarose (BioLog) with the purified proteins. Additionally, intracellular c-di-AMP levels were modulated to evaluate their impact on transporter activity and *kdpFABC* operon expression, aiming to elucidate the role of c-di-AMP in potassium homeostasis in mycobacteria.

Results:
Among all the potassium transport-related proteins analyzed, we identified specific binding of cyclic di-AMP exclusively with KdpD from *Mycobacterium tuberculosis*. Mutational analysis confirmed the specificity of this interaction. Furthermore, using Δpde and ΔdisA strains with altered intracellular c-di-AMP levels, we demonstrated that this binding directly affects the expression of the *kdpFABC* operon, linking c-di-AMP signaling to potassium homeostasis regulation in Mtb.

Conclusion:
Our study reveals that c-di-AMP specifically binds to KdpD in *Mycobacterium tuberculosis*, and this interaction negatively affect the expression of the *kdpFABC* operon in potassium limited condition. These findings establish a direct link between c-di-AMP signaling and potassium homeostasis in Mtb, offering potential targets for new tuberculosis therapies.

Keywords:
Cyclic-di AMP signaling, KdpD, potassium transport system, *Mycobacterium tuberculosis*

GDDB-PP-17

Longitudinal analysis of the composition and dynamics of the respiratory tract microbiome in ICU patients undergoing antibiotic treatment

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Background:
Patients admitted to Intensive Care Units (ICUs) are frequently administered broad-spectrum prophylactic and empiric antibiotics soon after admission to treat or prevent infection. However, antibiotic use can unintentionally disrupt the commensals of the respiratory tract. Despite its clinical relevance, the dynamics of microbial composition in this setting, and its potential association with disease progression, remain poorly understood. This study investigates the longitudinal changes in microbial diversity and taxonomic composition of the upper respiratory tract microbiota in ICU patients diagnosed with conditions such as chronic obstructive pulmonary disease (COPD) and pneumonia, across defined antibiotic treatment windows. Understanding microbiome responses to antibiotic regimens may offer valuable insights for optimizing therapy and managing complications in critically ill patients with respiratory illnesses.

Methods:
Throat swab samples (n = 86) were collected longitudinally from 21 ICU patients across three antibiotic treatment stages: (a) prior to antibiotic administration (Stage-0), (b) 1–3 days after antibiotic initiation (Stage-1), and (c) 4–15 days post-initiation (Stage-2). DNA was extracted, and the V3–V4 region of the 16S rRNA gene was sequenced for 52 samples. Taxonomic profiling and alpha/beta diversity analyses were performed using QIIME2, while DESeq2 was used to identify differentially abundant taxa across stages.

Results:
Microbial diversity showed a significant decline over time following antibiotic administration. A comparison between Stage-0 (n = 21) and Stage-2 (n = 14) revealed a marked reduction in alpha diversity (Shannon index, $p = 0.001$), and significant changes in community composition (weighted UniFrac beta diversity, $p = 0.003$). At the genus level, *Haemophilus*, *Ligilactobacillus*, and *Aggregatibacter* were significantly more abundant in Stage-0, while *Acetobacter* and *Fannyhessea* were enriched in Stage-2.

A focused analysis of a subset of 10 patients with samples across nearly all stages (n = 27) supported these trends. A progressive decline in diversity was observed from Stage-0 to Stage-1 ($p = 0.033$) and Stage-0 to Stage-2 ($p = 0.018$), along with significant shifts in microbial composition from Stage-0 to Stage-2 ($p = 0.002$). Differential abundance analysis revealed that taxa such as *Haemophilus*, *Nanogingivalis*, *Gemella*, and *Eubacterium* were more prevalent at Stage-0, while *Acetobacter*, *Fannyhessea*, *Lactobacillus*, and *Klebsiella* were enriched in Stage-1 and Stage-2.

Conclusion:
This longitudinal study highlights that antibiotic treatment in ICU patients leads to significant, time-dependent alterations in the upper respiratory tract microbiota. The observed reduction in microbial diversity and shifts in key taxa suggest that antibiotic exposure disrupts microbial balance, potentially affecting disease trajectory and patient outcomes. These findings underscore the importance of microbiome-aware clinical practices in critical care settings.

Keywords:
16S rRNA sequencing, ICU patients, respiratory microbiota, antibiotics, microbial diversity, differential abundance, DESeq2, QIIME2, PICRUSt2.

GDDB-PP-18

TransBGC: A transformer-driven deep learning framework for high-accuracy classification of microbial biosynthetic gene clusters in genomes

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Background:
Microbial Biosynthetic Gene Clusters (BGCs) encode pathways for diverse bioactive compounds, including antibiotics, anticancer agents, and immunomodulators. Despite advances in genome sequencing, current BGC prediction tools face challenges in accurate classification, discovery of novel cluster types, and biological interpretability. Specifically, existing methods often fails to identify conserved sequence features critical for distinguishing BGC classes.

Methods:
In order to address these limitations, we developed TransBGC, a transformer-based deep learning framework for interpretable classification of microbial BGCs. The sequences of BGCs were retrieved from the MIBiG database, comprising 1,910 reference entries. These were curated to retain 1,582 single-class BGCs to ensure unambiguous class labelling. Each sequence was encoded as 4,096-dimensional 6-mer frequency vectors. These vectors were used to train a deep neural network for multi-class classification across six major BGC types: Alkaloid, Nonribosomal Peptide (NRP), Polyketide, RiPP, Saccharide, and Terpene. Post-training, SHAP (SHapley Additive exPlanations) analysis was employed to interpret the model and identify discriminative k-mer features underlying class predictions.

Results:
TransBGC achieved high classification performance across all BGC classes. Alkaloid BGCs showed the highest precision (0.97), recall (0.96), and F1-score (0.98), followed by Saccharide (F1-score: 0.89) and RiPP (F1-score: 0.82). Moderate results were observed for NRP (F1-score: 0.83) and Polyketide (F1-score: 0.74), while Terpene BGCs presented the greatest challenge (F1-score: 0.75). The results demonstrate effective classification for conserved BGC classes like Alkaloids and Saccharides, with variability in performance reflecting inherent challenges in structurally diverse classes such as Terpenes and Polyketides. Subsequently, each class specific key sequence patterns were also identified.

Conclusion:
Our study introduces a robust and interpretable deep learning framework for accurate BGC classification from genomic data. By identifying key sequence patterns associated with BGC types, TransBGC enhances both prediction accuracy and biological insight. This approach offers valuable tools for genome mining and natural product discovery, with future directions including application to metagenomes, uncharacterized genomes and integration of functional annotations for improved classification.

Keywords:
Biosynthetic Gene Clusters (BGCs), Deep Learning, Multi-Class Classification, Transformer Model, SHAP, MIBiG Dataset

GDDB-PP-19

Dual-channel fluorescent probe for simultaneous monitoring of polarity and viscosity in live-cell nucleus

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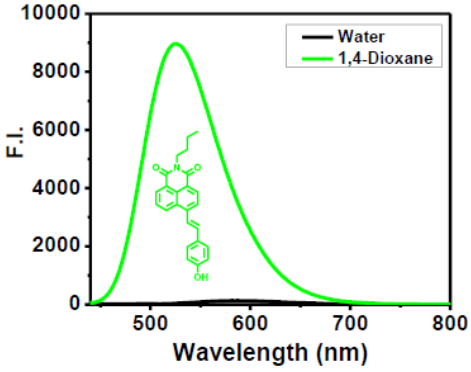
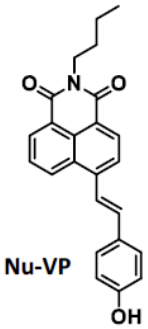
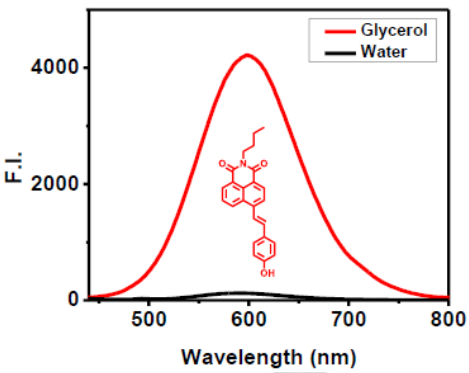
Abstract:
Nuclear polarity and viscosity are critical for maintaining proper nuclear function, including gene expression, chromatin organization, and nucleocytoplasmic transport. Disruptions in these physical properties are associated with various pathological conditions such as cancer, neurodegeneration, and apoptosis. Here, we report the development of ***Nu-VP***, a naphthalimide-based fluorescent molecular rotor capable of sensing both viscosity and polarity within the cellular environment. The probe exhibits dual sensitivity: it emits at 525 nm in nonpolar solvents such as dioxane (ET30 scale), while displaying a red-shifted emission maximum at 600 nm in highly viscous media like glycerol. ***Nu-VP*** is chemically stable, non-toxic, and non-reactive toward common intracellular analytes, making it suitable for live-cell imaging. In cellular studies, ***Nu-VP*** selectively localizes within the nucleus and effectively detects changes in its microenvironment. Notably, the probe revealed a decrease in nuclear polarity under stress conditions including oxidative stress, starvation, and apoptosis. These results suggest that ***Nu-VP*** serves as a powerful tool for real-time monitoring of nuclear biophysical changes in live cells. This capability may contribute to a better understanding of nuclear dysfunction in disease progression and facilitate the development of targeted therapeutic strategies.







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GDDB-PP-20

Advancing type 2 diabetes mellitus therapy through epigenetics

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Abstract:
Type 2 diabetes mellitus (T2DM), a multifactorial metabolic disorder with the most rapidly increasing global prevalence, is increasingly being understood through the lens of epigenetics, that is, heritable changes in gene expression without alterations in DNA sequence. Epigenetic modifications, including DNA methylation, histone modifications, and non-coding RNA regulation, have been implicated in the development and progression of T2DM [1]. These changes affect pancreatic β-cell function, insulin resistance, and inflammatory pathways, potentially influenced by environmental factors such as unhealthy diet, stress, physical inactivity, ageing, lifestyle and obesity.

Recent advances in allopathic medicines have begun to address these reprogrammable epigenetic mechanisms as novel therapeutic targets. Several drugs, including metformin and thiazolidinediones, have demonstrated epigenetic modulatory effects, influencing gene expression patterns that contribute to glycemic control [2]. Emerging therapies targeting specific epigenetic enzymes such as histone deacetylases (HDACs), histone acetyltransferase (HAT) and DNA methyltransferases (DNMTs) are under investigation for their potential to reverse adverse epigenetic marks associated with diabetic pathology [3, 4]. DNMT inhibitors such as 5-azacytidine and 5-aza-2′-deoxycytidine have already been approved by The United States Food and Drug Administration (US FDA) for use in diabetic retinopathy, a T2DM microvascular complication [1].

The epigenetic marks may potentially be used as biomarkers for prediction of T2DM, risk of vascular complications, and response to therapy and lifestyle interventions, thereby offering a tool for precision medicine. This may also offer a promising direction for personalized diabetes management via the use of epidrugs as a valuable adjunct to pharmacotherapy and immunotherapy.

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GDDB-PP-21

Isolation and characterization of a novel probiotic

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Background:
Probiotics are microorganisms (bacteria/yeast) which help in metabolism and growth in human as well as animals. Probiotics were originally used to improve animal and human health by modulating the gut microbiome. Probiotics have been observed to provide health benefits when consumed generally by improving or restoring gut flora. The most common of the Probiotics belong to groups called *Lactobacillus* and *Bifidobacterium*. Other bacteria may also be used as probiotics. Yeasts such as *Saccharomyces boulardii* may also be used as probiotics. Different types of probiotics have different effects. Probiotics are used to prevent antibiotic-associated diarrhea (including diarrhea due to *Clostridium difficile*), prevent necrotic enteritis and sepsis in preterm infants, treat infantile colic, treat periodontal disease, and treat induced or ulcerative colitis. As our knowledge of gut micro biota, nutrition, immunity, and genetics in health and disease has increased in recent years, such information will certainly aid in the development of new probiotic strains with disease-specific functions. It may also help to understand when and how to use probiotics.

Methods:
Isolation of prospective probiotic organism was done from fermented food products through serial dilution plating on MRS agar. Commercially available probiotic drink Yakult was used as a control. The standardized procedure with Yakult was followed for local yogurt. The isolated organism was tested for growth inhibition assay of bacterial pathogens *E. coli*, *P. aeruginosa,* and *S. aureus*, by agar diffusion method. *E. coli*, *P. aeruginosa* and *S. aureus* suspensions were plated on Mueller-*Hinton* agar plates. The prospective probiotic bacteria culture was spotted in the wells and zone of inhibition around the spotted culture was measured.

Results & Conclusion:
In the present study, *Lactobacillus* species were isolated from local yogurt and commercially available Yakult, their antibacterial effects were observed against the bacteria *P. aeruginosa, E. coli,* and *Staphylococcus aureus.* Results indicated that organism isolated from local curd showed growth inhibition for *P. aeruginosa*, *E. coli,* and *Staphylococcus aureus.* The potential probiotic organism was isolated from local yogurt and characterized by amplifying the 16S rRNA and sequencing the 1500 bp amplicon. Molecular Phylogeny identified the organism to be 94% homologous to *Lactobacillus delbruckei, sp.indicus*.

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