ICAMMIT '25

International Conference on Advances in Molecular Medicine & Multiomics Technology

19th & 20th September 2025

Conference Proceedings

Editors
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PSG College of Technology
Peelamedu, Coimbatore-641004,
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Supported by

ISBN: 978-93-95105-97-2

ISBN Number : 978-93-95105-97-2
Published by: PSG Center for Academic Research and Excellence, Coimbatore
Language: English
Country of Publication: INDIA
Date of Publication: 19/09/2025
Edition: First Edition
Editors: Dr M Ananthasubramanian
Dr S Vidyalakshmi
Dr S Siddhi

ICAMMT '25

Department of Biotechnology

The Department of Biotechnology at PSG College of Technology, established in the year 2000, is dedicated to advancing education and research in Biotechnology. This International Conference is a part of PSG Tech's Platinum Jubilee and the Department's Silver Jubilee celebrations. The department offers a comprehensive range of programs, including B. Tech., M. Tech., and Ph.D., aimed at nurturing a new generation of biotechnology professionals. The research environment in the Department is excellent due to its well-equipped laboratories and excellent infrastructure. The Department has received significant funding from national and international agencies, recognizing its commitment to excellence in teaching and research. The Department's research areas include bioinformatics, bioprocess technology, biofuel technology, cancer biology, environmental engineering, drug discovery, and medical biotechnology. The Department also hosts two specialized centres – the Centre for Biological Big Data Analysis and the Centre for Environmental Analyses and Solutions to encourage interdisciplinary research and innovation. In addition, it houses an incubation facility, the PSG-STEP: BIRAC BioNEST Bioincubation Centre, which supports entrepreneurial ventures and translational research in biotechnology.

Organisers' Note

We are delighted to welcome you to the International Conference on Advances in Molecular Medicine & Multi-Omics Technology (ICAMMT 2025), organized by the Department of Biotechnology, PSG College of Technology, as part of our College's Platinum Jubilee and Department's Silver Jubilee celebrations.

The conference, scheduled for 19–20 September 2025, is preceded by a Pre-Conference Workshop on Multi-Omics Data Analysis and Integration on 18 September 2025. ICAMMT 2025 aims to create a dynamic platform for researchers, clinicians, and industry professionals to exchange ideas and discuss cutting-edge advances in molecular medicine, drug development, biomarker discovery, systems biology, pharmacogenomics, and precision medicine.

Our program features special invited talks, oral and poster sessions, offering excellent opportunities not only for knowledge sharing but also for nurturing collaborations.

We also encourage you to take some time to explore Coimbatore, fondly known as the "Manchester of South India", with its distinctive culture, thriving industries, and scenic surroundings.

We wish you an intellectually stimulating and truly memorable conference experience.

Dr. M. Ananthasubramanian, Convenor
Dr. S. Vidyalakshmi, Organising Secretary

ICAMMT '25

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Dr. S. Shobana	Assistant Professor, Department of Biotechnology PSG College of Technology, Coimbatore
Mr. V. Venkatramanan	Assistant Professor, Department of Biotechnology PSG College of Technology, Coimbatore

Invited Speakers

Dr. C. Ron Geyer
Professor, Department of Pathology
University of Saskatchewan, Canada

Dr. Radhakrishna Rao
Professor, Department of Physiology
University of Tennessee, USA

Dr. Sachdev S. Sidhu
CEO Simisco Biosciences
Anvil Institute for Systems Biologics
Toronto, Canada

Dr. Arun Bandyopadhyay
Director
Gujarat Biotechnology University,
India

Dr. Shane Miersch
Chief Scientific Officer
Simisco Biologics East

Dr. Vinod Scaria
Chief Data Officer
Karkinos Healthcare, India

Dr. C N Ramchand
Co-founder and CSO
MagGenome Labs Pvt. Ltd, Chennai

Dr. Raju Rhee
Director
Kerala Genome Data Centre, India

Dr. Krishnakumar Kandaswamy
Chief Technology Officer
Centogene GmbH, Germany

Dr. Nithya Baburajendran
Head of Protein Structure & Biophysics
EDDC, Singapore.

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Programme Schedule

Time	Programme
Day 1: 19th September 2025	
8:30 AM – 9:00 AM	Registration
9:00 AM - 9:30 AM	**Inauguration** Venue: D Block Conference Hall PSG College of Technology
Session I **Applications of Omics technology for the development of novel therapeutic antibodies** **Session Chair: Dr. Radhakrishna Rao,** *Professor, Department of Physiology, University of Tennessee, USA* **Co-Chair: Dr. M. Ananthasubramanian,** *Head, Department of Biotechnology, PSG CT*	
9:30 AM – 10:05 AM	Invited Talk – 1 **Dr. Sachdev S. Sidhu** *CEO Simisco Biosciences, Anvil Institute for Systems Biologics, Toronto, Canada* Title: Large-Scale Engineering of Modulators of Protein Networks—Exploring High-Throughput Strategies for Manipulating Protein Interactions to Develop Novel Biological Agents.
10:05 AM – 10:40 AM	Invited Lecture – 2 **Dr. C. Ron Geyer** P*rofessor, Department of Pathology, University of Saskatchewan, Canada* Title: Post-Translational Engineering of Therapeutic Devices—Highlighting Innovations in Modifying Biological Therapeutics at the Post-Translational Level.
10:40 AM – 11:00 AM	**High Tea**
11:00 AM – 11:35 PM	Invited Lecture – 3 **Dr. Shane Miersch** *Chief Scientific Officer, Simisco Biologics East* Title: “Novel Multivalent and Multi Specific Antibodies for Therapeutic Applications" - Discussing Antibody Engineering Techniques for Complex Disease Models, Including Cancer and Infectious Diseases.
11:35 PM – 12:10 PM	Invited Lecture – 4 **Dr. C N Ramchand** *Co-founder and CSO, MagGenome Labs Pvt. Ltd, Chennai* Title: Discovery, Development, and Preclinical Evaluation of Monoclonal Antibody Therapeutics to Treat Wet AMD and Diabetic Retinopathy—Showcasing the Translational Journey of a Monoclonal Antibody Therapeutic Candidate for Vision-Threatening Diseases.
12:10 PM – 1:10 PM	**Technical Paper Presentation – Session 1**
1:10 PM – 2:00 PM	**Lunch**
Session II **Applications of Omics technology in biologicals discovery and development** **Session Chair: Dr. Sachdev S. Sidhu,** *CEO Simisco Biosciences, Anvil Institute for Systems Biologics, Toronto, Canada* **Co - Chair: Dr. P. Rani,** Professor, Department of Biotechnology, PSG CT	
2:00 PM – 2:35 PM	Invited Lecture – 5 **Dr. Vinod Scaria** *Chief Data Officer, Karkinos Healthcare, India* Title: Personal Genomes to Precision Medicine
2:35 PM – 3:10 PM	Invited Lecture – 6 **Dr. Radhakrishna Rao**

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	Professor, Department of Physiology, University of Tennessee, USA Title: Multiomics and Molecular Approaches in Identifying the Gut as a Therapeutic Target for Multiple Diseases
3:10 PM – 3:30 PM	**Tea Break**
3:30 PM – 4:05 PM	Invited Lecture – 7 **Dr. Arun Bandyopadhyay** *Director, Gujarat Biotechnology University, India* Title: Inflammation Markers for Risk Assessment of Coronary Artery Disease
4:05 PM – 4:40 PM	Invited Lecture – 8 **Dr. Romsha Kumar** *Senior Application Specialist - Single Cell MultiOmics, BD Lifesciences - Biosciences India/South Asia* Title: Unraveling Cellular Complexity: A Journey Through Single-Cell Multi-Omics
4:40 PM – 5:50 PM	**Technical Paper Presentation – Session 2**

Day 2: 20th September 2025	
Session III **Molecules to markets: Case studies** **Session Chair: Dr. Krishnaveni N,** *Head, Department of Biotechnology, PSG College of Arts and Science* **Co-Chair: Dr. J. Hema,** Associate Professor, Department of Biotechnology, PSG CT	
9:30 AM – 10:05 AM	Invited Lecture – 8 **Dr. Nithya Baburajendran** *Head of Protein Structure & Biophysics, EDDC, Singapore.* Title: From Crisis to Candidate: Discovery of A Reversible Covalent SARS-CoV-2 Mpro Inhibitor with Pan-Coronavirus Activity
10:05 AM – 10:40 AM	Invited Lecture – 9 **Dr. Raju Rhee** *Director, Kerala Genome Data Centre, India* Title: Beyond the Microscope: Genome Data centre as a Blueprint for Holistic Genomics—Connecting Human, Animal, Plant, and Microbial Health
10:40 AM – 11:00 AM	**High Tea**
11:00 AM – 11:35 AM	Invited Lecture – 10 **Dr. Krishnakumar Kandaswamy** *Chief Technology Officer, Centogene GmbH, Germany* Title: Next-Gen Rare Disease Diagnostics Powered by Multiomics
11:35 AM – 12:45 PM	**Technical paper presentations – Session 3**
12:45 PM – 1:35 PM	**Poster Sessions**
1:35 PM – 3:00 PM	**Lunch**
3:00 PM – 3:30 PM Valedictory Function	
3:30 PM	**High Tea**

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KT-01

Dr. C. Ron Geyer
Professor, Department of Pathology
University of Saskatchewan, Canada

Dr. C. Ron Geyer is a Professor of Pathology and Laboratory medicine at the University of Saskatchewan and a leading biomedical researcher. He is best known for his work in developing NeuroEPO, a modified hormone that shows promise in repairing brain neurons in Alzheimer's patients. At the University of Saskatchewan, he established several research entities, including the Saskatchewan Therapeutic Antibody Resource, the Advanced Diagnostics Research Laboratory, and the Centre for Biologic Imaging Research and Development. Beyond his groundbreaking work on NeuroEPO, which has entered Phase II clinical trials, his research focuses on creating therapeutic antibodies and improving diagnostic techniques for various diseases, advancing biomedical science in the region. His teams collaborate with other institutions to create a robust research environment for developing new treatments and diagnostics.

Title: Post-Translational Engineering of Therapeutic Devices —Highlighting Innovations in Modifying Biological Therapeutics at the Post-Translational Level

Exploiting the innate modularity of proteins has allowed advances across the fields of synthetic biology and biotechnology. By using standardized protein components as building blocks, complex, multiprotein assemblies with sophisticated functions can be generated; feats previously not possible with strictly genetic-engineering approaches. The development of strategies for protein assembly is accelerating, pushing the boundaries of protein architecture. Here, we review the use of protein ligases, SPAAC chemistry, and nanovesicles for the generation of antibody and antibody-like “devices”.

KT-02

Dr. Sachdev S. Sidhu
CEO Simisco Biosciences
Anvil Institute for Systems Biologics Toronto, Canada

Dr. Sachdev S. Sidhu is a Canadian scientist and Professor at the University of Toronto's Donnelly Centre, known for his innovative work in protein and antibody engineering. After a decade as a principal investigator at the biotechnology company Genentech, he joined the university in 2008 and is recognized for developing synthetic antibody libraries using phage display technology. His research focuses on creating therapeutic antibodies for a wide range of diseases, including cancer, infectious diseases, and chronic disorders. Beyond academia, Dr. Sidhu has co-founded organizations to bridge the gap between academic research and commercial applications, successfully translating his scientific innovations into potential drug candidates for clinical use. For his significant achievements, he was awarded the prestigious Christian B. Anfinsen Award in 2015 and was elected a Fellow of the National Academy of Inventors in 2020.

Title: Large-Scale Engineering of Modulators of Protein Networks—Exploring high-throughput strategies for manipulating protein interactions to develop novel biological agents.

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KT-03

Dr. Shane Miersch
Chief Scientific Officer
Simisco Biologics East

Dr. Shane Miersch is a protein engineer and research associate at the University of Toronto who specializes in designing and optimizing antibodies for therapeutic use, notably developing potent antibodies to combat the SARS-CoV-2 virus during the COVID-19 pandemic. His work, often conducted at the university's Donnelly Centre, focuses on engineering antibodies to be more effective and versatile, with research also extending to creating antibodies for other applications, such as neutralizing snake venom toxins. An inventor on multiple patents and an author of numerous publications in scientific journals, Dr. Miersch is known for developing advanced multivalent and multi-specific antibody formats. He holds a Ph.D. and has conducted postdoctoral work at the Harvard Institute of Proteomics.

Title: "Novel Multivalent and Multi specific Antibodies for Therapeutic Applications" -Discussing antibody engineering techniques for complex disease models, including cancer and infectious diseases.

This talk will detail an antibody engineering platform that exploits the modularity of antibody variable regions to generate novel multivalent antibodies that neutralize viruses with enhanced potency and breadth of specificity as means of optimizing therapeutic efficacy. Using SARS-CoV-2 as a test scenario, we show proof of concept that tetravalent antibodies comprised of one or more paratopes can be expressed at levels and with biophysical properties equivalent to conventional bivalent IgGs using the exact same purification techniques. Furthermore, direct comparison to the neutralization activity of conventional clinical stage antibodies used under emergency authorization during the pandemic, confirmed that multivalent and multi-specific antibodies exhibited enhanced resistance to mutational escape when evaluated against a panel of SARS-CoV-2 variants that rendered other therapeutic antibodies ineffectual. Lastly, successful application of these techniques to generate higher valency antibodies that improve neutralization and specificity against viruses other than SARS-CoV-2, suggest broad-based applicability of this platform to improving antibody-based therapies against virtually any virus.

KT-04

Dr. Radhakrishna Rao
Professor, Department of Physiology
University of Tennessee, USA

Dr. Radhakrishna Rao is a Professor and Vice-Chair of Physiology at the University of Tennessee Health Science Center (UTHSC). He is also a research biologist at the Veterans Administration Medical Center and a Fellow of the American Gastroenterological Association. A leading expert in intestinal epithelium research, Dr. Rao's work focuses on the structure and regulation of the gut barrier. His investigations explore how the gut microbiome and barrier dysfunction contribute to diseases such as inflammatory bowel diseases, alcoholic liver disease, colon cancer, and conditions resulting from chronic stress. Dr. Rao's research has received substantial funding from the National Institutes of Health and the Veterans Administration. Beyond his research, he is heavily involved in teaching and mentoring students at UTHSC at all levels, from undergraduate to postdoctoral.

Title: Multiomics and molecular approaches in identifying the gut as a therapeutic target for multiple diseases

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KT-05

Dr. C N Ramchand
Co-founder and CSO
MagGenome Labs Pvt. Ltd, India

Dr. C. N. Ramchand is a distinguished scientist, entrepreneur, and leader in the biotech and pharmaceutical industries with over four decades of experience. He holds key positions as the co-founder, CEO, and CSO of MagGenome Technologies, a company specializing in magnetic nanoparticle-based diagnostics, and as the CEO of Saksin Lifesciences, a biotech startup focused on novel biologics. His extensive career includes leadership roles at major companies like Sun Pharmaceuticals, where he headed drug discovery, and Kemin Industries, where he served as a Director of R&D. An accomplished academic, Dr. Ramchand holds a Ph.D. in Biochemistry, has published numerous peer-reviewed papers and books, holds many patents, and is an honorary visiting professor at the University of Newcastle. He is known for his innovative contributions to nanotechnology, drug discovery, and the commercialization of scientific advancements.

Title: Discovery, Development, and Preclinical Evaluation of Monoclonal Antibody Therapeutics to Treat Wet AMD and Diabetic Retinopathy—Showcasing the translational journey of a monoclonal antibody therapeutic candidate for vision threatening diseases.

Saksin Lifesciences Pvt. Ltd., in collaboration with MagGenome Technologies Pvt Ltd, India, is focused on the discovery and development of a synthetic humanized anti-VEGF Fab (Fragment Antigen Binding) for the treatment of Diabetic Retinopathy. Diabetic Retinopathy (DR) is caused by irreversible blood vessel damage in the retina due to diabetes and remains one of the leading causes of visual impairment worldwide. The global prevalence is estimated at 2.1% (approximately 160 million people). With the rising burden of diabetes mellitus, the DR patient population is projected to increase to nearly 220 million by 2040. The synthetic anti-VEGF Fab developed by our team demonstrates superior affinity and stability compared to existing therapies, which may translate into a larger responder population and reduced frequency of intravitreal injections. Importantly, the molecule is being designed to be cost-effective and widely accessible, addressing limitations of current market drugs such as Lucentis and Eylea. Our vision is to make efficacious and affordable treatment a reality for DR patients across diverse populations.

Development Details

The anti-VEGF Fab was engineered with unique complementarity-determining regions (CDRs) in both light and heavy chains and was expressed in *E. coli* BL21 using a phage display library coupled with affinity maturation strategies. The gene was inserted into a modified pBR322 vector under the control of a pho A promoter. Downstream processing was optimized to achieve high yield and purity. Preclinical development encompassed:

- Biochemical and biophysical characterization
- In vitro binding affinity studies
- In vivo efficacy models
- Toxicology evaluation as per regulatory guidelines

Furthermore, current Good Manufacturing Practice (cGMP) material has been successfully generated to enable clinical studies.

Current Status

The U.S. FDA Investigational New Drug (IND) filing has been completed. The molecule is ready to progress into Phase 1 clinical trials, both as Investigator-Sponsored Trials (ISTs) in the United States and clinical trials in India.

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KT-06

Dr. Arun Bandyopadhyay
Director
Gujarat Biotechnology University, India

Dr. Arun Bandyopadhyay is the Director of the Gujarat Biotechnology University (GBU), a position he has held since 2023. Before joining GBU, he was the Director of the Indian Institute of Chemical Biology (CSIR-IICB) from 2020 to 2023, where he also worked as a scientist for over two decades. He is known for his extensive research in chemical biology and biotechnology, focusing on areas like cardiovascular and respiratory diseases. His work, which includes patents for new therapeutic compounds, has earned him several honors, including fellowships from the Indian National Science Academy and the National Academy of Sciences.

Title: Inflammation markers for Risk Assessment of Coronary Artery Disease

Cholesterol is a critical component of the cell, and its homeostasis is one of the tightly regulated processes. Although Reverse cholesterol transport (RCT) plays a crucial role in removing cholesterol from the arterial wall, very few studies directly relate chronic inflammation and RCT with atherosclerosis. High resolution Mass spectrometric analysis of the human plasma identified about 2500 proteins in subjects with myocardial infarction. Network analysis revealed that most of the identified proteins were related to chronic inflammation, atherosclerosis and RCT. Few differentially expressed proteins (3, upregulated and 3, down regulated) were found to be clinically correlated with various risk factors of CAD in asymptomatic subjects as well as subjects with Acute coronary syndrome or heart attack. These proteins are ABCA1, ABCA5, sTLT1, AZGP1, vitronectin and PGLYRP2. The mechanistic significance and clinical significance were established using genetically altered mice (Apoe-/-) model and human macrophage cell culture system. In overall, we have narrowed down to 6 differentially expressed proteins for detail analysis in the context of cholesterol mobilization, inflammation and for longitudinal validation in human cohort. Eventually, these 6 proteins will be established as possible signature profile for risk assessment of athero-inflammation, atheroprogression, disease severity and predicting acute coronary event.

KT-07

Dr. Vinod Scaria
Chief Data Officer
Karkinos Healthcare, India

Dr. Vinod Scaria is a leading Indian clinician-turned-computational biologist and pioneer in clinical genomics and precision medicine, currently serving as the Chief Data Officer at Karkinos Healthcare. A former Senior Principal Scientist at the CSIR Institute of Genomics and Integrative Biology (CSIR-IGIB), he is best known for his work on the first Indian human genome and for co-founding the GUaRDIAN network for rare genetic diseases. During the COVID-19 pandemic, he led genomic surveillance programs that were critical for understanding the spread of the virus and its variants in India. He has authored numerous publications and holds several prestigious recognitions, including being an elected Fellow of the Royal Society of Biology and the Royal Society of Public Health. At Karkinos Healthcare, he focuses on leveraging data and genomic insights for advanced oncology care.

Title: Personal Genomes to Precision Medicine

ISBN: 978-93-95105-97-2

KT-08

Dr. Raju Rhee
Director
Kerala Genome Data Centre, India

Dr. Raju Rhee is a pharmaceutical physician and health tech innovator who serves as a consultant and project leader for the Kerala Genome Data Centre (KGDC). The KGDC is a significant government initiative in Kerala, overseen by the Kerala Development and Innovation Strategic Council (K-DISC), to establish the state as a genomics and bioeconomy leader. With extensive experience from major pharmaceutical companies like Pfizer and AstraZeneca and leadership roles at digital health firms like Twin Health, Rhee leverages his deep industry knowledge to drive this ambitious project. In his role at the KGDC, Dr. Rhee oversees the collection and analysis of genomic data from human, animal, and plant sources to advance precision medicine and other sectors. He has specifically highlighted the center's focus on diseases like sickle cell anemia and dengue fever, aiming to develop more effective treatments through genomic sequencing. Rhee's work blends scientific innovation with a commitment to improving public health, using genomics to make healthcare more affordable and accessible, particularly for chronic conditions like diabetes. By positioning the KGDC as India's first center of its kind, he is helping to harness Kerala's biodiversity and medical expertise to drive significant health and economic development.

Title: Beyond the Microscope: Genome Data centre as a Blueprint for Holistic Genomics—Connecting Human, Animal, Plant, and Microbial Health

Genomics is no longer confined to disease diagnostics or human health—it has become a holistic science that connects humans, animals, plants, and microbes under the broader vision of One Health. The Kerala Genome Data Centre is presented as a blueprint for such integration, demonstrating how genomic data can power solutions in public health, agriculture, biodiversity conservation, and microbial surveillance. Globally, initiatives like the Human Genome Project, UK Biobank, Genome Asia 100K, and the Earth BioGenome Project reflect the same ethos—linking diverse genomic resources to improve health, food security, and environmental resilience. By drawing from these models, genome data centres in emerging regions can create scalable, collaborative ecosystems that serve both local populations and global science.

ISBN: 978-93-95105-97-2

KT-09

Dr. Krishnakumar Kandaswamy
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Dr. Krishna Kumar Kandaswamy is the Chief Technology Officer of Bioinformatics at Centogene, a diagnostics company focused on rare hereditary diseases. With over 19 years of experience, his expertise lies at the intersection of bioinformatics and genomics. He previously served as the Director of R&D for Bioinformatics at Centogene. A key focus of his work is on advancing the company's bioinformatics applications and tools, including the CentoCloud platform. Dr. Kandaswamy's academic background includes a doctorate from Universität zu Lübeck and research affiliations with institutions like the Max Planck Institute for Biology of Ageing. His publications reflect his research interests in machine learning for protein analysis and using genomic data, like clinical exome sequencing, to improve diagnostics for genetic conditions.

Title: Next-Gen Rare Disease Diagnostics Powered by Multiomics

Rare diseases remain a major diagnostic challenge due to their genetic heterogeneity and the limitations of conventional single omics testing. To address this, we applied a multi-omics framework that integrates genomic, transcriptomic, proteomic, and metabolomic data to capture a comprehensive molecular profile of patients. Leveraging a large-scale rare disease Biodatabank and advanced bioinformatics pipelines, this approach demonstrated clear advantages over genome or exome sequencing alone. It enabled the reclassification of variant of uncertain significance (VUS), provided functional validation, and uncovered disease mechanisms that would otherwise remain hidden. Case analyses involving patients with neurometabolic and neurodegenerative disorders highlighted the clinical relevance of integrating multiple molecular layers, both for improving diagnostic yield and for guiding patient management. These findings illustrate the power of multi-omics to shorten the diagnostic odyssey, enhance precision medicine, and ultimately improve outcomes for patients affected by rare diseases.

ISBN: 978-93-95105-97-2

KT-10

Dr. Nithya Baburajendran
Head of Protein Structure & Biophysics
EDDC, Singapore.

Dr. Nithya Baburajendran is a translational scientist and the Head of Protein Structure & Biophysics at Singapore's Experimental Drug Development Centre (EDDC). With over a decade of experience, she is an expert in structural biology and drug discovery, using techniques like cryo-EM and X-ray crystallography to analyze protein and ligand interactions. In her role since 2022, she leads an ISO 9001-certified lab and has contributed to advancing multiple drug candidates toward commercialization. Her work is focused on identifying new drug candidates by studying how proteins interact with small molecules, peptides, and fragments. She earned her PhD in Structural Biochemistry from the National University of Singapore and previously conducted postdoctoral research at Columbia University.

Title: From crisis to candidate: Discovery of a reversible covalent SARS-CoV-2Mpro inhibitor with pan-coronavirus activity

The global COVID-19 pandemic highlighted the urgent need for effective antiviral agents. In response, we targeted the SARS-CoV-2 main protease, M pro , a highly conserved and essential enzyme in the viral life cycle. Leveraging structure-based drug design, we rapidly advanced from initial hit molecules to the discovery of compound 18, a reversible covalent inhibitor with potent antiviral activity across several clinical variants of SARS-CoV-2. Iterative optimization guided by high-resolution co-crystal structures was critical to fine-tuning key interactions within the active site, improving potency, selectivity, and pharmacokinetic properties. Compound 18 demonstrated dose-dependent efficacy in a mouse-adapted SARS-CoV-2 infection model and favourable pharmacokinetics in multiple species including mouse, rat, dog, and monkey. This work illustrates how structural insights transformed a global health crisis into a therapeutic opportunity, culminating in a viable preclinical candidate.

ISBN: 978-93-95105-97-2

OP-01

Upregulated MMP Family Genes as Ideal Biomarkers in Oral Squamous Cell Carcinoma: Insights from Computational Approach

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Abstract
Matrix metalloproteinases (MMPs) are a family of zinc-dependent proteolytic regulators that degrade various components of the extracellular matrix and plays critical role in tumor invasion and metastasis. In this study, the transcriptomic profiles of 23 human MMPs with a specific focus on evaluating the prognostic relevance of their combined expression patterns were investigated. From the Gene Expression Omnibus (GEO) and The Cancer Genome Atlas (TCGA) databases six overlapping differentially expressed genes (DEGs) of the MMPs were identified through integrated analysis. The prognostic value of these MMPs were examined using the receiver operating characteristic curves, survival analyses, and nomograms, which showed that the combination of 6 selected MMPs had moderate predictive ability, whereas the Tumor-Node-Metastasis (TNM) prediction model demonstrated higher accuracy. Followed by it, Gene Set Enrichment Analysis (GSEA) was performed to explore the potential mechanisms associated with the role of MMPs in Oral Squamous Cell Carcinoma (OSCC). The in-silico approach also revealed that the MMPs are associated with proliferative signalling, metabolic reprogramming and Serine/threonine protein kinase (STK33) related oncogenic pathways, highlighting their contribution to the development and progression of OSCC. Therefore, MMPs may serve as diagnostic biomarkers for OSCC, and their inhibition could offer a promising therapeutic strategy for patients with OSCC.

Keywords: Matrix Metalloproteinases, Oral Squamous Cell Carcinoma, Diagnostic, Biomarkers, Tumor Invasion, Metastasis.

1. Background
Oral cancer (OC), a prevalent type of head and neck cancer that affects regions such as, lips, tongue, cheeks, floor and roof of the mouth [1] . It ranks as the 16th most common type of cancer in Asian population and contributes to 66.3% of the total global OC burden [2] . Men are more frequently affected than women, due to the behavioural influences, such as, tobacco use and alcohol consumption. OSCC is a typical histological type of OC, strongly linked with activity of Matrix metalloproteinases (MMPs), proteolytic enzymes that degrades the components of the extracellular matrix, facilitating invasion and metastasis [3, 4] . Despite their recognized role in OC, the comprehensive prognostic significance of their combined expression patterns of all human MMPs in OSCC remains unclear.

2. Objective
This study aimed to systematically investigate the transcriptomic profiles of all 23 human MMPs in OSCC, with a specific emphasis to assess the prognostic relevance of their collective expression patterns using large-scale genomic datasets.

3. Methods
Transcriptomic data were obtained from publicly available repositories, including the Gene Expression Omnibus (GEO) and The Cancer Genome Atlas (TCGA) [5] . Through the integrated bioinformatic approach, 6 overlapping differentially expressed MMP genes were identified across the chosen 12 microarray datasets and 1 RNA-seq data from GEO and TCGA respectively [6, 7] . The prognostic value of all the 23 MMPs was evaluated using receiver operating characteristic (ROC) curves, survival analyses, and nomogram modelling. Additionally, the predictive performance of the MMP-based model was compared with traditional Tumor-Node-Metastasis (TNM) staging system. To explore the biological pathways associated with MMP expression, gene set enrichment analysis (GSEA) was conducted [8] , focusing on oncogenic mechanisms in OSCC.

ISBN: 978-93-95105-97-2

4. Results

Integrated analysis identified 6 consistently upregulated MMPs in OSCC, whose combined expression moderately predicted patient outcomes. However, the TNM model showed higher prognostic accuracy. GSEA linked these MMPs to key oncogenic pathways, highlighting their role in OSCC progression. These findings altogether underscore the multifaceted role of MMPs in OSCC development.

5. Conclusion

This study specifically highlights the potential role of MMPs as novel biomarkers in OSCC. MMPs expression is positively associated with higher TNM stage, suggesting a link between MMPs activity and oral tumor progression. Overall, the in-silico analysis confirms that the upregulated MMPs can be ideal biomarkers in OSCC.

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OP-02

Biomimetic Spray-Dried Formulations of Garlic-Derived Alliinase and Allicin Precursors for Pulmonary Biofilm Eradication

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Abstract

Allicin, a sulfur-containing compound derived from garlic, is known for its strong antibacterial activity but suffers from extreme instability, restricting its therapeutic application. In garlic, allicin is generated only upon tissue disruption, where alliin and the enzyme alliinase are compartmentalised and rapidly react. Inspired by this natural mechanism, we developed a biomimetic spray-dried formulation to stabilise the enzyme and precursor and enable in situ allicin formation for pulmonary delivery. Alliinase was extracted from garlic and stabilised with polyethylene glycol (PEG), while allicin was synthesised via a two-step method. Encapsulation was performed using spray drying with two-fluid and three-fluid nozzles, employing chitosan, maltodextrin, and lactose monohydrate as shell materials. Particle morphology and size distribution were characterised using scanning electron microscopy (SEM) and dynamic light scattering (DLS), while aerodynamic performance was evaluated

ISBN: 978-93-95105-97-2

by cascade impaction. Enzyme stability and solubility were assessed pre- and post-encapsulation. Antibiofilm activity was tested against *Escherichia coli* and *Pseudomonas aeruginosa* biofilms cultivated for 24–72 hours, including those in mucus-like media that mimic lung conditions. Biofilm eradication was quantified using the MTT assay, with kanamycin as a control. Among the formulations, chitosan-based particles demonstrated superior enzyme stability, favourable aerodynamic diameters for lung deposition, and the highest antibiofilm efficacy, significantly reducing biofilm biomass compared to other carriers. The study establishes that chitosan-encapsulated garlic-derived precursors as a promising pulmonary drug delivery strategy, combining natural biomimicry with modern spray-drying technology to target multidrug- resistant respiratory infections.

Keywords: Allicin, Alliinase, Spray drying, Pulmonary drug delivery, Biofilm eradication.

Introduction

Respiratory infections caused by multidrug-resistant (MDR) bacteria remain a critical public health concern, particularly in chronic conditions such as cystic fibrosis and chronic obstructive pulmonary disease. These diseases are frequently associated with persistent microbial colonisation in the form of biofilms, which confer tolerance to antibiotics and render conventional therapies largely ineffective. Escherichia coli and Pseudomonas aeruginosa are two pathogens frequently implicated in chronic pulmonary infections, and their biofilm-forming ability presents additional therapeutic barriers. Biofilms protect bacterial communities through an extracellular polymeric substance matrix, limit penetration of antibiotics, and promote survival under hostile conditions. Consequently, eradication requires novel strategies that can circumvent resistance while directly targeting the biofilm microenvironment. Allicin, a thiosulfinate compound produced in freshly crushed garlic, has long been recognised for its potent antimicrobial and antibiofilm activities. Its mechanism of action involves disruption of quorum sensing, oxidative inactivation of thiol-containing enzymes, and destabilisation of bacterial membranes. These properties extend to both Gram-positive and Gram-negative organisms, including MDR strains. Despite its potential, the therapeutic application of allicin is severely limited by its chemical instability, as it readily decomposes in aqueous environments and loses activity before reaching infection sites. Interestingly, in intact garlic tissue, allicin is never stored as such. Instead, its precursor, alliin, and the enzyme alliinase are compartmentalised in separate cellular structures. Upon mechanical disruption, they interact rapidly to generate allicin in situ. This natural mechanism of on-demand synthesis inspired the present study. Spray-dried microparticles were chosen as carriers due to their suitability for inhalation, stability, and ability to encapsulate sensitive biomolecules. Chitosan, maltodextrin, and lactose monohydrate were employed as encapsulating agents, each with distinct physicochemical and biomedical properties. The formulated microparticles were tested for their aerodynamic behaviour, enzyme stability, and antibiofilm efficacy against MDR pathogens under conditions mimicking the pulmonary environment.

Methodology

The precursor alliin was synthesised via a two-step procedure based on Stoll and Seebeck's method. Initially, L-cysteine was alkylated with allyl bromide in ammonium hydroxide at low temperature, yielding S-(2-propenyl)cysteine. This intermediate was subsequently oxidised with hydrogen peroxide at ambient conditions to form (±)-L-alliin, which was precipitated, purified, and vacuum-dried for further use. Alliinase was extracted from fresh garlic cloves homogenised in a phosphate buffer containing glycerol, EDTA, NaCl, and pyridoxal phosphate to stabilise enzymatic activity. The homogenate was clarified through filtration and subjected to protein precipitation with polyethylene glycol (PEG 6000). Following centrifugation at 30,000 g, the protein-rich pellet was resuspended, filtered, and lyophilised. Protein concentration was determined by the Bradford method, and enzyme activity was quantified using a lactate dehydrogenase-linked assay. Among the obtained fractions, the F2-3 fraction displayed the highest specific activity, making it the most suitable for encapsulation. Encapsulation of the enzyme and precursor was performed by spray drying with a Büchi B-290 instrument. Both two-fluid and three-fluid nozzle systems were employed to achieve optimal entrapment and minimise enzyme denaturation. Chitosan, maltodextrin, and lactose monohydrate were selected as encapsulating matrices based on their biocompatibility and ability to form respirable microparticles. The resulting powders were collected and stored under vacuum to prevent moisture absorption. Particle morphology was characterised by scanning electron microscopy, revealing that chitosan-based particles were spherical with smooth surfaces, while maltodextrin and

ISBN: 978-93-95105-97-2

lactose produced irregular morphologies. Size distribution and aerodynamic properties were analysed using dynamic light scattering and Anderson cascade impaction. Parameters such as mass median aerodynamic diameter (MMAD), fine particle fraction (FPF), and geometric standard deviation were determined to assess deposition potential within the respiratory tract. To evaluate biological efficacy, antibiofilm assays were conducted against *E. coli* and *P.aeruginosa* biofilms grown for 24–72 hours in both nutrient-rich LB medium and mucus-like medium supplemented with mucin, closely simulating lung conditions. Particles were compared with free synthetic allicin, free enzymatic allicin, and the antibiotic kanamycin. Biofilm biomass was quantified using the MTT reduction assay, which measures metabolic activity of viable cells. Enzyme stability and activity were measured before and after spray drying to confirm preservation of functional alliinase.

Results and Discussion

Encapsulation efficiency varied across matrices, with chitosan achieving the highest efficiency at 88%, followed by maltodextrin at 82% and lactose at 68%. PEG provided lower stability and was not as effective in retaining activity. Enzyme activity assays confirmed that chitosan and maltodextrin were superior carriers for maintaining alliinase stability during spray drying, while lactose resulted in partial activity loss. Cascade impaction studies demonstrated that chitosan particles had an MMAD of approximately 2.5 µm with a fine particle fraction exceeding 70%, favouring deposition in the deep alveolar region. Maltodextrin particles exhibited slightly larger MMAD values around 3.2µm, corresponding to deposition in central bronchial regions. Lactose particles presented intermediate characteristics but with lower deposition efficiency. These findings highlight the ability of carrier choice to dictate pulmonary targeting, with chitosan being particularly advantageous for treating infections in alveolar spaces. Antibiofilm testing revealed striking differences between formulations. In mucus-like media, which more accurately reflect the environment of cystic fibrosis and COPD lungs, chitosan-based particles showed the strongest biofilm eradication, significantly outperforming both maltodextrin and lactose carriers. The activity of chitosan-encapsulated formulations was even greater than that of free synthetic allicin, underscoring the importance of in situ enzymatic generation. Maltodextrin formulations displayed strong activity in LB medium, while lactose produced moderate inhibition. PEG-based formulations were the least effective across all conditions. Importantly, cytotoxicity assays confirmed that all encapsulated formulations were non-toxic to mammalian cells at therapeutically relevant doses. These findings collectively demonstrate that biomimetic spray-dried microparticles can effectively stabilise enzyme and precursor systems, provide suitable aerodynamic properties for inhalation, and deliver potent biofilm eradication in conditions mimicking chronic lung infections.

Conclusion

This study presents a novel biomimetic formulation strategy for pulmonary delivery of garlic-derived alliinase and alliin, enabling in situ allicin generation at the site of infection. Through spray drying with biocompatible carriers, we successfully stabilised the enzyme, maintained activity, and achieved favourable aerodynamic properties. Among the tested carriers, chitosan proved superior in terms of enzyme retention, alveolar deposition, and antibiofilm efficacy in mucus-like conditions. Maltodextrin also demonstrated strong potential, particularly for central bronchial delivery, whereas lactose showed moderate performance and PEG was inadequate for this application. The implications of this work extend beyond garlic-derived antimicrobials. The combination of natural biomimicry and modern spray-drying technology provides a platform for developing enzyme-substrate systems that generate unstable but therapeutically valuable compounds at the target site. By overcoming the challenges of instability and biofilm tolerance, the present formulation represents a promising antibiotic-free approach for managing multidrug-resistant respiratory infections. Further research will focus on in vivo validation, optimisation of enzyme-substrate ratios, and large-scale production to enable translation into clinical applications. If successful, this approach may provide patients suffering from chronic lung infections with a novel, sustainable therapy that harnesses nature’s own defence mechanisms.

ISBN: 978-93-95105-97-2

OP-03

Aptamer for the Recognition of Whole Cell MRSA from Food Sources

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Abstract

Introduction: Methicillin-resistant *Staphylococcus aureus* (MRSA) poses a significant threat to public health due to its resistance to β-lactam antibiotics and its growing prevalence in foodborne outbreaks. Rapid and specific detection strategies are essential for effective monitoring of MRSA contamination in food sources.

Methods: In this study, a whole-cell Systematic Evolution of Ligands by Exponential enrichment (SELEX) approach was employed to identify single-stranded DNA aptamers with high affinity and specificity for MRSA strains isolated from contaminated food samples. The SELEX process involved iterative rounds of binding, partitioning, and amplification using live MRSA cells as target and non-target bacterial species for counter-selection to enhance specificity. The enriched aptamer pool was characterized for binding kinetics and selectivity using various assays.

Results: These findings suggest that whole-cell SELEX-derived aptamers can serve as promising molecular recognition elements for developing rapid, portable biosensors for MRSA detection in food safety monitoring and outbreak prevention.

1. Introduction

Staphylococcus aureus is a common opportunistic pathogen found on the skin, nose, and respiratory tract of humans and other animals. They are contributory agents of small skin infections to life-threatening septic shock. They are armoured with numerous virulence factors such as staphylococcal enterotoxins (SE), leucocidin, exfoliative toxins, haemolysin, toxic shock syndrome toxins (TSST), and less explored staphylococcal enterotoxin-like proteins (SEl) that can trick the human innate and humoral immune responses and cause numerous health hazards in humans. They are highly heat-stable and low pH-resistant superantigenic enterotoxins that are the reason for food poisoning and toxic shock. In 1960s, the antibiotic-resistant strain of *S. aureus* was first reported, which was nosocomial in origin and termed as HA-MRSA (Hospital-associated-MRSA). Later, other lineages were identified, which showed higher invasive ability termed as CA-MRSA (Community Associated-MRSA) and LA-MRSA (Livestock associated-MRSA). Methicillin-resistant *Staphylococcus aureus* (MRSA) poses a significant threat to public health due to its resistance to β-lactam antibiotics and its growing prevalence in foodborne outbreaks. International travel and global food trading play a vital role in the transmission of bacteria across the world. Rapid and specific detection strategies are essential for effective monitoring of MRSA contamination in food sources. Aptamers, single-stranded nucleic acid (DNA or RNA) ligands, have emerged as auspicious molecular recognition elements in the field of diagnostics and detection. Selected through an in vitro process known as Systematic Evolution of Ligands by Exponential Enrichment (SELEX), aptamers exhibit high specificity and affinity for a diverse range of targets, from small molecules to proteins and whole cells. They were first reported in the early 1990s; their development through the SELEX process allows for the generation of target-specific ligands against a broad spectrum of analytes, including those that are non-immunogenic or toxic. Contrasting antibodies, which are biological products, aptamers are chemically synthesised, which is the fundamental difference that significantly lowers production costs. In this study, we have used aptamers as biosensors that target whole-cell Methicillin-resistant Staphylococcus aureus (MRSA). These platforms offer advantages such as rapid response times, high sensitivity, and the potential for miniaturisation and point-of-care (POC) testing.

2. Methodology

2.1 Isolation of Food samples: Various food samples were collected from parts of Mysore city. All samples were pre-enriched in BHI broth overnight at 37ºC. Serially diluted overnight cultures in peptone water were then plated onto Baird Parker (BPA) agar plates supplemented with egg yolk–tellurite emulsion (HiMedia, Mumbai, India), and plates were incubated for 24 - 48 hrs.

2.2 Biochemical Characterisation: Characteristic black centered colonies with an opaque zone on Baird-Parker agar were biochemically characterized. Presumptive colonies were subjected to the Indole, Methyl red-Voges-Proskauer, Mannitol fermentation, Coagulase, Catalase, Phosphatase production and DNase test. The positive

ISBN: 978-93-95105-97-2

colonies were then further screened with MeReSa agar (HiMedia, Mumbai, India), which is a chromogenic agar specific for the MRSA.

2.3 Genomic identification and Automated identification and Antibiotic Resistance study: Extracted DNA was screened for the presence of nuclease gene (*nuc*), Methicillin resistance determinant (*mec*A), SEB toxin coding gene (*seb*), and Panton-valentine leukocidine S and F fraction encoding genes (l*uk*F and *luk*S). All the PCR-positive isolates were then loaded onto the BD PHOENIX M50 system installed in the Defence Food Research Laboratory, Mysore, Karnataka. Positive isolates were identified and studied for their antibiotic resistance pattern using this system.

2.4 Multiplex PCR: A Total of two multiplex PCRs were standardized for this study. First set for the determination of MRSA with *nuc* and *mec*A primers. Another set was optimized with *luk*F, *luk*S (*pvl*), and *nuc* primers for the characterization of CA-MRSA.

2.5 Whole cell SELEX: The SELEX protocol for the enrichment of aptamer pool against MRSA was done as whole cell SELEX. Twelve SELEX rounds were done initially to enrich the aptamer pool to an optimum level. Counter SELEX was done after every four SELEX rounds to increase the specificity. All SELEX and counter SELEX rounds were performed by keeping the concentration of target and aptamer constant. The bound and unbound aptamers were purified and suspended in the same volume of water, and the concentration was measured.

2.6 TA- Cloning, Multiple Sequence Alignment Studies: The final aptamer pool was cloned, and the transformants were studied for specificity using ELONA. The specific aptamer sequences were sequenced, and their multiple sequence analysis was done. Secondary structures were predicted using software tools.

2.7 Enzyme-Linked Oligonucleotide Assay: The enriched aptamer pool was characterized for binding kinetics and selectivity using various assays. Enzyme-linked Oligonucleotide Assay (ELONA) (Mondal et al., 2015) was performed to monitor the MRSA whole cell binding affinity of the aptamer pool obtained after every round of SELEX, and at the end, to study the specificity. The absorbance of the colour developed was measured using a spectrophotometer at 490 nm wavelength.

2.8 Enzyme-linked aptamer sedimentation assay (ELASA): This technique uses the same chemistry as ELONA. Here, the specificity and sensitivity are observed colorimetrically.

3. Results

From a total of 70 samples, 18 were found to be MRSA. Four aptamer sequences were found to be specific to MRSA after the ELONA study. The Multiple sequence alignment study showed a very few conserved sequences. Two aptamer sequences showed exceptional binding affinity. They were used for the development of a bioassay using ELASA. These findings suggest that whole-cell SELEX-derived aptamers can serve as promising molecular recognition elements for developing rapid, portable biosensors for MRSA detection in food safety monitoring and outbreak prevention.

ISBN: 978-93-95105-97-2

OP-04

A Comparative Analysis of Microbiome Diversity in Coastal and Non-Coastal Populations of Tamil Nadu by 16S rRNA Gene Sequencing

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Abstract

Introduction: The gut microbiome plays a crucial role in various aspects of human health, including immune function, cardiovascular health, and cognitive abilities. This study explores how environmental pollutants affect human health because microbial diversity in coastal and non-coastal populations of Tamil Nadu.

Objectives: This study aims to investigate the bacterial diversity in coastal and non-coastal populations of Tamil Nadu, India, using metagenomic analysis, providing insights into the impact of pollution on gut microbiome and disease development using 16S rRNA gene sequencing. Further, this study will help to assess the potential health risks associated with prevalence of pathogenic bacteria in feces, urine, and skin swab samples in non-coastal and coastal populations.

Methods: This study involved 507 participants from coastal areas and 92 from non-coastal regions of Tamil Nadu, India. Stool and skin swabs from coastal and non-coastal populations were collected for 16S rRNA sequencing using the Illumina MiSeq platform. Additionally, bacterial isolation and identification were done through aerobic and anaerobic cultures.

Results: The study showed that Firmicutes were the dominant phylum in the gut microbiota of both coastal and non-coastal populations, with the coastal group exhibiting a higher abundance of *Bacilli* and *Clostridia*. In skin swab samples, *Firmicutes* and *Proteobacteria* were the predominant phyla. The study revealed distinct microbial communities across coastal and non-coastal populations, with coastal groups exhibiting a higher prevalence of opportunistic and pathogenic bacteria such as *Escherichia coli, Klebsiella,* and *Clostridium perfringens*. But it's a preliminary report, and we are studying with a larger cohort. We also correlated the prevalence of pathogenic bacterial populations and disease outcome in non-coastal and coastal regions.

Conclusion: This preliminary study highlights significant differences in gut and skin microbiota in both populations. These findings emphasize the need for further research to understand the potential implications for human health, particularly with environmental influences on microbial diversity.

Keywords: Coastal pollution, Microbiome, 16S rRNA sequencing, Taxonomic profiling, Human health

1. Introduction

The human microbiome plays a pivotal role in maintaining health, influencing immunity, metabolism, and disease susceptibility. Among its components, the gut and skin microbiota are highly sensitive to environmental factors such as diet, lifestyle, pollution, and occupational exposure. Coastal populations are uniquely vulnerable due to close interaction with the marine environment, where pollutants, heavy metals, and pathogenic microbes can affect human health. In contrast, non-coastal groups have distinct exposures shaped by urban or inland settings. Despite increasing evidence of environmental effects on microbiota, few studies have compared human microbial profiles between coastal and non-coastal communities in India. This study investigates gut and skin microbiome diversity in populations residing near the East Coast of Tamil Nadu, aiming to identify distinct microbial patterns and potential health implications.

2. Materials and Methods

2.1 Study Population

A prospective cohort design was adopted, enrolling 600 participants: 400 from coastal regions (Sadras, Pattinampakkam, Ennore, Pudupattinam, Mahabalipuram, Uyyalikuppam) and 200 from the non-coastal area of Oragadam. Participants included both healthy individuals and those with clinical conditions, ensuring diversity. Written informed consent was obtained, and ethical approval was secured.

ISBN: 978-93-95105-97-2

2.2 Sample Collection and Processing

From each participant, stool samples (for gut microbiome) and skin swabs (forearm) were collected under sterile conditions. Samples were stored at –20°C and transported for sequencing.

2.3 Sequencing and Data Analysis

Microbial DNA was extracted, and the 16S rRNA V3–V4 region was amplified using universal primers. Sequencing was performed on the Illumina MiSeq platform. Bioinformatics processing included quality filtering, OTU clustering at 97% similarity, and taxonomic assignment against the SILVA database. Diversity analyses (alpha and beta) and community composition comparisons were performed using QIIME2 and R packages.

3. Results

3.1 Gut Microbiome

The gut microbiota was dominated by *Firmicutes* and *Bacteroidetes*, consistent with global patterns. Coastal participants showed significantly higher relative abundance of *Firmicutes* (45%), particularly *Clostridia* and *Bacilli*, whereas non-coastal groups had increased *Bacteroidetes* (40%) and *Prevotella* species. *Proteobacteria*, often linked to dysbiosis, were enriched in the coastal group. Alpha diversity was higher in non-coastal individuals, indicating richer microbial communities. Beta diversity analysis revealed distinct clustering between coastal and non-coastal groups, suggesting environmental shaping of gut microbiota.

3.2 Skin Microbiome

The skin microbiome was primarily composed of *Firmicutes* and *Proteobacteria*. Coastal participants exhibited greater colonization by *Staphylococcus, Bacillus,* and *Vibrio* species, likely reflecting occupational exposure to seawater and fishing activities. In contrast, non-coastal individuals showed enrichment of *Corynebacterium* and *Streptococcus*. Skin microbiome diversity was lower among coastal participants, particularly in fishermen/women, suggesting reduced resilience of the skin ecosystem under polluted or high-exposure conditions.

3.3 Comparative Insights

Both gut and skin microbiomes showed clear environment-specific patterns. Coastal exposure correlated with higher prevalence of opportunistic and potentially pathogenic taxa, while non-coastal groups maintained a more balanced community structure.

4. Discussion

This study highlights the significant influence of coastal environments on the human microbiome. The dominance of *Firmicutes* in the coastal gut microbiome aligns with studies linking this phylum to energy harvesting and metabolic disorders, potentially increasing susceptibility to obesity and related conditions. Elevated *Proteobacteria* further raise concerns, as this group is associated with inflammatory responses and gut barrier dysfunction. In the skin microbiome, the abundance of *Staphylococcus* and *Vibrio* among coastal participants is consistent with findings from marine-exposed populations, where seawater acts as a reservoir of both commensal and pathogenic bacteria. Such alterations may predispose individuals to skin infections, dermatitis, or delayed wound healing, especially among fishermen frequently in contact with seawater and pollutants. The reduced microbial diversity in coastal groups suggests ecological stress on the microbiome, possibly driven by heavy metal exposure, marine pollution, or antibiotic use in fishing communities. Lower diversity is widely recognized as a marker of reduced resilience and higher disease risk. Comparatively, non-coastal populations showed more stable microbial communities, dominated by *Prevotella*. These findings underscore the importance of environmental and occupational factors in shaping microbiomes. Coastal communities may face increased risk of gut dysbiosis, skin infections, and pollution-related health outcomes. Integrating microbiome monitoring into coastal health surveillance could provide early indicators of disease susceptibility and guide targeted interventions.

5. Conclusion

Our study demonstrates clear differences in gut and skin microbiota between coastal and non-coastal populations of Tamil Nadu. Coastal participants exhibited a higher abundance of *Firmicutes, Proteobacteria, Staphylococcus,* and *Vibrio*, coupled with reduced microbial diversity, suggesting vulnerability to metabolic and skin-related health risks. Non-coastal populations maintained richer, more balanced microbial communities. These findings highlight

ISBN: 978-93-95105-97-2

the microbiome as a sensitive biomarker of environmental exposure and reinforce the need for microbiome-based strategies in environmental health assessment.

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ISBN: 978-93-95105-97-2

OP-05

A Combinational Effect of Statin with Conventional ChemoRadiotherapy in Colorectal Cancer

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Abstract:
Colorectal cancer (CRC) is the third most common malignancy worldwide and a major cause of cancer-related mortality. Although chemoradiotherapy (CRT) with 5-fluorouracil (5-FU), oxaliplatin, and radiation is the current standard, nearly 20% of patients show poor responsiveness due to intrinsic or acquired resistance, underscoring the need for novel adjunctive strategies. Statins, widely prescribed lipid-lowering drugs, have recently gained attention for drug repurposing in oncology owing to their pleiotropic effects on proliferation, apoptosis, and inflammation. In this study, we investigated the effect of Simvastatin and Atorvastatin in combination with CRT in an *in vivo* CRC model. BALB/c mice were treated with azoxymethane (AOM) and dextran sulfate sodium (DSS) to induce colorectal cancer and subsequently treated with standard CRT with or without statins. Further, histopathological analyses of colon tissues revealed that animals receiving statin-CRT combination exhibited marked restoration of normal crypt architecture, reduced inflammatory cell infiltration, and significantly lower incidence of dysplastic lesions compared to CRT alone. Moreover, statin co-treatment appeared to mitigate CRT-induced mucosal injury, indicating a protective effect on normal tissues. Importantly, features associated with resistance, such as persistent dysplasia and crypt distortion, were reduced in the statin-treated groups, suggesting that statins may overcome resistance mechanisms. These findings demonstrate that statins, particularly Simvastatin and Atorvastatin, enhance the therapeutic efficacy of CRT while reducing treatment-related toxicity in an AOM+DSS-induced CRC model. Given their established safety profile and clinical accessibility, showing strong potential to be repositioned as adjuvant agents in CRC therapy, offering a dual advantage of enhanced efficacy and reduced toxicity. Their integration into existing treatment regimens could represent a significant advancement in CRC management.

Keywords: colorectal cancer, chemoradiotherapy, Statins, Simvastatin, Atorvastatin

1. Introduction

Colorectal cancer (CRC) represents one of the most significant global health burdens, ranking as the third most common malignancy and the second leading cause of cancer-related mortality worldwide. Standard treatment regimens for locally advanced CRC typically involve multimodal approaches, of which chemoradiotherapy (CRT) is a cornerstone. CRT commonly includes systemic administration of 5-fluorouracil (5-FU) and oxaliplatin, in combination with localized radiation therapy. Drug repurposing has emerged as an attractive approach to improve cancer therapy outcomes by utilizing existing, clinically approved drugs for new indications. Statins, inhibitors of 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase, are primarily prescribed for the management of hypercholesterolemia and cardiovascular disease prevention. However, increasing preclinical and clinical evidence suggests that statins may possess anticancer properties. Beyond their lipid-lowering activity, statins exhibit pleiotropic effects, including modulation of cell proliferation, induction of apoptosis, reduction of inflammation, and interference with signaling pathways implicated in tumor progression and metastasis. Importantly, statins are safe, inexpensive, and widely accessible, making them highly suitable candidates for integration into oncological treatment regimens. The present study was designed to evaluate the efficacy of Simvastatin and Atorvastatin in combination with CRT in a murine model of inflammation-driven CRC. By employing a well-established azoxymethane (AOM) and dextran sulfate sodium (DSS) protocol to induce colitis-associated cancer, we sought to investigate whether statin co-administration could enhance CRT efficacy, reduce treatment-induced mucosal injury, and overcome resistance-related features.

2. Methods

An experimental in vivo study was conducted using BALB/c mice, chosen for their susceptibility to colitis-associated tumorigenesis. CRC was induced via a single intraperitoneal injection of azoxymethane (AOM), followed by repeated cycles of dextran sulfate sodium (DSS) dissolved in drinking water, a protocol known to

ISBN: 978-93-95105-97-2

mimic human inflammation-associated colorectal cancer. After confirmation of tumor induction, mice were randomized into different groups. Drug dosages were selected based on murine equivalents of clinically relevant concentrations. At the conclusion of treatment, animals were sacrificed, and colonic tissues were harvested for histopathological analysis. Parameters assessed included crypt morphology, epithelial regeneration, extent of inflammatory cell infiltration, incidence of dysplastic lesions, and evidence of CRT-induced mucosal injury. Comparisons between groups were made to assess therapeutic efficacy and tissue protective effects of statins.

3. Results

Histopathological analysis revealed that mice treated with CRT alone showed partial tumor regression but continued to exhibit significant pathological abnormalities. These included distorted crypt architecture, severe mucosal injury, persistent dysplastic lesions, and high levels of inflammatory infiltration. Such findings reflected the dual limitation of CRT: while partially effective against tumor burden, it inflicted collateral damage on normal colonic tissue. By contrast, groups receiving statin-CRT combinations demonstrated pronounced improvements in colonic tissue integrity and tumor suppression. Both Simvastatin and Atorvastatin co-treatment restored crypt architecture closer to normal, reduced inflammatory cell infiltration, and markedly decreased the incidence of dysplasia. Furthermore, mucosal damage typically induced by CRT—such as epithelial denudation and ulceration—was attenuated in the statin-treated groups. Comparative analysis indicated subtle differences between the two statins. Atorvastatin provided superior protection against mucosal injury, thereby preserving tissue integrity during CRT. Simvastatin, on the other hand, demonstrated stronger effects in limiting dysplastic transformation, suggesting a more pronounced role in suppressing tumor progression. Together, these findings underscore the dual advantage of statins as both sensitizers to CRT and protectors of normal tissue.

4. Discussion

This study provides preclinical evidence that statins can serve as effective adjuvant agents in colorectal cancer management when combined with conventional CRT. Statin co-administration enhanced treatment efficacy, reduced pathological features associated with resistance, and minimized treatment-induced mucosal toxicity. Such dual effects are of significant clinical importance, as they address two major limitations of CRT: incomplete tumor suppression and collateral tissue damage. The mechanisms underlying these outcomes are likely multifactorial. Statins inhibit the mevalonate pathway, reducing the availability of intermediates required for the prenylation of oncogenic proteins such as Ras and Rho. This disruption impairs tumor cell proliferation, survival, and metastasis. Statins also exert anti-inflammatory effects by downregulating NF-κB signaling and reducing pro-inflammatory cytokines, thereby creating a tumor microenvironment less conducive to cancer progression. Additionally, statins promote apoptosis and sensitize tumor cells to DNA damage induced by chemotherapy and radiation, potentially overcoming resistance mechanisms. CRT-associated gastrointestinal toxicity often limits patient compliance and can necessitate dose reduction or treatment discontinuation. By preserving tissue integrity and reducing mucosal injury, statins may enable more effective and sustained delivery of CRT, ultimately improving therapeutic outcomes.

5. Conclusion

In conclusion, the present study demonstrates that Simvastatin and Atorvastatin, when combined with CRT, significantly enhance therapeutic efficacy and reduce treatment-associated toxicity in a murine model of colorectal cancer.

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OP-06

Anthropogenic Shaping of the Poultry Litter Resistome: Metagenomic Insights into ARG-Pathogen Co-occurrence

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Abstract

Poultry litter serves as a critical hotspot for the emergence and dissemination of antimicrobial resistance genes (ARGs), driven by the widespread use of antibiotics in intensive farming. In this study, we employed shotgun metagenomic sequencing to profile the resistome and taxonomic composition of broiler litter microbiota from Namakkal, India. The analysis revealed a diverse community dominated by *Brevibacteria, Corynebacteria* and *Staphylococci* with notable representation of zoonotic and human pathogens such as *Staphylococcus aureus*, *Klebsiella pneumoniae,* and *Enterococcus faecium*. ARG profiling demonstrated a high prevalence of aminoglycosides, tetracyclines and MLS resistance determinants, with multidrug and aminoglycoside-modifying enzymes and efflux pumps being the predominantly adopted mechanisms of resistance. Co-occurrence analysis highlighted that ARGs were frequently associated with virulence-encoding taxa, underscoring the risk of resistant pathogens. The findings highlight poultry litter as a reservoir of clinically relevant ARGs, shaped by anthropogenic antibiotic pressure, with implications for environmental dissemination and potential transmission to humans and livestock.

Keywords: Metagenomics, Namakkal, Resistome, Virulome, Co-occurrence.

1. Introduction

Antibiotics have been used in poultry farming since the 1950s (1). They have been used to treat bacterial infections and for their prophylactic actions. The unregulated and widespread use of antibiotics in poultry has led to the global catastrophe of antimicrobial resistance (AMR). Poultry litter is one of the major by-products of broiler production. Nearly 90% of the unmetabolized antibiotics are released into the poultry litter as parent compounds. Hence, poultry litter acts as a reservoir of residual antibiotics and antibiotic-resistant bacteria with mobile genetic elements that facilitate the spread of antibiotic resistance genes (2). Namakkal district, in Tamil Nadu, is one of the major poultry production hubs of Asia, and has undergone intensive antibiotic usage to support its poultry farming sector through growth promotion and disease prevention. Besides being an economically significant hotspot, limited metagenomic studies have been conducted in the area to explore the resistome and virulome anchored by the poultry litter microbiomes. Metagenomic analysis bestows a comprehensive approach to explore the taxonomical composition, resistance profile and virulence nature of the complex microbial population that prevails within the environment (3). The present study correlates the co-occurrence of ARGs and VFs in bacterial taxa within the community and interprets their evolution amidst the prevailing selective pressure in the farm setup. Also, the study highlights the One Health implications associated with the AMR dissemination through the poultry farming industry in Namakkal.

ISBN: 978-93-95105-97-2

2. Materials and Methods

Poultry litter samples were collected from multiple commercial broiler farms in Mohanur village in Namakkal district, Tamil Nadu, and stored under sterile conditions until processing. Metagenomic DNA was extracted using a soil DNA kit with minor modifications to optimize yield and purity. DNA integrity was checked by electrophoresis, while concentration and quality were assessed spectrophotometrically. Shotgun metagenomic sequencing libraries were prepared following Illumina protocols and sequenced on a high-throughput platform to generate paired-end reads. Raw reads were subjected to quality control for adaptor contamination and low-quality bases before downstream analysis (4). High-quality reads were assembled de novo, and contigs above a defined length threshold were retained for further downstream processing. Taxonomic assignments were carried out using Kraken 2 (5). Functional screening for antibiotic resistance genes (ARGs) and virulence factors (VFs) was performed against multiple curated databases. Identified ARGs were annotated by antibiotic class and resistance mechanism, while VFs were mapped to potential bacterial hosts (6). Relative abundances were estimated by normalizing mapped read counts to contig length and total dataset size, providing quantitative insights into the resistome and virulome profiles.

3. Results

Metagenomic sequencing generated a high volume of quality-filtered reads, which assembled into contigs suitable for downstream analysis. Taxonomic profiling revealed a diverse microbial community, with a dominance of *Brevibacterium, Corynebacterium,* and *Staphylococcus* genera. These taxa, often associated with poultry environments, reflected selective enrichment under prolonged antibiotic exposure. Resistome analysis identified a broad spectrum of antibiotic resistance genes (ARGs) spanning major classes such as β-lactams, tetracyclines, macrolides, aminoglycosides, and fluoroquinolones. Notably, multi- drug resistance determinants were also detected, highlighting the complex adaptive capacity of the poultry litter microbiome. The mechanisms represented included enzymatic inactivation, efflux pumps, target modification, and ribosomal protection. Virulome profiling further revealed virulence factors linked to stress tolerance, adhesion, and capsule formation. These were anchored predominantly by opportunistic pathogens within *Staphylococcus* and *Corynebacterium*, indicating potential risks of pathogenicity. Integration of taxonomic and functional profiles highlighted specific bacterial hosts acting as reservoirs for both ARGs and VFs, indicating their ecological role in persistence and dissemination. These findings suggest that the generally overlooked poultry litter acts as a hotspot for the dissemination of antibiotic-resistant clinically significant pathogens into the environment.

4. Conclusion

This study highlights poultry litter from Namakkal poultry farms as a critical source anchoring both antibiotic resistance genes and virulence factors. The co-localization of these traits within dominant bacterial genera explains the impact of unregulated antibiotic use in poultry production. Such reservoirs not only cause zoonotic infections but also pose risks to farm workers, residents, and meat product consumers through environmental release and infiltration into the food chain. The findings point to the urgent need for integrated One Health surveillance and stricter regulations on antibiotic usage in poultry farming. By combining taxonomic and functional annotations, this study provides evidence for the role of poultry litter in shaping antibiotic resistance dynamics in humans, animals and the environment.

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ISBN: 978-93-95105-97-2

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OP-07

Genetic Variations in Folate Metabolism Genes: Precision Medicine for Type 2 Diabetes Mellitus

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Abstract

Folate metabolism plays a crucial role in one-carbon metabolism, DNA synthesis, repair, and methylation processes that regulate gene expression and metabolic homeostasis. Genetic polymorphisms in key folate metabolism genes such as MTHFR, MTR, GCPII, and RFC have been strongly associated with altered folate status, homocysteine levels, and impaired glucose metabolism. The case-control study consisted of 300 study subjects, of which, hundred were healthy controls, and hundred were type 2 diabetes mellitus (T2DM) with complications and hundred without complications, who belonged to south Indian population. The genotyping (PCR-RFLP) analysis suggests that these genetic variants MTHFR A1298C and RFC1 A80G were associated with the development of Type 2 diabetes and its complications among south Indians through mechanisms involving increased oxidative DNA damage and decreased antioxidative defense relative to the overproduction of free radicals (oxidative stress). Single Cell Gel Electrophoresis (SCGE) reveals that significant differences were observed in the DNA damage in diabetic patients with and without complications compared to controls ($P < 0.001$). Also, increased DNA damage along with the occurrence of a mutant genotype of the MTHFR 1298A→C and MTR 2756A→G polymorphisms in an individual with diabetes may be at an increased risk for the development of chronic complications. Precision medicine approaches that integrate genetic screening of folate metabolism variants with dietary interventions, nutraceutical supplementation, and individualized treatment strategies hold promise for optimizing glycemic control, homocysteine level and preventing diabetes-related complications.

Keywords: Folate metabolism, Genetic polymorphism, Single Cell Gel Electrophoresis, Type 2 Diabetes Mellitus and Precision medicine

1. Introduction

Folate metabolism is central to one-carbon metabolism, which supports DNA synthesis, repair, and methylation processes that influence gene expression, metabolic regulation, and oxidative balance. Genetic polymorphisms in folate metabolism genes such as methylenetetrahydrofolate reductase (MTHFR), methionine synthase (MTR), glutamate carboxypeptidase II (GCPII), and reduced folate carrier (RFC) have been strongly implicated in altered folate status, elevated homocysteine levels, and dysregulated glucose metabolism. Accumulating evidence suggests that these genetic variants contribute to type 2 diabetes mellitus (T2DM) risk and progression, particularly through mechanisms involving oxidative stress, DNA damage, and impaired antioxidative defense (Rehman and Akash, 2017, Rawi et al., 2011, Sharaf et al.,2012). Given the high prevalence of T2DM and its complications in the South Indian population, understanding gene–disease associations in folate metabolism is crucial for advancing precision medicine strategies.

ISBN: 978-93-95105-97-2

2. Methods

A case-control study was conducted among 300 South Indian subjects categorized into three groups: healthy controls (n = 100), T2DM without complications (n = 100), and T2DM with complications (n = 100). Genotyping of candidate folate metabolism variants—MTHFR C677T, MTHFR A1298C, MTR A2756G and RFC1 A80G was performed using polymerase chain reaction–restriction fragment length polymorphism (PCR-RFLP) analysis (Frosst et al., 1995, Friedman et al., 1999, Zara-Lopes et al., 2016, Neagos et al., 2010). SCGE was employed to assess the level of DNA damage in peripheral lymphocytes by a two-layer method according to Tice et al., (2000) with slight modifications of the method adopted by Singh et al., (1988). Statistical analyses were applied to determine genotype distribution, allele frequency differences, and their association with T2DM and its complications.

3. Results

The genotypic analysis revealed a significant association between MTHFR A1298C and RFC1 A80G polymorphisms with susceptibility to T2DM and its complications in the studied subject. The occurrence of the mutant genotypes correlated with increased oxidative DNA damage as indicated by SCGE results, which showed highly significant differences in DNA damage among diabetic patients compared with controls ($P < 0.001$). Furthermore, individuals carrying the mutant MTHFR 1298C allele and MTR 2756G allele exhibited higher DNA damage profiles, indicating synergistic effects between genetic susceptibility and oxidative stress in the pathogenesis of diabetic complications.

4. Conclusion

This study demonstrates that genetic polymorphisms in folate metabolism genes, particularly MTHFR A1298C, MTR A2756G, and RFC1 A80G, are associated with an elevated risk of T2DM and its complications among South Indians, potentially mediated by oxidative DNA damage and impaired redox defense mechanisms. These findings underscore the importance of incorporating genetic screening into clinical risk assessment for T2DM. Precision medicine approaches integrating genetic profiling with dietary modulation, folate supplementation, and targeted nutraceutical interventions may improve glycemic regulation, reduce homocysteine accumulation, and mitigate the development of chronic complications.

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ISBN: 978-93-95105-97-2

OP-08

Multiomics Insights into Microbial Community Structure and Soil Diversity Across *Fabaceae* and *Poaceae* Crops Families

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Abstract

Microbial inoculants are increasingly applied in sustainable agriculture to improve crop productivity and resilience, yet their impacts on native rhizosphere microbiomes across crop families remain unclear. In this study, we investigated the microbial diversity, composition, and structure in *Fabaceae* (soybean) and *Poaceae* (wheat) rhizospheres using publicly available genomic and metagenomic datasets. The microbial community profiles were analyzed for taxonomic composition, alpha and beta diversity, and differential abundance. In both families, bacteria dominated (>85–90%) with minimal contributions from *Archaea* and *Eukaryota*. *Fabaceae* (FA) rhizospheres were enriched with nitrogen-fixing symbionts such as *Rhizobium* and *Bradyrhizobium*, exhibiting lower microbial diversity (Shannon 2.5–3.0; Simpson 0.65–0.72) but greater ecological specialization. In contrast, *Poaceae* (PO) rhizospheres supported more diverse and evenly distributed microbial communities (Shannon 3.5–4.2; Simpson 0.80–0.88), dominated by *Gammaproteobacteria, Bacilli, Massilia,* and *Dyella*. Alpha diversity indices and unique OTUs were consistently higher in PO, while beta diversity analysis ($p < 0.05$) revealed clear family-level clustering, confirming host phylogeny as a major driver of microbial assembly. Together, our results highlight FA supports specialized symbiosis-driven assemblages, whereas PO fosters diverse, resilient microbiomes. To conclude, integrating genomic and metagenomic approaches highlights crop-specific microbiome assembly processes, providing a framework for designing precision microbial inoculants that optimize soil health, enhance crop resilience, and advance sustainable agricultural practices.

Keywords: Rhizosphere microbiome, *Fabaceae* and *Poaceae*, microbial diversity, alpha and beta diversity, QIIME2, sustainable agriculture, plant-microbe interactions.

1. Background

Microbial communities are vital to plant health, growth, and stress resilience. They influence nutrient cycles, disease suppression, and overall crop productivity. Current research leverages high-throughput sequencing (genomics and metagenomics) to examine the functional potential, diversity, and dynamics of plant-associated microbiomes, with particular focus on two globally important crop families: *Poaceae* (wheat, rice) and *Fabaceae* (soybean, peanut) (1-3). The study aims to compare these groups through meta-analytical approaches, assessing taxonomic composition, alpha and beta diversity, and differentially abundant microbial taxa.

2. Methods

Publicly available datasets from the NCBI Sequence Read Archive (SRA) were selected using specific criteria, focusing on soil and root-associated microbiomes from crops in both families. Data processing utilized the SRA Toolkit, followed by quality control with FastQC and Cutadapt for adapter trimming and filtering. Subsequent analysis was performed using QIIME2, a reproducible and modular bioinformatics framework, with denoising and chimera removal via DADA2, followed by taxonomy assignment using a Naive Bayes classifier trained on the SILVA rRNA database. Diversity and abundance metrics were calculated and visualized with RStudio and related statistical packages, ensuring robust ecological and statistical insights (4-6). A detailed step-by-step workflow is included in the methodology section and illustrated in the workflow Figure 1.

ISBN: 978-93-95105-97-2

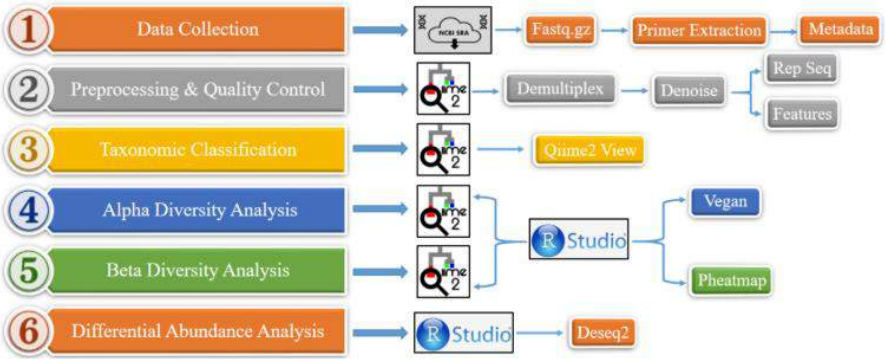

Figure 1: Overall Workflow

3. Results

Diversity Metrics and Taxonomic Composition

Alpha Diversity

Alpha diversity metrics consistently showed that *Poaceae* samples possess significantly greater microbial diversity compared to *Fabaceae*. Shannon diversity indices ranged from 3.5 to 4.2 for *Poaceae*, compared to 2.5 to 3.0 for *Fabaceae*, and Simpson indices indicated higher evenness in *Poaceae* (0.80–0.88) than in *Fabaceae* (0.65–0.72). Correspondingly, richness indices such as ACE and Chao1 were significantly greater in *Poaceae*, supported by rarefaction analyses revealing saturation of diversity estimates across samples. Furthermore, *Poaceae* rhizospheres harbored nearly twice as many unique operational taxonomic units (OTUs) as observed in Fabaceae, although both families shared a large core microbiota of approximately 6,000 OTUs.

Beta Diversity

Beta diversity analyses using Bray-Curtis and Manhattan distance metrics revealed robust clustering of samples according to host plant family ($p < 0.05$). Ordination plots from Principal Coordinates Analysis (PCoA) and Non-metric Multidimensional Scaling (NMDS) showed clear separation of *Fabaceae* and *Poaceae* samples. Bray-Curtis-based ordinations provided higher resolution and lower stress values, indicating stronger discrimination between microbial communities shaped by host identity.

Differential Abundance and Taxonomic Patterns

Compositional analysis reflected distinct ecological roles and evolutionary histories. At the highest taxonomic levels, Bacteria dominate both families, but *Fabaceae* exhibited greater proportions of unassigned and potentially novel lineages, supporting the idea of unique symbiotic adaptations. At the phylum level, *Proteobacteria, Actinobacteriota, Firmicutes,* and *Bacteroidota* were prevalent, though grasses generally displayed a broader, more even phylum distribution.

Finer taxonomic resolutions revealed the following:

Taxonomic composition at the phylum level showed dominance of:

- *Proteobacteria, Actinobacteriota, Firmicutes* and *Bacteroidota*

Fabaceae samples contained a higher proportion of unclassified taxa, suggesting novel microbial lineages possibly linked to symbiotic relationships.

At the class level:

- *Alphaproteobacteria* and *Actinobacteria* were more abundant in *Fabaceae*.
- *Gammaproteobacteria* and *Bacilli* predominated in *Poaceae*.

At the order level:

- *Rhizobiales, Rickettsiales,* and *Sphingomonadales* were enriched in *Fabaceae*, reflecting their nitrogen fixation roles.
- *Burkholderiales, Bacillales,* and *Enterobacterales* were more diverse and abundant in *Poaceae*.

At the genus level:

- *Fabaceae* was characterized by *Sphingomonas* and uncultured *Rhizobiaceae*.
- *Poaceae* had dominant genera such as *Dyella* and *Massilia*.

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4. Discussion and conclusion

The microbial communities in the rhizospheres of *Fabaceae* (legumes) and *Poaceae* (grasses) crops show clear and significant differences in diversity and composition. Alpha diversity analyses indicate that *Poaceae* hosts a richer and more diverse microbial community, with greater species richness and evenness, compared to the more specialized and less diverse communities associated with *Fabaceae* (7, 8). This suggests that *Poaceae* rhizospheres support a broad range of microbes adapted to varying environmental conditions, whereas *Fabaceae* tend to recruit specific symbiotic microbes, such as nitrogen-fixing bacteria (9). Beta diversity analyses further reinforce this distinction, demonstrating strong clustering of microbial communities by plant family, with a clear separation between *Fabaceae* and *Poaceae* microbiomes (8, 10). Such host- dependent structuring highlights the role of plant identity as a key determinant of microbial community assembly. These observed patterns reflect differences in ecological niches and plant-microbe interactions influenced by host traits and environmental factors. Taxonomic profiling revealed that bacterial communities dominate both families but vary in their composition. *Fabaceae* soils are enriched with groups like *Alphaproteobacteria* and key symbiotic taxa such as *Rhizobiales* and *Rhizobiaceae* reflecting their role in nitrogen fixation and symbiotic relationships (7,9). In contrast, *Poaceae* soils contain a more diverse array of taxa, including *Gammaproteobacteria*, *Bacilli*, and genera like *Dyella* and *Massilia*, indicative of greater environmental adaptability and functional versatility in fluctuating soil environments. In summary, *Fabaceae* crops harbor specialized, symbiotic microbial communities, while *Poaceae* crops sustain diverse, resilient microbiomes. These findings highlight the importance of plant family and ecological strategies in determining rhizosphere microbial structure and suggest potential for targeted microbiome management in sustainable agriculture. This meta-analysis provides a robust, reproducible foundation for ongoing research into the ecological and functional roles of microbial communities in agriculture, emphasizing the importance of integrating omics technologies and advanced bioinformatics for crop improvement.

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OP-09

Gut–Brain Digital Twins: Revolutionizing Precision Medicine in Adult Neurodegeneration

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Abstract

Background: Neurodegenerative disorders such as Alzheimer's, Parkinson's, amyotrophic lateral sclerosis, Huntington's, and multiple sclerosis have been regarded as brain diseases. The new evidence has revealed the role of a more systemic aspect of the enteric-brain connection (GBA). This multifactorial network connects the enteric nervous system, immune mediators, hormones, vagal pathways, and the microbiome within the gastrointestinal system.

Purpose: The paper provides an overview of the potential of gut microbes and their products to contribute to neurodegeneration and introduces a breakthrough in personalized medicine through the implementation of AI-based and digital twin models of adults impaired by the condition.

Approaches: Analysis of integrative models that integrate the information collected by longitudinal multi-omics, neuroimaging, and real-time biosensors in order to create a digital twin of the patient. Superior AI systems imitate GBA biochemical and network processes, allowing for the detection of early disease markers and the testing of precise interventions before applying them to patients, including dietary adjustments, microbiome editing, and neuromodulation.

Intervention: Digital twins can enable precise medicine by forecasting how a disease will progress in a patient and by optimizing multi-parametric treatment regimes through reinforcement learning and uncertainty measurement. With such an introduction, the possibility of earlier detection becomes a reality with adaptive treatment, and issues related to data standardization, regulatory control, and equal opportunities are brought into perspective.

Conclusions: The digital twin, enhanced by AI technology, alters the paradigm of therapeutic neuromedicine in neurodegenerative disorders by introducing dynamic, personalized, and rationalized interventions that adapt to a patient's specific condition over time—the future of precision neuromedicine has arrived.

Keywords: Gut–brain axis, neurodegeneration, digital twin, precision medicine, microbiome, artificial intelligence, multi-omics

1. Introduction

Neurodegenerative diseases represent a growing global health crisis, affecting millions worldwide with enormous social and economic implications. Traditional brain-centric approaches have yielded limited clinical success, with numerous late-stage therapeutic failures highlighting the need for paradigm shifts in understanding these complex disorders. Recent cross-disciplinary research in metagenomics, immunology, and systems neuroscience has revolutionized our comprehension of neurodegeneration as systemic conditions involving the gut-brain axis. The gut-brain axis constitutes a sophisticated bidirectional communication network encompassing the enteric nervous system, vagal and spinal pathways, hormonal signaling, and immune responses. This complex system directly influences neuroinflammation, protein aggregation, and cognitive function through multiple interconnected mechanisms. Compelling evidence from germ-free mouse models demonstrates that gut microbiota manipulation can prevent or induce neurodegeneration, establishing causality between microbial communities and brain pathology. Disease-specific gut dysbiosis patterns emerge years before clinical manifestations, presenting unprecedented opportunities for early intervention. Alzheimer’s disease exhibits reduced butyrate-producing bacteria and elevated lipopolysaccharides, while Parkinson’s disease shows increased Enterobacteriaceae and

ISBN: 978-93-95105-97-2

compromised gut barrier integrity. These dynamic biomarker cascades precede cognitive and motor symptoms, offering novel therapeutic targets for precision medicine approaches. Current literature remains fragmented, focusing on static mechanistic pathways or general interventions while overlooking temporal dynamics and individual heterogeneity. Existing studies predominantly examine pediatric populations with limited applicability to adult neurodegeneration. Cross-sectional omics approaches miss critical biomarker evolution patterns, while patient diversity challenges identification of universal microbiome signatures.

Recent advances in artificial intelligence and computational systems biology provide powerful tools to address these limitations. Models like SIMBA and GIM integrate genome-scale metabolic networks with graph neural networks, enabling mechanistic predictions while maintaining interpretability. Digital twin technology, successfully deployed in cardiology and oncology, now emerges in neuroscience through projects like EU-funded Neurotwin, demonstrating feasibility for virtual brain stimulation applications.

2. Methods

2.1 Digital Twin Architecture: We developed a modular four-layer digital twin framework integrating multi-source data streams. The molecular layer employs SIMBA-GNN algorithms to predict microbial community evolution using genome-scale metabolic models combined with graph neural networks. The cellular layer implements agent-based models simulating microglial activation and neuronal survival guided by cytokine and short-chain fatty acid signals. The network layer utilizes EEG and neuroimaging data to model neural circuit dynamics and gut-brain signal integration. The clinical phenotype layer translates molecular changes into clinically meaningful outcomes using reinforcement learning for personalized treatment optimization.

2.2 Multi-Omics Integration: Longitudinal data collection encompasses metagenomics, metabolomics, transcriptomics, and proteomics analysis using Multi-Omics Factor Analysis (MOFA) for dimensionality reduction and temporal tracking. Mixed-effects models establish relationships between molecular changes and cognitive/motor outcomes over time. Neuroimaging protocols include MRI volumetry, diffusion tractography, and PET amyloid/tau imaging following FAIR and BIDS standards for interoperability.

2.3 Real-Time Biosensing: Implementation of enzyme-powered, battery-free ingestible capsules containing electrochemical sensors for continuous small-intestinal metabolite monitoring including glucose and short-chain fatty acids. Data transmission utilizes magnetic coupling to eliminate invasive procedures. Complementary wearable devices include mobile EEG headsets and epidermal sweat sensors for brain activity and peripheral metabolite assessment.

2.4 AI Model Development: Graph neural networks map microbe-metabolite-brain interactions with mechanistic interpretability. Bayesian updating algorithms continuously incorporate new biosensor, omics, and imaging data for real-time parameter estimation and prognostic forecasting. Federated learning enables distributed model training across multiple centers while preserving patient privacy under HIPAA and GDPR compliance.

2.5 Validation Framework: Verification, validation, and uncertainty quantification (VVUQ) protocols ensure model reliability. Retrospective validation compares digital twin predictions against longitudinal cohort biomarkers. Prospective validation tracks new patient cohorts with real-time digital avatar updates. Advanced uncertainty quantification methods including Bayesian approaches and conformal prediction identify out-of-distribution data and trigger assistance-only modes when prediction confidence is insufficient.

3. Expected Results

Biomarker Discovery and Validation: The digital twin framework is anticipated to identify novel gut-brain biomarker signatures specific to each neurodegenerative disease. Alzheimer’s disease models should demonstrate reduced butyrate and propionate levels with elevated lipopolysaccharides and bacterial amyloids preceding mild cognitive impairment by 6-12 months. Parkinson’s disease patterns are expected to show increased Enterobacteriaceae abundance with compromised gut barrier integrity markers including zonulin and LPS-binding protein correlating with disease progression rates.

3.1 Personalized Intervention Optimization: In silico clinical trials demonstrate significant therapeutic potential across multiple intervention modalities. Mediterranean-ketogenic dietary interventions optimized through reinforcement learning algorithms show predicted 18% cognitive improvement in Alzheimer's patients through enhanced short-chain fatty acid production and reduced neuroinflammation. CRISPR-engineered

ISBN: 978-93-95105-97-2

butyrate-producing bacterial strains demonstrate 22% amyloid clearance enhancement with concurrent LPS reduction. Adaptive vagus nerve stimulation protocols achieve 40% reduction in Parkinson's motor fluctuations with normalized heart rate variability.

3.2 Early Detection Capabilities: Predictive models identify abnormal biomarker patterns weeks to months before clinical symptom onset. Sudden short-chain fatty acid decreases or lipopolysaccharide elevation trigger early intervention protocols, potentially delaying disease progression through proactive therapeutic adjustments. Real-time monitoring enables dynamic treatment modifications based on individual patient responses.

3.3 Clinical Translation Outcomes: Digital twin integration with electronic health records provides clinicians with personalized risk profiles and treatment recommendations. Dashboard interfaces display gut-brain biomarker trends, cognitive assessments, and individualized intervention suggestions based on evolving molecular patterns. Learning health systems enable continuous refinement of personalized gut-brain axis interventions through feedback loops between clinical practice and research.

4. Conclusion

This comprehensive digital twin framework represents a transformative approach to neurodegenerative disease management through personalized gut-brain axis medicine. By integrating cutting-edge AI technologies with multi-omics analysis and real-time biosensing, the system enables unprecedented precision in understanding individual disease trajectories and optimizing therapeutic interventions. The modular architecture successfully addresses critical limitations in current neurodegenerative research by providing mechanistic insights into gut-brain interactions while maintaining clinical interpretability. The framework's ability to predict disease progression and simulate intervention outcomes offers substantial advantages over traditional one-size-fits-all therapeutic approaches. Key innovations include the integration of ingestible biosensors with federated learning algorithms, enabling continuous monitoring while preserving patient privacy. The system's capacity for real-time adaptation through Bayesian updating ensures treatment recommendations evolve with changing patient conditions, maximizing therapeutic efficacy. Expected clinical impacts include earlier disease detection through dynamic biomarker monitoring, personalized treatment optimization achieving 18-40% improved outcomes, and reduced healthcare costs through precision medicine approaches. The framework's scalability through federated learning enables global validation while addressing data privacy concerns. Future implementations require collaborative efforts across multiple domains including regulatory approval pathways, healthcare system integration, and clinician training programs. Addressing challenges in data standardization, AI explainability, and digital divide issues will be critical for equitable deployment. This AI-empowered digital twin approach heralds a new era in precision neuromedicine, transforming neurodegenerative disease management from reactive treatment to proactive, personalized interventions that adapt to individual patient needs over time, ultimately improving outcomes and quality of life for millions affected by these devastating conditions.

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ISBN: 978-93-95105-97-2

OP-10

Characterization of the Intervertebral Disc Microbiome Through Shotgun Sequencing: Evidence for Infection-Mediated Intervertebral Disc Degeneration

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Abstract

Low back pain (LBP) affects approximately 619 million people globally as of 2020, with projections reaching 843 million by 2050, making it the leading cause of years lived with disability worldwide. This condition predominantly arises from intervertebral disc (IVD) degeneration, a multifactorial process where emerging evidence supports subclinical infection as a key contributing factor. Recent research has evidenced that human discs harbor unique microbiomes, with dysbiosis determining health and disease outcomes. Our previous studies have provided substantial evidence for microbial colonization rather than contamination in disc tissue. Based on this evidence, the present study aims to characterize microbial communities through shotgun metagenomic sequencing to understand the role of the disc microbiome in degeneration processes. Our approach involved a cohort comprising healthy IVD tissue from brain-dead organ donors and diseased tissue from patients undergoing surgery. Shotgun sequencing was performed using the Illumina NovaSeq platform and analyzed with Kraken 2 (v-2.2.3) and Bracken tools. The relative abundance of bacterial species and species diversity were assessed using standard statistical analyses. The results revealed rich species diversity with significant differences among control and diseased samples ($p=0.03$), and beta diversity analysis showed significant variation ($p=0.002$) between control and diseased groups. The most abundant bacterial species identified were *Klebsiella pneumoniae, Pseudomonas aeruginosa, Corynebacterium amycolatum*, and *Acidovorax sp*. BLS4, though the order of abundance varied according to phenotypes. Furthermore, the bacteria *Pseudomonas aeruginosa, Stenotrophomonas maltophilia, Burkholderia gladioli, Stutzerimonas stutzeri, Ralstonia insidiosa, Leifsonia shinshuensis,* and *Leifsonia sp*. WHRI.6310E showed positive correlation with diseased condition, providing evidence of dysbiosis. These findings strengthen our previous studies demonstrating that IVDD is caused by infection, particularly due to *P. aeruginosa* abundance, which facilitates polymicrobial infection that may influence IVDD progression by altering host immunity and the IVD microbiome.

Keywords: Intervertebral disc degeneration, Low back pain, microbiome, infection

1. Background

Low back pain (LBP) affects approximately 619 million people globally as of 2020, with projections reaching 843 million by 2050, making it the leading cause of years lived with disability worldwide (Ferreira et al., 2023). This condition predominantly arises from intervertebral disc (IVD) degeneration, a multifactorial process where emerging evidence supports subclinical infection as a key contributing factor. Recent research has evidenced that human discs harbor unique microbiomes, with dysbiosis determining health and disease outcomes. Our previous studies have provided substantial evidence for microbial colonization rather than contamination in disc tissue (Rajasekaran et al., 2017, 2020a,b; 2021,2022a,b; 2023;Chen et al., 2018)).

2. Aim

The present study aims to characterize microbial communities through shotgun metagenomic sequencing to understand the role of the disc microbiome in degeneration processes.

3. Methodology

The approach involved a cohort comprising healthy IVD tissue from brain-dead organ donors and diseased tissue from patients undergoing surgery. Shotgun sequencing was performed using the Illumina NovaSeq platform and

ISBN: 978-93-95105-97-2

analyzed with Kraken 2 (v-2.2.3) and Bracken tools. The relative abundance of bacterial species and species diversity were assessed using standard statistical analyses.

4. Results

The results revealed rich species diversity with significant differences among control and diseased samples (p=0.03), and beta diversity analysis showed significant variation (p=0.002) between control and diseased groups. The most abundant bacterial species identified were *Klebsiella pneumoniae, Pseudomonas aeruginosa, Corynebacterium amycolatum,* and *Acidovorax sp. BLS4,* though the order of abundance varied according to phenotypes. Furthermore, the bacteria *Pseudomonas aeruginosa, Stenotrophomonas maltophilia, Burkholderia gladioli, Stutzerimonas stutzeri, Ralstonia insidiosa, Leifsonia shinshuensis,* and *Leifsonia sp. WHRI.6310E* showed positive correlation with diseased condition, providing evidence of dysbiosis.

5. Conclusion

The findings strengthen our previous studies demonstrating that IVDD is caused by infection, particularly due to *P. aeruginosa* abundance, which facilitates polymicrobial infection that may influence IVDD progression by altering host immunity and the IVD microbiome.

6. Acknowledgments

I acknowledge Ganga Orthopaedic Research and Education Foundation (GOREF), Coimbatore, India, for providing grant, infrastructure and facilities for this study.

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OP-11

Mapping Epigenomic modifications in Rheumatoid Arthritis Synovial Fibroblasts and Monocytes via ChIP-Seq Meta-Analysis

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Abstract
Rheumatoid Arthritis (RA) is a chronic autoimmune disorder driven by synovial inflammation. Fibroblast-like synoviocytes (FLS) and circulating monocytes play key roles, with monocytes differentiating into macrophages which produce elevated pro-inflammatory cytokines and maintaining hyper-inflammatory memory, accelerating joint damage and disease progression. Epigenomic alterations and chromatin structure rearrangement affect and regulate the characteristic and expression of genes in RA development through mechanisms such as DNA methylation, histone modification and chromatin remodeling. In this study, ChIP-seq data of FLS and monocytes were explored using a meta-analysis workflow to identify histone modifications and meta-Differential Binding Sites (meta-DBS). The retrieved data was pre-processed and peak calling was carried out. Then, Meta-analysis was performed and results were compared for more precision. Further, enrichment analyses were also employed to find the functional importance of common meta-differentially bound sites and associated genes in RA. Meta-analysis revealed shared meta-DBSs among FLS and monocytes. Histone marks such as H3K27ac, H3K36me3, H3K4me3, H3K27me3, H3K4me1 and H3K9me3 were found across the binding sites highlighting both active regulatory regions and repressive chromatin domains. Specifically, H3K27me3 and H3K4me3 were observed in monocyte associated meta-DBSs. These findings underscore the role of epigenetic dysregulation of histone modifications on binding site in maintaining chronic synovial inflammation and joint deterioration in RA.

Keywords: Meta-analysis, Rheumatoid arthritis, Synovial fibroblast, Monocytes, ChIP-seq and Differential Binding Sites.

1. Introduction
Rheumatoid Arthritis (RA) is a chronic auto-immune disorder characterized by synovial inflammation and leading to joint damage. As the understanding of RA pathogenesis deepens, fibroblast-like synoviocytes (FLS)/Synovial fibroblasts are increasingly recognized for their crucial role in RA pathogenesis. Circulating monocytes also strongly contribute to RA progression due to production of pro-inflammatory molecules. Studies demonstrate that the proportion of circulating monocytes positively correlates with RA disease activity. Monocytes migrate into RA synovial tissues where they differentiate into macrophages. RA monocyte- derived macrophages maintain the hyper-inflammatory memory bias of their precursor cells, with significantly higher expression of pro-inflammatory cytokines. Metabolic alterations such as Glycolytic dependence, Elevation of Iron and Ferroptosis, Leucine Transport, Arginine Biosynthesis, Hyper-Inflammatory phenotype, abnormal lipid metabolism are found

ISBN: 978-93-95105-97-2

to be prominent in RA-FLS and monocytes. RA monocytes and FLS show transcriptional and metabolic changes, but epigenetic pattern and mechanisms driving pro-inflammatory memory remain unclear. By integrating Monocytes and FLS ChIP-seq datasets, this study aim to uncover conserved regulatory signatures and to construct a comprehensive regulatory network bridging monocyte and FLS in RA.

2. Methods

The datasets for FLS and monocytes were retrieved from NCBI-SRA and then subjected to preprocessing. The quality check using Falco revealed presence of adapters which were removed using trimmomatic. The trimmed sequences were aligned using Bowtie2 with reference genome hg38. The aligned sequences were sorted and deduplicated using samtools. The refined BAM files were used for peak calling using MACS2. The peaks across all samples and conditions were merged using BEDtools merge. All the tools were implemented in linux platform. Gene ontology (GO) and pathway enrichment analyses were performed with Cistrome-GO. The adjusted p-value ≤ 0.05 was applied to select significant GO terms and KEGG pathways. The general steps involved in the present study are summarized in Fig. 1.

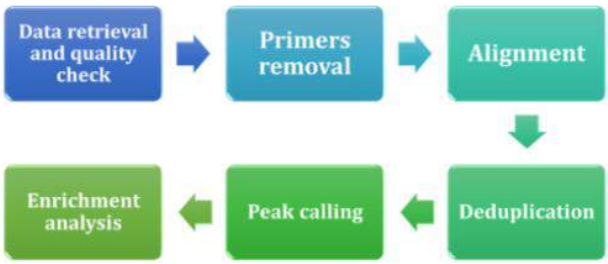

Figure 1: An overview of the steps performed in the present study.

3. Results and Discussion

The Chip-seq datasets were explored and subjected to meta-analysis in this study. Six histone modifications were observed in the FLS dataset, including H3K27ac, H3K36me3, H3K4me3, H3K4me1 H3K27me3, H3K9me3 and two in monocyte dataset which were H3K4me3 and H3K27me3. Raw Fastq files were downloaded and quality check reported the presence of primers. Trimmomatic was utilized to remove the primer sequences. The aligned BAM files were utilized for peak calling. For more stringent results, the q-value was set to 0.01. The peaks were merged for each histone modification separately. Majority of the peaks were located in the promoter region. The GO analysis of FLS dataset revealed genes associated with cell differentiation, anchoring junction, and collagen-containing extracellular matrix while the monocytes disclosed genes that are specific to monocyte-macrophage differentiation that could emerge as potent pro-inflammatory mediators. Pathway analysis highlighted the pathways such as pathways in cancer, axon guidance, calcium signalling pathway and TNF signaling pathway indicating shared regulatory mechanisms underlying aberrant cell proliferation, migration, and pro-inflammatory signaling in RA.

4. Conclusion

This study provides an integrative meta-analysis of FLS and monocyte ChIP-seq datasets in rheumatoid arthritis, highlighting conserved regulatory signatures across distinct cell types. Promoter-associated peaks and enrichment of pathways related to cancer, axon guidance, calcium signaling, and TNF signaling highlight shared mechanisms driving aberrant proliferation, migration, and inflammation. These findings suggest that transcriptional and epigenetic reprogramming in both stromal and immune compartments contribute to RA pathogenesis, and emphasize the importance of targeting cross-cell regulatory networks for therapeutic intervention.

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OP-12

Redox-Sensitive and Receptor-Targeted Delivery of Erlotinib using Amino Acid-Functionalized BNNTs: In Vitro Cytocompatibility Studies towards A549 and HEK293

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Abstract

Advancing precision oncology requires nanocarriers that enable receptor-specific, stimuli-responsive drug delivery with high biocompatibility and efficacy for a successful selective cancer therapy. In this study, we developed a redox- and receptor-responsive drug delivery system based on boron nitride nanotubes (BNNTs) functionalized individually with amino acids Arginine (Arg) and Aspartic acid (Asp) to enhance dispersion, introduce reactive functional groups and improve the biological interaction. The functionalized BNNTs were further conjugated with folic acid (FA) and hyaluronic acid (HA) to target folate and CD44 receptors, respectively, which are overexpressed in various cancer types. Erlotinib, a tyrosine kinase inhibitor, was loaded into the nanocarriers, achieving high drug encapsulation efficiencies (Arg: 77.1 ± 0.02%, Asp: 82.6 ± 0.04%) and loading capacities (Arg: 33.16 ± 0.12%, Asp: 35.5 ± 0.09%) to deliver at the targeted area. The dual-ligand decorated system demonstrated efficient drug encapsulation (Arg - 77.1±0.02% and Asp - 82.6±0.04%) and loading capacities (Arg - 33.16±0.12% and Asp - 35.5±0.09%). Drug release studies revealed redox- and pH-responsive behaviour, with accelerated release under tumor-mimicking acidic and reductive conditions. Among the two formulations, BNNT–Arg–HA–FA showed faster release and higher tumor-specific uptake, while BNNT–Asp–HA–FA released drug more slowly, contributing to reduced off-target cytotoxicity. In vitro cytotoxicity (MTT assay) against A549 lung cancer cells confirmed superior anticancer efficacy of both nanocarriers compared to free erlotinib, with minimal toxicity observed in HEK293 normal cells. Hemocompatibility assays indicated negligible hemolytic activity, supporting systemic safety for delivery platform. These amino acid-assisted BNNTs with dual stimuli responsive and receptor-targeted nanocomposite material offers a promising approach for selective, redox-triggered delivery of Erlotinib with favourable in vitro performance, making it a potential nanomedicine candidate for future clinical translation in targeted cancer therapy.

Keywords: Nanocarrier, Targeted drug delivery, Hyaluronic acid, CD44 receptor, Redox response, Erlotinib.

1. Introduction

Targeted nanocarriers have emerged as a successful and promising material to improve the therapeutic efficacy of anticancer drugs while minimizing systematic toxicity and off-target effects. Boron nitride nanotubes (BNNTs)

ISBN: 978-93-95105-97-2

offer a versatile platform for drug delivery systems due to their high surface area, biocompatible to living cells, excellent chemical and thermal stability and more efficient to load drug molecules (1,2). Their hydrophobic surface enables to encapsulate even poorly soluble drugs such as erlotinib, a tyrosine kinase inhibitor which is widely used to treat various cancer type but limited by low aqueous stability (3). To improve tumor specific uptakes, BNNTs can be functionalized with receptor targeting ligands to target overexpressed receptors onto cancer cells. In additional, amino acid conjugation with BNNTs allows modulation of surface charge and hydrophobicity, which can influence drug encapsulation efficiency, drug release behavioural kinetics, and interactions with biological systems. Furthermore, the cationic and anionic nature of the amino acid functionalized nanocarriers may have an effective antibacterial property for reducing opportunistic infections in immunocompromised cancer patients (4). This present research work focus on the development of ARG and ASP amino acid conjugated BNNT along with folic acid and hyaluronic acid nanocomposites as a multifunctional drug carrier for erlotinib drug, evaluating their drug loading capacities, redox stimuli release behaviour, Drug release kinetics under different pH environment, anti-bacterial activity, and biocompatibility towards living cells.

2. Methods

BNNTs were initially functionalized with amino groups using APTES (3-aminopropyl triethoxysilane), which create a reactive site for subsequent bioconjugation with amin acids (5). Arginine and aspartic acid are the two amino acids which were conjugated to BNNTs separately using carbodiimide-mediated coupling facilitated by EDC (1-Ethyl-3-(3-dimethylaminopropyl) carbodiimide) and DMAP (4-Dimethylaminopyridine) as nucleophilic catalyst along with Folic acid (FA) and Hyaluronic acid (HA). The drug Erlotinib was loaded onto the synthesized nanocomposites through electrostatic interaction under controllable conditions to maximize the encapsulation while maintaining at colloidal stability. The resulting drug nanocomposites were characterized for morphological size, functional group, drug loading and encapsulation and in-vitro studies. Drug release studies were carried out under normal physiological (pH-7.2) and tumor-mimicking acidic condition (pH – 6.5), with or without hyaluronidase (HAase) and glutathione (GSH) to evaluate redox-stimuli response drug release profiles. Antibacterial activity was also evaluated against *Escherichia coli* (gram-negative) and *Bacillus subtilis* (gram-positive) using standard inhibition assays (6). Hemolysis and coagulation assays were conducted to determine the blood compatibility of the nanocomposites and preliminary cytotoxicity studies were performed against A549 and normal cell lines.

3. Results

The synthesized two nanocomposites were characterized using Field-emission scanning electron microscope (FE-SEM) confirming a successful conjugation of amino acids onto BNNTs surface, showing a uniform coating and reduced aggregation. FTIR spectra confirms the functionalization process of BNNTs with amino group, conjugation of amino acids, folic acid and hyaluronic acid using amidation process, and encapsulation of Erlotinib drug onto the synthesized nanocomposites. Drug encapsulation and loading percentage of BNNTs-ASP nanocomposites showed slightly higher encapsulation (82.6±0.04%) and drug loading (35.5±0.09%) compared to BNNTs-ARG nanocomposites (77.1±0.02% and 33.16±0.12%, respectively). This happens due to the difference in the surface charge and hydrophilic interactions affecting drug adsorption. Redox-stimuli and pH responsive drug release studies revealed that BNNTs-ARG nanocomposites exhibited minimal release at pH-7.2 under controlled conditions, with a gradual increase upon Haase treatment, moderate enhancement with GSH, and maximum release in the presence of HAase and GSH. Under pH-6.5 tumor-environment, drug release was accelerated under controlled and sustained release to cancerous tissue (7). In comparison, the BNNTs-ASP nanocomposites showed a higher baseline release at pH-7.2, moderate HAase induced release, stronger GSH-mediated release, and maximum release with HAase and GSH. At pH-6.5, this nanocomposite exhibited a rapid and sustained drug release, suggesting faster release under normal cell conditions (8). These observations confirms that BNNT-ARG nanocomposites achieve more controlled drug release profile when compared with BNNT-ASP nanocomposites particularly in tumor microenvironment. Anti-bacterial activities for both the samples demonstrated a significant activity against *E.coli* and *B.subtilis* with BNNT-ARG nanocomposites shows slightly higher efficacy due to cationic surface promoting stronger interaction with bacterial cell membranes. Hemolysis and coagulation assay confirmed that these both the nanocomposites were within safe limits, indicating excellent

ISBN: 978-93-95105-97-2

blood cell compatibility. Cytotoxicity assays also confirms that these two nanocomposites show a good toxicity effect on A549 cancer cells while showing minimal effect on normal cell lines.

4. Conclusion

In summary, these research work findings suggest that BNNT-ARG nanocomposites serves as a superior multifunctional nanocarrier, providing controlled, redox-responsive, and pH-sensitive drug release, while both the nanocomposites offer a good antibacterial activity, highly biocompatible, proving them a promising nanocarrier for targeted cancer therapy.

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ISBN: 978-93-95105-97-2

OP-13

UHPLC-MS/MS Based Metabolomic Profiling Revealed Alteration in Amino Acids During Intervertebral Disc Disease

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1. Background

Intervertebral disc degeneration (IVDD) is a multifactorial disease that causes structural and biochemical changes that lead to disability. Understanding the molecular signatures of intervertebral disc (IVD) during degeneration is crucial to understand the disease pathogenesis. Metabolomic profiling of control and modic intervertebral disc tissue will elucidate the metabolic alteration during disease which supports the development of metabolite markers for the early diagnosis of disease and treatment.

2. Objective

To characterize the metabolic alteration in modic IVD tissue compared to control IVD tissue and to decipher the altered pathway associated with disc degeneration.

3. Methods

Intervertebral disc tissues were obtained from 21 brain-dead organ donors as controls and 20 IVDD patients (Modic & non modic) undergoing surgery. Metabolites were extracted from the nucleus pulposus tissue of the control, modic and non modic group using a triple solvent mixture (Methanol: Acetonitrile: Water with 2:2:1 ratio) for UHPLC-MS/MS analysis. The raw data obtained were analyzed using Compound Discoverer 3.7 and the metabolic features were further analyzed using mzCloud and ChemSpider databases. Further, differential and pathway analysis was performed by MetaboAnalyst 6.0 to detect the significantly altered metabolites and associated pathways in the disease.

4. Results

The study identified 832 metabolites from control and disease samples by Untargeted metabolomics. Metaboanalyst 6.0 software analysis revealed alteration of 224 metabolites in disease among which 141 metabolites were upregulated and 83 metabolites were downregulated. Chemical classification of altered metabolites indicated that amino acids, peptides, and analog group was enriched during disease. Among the amino acids, L-phenylalanine, and L-Proline were upregulated in disease and peptides such as dipeptides and tripeptides were also significantly altered in the disease group. Further, the KEGG pathway analysis revealed that phenylalanine metabolism was the top pathway enriched during disease and downregulated metabolites were associated with histidine metabolism and retinol metabolism.

5. Conclusion

Untargeted metabolomic analysis highlights the changes in the metabolic profiles of disease and normal intervertebral disc tissue and reveals altered amino acids including phenylalanine and proline in disease. Our study hypothesizes that the breakdown of aggrecan and collagen alpha 1 type II leads to the elevated level of phenylalanine and proline amino acids and few dipeptides in the disease group. Further large-scale analyses are necessary to validate the findings.

ISBN: 978-93-95105-97-2

6. Acknowledgments
I acknowledge Ganga Orthopaedic Research and Education Foundation (GOREF), Coimbatore, India, for providing infrastructure and facilities and AO Spine grant for this study (Grant ID: AOS-Startup-23-024).

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OP-14

Leveraging Machine Learning Models to Elucidate Microbiota-Mediated Biotransformation

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Abstract
A single chemical compound can be transformed into numerous products depending on the enzymes that catalyse it. Identifying enzyme-substrate relationships is crucial for drug development and prescription. The gut microbiome has gained attention because it plays a significant role in transforming pharmaceutical drugs, leading to altered drug responses. Similarly, skin microbial enzymes act on topical pharmaceuticals, cosmetics, and other xenobiotics. Recent advancements in machine learning approaches have been extended to predict xenobiotic biotransformation mediated by microbial enzymes. The microbial community is highly diverse and shaped by geographical location, genetics, diet, etc. Existing models often focus on specific microbial communities, limiting their generalizability. To address this gap, we have developed a robust machine learning model capable of predicting the susceptibility of chemical compounds to microbial enzymes across diverse environments.

1. Introduction
A single chemical compound can be transformed into numerous products depending on the enzymes that catalyse it. Identifying enzyme-substrate relationships is crucial for drug development and prescription. The gut microbiome has gained attention because it plays a significant role in transforming pharmaceutical drugs, leading to altered drug responses. Similarly, skin microbial enzymes act on topical pharmaceuticals, cosmetics, and other xenobiotics. Recent advancements in machine learning approaches have been extended to predict xenobiotic biotransformation mediated by microbial enzymes. The microbial community is highly diverse and shaped by geographical location, genetics, diet, etc. Existing models often focus on specific microbial communities, limiting their generalizability. To address this gap, we have developed a robust machine learning model capable of predicting the susceptibility of chemical compounds to microbial enzymes across diverse environments.

2. Methodology
A comprehensive dataset encompassing enzymatic reactions, protein sequences, and substrate information was sourced from publicly available databases. These data were processed using molecular fingerprinting techniques

ISBN: 978-93-95105-97-2

to facilitate machine learning applications. Six machine learning classifiers: i) Logistic Regression, ii) K-Nearest Neighbours, iii) Decision Trees, iv) Random Forest, v) Naive Bayes, and vi) Gradient Boosting have been constructed. Model performance was assessed using metrics such as accuracy, precision, recall, F1-score, and ROC-AUC. To ensure the robustness of our findings, we employed 10-fold cross-validation and reserved 10% of the positive dataset as a blind test set, which was excluded from the training phase.

3. Results

The dataset comprised enzyme-substrate interaction data from 1,687 species across 854 genera, encompassing 3,224 unique Enzyme Commission (EC) numbers. Approximately 70% of this data was utilised for model training, with the remaining 30% allocated for testing. Among the classifiers assessed, the Random Forest model exhibited superior performance, achieving a score of 0.99 across all evaluation metrics. The Logistic Regression model followed closely with an accuracy of 0.92. The Random Forest model demonstrated a mean accuracy of 0.831 (±0.068) during 10-fold cross-validation. Notably, the model accurately predicted 96% of the blind set data.

4. Conclusion

The Random Forest classifier developed in this study offers a high-accuracy tool for predicting the susceptibility of chemical compounds to microbial enzymes. This model can identify potential enzymes and microbial species involved in the biotransformation of a given compound. Its applications can be extended to personalising pharmaceutical treatments based on individual gut microbiome profiles and optimising topical formulations by considering skin microbial compositions. By integrating such predictive models, we can advance precision medicine and enhance the efficacy and safety of therapeutic interventions.

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ISBN: 978-93-95105-97-2

OP-15

Metagenomic Insights into Resistome–Microbiome Interactions Across Biotic and Abiotic Environment

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Abstract:
Antibiotic resistance is a global threat that spreads across diverse environments, posing risks to human, animal, and ecosystem health while causing significant economic losses. This study examined resistome profiles, microbial diversity, and associations between antibiotic resistance genes (ARGs) and bacterial taxa using machine learning models. Shotgun metagenomic datasets from six ecosystems were classified into biotic (healthy individuals, livestock) and abiotic (freshwater, ocean sediment, agricultural soil, wastewater) environments for analysis. A total of 1,686 ARG subtypes across 28 ARG types were identified, with beta-lactam resistance genes being the most abundant in abiotic environments and tetracycline resistance genes dominating biotic environments. Key bacterial genera influencing ARGs included *Escherichia, Lactobacillus,* and *Subdoligranulum* in biotic environments and *Pseudomonas, Chryseobacterium,* and *Brevundimonas* in abiotic environments. Model validation showed moderate to strong correlations ($0.400 \leq R^2 < 0.600$ to $R^2 > 0.600$) between predicted and observed ARG abundances across bacterial genera. The RF model demonstrated high accuracy in predicting resistance risk. These findings provide crucial insights into antimicrobial resistance dynamics, facilitating early detection of resistance hotspots and enhancing risk prediction across ecosystems.

1. Introduction

Antimicrobial resistance (AMR) represents a critical global challenge that transcends ecological boundaries, threatening human, animal, and environmental health while imposing substantial economic burdens. Antibiotic resistance genes (ARGs) are pervasive in both biotic and abiotic environments, primarily driven by bacterial proliferation and horizontal gene transfer (HGT) mediated by mobile genetic elements (MGEs). While numerous studies have examined ARG distribution across ecosystems, there has been limited emphasis on differentiating resistome patterns between biotic and abiotic domains. Such comparative understanding is essential for developing predictive frameworks to monitor and mitigate the dissemination of AMR under the One Health paradigm. This study aims to characterize resistome and microbiome profiles across biotic and abiotic environments using metagenomic sequencing, and to develop predictive models that elucidate their interactions, thereby generating insights to inform AMR management strategies.

2. Methodology

Sixty shotgun metagenomic datasets representing six ecosystems—healthy individuals, freshwater, ocean sediment, agricultural soil, livestock, and wastewater were retrieved from public repositories. Resistome profiles were annotated using the CARD database, while bacterial community composition was determined through Kraken2. To investigate microbiome–resistome interactions, multiple machine learning approaches were employed, with a focus on model validation and predictive performance.

3. Results

Across the datasets, 1,686 ARG subtypes spanning 28 ARG types were identified. Abiotic environments were enriched in beta-lactam resistance genes, whereas tetracycline resistance genes predominated in biotic environments. Microbial genera strongly associated with ARGs differed by ecosystem type: *Escherichia, Lactobacillus,* and *Subdoligranulum* were major contributors in biotic environments, while *Pseudomonas, Chryseobacterium,* and *Brevundimonas* dominated in abiotic settings. Model validation demonstrated moderate to strong correlations between observed and predicted ARG abundances (R^2 ranging from 0.400 to 0.600). Among tested models, the Random Forest (RF) classifier consistently outperformed others, exhibiting high predictive accuracy in distinguishing resistome risks across both biotic and abiotic environments.

4. Conclusion

This study demonstrates that resistome profiles differ between biotic and abiotic environments, driven by distinct patterns of ARG prevalence and microbial associations. The superior predictive capacity of machine learning

ISBN: 978-93-95105-97-2

models, especially Random Forest, highlights their value in assessing resistome risk with high reliability. These findings strengthen the One Health perspective, supporting evidence-based surveillance and guiding targeted strategies to curb the spread of antibiotic resistance across interconnected ecosystems.

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OP-16

Identification of Molecular Signatures in Rheumatoid Arthritis-Associated Atherosclerosis- A Transcriptomics Approach

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Abstract

Rheumatoid arthritis is a rising health issue that results in mobility-related functionality loss in the global population. This auto-immune disorder triggers the progression of atherosclerosis, a condition where blood flow is obstructed by plaques deposited on the blood vessel walls. Studies have shown that the increase in mortality of the RA patients is contributed by the comorbidity of cardiovascular diseases. Therefore, the identification of molecular signatures that indicates the chances of progression of cardiovascular diseases in the RA patients would increase the patient's life span by providing early treatment to the imminent cardiovascular disease. In this study, the gene expression profiles of the blood cells of RA and atherosclerosis patients were compared and analyzed to find the marker genes, such that these gene expression signatures, from the blood cells of the patient, could serve as potential biomarkers in the less-invasive diagnosis of the occurrence of RA-associated atherosclerosis. About 2880 differentially expressed genes were identified by DESeq2 analysis and were classified based on the 3 gene ontology terms and the DEGs from both the disease datasets group together in various GO terms, indicated that they have interrelated genes. The pathways that the DEGs involve in were found to be those that trigger immune responses, pointing out to the immunological relation between the two diseases. PPI network and clustering analysis yielded the hub genes in which the PDGFR-A, TNF and IL1 was found to indicate the progression of atherosclerosis in RA patients. The detection of expression level of these genes could help in early diagnosis of progression of atherosclerosis in RA patients.

Keywords: Rheumatoid arthritis, Atherosclerosis, RNA-sequencing, differential gene expression analysis, biomarkers.

1. Introduction

Rheumatoid arthritis is an autoimmune disorder which is characterized by chronic inflammation and damage of the synovial fluid cells present in the joints (1). The mortality of RA is majorly due to occurrence of cardiovascular diseases, which compromises the functioning of the circulatory system. One among this is atherosclerosis, a condition where plaques form on the inner walls of blood vessels, resulting in obstructed blood flow to tissues and leads to stroke and myocardial infarction (2). In order to predict the progression of atherosclerosis in RA patients, less invasive diagnostic methods are required, so as to prevent the RA-associated atherosclerosis progression. This study aims to unravel the molecular relationships at gene expression level between rheumatoid arthritis and atherosclerosis to identify potential biomarkers that could indicate of atherosclerosis progression in RA-patients through their blood sample, using differential gene expression analysis (3).

ISBN: 978-93-95105-97-2

2. Materials and Methods

The RNA sequencing experiment data on blood cells, collected from patients diagnosed with rheumatoid arthritis and atherosclerosis were identified in the GEO database of the NCBI server. The counts data and metadata from each of the GEO dataset retrieved were given as input to the R studio interface and were converted to DESeq object. The differential gene expression analysis was performed using DESeq2 software package. The results were the written in .csv files and were retrieved to respective folder of interest. The DEGs were filtered out based on their log_2FC value ($-1.5>log_2FC>1.5$) the p-value (<0.1) (3). The DEGs thus selected were given as input list to the DAVID website and was subjected to gene ontology classification based on cellular component, molecular function and biological process (5). The significant classifications were chosen based on their fold enrichment value being close to one. The pathway enrichment was done for the same, using the KEGG database (6), and the possible pathways which the DEGs play a role in were identified and screened, based of p-value and fold enrichment score. The DEGs list was given to the stringapp plugin of the Cytoscape software version 3.10 (7), which constructs a protein-protein interaction network, using which cluster analysis was performed using MCODE app plugin that puts highly connected nodes together as a cluster. The clusters were further analyzed for it functions by functional enrichment. The hub genes in the cluster network were identified using the cytoHubba app plugin, based on the degree of the node in the cluster. These genes were then studied for their potential as biomarker for atherosclerosis progression in rheumatoid arthritis patients.

3. Results

From the DESeq2 output, about 2880 differentially expressed genes were identified, based on their log2foldchange and p-value. On performing GO classification, 5 BP terms, 4 CC terms and 5 MF terms were identified to have the most genes grouped together. The 6 pathways which the DEGs take part in were identified and further studied for their relevance to the diseases. From the undirected PPI network comprising 1801 proteins and 11614 interactions, 44 subnetworks were identified, from which six clusters were chosen for functionality studies to identify its importance and narrow down the hub genes that can be used as biomarkers.

4. Discussion

The GO classification of the DEGs indicates that these genes reside in the cell membrane and act as receptors for receiving signals and involve in intracellular signaling as protein kinases. The pathways the DEGs take part in, were found to be those that involve in immune system regulation such as the Wnt signaling pathway, NF-kappaB signal transduction and JAK-STAT signaling pathway and thereby, signifying the link between the two diseases in immunological terms. From the hub gene analysis of the clusters, the platelet-derived growth factor receptor-A (PDGFRA) was found to be one of the hub genes in the cluster 2. It was present in the neutrophil RNA sequencing dataset in RA-patients, with a log_2FC value of 1.77. As the overexpression of this gene has been reported to cause cardiovascular diseases (8), the overexpression of this gene in RA patients, could give an insight to high propensities of the patient to develop atherosclerosis. Similarly, the hub genes interleukin 1 and tumor necrosis factor-α were found to be differentially expressed in the $CD14^+$ cells RNA sequencing data and is widely for its involvement in inflammatory responses in both RA and atherosclerosis (9,10).

5. Conclusion

From this study the DEGs from the blood cells were identified and studied for its involvement in the RA-associated atherosclerosis. The immune response and regulation pathways were major areas where the commonalities between two diseases were found through GO analysis, PPI network and cluster analysis. Potential biomarkers identified through this study are PDGFRA, IL1 and TNF-α. These give a way for a less-invasive, blood-based detection of RA-associated atherosclerosis progression, which would contribute to the reduction in the mortality of rheumatoid arthritis.

ISBN: 978-93-95105-97-2

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OP-17

Transcriptome analysis of Duchenne Muscular Dystrophy

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Abstract

Duchenne Muscular Dystrophy (DMD) is an X-linked disorder due to dystrophin deficiency causing sarcolemmal fragility, chronic inflammation, and progressive myofiber loss. Despite advances in gene-targeted and steroid therapies, early biomarkers and molecular targets for monitoring progression or classifying patients remain challenging. Five public DMD RNA-Seq datasets' (skeletal muscle and hiPSC-derived cardiac fibroblasts) counts were normalized with DESeq2 (R), and PCA showed DMD–control separation, indicating consistent transcriptomic differences between samples. Differential expression analysis revealed some significantly dysregulated genes - upregulated genes linked to synaptic vesicle cycling and neurotransmission, including SYT1 (Synaptotagmin-1), SLC6A1 (a GABA transporter), and NPTX1 (Neuronal Pentraxin-1), indicating possible involvement of neuromuscular junction remodeling and excitatory/inhibitory imbalance in DMD pathology. Downregulation of genes such as IL18R1 (Interleukin-18 Receptor 1), GABRA5, and other GABA-A receptor subunits suggest the immune regulation and inhibitory neurotransmission. Gene Ontology and KEGG enrichment analyses confirmed the involvement of vesicle-mediated transport, synaptic signaling, neurodevelopmental processes, immune response modulation, cytokine signaling, and cell cycle regulation. Particularly, IL18R1 was found to be a candidate downregulated gene across multiple datasets, indicating a probable biomarker role for immune dysfunction in DMD. SYT1 upregulation reflected altered presynaptic activity and neuromuscular interface stress, indicating its potential as a marker of NMJ pathology. Together, these findings indicate that neuroimmune interactions play an important role in DMD progression with SYT1 and IL18R1 genes as prospective biomarkers. This transcriptome-wide analysis highlights the importance of integrating skeletal and cardiac tissue datasets to find convergent molecular signatures, and for precise diagnostics in DMD.

Keywords: Duchenne Muscular Dystrophy, RNA-Seq, Differential gene expression

ISBN: 978-93-95105-97-2

1. Introduction

DMD is caused due to DMD gene mutations that affect the production of functional dystrophin, affecting young boys around the age 2-3. This affects the dystrophin-glycoprotein complex [4] and contributes to sarcolemmal instability, calcium dysregulation, chronic inflammation, and muscle fiber loss; further leading to diaphragm and cardiac involvement along with skeletal muscle resulting in mortality [1-3]. Current diagnostics for DMD include monitoring creatine kinase levels, electromyography (EMG) and tissue biopsy, and these methods help in detecting the disease only after the symptoms appear [2], countering early detection which is essential to prevent mortality. These methods also lack specificity for molecular alterations. Transcriptomic profiling can be carried out to characterize these molecular signatures and to identify biomarkers for early detection. Previous transcriptomics studies have highlighted dysregulation in inflammation, fibrosis and metabolic pathways focusing on single tissue type. Mortalities have also been caused due to DMD in cardiac muscles leading to cardiac arrest through progressive cardiomyopathy, which is the leading cause of death in DMD patients [4]. By combining the datasets for both DMD skeletal muscle and cardiac muscle, convergent molecular signatures can be elucidated to identify biomarkers for DMD progression.

2. Objective

To combine five publicly available DMD RNA-Seq datasets for different tissues, to identify DEG using DESeq2, perform GO/KEGG enrichment, find out hub genes using cytoHubba and to find out suitable biomarkers for DMD.

3. Methods

3.1 Datasets

Five gene expression omnibus (GEO) datasets (skeletal muscle biopsy, hiPSC-derived cardiac fibroblasts, myogenic cells and myotubes) sequenced by Illumina (paired end) were used and these data were combined irrespective of tissue and batch variations.

3.2 Differential gene expression analysis

Differential gene expression was carried out using DESeq2 (R 4.4.2) and gene annotation was merged after this process. The results were visualized using PCA and volcano plots.

3.3 Enrichment analysis

The upregulated and downregulated genes were sorted and analysed with clusterProfiler enrichGO (Biological Process/Molecular Function/Cellular Component) and enrichKEGG separately.

3.4 Network analysis and hub gene identification

Network analysis was performed using Cytoscape by loading the DEGs into STRING, and hub genes were identified by cytoHubba's MCC (Maximal Clique Centrality) ranking.

4. Results & Discussions

Visualization: The PCA plot shows that there is a difference between DMD samples and controls. The overlap of some control and samples might be due to tissue heterogeneity or disease stage. The volcano plot showed a large number of differentially expressed genes which includes many with very strong fold changes.

GO and KEGG Enrichment: Upregulated processes included secretion, epithelial-mesenchymal transition, cardiac development, synaptic signaling, vesicle transport, and antigen processing. On the other hand, downregulation involved enriched in pathways related to the cell cycle, mitotic checkpoints, neuroactive ligand-receptor interactions, cytokine signaling, and calcium signaling. These signals correlated with the fibrosis and neuromuscular dysfunction which is seen in DMD.

PPI and Hub genes: From protein-protein interaction analysis two hub genes were identified, the upregulated one related to synaptic neurotransmission (e.g., SYT1, NPTX1, SLC6A1/4, SLC17A7, SLC18A2, GABRA4, HLA-G, TOR1A) and the downregulated one was linked to immunomodulation and inhibitory signaling (e.g., GABRA5, GABRR1/2/3, GABRG3, IL18R1, IRAK3, SIGIRR, ORMDL3, COL2A1. This shows a neuro-immune imbalance in DMD.

Potential Biomarkers: Two potential biomarkers were identified: SYT1 - a calcium-sensing protein which is upregulated and involved in synaptic vesicle exocytosis and neuromuscular junction function; and IL18R1 - a

ISBN: 978-93-95105-97-2

receptor involved in Th1/IFN-γ-mediated cytokine signaling, was consistently downregulated. These suggests that the identified biomarkers indicate the neuromuscular-immune interface in DMD.

5. Conclusion

Transcriptomics analysis across skeletal and hiPSC-derived tissues revealed a convergent neuroimmune signature in DMD with upregulated synaptic/vesicle pathways and downregulated immune modulation and cell cycle programs. Network and hub analysis results show that SYT1 and IL18R1 are candidate biomarkers indicating NMJ stress and immune dysregulation.

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ISBN: 978-93-95105-97-2

OP-18

CRISPR-Cas12a–Based Molecular Diagnostics for Ocular Fungal Infections: Journey from Lab-towards-Bedside

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Abstract
Ocular fungal infections are one of the major causes of vision loss, yet their diagnosis remains challenging due to the poor sensitivity and prolonged turnaround of smear microscopy and culture. To address this unmet need, we developed a CRISPR-Cas12a–based molecular diagnostic assay, beginning with laboratory optimization and advancing toward a clinically deployable point-of-care (POC) format. In the initial phase, DNA extracted from clinical samples underwent recombinase polymerase amplification (RPA) followed by CRISPR-Cas12a detection. In keratitis (n = 123), the assay achieved 93% sensitivity and 89% specificity, while in endophthalmitis (n = 133), it demonstrated 93% sensitivity and 88% specificity. Validation with Sanger sequencing and metagenomic next-generation sequencing (NGS) in three endophthalmitis samples confirmed complete concordance with both culture and RID-MyC results. Although accurate, the need for DNA extraction and multi-step handling limited translation to bedside use. To streamline the workflow, we implemented an extraction-free protocol tested on eight samples, which showed 100% concordance with the DNA-based assay. To further simplify, RPA amplification and CRISPR-Cas12a detection were integrated into a single-tube format, reducing contamination risk and operator dependency. This single-tube assay, evaluated in 61 samples, yielded 90% sensitivity and 90% specificity. Analytical sensitivity studies with the RPA-Exo fluorescence readout confirmed reliable detection of as few as 10^4 genomic copies of *Candida albicans*. The potential of CRISPR-based diagnostics, supported by omics validation, to become a rapid, accurate, and practical POC tool for ocular fungal infections in the future.

Keywords: Ocular Fungal Infection, Crispr based detection, Point of care diagnosis, Metagenomics NGS, RIDMyC

1. Introduction

Ocular fungal infections, including keratitis and endophthalmitis, represent a significant cause of vision loss worldwide. These infections are particularly challenging to diagnose due to their nonspecific clinical presentation and the limitations of conventional diagnostic tools. Microscopy and culture, the standard frontline methods, often suffer from low sensitivity and specificity[1] . Culture-based confirmation is further hindered by the slow growth of fungi, with many samples remaining negative despite clinical evidence of infection. This diagnostic gap frequently delays treatment, leading to poor visual outcomes. Molecular diagnostics, particularly those leveraging omics-based technologies, have shown promise in overcoming these limitations. CRISPR-based diagnostics, which utilize the collateral cleavage activity of Cas nucleases upon recognition of a target sequence, offer rapid, highly specific, and sensitive detection of nucleic acids. Cas12a[2], when coupled with recombinase polymerase amplification (RPA)[2] , enables isothermal amplification and real-time detection within minutes, making it an attractive candidate for translation into point-of-care (POC) applications. In this study, we describe the complete trajectory of developing a CRISPR-Cas12a–based assay for the diagnosis of ocular fungal infections. Beginning with laboratory optimization using DNA-extracted samples, we systematically improved the workflow through omics validation, eliminated DNA extraction, and simplified the assay into a single-tube format[3]. We also explored analytical sensitivity and initiated the design of a portable fluorescence detection device to enable bedside application.

2. Methodology

2.1 Conventional RID-MyC Assay (Laboratory Level)

For the initial laboratory assay, DNA was extracted from clinical samples using the Qiagen Blood Mini Kit, with final elution in 40 µL. A 10 µL aliquot of the extracted DNA was used as template for recombinase polymerase amplification (RPA), targeting the conserved 18S rRNA fungal region. The amplified products were detected using CRISPR-Cas12a, which recognizes the specific RPA amplicon and activates collateral trans-cleavage of a single-stranded DNA reporter labeled with 5′-FAM and 3′-quencher. Fluorescence was monitored in real time on a qPCR machine for 120 minutes. Normalization was performed against a no-template control (NTC), and baseline

ISBN: 978-93-95105-97-2

thresholds were determined using negative samples, calculated as the mean + 3 × standard deviation (SD). This workflow constituted the conventional laboratory-level RID-MyC assay[2] .

2.2 Clinical Sampling

For suspected fungal keratitis, corneal scrapes were obtained, while for endophthalmitis, intraocular samples were collected. All samples underwent parallel microbiological testing (microscopy and culture) and molecular analysis using the RID-MyC workflow described above.

2.3 Sequencing Validation

Selected positive samples underwent confirmatory sequencing. RPA amplicons were analyzed by Sanger sequencing, while a subset of endophthalmitis samples (n = 3) were subjected to metagenomic next-generation sequencing (NGS). Sequencing results were compared against culture and RID-MyC outcomes for concordance.

2.4 POC Assay Development Strategies

To adapt the assay for point-of-care (POC) application, two key strategies were explored:

1. DNA Extraction-Free Workflow: Corneal impressions were collected using Schirmer's sheets and directly subjected to the RID-MyC assay without DNA extraction. This approach was tested on eight samples.

2. Workflow Simplification: To reduce hands-on steps and risk of contamination, RPA amplification and CRISPR-Cas12a detection were integrated into a single-tube format[3] . In parallel, an exonuclease-based fluorescence detection system (RPA-Exo) was evaluated to determine analytical sensitivity and to further simplify readouts.

2.5 Device Development

To consolidate these advances into a deployable diagnostic platform, a portable fluorescence detection device was conceptualized. The device is designed to accommodate the single-tube extraction-free RID-MyC assay, integrate isothermal amplification and CRISPR detection, and provide real-time fluorescence output at the bedside. Development of this prototype device is ongoing, with the ultimate aim of translating the assay from laboratory optimization to a clinically practical POC diagnostic tool.

3. Results

3.1 Conventional RID-MyC Assay Performance

The laboratory-level RID-MyC assay demonstrated strong diagnostic accuracy when applied to clinical samples. In keratitis samples (n = 123), the assay achieved 93% sensitivity and 89% specificity compared to conventional microbiological methods[2] . In endophthalmitis samples (n = 133), sensitivity was 93% and specificity was 88%[4] . Fluorescence signals were clearly distinguishable from baseline (mean + 3 × SD of negatives), and no-template controls remained consistently negative.

3.2 Sequencing Validation

Sanger sequencing of selected RPA amplicons confirmed the presence of fungal DNA in samples identified as positive by RID-MyC. Metagenomic NGS of three endophthalmitis samples demonstrated complete concordance with both culture and RID-MyC results, further validating assay accuracy. Sequencing also confirmed species-level identification consistent with microbiological findings.

3.3 Extraction-Free Workflow

Eight corneal impression samples collected on Schirmer's sheets and directly tested by RID-MyC showed 100% concordance with the DNA-extracted assay results. This demonstrated the feasibility of eliminating DNA purification without compromising diagnostic accuracy.

3.4 Single-Tube RID-MyC Assay

Integration of RPA amplification and CRISPR-Cas12a detection into a single-tube format simplified the workflow and reduced hands-on time. Evaluation on 61 clinical samples yielded 90% sensitivity and 90% specificity[5], comparable to the conventional multi-step RID-MyC workflow. This approach also minimized contamination risk and improved suitability for bedside use.

3.5 Analytical Sensitivity (RPA-Exo Assay)

Analytical sensitivity assessment using RPA-Exo fluorescence readout showed that the assay could reliably detect as few as 10^4 genomic copies of Candida albicans. This detection limit is consistent with clinically relevant pathogen loads.

3.6 Device Development

The conceptual design of a portable fluorescence detection device was initiated to support clinical translation. The device aims to integrate the single-tube RID-MyC assay, incorporate real-time fluorescence monitoring, and

ISBN: 978-93-95105-97-2

provide a compact, user-friendly platform for bedside testing. Although development is ongoing, this step demonstrates the feasibility of translating the assay into a true point-of-care diagnostic tool.

4. Conclusion and Discussion

This study demonstrated the progressive development of the RID-MyC assay for the rapid diagnosis of ocular fungal infections. The conventional laboratory workflow, which relied on DNA extraction, RPA amplification, and CRISPR-Cas12a–based detection with fluorescence readout, showed high accuracy and concordance with both culture and sequencing results. To simplify and accelerate the workflow for point-of-care (POC) application, we developed two key modifications: (i) an extraction-free protocol using Schirmer's strip–based corneal impressions, and (ii) a single-tube RPA–CRISPR assay and/or RPA-Exo chemistry 6 . Both approaches significantly reduced assay time and operator steps, moving the system closer to a deployable POC platform. The prototype portable fluorescence detection device 7 , designed for real-time bedside readouts, is currently in the testing stage. Once fully optimized and validated with patient samples, it is expected to complement the RID-MyC assay and enable deployment in rural and resource-limited clinical centers. Further validation, particularly of the RPA-Exo single-tube strategy with patient-derived specimens, will be crucial to establishing this assay as a reliable and commercially viable diagnostic tool. Together, these efforts highlight the potential of CRISPR-based diagnostics to bridge laboratory accuracy with field-ready utility for fungal keratitis and endophthalmitis.

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ISBN: 978-93-95105-97-2

PP-01

Integration of Genomics and Transcriptomic Data for PBMC, Pancreatic Tissue-Specific Pathway Identification in Type 1 Diabetes Mellitus

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Abstract

Type 1 diabetes mellitus (T1DM) is characterized by autoimmune destruction of insulin-producing beta cells in the pancreas, leading to hyperglycemia and associated complications. Recent advancements in genomics and transcriptomics provide unprecedented opportunities to unravel the complex pathways underlying this disease. In this study, we integrated genomic and transcriptomic datasets from peripheral blood mononuclear cells (PBMCs) and pancreatic tissues of T1DM patients to facilitate the identification of tissue-specific pathways that contribute to the pathogenesis of T1DM. Through a comprehensive analysis utilizing next-generation sequencing and bioinformatic tools, we highlighted key differentially expressed genes and their associated pathways relevant to immune response, inflammation, and beta-cell function. Our results suggest significant dysregulation in pathways such as the JAK-STAT signalling pathway, which plays a crucial role in mediating immune responses, and insulin signalling pathways implicated in beta-cell survival. Furthermore, the integration of PBMC and pancreatic tissue data revealed potential biomarkers indicative of disease progression and therapeutic targets. These findings advance our understanding of the molecular mechanisms contributing to T1DM and emphasize the utility of a multi-omics approach in elucidating disease-specific biological processes. The insights gained from this integration are pivotal for developing targeted therapies aimed at modulating immune responses and preserving pancreatic functions in patients with T1DM.

Keywords: PBMC, Pancreatic Tissues, JAK-STAT signalling pathway, Beta-cell survival, multi-omics

PP-02

Prediction of Risk Score in Type 1 Diabetes Mellitus from Genomic and RNA-Seq Expression using Machine Learning

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Abstract

Type 1 Diabetes Mellitus is a chronic autoimmune disease characterized by the immune system's targeted destruction of insulin-producing pancreatic beta cells, resulting in an absolute insulin deficiency and persistent hyperglycemia that affects individuals of all ages. Typically, the disease presents with classical symptoms polyuria, polydipsia, unexplained weight loss, fatigue, and increased hunger, and if left untreated, can progress rapidly to acute, life-threatening complications including diabetic ketoacidosis. The pathophysiology of T1D is highly complex and multifaceted, involving a dynamic interplay between genetic predisposition, environmental triggers, and immune system dysfunction. Unlike monogenic disorders, T1D is polygenic in nature, with susceptibility driven by the collective influence of numerous genetic variants distributed across the genome, complicating efforts to predict disease risk using traditional genetic models alone. While advances in polygenic risk scores have markedly improved T1D risk prediction accuracy, achieving robust area under the curve values exceeding 0.90, these models primarily rely on static genetic markers derived from genome-wide association studies and do not account for the dynamic transcriptomic changes that underpin cellular responses during disease initiation and progression. Transcriptomic profiling through RNA sequencing offers a powerful approach to capture the active gene expression landscape in immune cells and pancreatic beta cells, thereby providing crucial insights into the molecular and cellular mechanisms of autoimmunity and beta-cell destruction that genetic data

ISBN: 978-93-95105-97-2

alone cannot reveal. This project proposes to develop a pioneering computational model that seamlessly integrates high-dimensional RNA-seq data with established genetic risk markers to substantially enhance the predictive power of existing PRS frameworks for T1D. The proposed framework integrates multi-omics data by combining static genetic risk markers with dynamic transcriptomic profiles derived from RNA sequencing of immune cells. It employs clustering using machine learning algorithms to analyze and integrate the multiomics datasets, uncovering key gene expression signatures linked to T1D susceptibility and disease progression. This integrative approach enhances the predictive accuracy of polygenic risk scores and facilitates the identification of novel biomarkers and therapeutic targets, ultimately advancing personalized risk stratification and precision medicine for Type 1 Diabetes.

Keywords: Type 1 diabetes, autoimmune disease, polygenic risk score, RNA sequencing, differential expression analysis, computational model.

PP-03

Genomics and Transcriptomics Studies on Regulatory mRNA – MicroRNAs Networks in Type 1 Diabetes Mellitus Through Multiomics Integration

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Abstract

Type 1 Diabetes Mellitus (T1DM) is a complex autoimmune disorder characterized by the destruction of pancreatic β-cells, driven by intricate regulatory networks and molecular mechanisms. Despite advances in omics technologies, the interplay between differentially expressed genes and their regulatory microRNAs (miRNAs) across multiple tissues remains incompletely understood. The present study aims to identify expression networks across tissues to identify disease states and molecular markers for disease diagnosis. The independent transcriptomics studies show significant differentially expressed genes and their target microRNAs. To comprehensive analysis the correlation the main objective of this approach involves integrating publicly available transcriptomic datasets from the Gene Expression Omnibus. Computational pipeline developed based on in-silico multiomics approach, we performed differential expression analysis, functional enrichment, and interactome mapping to uncover tissue-specific and shared molecular signatures. Publicly available datasets from Gene Expression Omnibus of subjects with Type 1 Diabetes Mellitus and healthy controls were analyzed to identify differentially expressed mRNAs using GEO2R by applying statistical threshold ($p<0.05$ and fold change expression > 0.5). Commonly dysregulated mRNAs were classified into upregulated, downregulated, and oppositely regulated groups, with their expression visualized by heat maps and violin plots. Protein–protein interaction networks, clustering, and enrichment analyses were performed using Gene Ontology and KEGG to identify hub genes and pathways. Integrative meta-analysis and miRNA enrichment were conducted to predict and validate regulatory miRNAs associated with the identified genes. Considering the total number of potential interactions between mRNAs and miRNAs, clustering of regulatory networks is crucial for elucidating disease mechanisms in Type 1 Diabetes Mellitus. To achieve this, both supervised and unsupervised machine learning models will be applied to the integrated datasets. Unsupervised models like k means or hierarchical clustering are employed to group mRNA-miRNA interactions and discover novel clusters. Supervised models will then be employed to prioritize and rank these clusters based on their biological relevance and association with T1DM-specific phenotypes. This integrative approach will facilitate the identification of high-confidence regulatory modules, enabling the discovery of hub regulators and potential therapeutic targets. Previous experimental results provided evidence for the regulation of oligoadenylate synthase mRNA stability in human β-cells.

Keywords: Type 1 Diabetes Mellitus (T1DM), gene expression, Multiomics, Interactomics, miRNA, mRNA, Protein-Protein Interactions, Machine Learning Models

ISBN: 978-93-95105-97-2

PP-04

Integrating Multi-Omics and Machine Learning Approach for Type 1 Diabetes Mellitus

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Abstract

Type 1 diabetes mellitus (T1DM) is a complex autoimmune disease that ultimately results in the destruction of pancreatic β-cells, necessitating exogenous insulin for glucose homeostasis. The genetic etiology of T1DM is complex with more than 200 genetic variants described to influence disease susceptibility, predominantly discovered by genome-wide association studies (GWAS). The pathophysiology of type 1 diabetes has been better understood through recent developments in molecular profiling, which have shown that 20–50 differently regulated miRNAs play a critical role in β-cell function, insulin secretion, and autoimmune reactions. Nevertheless, much remains to be known regarding the intricate associations between mRNA expression networks, miRNA regulation, and gene variants in T1DM. Type 1 diabetes mellitus (T1DM) is a disease where genetic heterogeneity impacts post-transcriptional regulation alongside perturbing protein-coding changes. Non-coding polymorphisms may influence miRNA maturation and expression, offering one potential mechanism whereby signals from genome-wide association studies localize to functional regulatory change. Coding transcript variants also have the potential to modulate miRNA–mRNA network interactions, thus reframing downstream gene expression. Computational modeling innovations provide a unifying platform for simultaneously taking into account genetic variation, miRNA dynamics, and mRNA targeting, and hence systematically interrogating the mechanisms by which inherited variation shapes T1DM regulatory pathways. The aim of the present effort is to bridge these critical gaps by applying an integrated analytical framework that integrates weighted gene co-expression network analysis (WGCNA) with cutting-edge machine learning algorithms. By using multi-omics data, we will methodically map the interplay of T1DM-associated genetic variants, miRNA regulatory networks, and mRNA expression profiles. Utilizing WGCNA will enable us to identify co-expression modules connecting genetic variants with both miRNA and mRNA networks, refining insights drawn from earlier successful uses in the research on diabetic complications. Simultaneously, machine learning algorithms will be used to predict miRNA expression levels with high accuracy on the basis of genetic variant profiles and mRNA expression data, building on recent progress that recorded high prediction accuracy for more than 350 miRNAs. Eventually, this integrative strategy is expected to bring important breakthroughs, such as the systematic discovery of genetic variants controlling miRNA expression, thorough mapping of variant-miRNA-mRNA interaction networks revealing new regulatory circuits, and the establishment of predictive machine learning models for cost-efficient inference of miRNA activity from genotypic and transcriptomic data. These results will not only add to personalized therapy approaches but also deepen risk stratification instruments for T1DM patients, pushing the field of precision medicine in diabetes therapy.

Keywords: Type 1 diabetes mellitus, genetic variants, microRNA, mRNA expression, WGCNA, machine learning, genome-wide association studies, miRNA-mRNA interactions, gene co-expression networks, multi-omics integration, precision medicine

ISBN: 978-93-95105-97-2

PP-05

Soil Metagenomics for Molecular Medicine: Mapping Resistance, Finding Cures

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Abstract

The rapid escalation of antimicrobial resistance (AMR) presents an urgent global health crisis, demanding advanced strategies for resistance surveillance and innovative pathways for the discovery of next-generation therapeutics. Soil, one of the planet's most complex microbial ecosystems, harbors an extensive diversity of microorganisms and resistance determinants, serving as a rich reservoir of both AMR genes and biosynthetic potential for bioactive compounds. In India, the widespread overuse of antibiotics in human health, livestock, and aquaculture has significantly accelerated the emergence of multidrug-resistant pathogens. The country's diverse soil microbiomes, influenced by intensive agriculture and pharmaceutical runoff, further contribute to the evolution and spread of AMR genes. In this study, we present a fully computational, multi-omics pipeline designed to analyze soil microbiomes and resistomes with precision and scalability. Publicly available shotgun metagenomic datasets from diverse soil niches are curated and subjected to rigorous quality control, followed by de novo assembly, binning, and reconstruction of high-quality metagenome-assembled genomes (MAGs). The Comprehensive Antibiotic Resistance Database (CARDdb) is systematically integrated to map known resistance determinants, identify putative novel resistance genes, and trace potential horizontal gene transfer networks that contribute to AMR dissemination. Parallel mining of biosynthetic gene clusters (BGCs) using antiSMASH and DeepBGC, coupled with functional annotation and metabolite structure predictions, enables the identification of unique antimicrobial scaffolds. This computational-only framework reduces experimental overhead, ensures scalability, and facilitates real-time AMR surveillance. By uniting resistome monitoring with secondary metabolite exploration, this integrative approach highlights the untapped potential of soil metagenomics in precision molecular medicine and positions it as a strategic tool in the fight against the AMR crisis.

Keywords: Soil microbiome, Antimicrobial resistance, Resistome analysis, Metagenome-assembled genomes, CARD database, Biosynthetic gene clusters, Computational metagenomics, Multi-omics pipeline, Horizontal gene transfer, Next-generation antibiotics

PP-06

A Machine Learning–Based Genetic Variant Analysis for Identifying T1DM-Associated Variants using IndiGenomes

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Abstract

Type 1 diabetes mellitus (T1DM) is a hereditary autoimmune disease caused by destruction of insulin-producing pancreatic beta-cells, leading to insulin deficiency. Its complex genetic basis largely involves variants in the HLA region on chromosome 6p, accounting for roughly half of the genetic risk. However, most genetic studies and risk models are based on European populations, limiting their relevance for South Asians. This study focuses on developing a machine learning pipeline to identify T1DM-associated variants within the 6p region of the chromosome by integrating Indian genomics data, patient cohorts, and targeted HLA analysis. Using whole-genome sequencing from over 1,000 healthy Indians in IndiGenomes, this framework filters variants using population-specific allele frequencies, providing insights to unique structural variants or genetic mutations. Advanced HLA imputation and association testing can reveal population-specific risk factors like HLA-DR3 and the AH8.1vhaplotype. This machine learning approach combines regularized logistic regression like LASSO, gradient boosting (XGBoost), and ensemble methods interpreted with SHAP which explains the contribution of each genetic feature to model predictions, improving transparency. It integrates diverse data including Indian allele frequencies, functional impact scores, notably CADD and REVEL—the latter being a robust ensemble score

ISBN: 978-93-95105-97-2

specifically predicting pathogenicity of missense variants. Evolutionary conservation, regulatory annotations, and pathway context can be elucidated by this framework. This IndiGenomes-based, population-tailored framework confirms known HLA risk genes common in Indian T1DM patients , thus providing a scalable and interpretable solution to improve precision medicine for South Asian populations.

Keywords: Type 1 diabetes mellitus, Autoimmune, Pancreatic Beta-Cells, 6p chromosome region, Genome-Wide Association Studies(GWAS), IndiGenomes, structural variants, genetic mutation, HLA imputation, Machine Learning.

PP-07

Data-driven Optimization of Electrocoagulation Process for Treating Restaurant Wastewater

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Abstract

Restaurant wastewater contains high concentrations of organic matter, fats, oils, grease (FOG), and chemicals, posing a serious environmental threat if discharged untreated. Electrocoagulation (EC) is an effective treatment method that removes pollutants through in-situ generation of coagulants. In this study, a Box-Behnken Design (BBD) of Response Surface Methodology (RSM) was applied to optimize key process parameters—initial pH, applied voltage, inter-electrode distance, and electrolysis time—with the goal of maximizing chemical oxygen demand (COD) removal and minimizing power consumption. The experimental dataset generated by RSM was further used to develop predictive models using different Machine Learning (ML) algorithms. Among the models, RFR showed the best performance in predicting both COD removal and power consumption, with high accuracy (R^2) and low error (RMSE). The RFR model was then used to suggest improved operating conditions, which led to a predicted increase in COD removal efficiency and a further reduction in power consumption compared to the RSM results. This improvement highlights the ability of the ML model to capture complex nonlinear interactions and provide better optimization than RSM alone. This study demonstrates that combining RSM with data-driven modeling provides an effective strategy for optimizing the electrocoagulation process, enabling energy-efficient and sustainable wastewater treatment solutions.

PP-08

Unraveling Degenerative Mechanisms in the Macula: An Integrin-Centered Approach

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Abstract

Age-related macular degeneration (AMD) is a leading cause of irreversible vision impairment in the elderly, primarily due to retinal pigment epithelium (RPE) degeneration. Integrins, transmembrane receptors crucial for cell adhesion and survival, may play a significant role in AMD progression through altered expression and signaling. In this study, RPE tissues from donor eyes with and without AMD were analyzed to assess integrin subunit expression at both the transcript and protein levels. Total RNA was isolated, quantified, and converted to cDNA for qPCR-based expression profiling of integrin subunits. Western blot analysis was further performed to validate protein-level expression. qPCR results revealed variable integrin expression, with AMD samples in some cases showing higher expression compared to normal tissues. Western blotting confirmed dysregulated integrin

ISBN: 978-93-95105-97-2

expression in AMD tissues. These findings suggest differential regulation of integrin subunits in AMD and RPE dysfunction. Further large-scale studies are required to delineate integrin-mediated pathways and their therapeutic potential in retinal degeneration.

Keywords: Age-related macular degeneration, Integrins, Retinal pigment epithelium, qPCR, Western blot.

PP-09

Differential Gene Expression in Breast Cancer using DESeq2

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Abstract

In recent decades, breast cancer becomes one of the major type of cancer diagnosed among women worldwide and continues to have a dominating number on the global death rates. Even though epidemiological and experimental researches are going on breast cancer, therapeutic concept in breast cancer still remains unsatisfactory. To increase survival rates and successful treatment plans, suitable and accurate diagnosis is crucial. New biomarkers and therapeutic targets were discovered using gene expression datasets which were publicly available. The goal of this research is to use high-throughput transcriptome analysis to find reliable gene-level biomarkers that can differentiate between breast tumor and healthy tissue which will be used for early detection of this disease. In order to identify genes that were differentially expressed, we used R packages to analyze three datasets that were obtained from NCBI-GEO and had accession numbers GSE164641, GSE228582, and GSE208731. To find differentially expressed genes (DEGs), RNA-Seq datasets were examined using R's DESeq2 package. Distinct gene expression patterns that distinguish tumor from non-tumor samples were discovered by visual analytics, such as heatmaps and volcano plots created with ggplot2. From these three datasets, top 30 upregulated and downregulated genes were selected and we have identified six upregulated hub genes such as SOX2, ASCL1, SHH, MUC5AC, NKX6-1, NKX6-2 and five downregulated hub genes such as KRT16, LAMB3, GJB5, KRT6A, and IL1A. Using Cytoscape, protein-protein interaction network was created to understand the functional interactions between these genes, underlying important regulatory nodes involved in the pathophysiology of breast cancer. To further clarify the biological mechanisms, cellular constituents, and molecular functions connected to these genes, KEGG pathway and Gene Ontology (GO) enrichment studies were performed. SOX2 was identified as potential oncogenic driver among the upregulated conditions, linked to epithelial-to-mesenchymal transition (EMT), therapeutic resistance, and stem cell characteristics. On the other hand, GJB5, one of the downregulated genes, seems to function by preserving epithelial integrity and intercellular communication. Making these molecular biomarkers potential for early detection.

Keywords: breast cancer; differentially expressed genes; upregulated genes; down related genes; DESeq2 package.

PP-10

Multi-Omics Analysis to Identify Epigenetic, Methylation and Microbiome Biomarkers in Alzheimer's Disease

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Abstract

Alzheimer's disease (AD) is a complex, progressive neurodegenerative disorder marked by cognitive impairment, memory loss, and behavioral dysfunction. While the disease has traditionally been characterized by amyloid-beta plaque deposition and neurofibrillary tangles, emerging evidence underscores the critical roles of systemic

ISBN: 978-93-95105-97-2

inflammation, epigenetic dysregulation, and gut-brain communication in its pathogenesis. In particular, the interplay between host gene expression, microbial metabolites, and DNA methylation patterns has been increasingly recognized as a key driver of early pathological changes preceding clinical symptoms. As such, integrative multi-omics approaches are vital to uncover the molecular networks underlying AD and to identify early-stage biomarkers for diagnosis and intervention. This study employs a systems biology framework to investigate Alzheimer's disease using a multi-omics strategy. Publicly available datasets were curated across three omics layers: transcriptomics, DNA methylation (epigenomics), and gut microbiome (metagenomics). Differential gene expression was assessed across five datasets using RNA-seq and microarray platforms, followed by functional enrichment and pathway analysis. In parallel, DNA methylation patterns were analyzed at CpG sites located in key AD- related, with methylation status annotated by genomic context and normalized using R packages such as minfi and limma. Metagenomic profiling was conducted using 16S rRNA sequence data and analyzed with tools like phyloseq, focusing on taxa known to influence neuroinflammatory processes. The analysis revealed recurrent upregulation of inflammatory genes (e.g., NFKB1, ITGAM, JMJD6, APBB3) and downregulation of mitochondrial and ribosomal genes (e.g., NDUFA1, COX17, RPL36AL). Methylation analysis showed hypermethylation in regulatory regions of APP, TREM2, and APOE, suggesting transcriptional repression of neuroprotective pathways. Microbiome profiling indicated enrichment of pro-inflammatory *Escherichia* and depletion of butyrate-producing *Faecalibacterium*, reflecting gut dysbiosis. Integration of these omics layers highlighted coordinated molecular cross-talk between microbial shifts, epigenetic remodeling, and gene expression changes, supporting the role of gut–brain–epigenome interactions in AD pathology. This approach aims to delineate the interconnected molecular pathways involved in AD and to propose candidate biomarkers and regulatory nodes that may inform future diagnostic or therapeutic strategies.

Keywords: Alzheimer's disease, gut-brain axis, DNA methylation, microbiome, multi-omics, transcriptomics, epigenetics, biomarker discovery, R programming

PP-11

Transcriptomic Analysis of Host Immune Response in Tuberculosis Condition

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Abstract

This study incorporates several transcriptomic databases GSE268366, GSE12297, GSE157657, GSE236156 and GSE203261, aimed at elucidating the key molecular actors of immune responses. The study technique includes differential gene expression, hub gene network analysis, enrichment analysis, and protein interaction networks. Results led to the identification of ten key hub genes: IFI44L, IFIT1, RSAD2, EPSTI1, SIGLEC1, TRIM22, AZU1, HBA2, RNASE3, and HBA1, being comprised of interferon stimulated genes (ISGs), immune effectors, and haemoglobin-associated proteins. The enrichment analyses by Gene Ontology (GO) and KEGG demonstrated enrichment in significant pathways related to viral defense and regulation of the viral life cycle, as well as relatedness to disease pathways including malaria, hepatitis C and African trypanosomiasis. Moreover, STRING bioinformatic network analyses illustrated substantial associations with protein-protein interactions, most notably with components of interferon signalling thereby establishing a cohesive type I IFN antiviral response. The elaborate network identifies antiviral mechanisms, including immune recruitment, and metabolic reprogramming as critical to overall strategy, leading to identification of potential therapeutic targets.

Keywords: Hub genes, interferon response, IFI44L, IFIT1, RSAD2, PPI network, viral defence, transcriptomic analysis, immune regulation

ISBN: 978-93-95105-97-2

PP-12

Comparative Genomic Architecture and Phylogenomic Profiling of Bacteriocin Biosynthetic Gene Clusters in Prokaryotic Strains

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Abstract

Bacteriocins are ribosomally synthesized antimicrobial peptides with growing potential as sustainable alternatives to conventional antibiotics. These bioactive compounds are typically encoded within bacteriocin biosynthetic gene clusters (BGCs), which are compact genomic loci containing genes for precursor peptides, post-translational modification enzymes, transport systems, and immunity proteins that safeguard the producing strain. BGCs are recognized as hotspots for natural product discovery, as their modular architecture supports the generation of chemically diverse antimicrobial metabolites. In this study, we developed a comprehensive in silico pipeline to identify, annotate, and compare bacteriocin BGCs across a diverse panel of prokaryotic strains. Previous genome-mining studies have demonstrated the value of such approaches in uncovering cryptic or silent clusters, which informed the design of our workflow. Whole genomes retrieved from NCBI were analyzed using antiSMASH for BGC prediction, followed by Clinker for gene synteny visualization and InterProScan for functional domain annotation. Multiple sequence alignment and phylogenetic reconstruction in R provided evolutionary insights and highlighted several clusters with minimal synteny to known bacteriocin families, consistent with earlier reports of uncharacterized bacteriocin diversity. Among 21 bacterial strains, 12 harbored conserved lantibiotic-related domains, whereas the remaining strains carried largely uncharacterized clusters, suggesting the presence of novel antimicrobial pathways. Comparative genomic analysis emphasized the diversity of gene organization, including precursor diversification and strain-specific hypothetical proteins. This integrative approach enables prioritization of cryptic clusters as promising candidates for experimental validation and synthetic biology-driven bacteriocin discovery, contributing to the development of next-generation therapeutics against antimicrobial resistance.

Keywords: Bacteriocins, biosynthetic gene clusters, comparative genomics, cryptic genes, antimicrobial resistance.

PP-13

Transcriptomic Signature Profiling of Pathophysiology in Multiple Sclerosis

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Abstract

Multiple Sclerosis (MS) is a chronic, progressive autoimmune disease that affects approximately 2.8 million people globally, and is associated with inflammatory, demyelinating and neurodegenerative events in the central nervous system. Understanding the molecular mechanism of MS pathology is still limiting. The present study applied a transcriptomic approach to MS, utilizing RNA-sequencing datasets from four independent cohorts obtained from the Gene Expression Omnibus (GEO). The datasets included MS patients and healthy controls white matter brain tissues. Differential expression analysis was performed using the DESeq2. As a result, significant dysregulation of gene expression was observed, and Principal Component Analysis (PCA) demonstrated clear separation between MS and control samples across datasets. Volcano plots further indicated key upregulated/downregulated genes as differentiators. Cross data set comparison revealed seven common genes that were differentially expressed (IGLL5, CPB2-AS1, ERLN, IGHV3-64D, PCAT14, PPP1R26-AS1, ACY3). Hub genes (USH1C, DAO, SH2D3A, HCN4, ASZ1, KCNE3, OLIG2, GJB6, MUC15, MUC12) were identified in protein-protein interaction (PPI) network analysis utilizing STRING database. Functional enrichment analysis was also performed using Gene Ontology (GO) and KEGG pathways and revealed significant associations with immune response pathways, myelination-associated processes, and neuronal signaling processes. Importantly, OLIG2, GJB6, and USH1C were found as potential biomarkers because they were part of the oligodendrocyte differentiation and myelination processes, and DAO and HCN4 were found as promising therapeutic targets for neurotransmitter metabolism and neuronal excitability. These findings provide new molecular contributions to MS pathophysiology and offer potential candidate genes for future diagnosis and therapeutics.

ISBN: 978-93-95105-97-2

Keywords: Multiple Sclerosis, Transcriptomics, White Matter, Biomarkers, Gene Expression, Neurodegeneration.

PP-14

Repurposing Drugs for Metabolic Disease Co-Morbidities: A Network Pharmacology Study of Diabetes Mellitus and Diabetic Retinopathy

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Abstract

Diabetes Mellitus (DM) and Diabetic Retinopathy (DR) are the two major chronic disease that causes a major public health challenges across the world. DM is a metabolic disorder that are characterized by hyperglycaemia due to insulin deficiency or resistance, and DR is a microvascular complication of DM which will lead to vision lost. Understanding of the shared molecular pathways involved in both disease states can ultimately result in the development of therapeutic strategies that are better efficacious, less invasive, and has efficient therapeutic strategies. In this report, we took an integrated bioinformatics approach using R - programming on RNA-seq datasets obtained from the Gene Expression Omnibus (GEO). Differentially expressed genes were identified using the DESeq2 package, and data visualizations were presented as volcano plots, MA plots and Principal Component Analysis (PCA). There are 103 common DEGs between DM and DR were identified and analysed for protein-protein interactions (PPI) network using the STRING and igraph. The analysis highlighted key hub genes, based on centrality measure values. The functional enrichment of these hub genes were analysed based on Gene Ontology (GO) and KEGG pathway database which shows a shared pathological process. Finally, drug-gene interaction analysis from the DGIdb indicated that Bevacizumab-awwb a biosimilar of Bevacizumab, can be a possible drug repurposing candidate, as it demonstrated strong interactions with multiple hub genes. Our results show that DM and DR, share similar molecular landscapes, which highlights the promise of bioinformatics and network pharmacology for discovering new therapeutic drugs. In addition, this work provides a basis for future experimental work, and supports the feasibility of repurposing existing drugs to address both metabolic and retinal diseases.

Keywords: Diabetes Mellitus, Diabetic Retinopathy, Drug repurposing, Bevacizumab-awwb, Metabolic Disease

PP-15

Gene Expression Profile Analysis for the Identification of Hub Genes and Their Significance in Blood Cancer

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Abstract

High-throughput sequencing technologies have transformed the advancement in the research area of hematologic malignancies into a complete molecular profile. This research uses the complete transcriptomic analysis of seven RNA-seq datasets made publicly available in the NCBI GEO database to assess gene expression profiles in blood cancer patients compared to healthy individuals. Our goal is to identify differentially expressed genes (DEGs) as well as biological pathways that were dysregulated in various hematological cancers (acute myeloid leukemia (AML), chronic lymphocytic leukemia (CLL), multiple myeloma (MM), and lymphomas). The DEGs were analyzed with DESeq2 for differential expression analysis, followed by gene ontology (GO) functional annotation, KEGG pathway mapping, and protein-protein interaction (PPI) network, which indicated substantial alterations in genes, including TP53, FLT3, NRAS, IDH1, and IDH2. These gene activity was linked to cellular processes such as cell cycle control, proliferative signaling, metabolic switching, immune evasion, and epigenetic

ISBN: 978-93-95105-97-2

remodeling. The uniquely expressed patterns of these genes highlighted the molecular mechanisms associated with hematologic tumorigenesis and tumor progression. Age-based stratification further revealed the expression trends linked to higher disease incidence in aged populations. Our analysis provides context for diagnosis, prognosis, and potential biomarkers across the cancer genome in disease-specific genomic contexts.

Keywords: High-throughput sequencing, hematologic malignancies, acute myeloid leukemia (AML), chronic lymphocytic leukemia (CLL), multiple myeloma (MM), lymphomas, differentially expressed genes (DEGs), transcriptomic profiling, cancer biomarkers.

PP-16

Multi-Disease Drug Repurposing for Autoimmune Disorders: A Network Pharmacology Study of Rheumatoid Arthritis and Systemic Lupus Erythematosus

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Abstract

Autoimmune diseases such as Rheumatoid Arthritis (RA) and Systemic Lupus Erythematosus (SLE) represent chronic inflammatory disorders which signifies immunological dysregulation and overlapping clinical features. The study establishes the value of a network-based drug repurposing strategy in identifying shared molecular characteristics and potential therapeutic interventions for rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE). Transcriptomic datasets for RA and SLE were obtained from the GEO database and analyzed using the DESeq2 package for DEG identification. A total of 148 genes were found to be commonly dysregulated across both disease conditions. Protein-protein interaction networks for these overlapping genes were then developed in STRINGdb, and hub genes were identified according to centrality measures of the network. Functional enrichment of biological processes and pathways through GO and KEGG indicates the possibility for immunological response, cytokine signalling, and interferon pathways. The drug-gene interaction analysis using DGIdb identified SB290157, a C3AR1 antagonist, as a potential multitarget therapy for both diseases. SB290157 was selected for its ability to interact with key hub genes and influence inflammatory pathways common for RA and SLE. This integrative pipeline combining transcriptomic meta - analysis and drug interaction data presents an efficient strategy for repurposing drugs across many diseases. This provides a cost effective and time efficient strategy to identify repurposable drugs and precision medicine in complex autoimmune diseases.

Keywords: Drug repurposing, Autoimmune diseases, Network analysis, Rheumatoid Arthritis, and Systemic Lupus Erythematosus.

PP-17

Network-Based Identification of Biomarkers in Rheumatoid Arthritis through Transcriptomic Approach

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Abstract

Rheumatoid arthritis (RA) is a chronic autoimmune disease associated with synovial inflammation, progressive joint destruction, and systemic effects. The heterogeneity of RA challenges the identification of effective safe and personalized therapeutic approaches. In this study, an integrative transcriptomic analysis was conducted to identify potential molecular markers linked to RA. Utilizing publicly available datasets (GSE181313, GSE217012 and GSE253495) Differentially Expressed Genes (DEGs) were identfied between RA and healthy controls. Using protein-protein interaction (PPI) network and centrality analysis, hub genes (CDH3, CDH18, CDH8, CHRNA1,

ISBN: 978-93-95105-97-2

CDX2, ENAM and CDKN2A) were identified. Functional enrichment analysis identified important immune related and inflammatory signalling pathways including NF-κB , PI3K-Akt and MAPK pathways. Immune cell infiltration analysis and gene expression profiling indicated altered immune conditions in RA synovial tissues consistent with previous reports, reinforcing the role of immune dysregulation in disease progression. In particular, hub genes CDKN2A and CHRNA1 are known to modulate fibroblast-like synoviocyte senescence and cholinergic anti-inflammatory signalling. Our findings highlight the hub genes and their pathway that could serve in diagnosis, monitoring of RA and provide a basis for future drug discovery.

Keywords: Rheumatoid arthritis, Transcriptomics, Biomarkers, Differential Gene Expression, Autoimmune Disorder.

PP-18

Integrative Multi-Omics Analysis for Identifying Epigenetic and Microbiome Biomarkers for Early Detection of Lung Cancer

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Abstract

Lung cancer remains one of the most lethal malignancies worldwide, due to poor prognosis largely attributed to late diagnosis, which limits precision in treatment. The identification of robust and clinically actionable biomarkers is a critical step for improving early detection, prognosis, and therapeutic decision-making in lung cancer. Traditional approaches have focused on individual molecular layers like gene mutations or mRNA expression; however, these often fail to capture the full biological complexity of disease. In this study, a multi-omics integration approach was applied, combining transcriptomic (RNA-seq), epigenomic (DNA methylation), and microbiome (16S rRNA/metagenomics) datasets from public repositories including TCGA and GEO. Data were preprocessed for quality control, normalized, and batch-corrected. Integration was carried out using advanced computational frameworks such as PCA, DIABLO, clustering, correlation, and network analyses, with validation through cross-cohort comparison, functional annotation, and literature mining. The results revealed distinct molecular signatures in lung cancer. Overall, the integrative approach demonstrates that multi-omics biomarkers outperform single-omic markers, offering improved sensitivity, specificity, and biological insights for early detection of lung cancer. Findings underscore the promise of multi-omics in advancing precision oncology, biomarker discovery, and personalized therapeutic strategies.

Keywords: Lung cancer, Multi-omics integration, Biomarkers, Genomics, Epigenomics, Transcriptomics, Proteomics, Metabolomics, Metagenomics, Precision oncology

PP-19

Analysis of Rhizospheric Microbial Communities and Drought-Responsive Genes in *Oryza sativa*

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Abstract

Drought remains one of the most serious challenges to rice cultivation, especially as climate change intensifies. In this study, we explored how rice (*Oryza sativa*) and its surrounding soil microbiome respond to drought stress by omics-based approach. We analyzed publicly available RNA-seq datasets to identify key genes involved in drought response in rice roots. Using metagenomic data from the rhizosphere of *Sorghum bicolor* as a model in order to understand how microbial communities shift under water-limited conditions.Our analysis uncovered a clear activation of several well-known drought-responsive genes, including NCED3, which drives ABA

ISBN: 978-93-95105-97-2

biosynthesis, PIP2.7 for water transport, and galactinol synthase, which helps in osmoprotectant production. These genes work together to manage water balance, stress signaling, and metabolic adjustments during drought. On the other hand, we also observed the downregulation of growth-related and energy-demanding processes, highlighting the plant's strategy to conserve resources during stress. From the microbial side, drought was found to dramatically reshape the rhizospheric community, especially by increasing the abundance of *Paenibacillus* species which are known for their drought-tolerant and plant-friendly traits. This suggests that not only do plants adapt on a genetic level, but their microbial partners also adjust, potentially playing a supportive role in the stress response.Together, these findings indicates a holistic picture of how rice plants and their microbial allies coordinate to survive drought. The insights gained here could be valuable for developing more drought-resilient rice varieties through breeding, microbial inoculation, or genetic engineering.

Keywords: Rhizospheric microbiome, drought stress, Oryza sativa, gene expression, omics analysis, microbial diversity, transcriptomics

PP-20

Predictive Modelling of Gene Signatures in Type II Diabetes Mellitus and their Functional Validation

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Abstract
Type II Diabetes Mellitus (T2DM) is a complex metabolic disorder characterized by chronic hyperglycemia resulting from insulin resistance and impaired insulin secretion. Identifying reliable molecular biomarkers is critical for early diagnosis and personalized treatment strategies. This study focuses on predictive modelling of gene signatures associated with T2DM using integrated transcriptomic datasets from RNA sequencing and microarray experiments and their functional validation. Differential expression analysis identified key upregulated and downregulated genes, followed by network-based hub gene identification using STRING and Cytoscape MCODE. A subset of 31 hub genes was selected for gene ontology enrichment to elucidate biological pathways involved in disease progression. A machine learning model was developed using gene expression data to accurately predict T2DM risk based on hub gene profiles. Additionally, a user-friendly Shiny web application was created to facilitate interactive prediction of diabetes risk from gene expression inputs, demonstrating translational potential in clinical diagnostics.

Keywords: Type II Diabetes Mellitus, Metabolic Disorder, Hyperglycaemia, Insulin, Biomarkers, Hub Genes

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Co-occurrence of carbapenemase and 16S rRNA methyltransferase genes in carbapenem resistance clinical isolates from a tertiary care hospital in Coimbatore, India

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Abstract
Antimicrobial resistance (AMR) has been a key research focus with increasing impact over time. The complications induced in hospitalized and chronically ill patients by drug resistant bacteria has resulted in millions

ISBN: 978-93-95105-97-2

of deaths and is estimated to rise to 10 million per year by 2050. Carbapenem resistant Enterobacteriaceae (CRE), along with *A.baumanii* and *P.aeruginosa* are principal causes of deadly infections and has raised concerns worldwide. Combination therapies are often used to manage drug-resistant bacteria. However, treatments that combine aminoglycosides with carbapenems are failing due to the increasing co-occurrence of resistance to both drug groups. This severely limits treatment choices and creates a significant clinical challenge. In this study, a total of 105 carbapenem-resistant isolates were analysed. Among which, 19 (18.1%) harbored both carbapenemase and 16S rRNA methyltransferase genes. Gentamicin resistance was seen in 24 isolates (22.9%) of which 19 (79.2%) were in co-occurrence with carbapenemase genes. The findings highlight, the concern in rising co-occurrence of aminoglycosides with carbapenem resistant genes which is a critical clinical challenge.

Keywords: Antimicrobial resistance, carbapenemase, gentamicin, co-occurrence, combination therapies

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Screening of MMV Pandemic response box library compounds against fluoroquinolone-resistant *Salmonella typhi* isolates

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ABSTRACT
Salmonella enterica serovar Typhi (*S. Typhi*) infections is estimated to cause millions of cases with thousands of deaths in low- and middle-income countries (LMICs) each year. Particularly a concern in Asia and Africa, where typhoid fever is prevalent and resistant strains continue to spread. Thus, the current study aimed identify the inhibitory compounds from the MMV pandemic Response box compounds against fluoroquinolone- resistant *S. typhi*. Fluoroquinolone-resistant *S. typhi* were from two patients (29y/F & 9m/M). Micro-broth dilution method was used to identify the hit compounds initially at 10µM in triplicates in 96 well flat bottom microtiter plate. The absorbance was recorded using Varioskan Multimode plate reader at 600nm (OD600nm). Suspensions from wells that showed initial inhibition was spread on to the drug free LB agar. Colonies were counted for Presister frequency (P*f*) in CFU/ml. Minimum inhibitory concentration determination on scale of 20µM to 0.062µM using micro-broth dilution method in triplicate and plates were read visually and photometrically. Suspensions from wells that showed initial inhibition was spread plated to determine minimum bactericidal concentration. Bacterial viability was assessed at various time points (0, 2, 4, 6, 8, and 24 hours) for the time-kill assay. Six out of 400 compounds were exhibiting potential antibacterial; activity against both stains. Persister ranged from 30 to 104 CF/ml. Alexidine was potent against both strains. Gepotidacin acted effectively against both fluoroquinolone-resistant S. typhi strains. MMV1580854 [2-(2-aminopyridin-3-yl)oxy-5-ethyl-4-fluorophenol] was inhibiting the strains but less MBC efficacy. MMV1580173 (Trimetrexate), MMV1580853 and MMV1579846 inhibited one of the strains.

Keywords : Typhoid fever, antimicrobial resistance, antibacterial, Gepotidacin, Alexidine

ISBN: 123-4567-890-1

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ISBN: 978-93-95105-97-2

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